THE EFFECTS OF A NUTRITION PROGRAM WITH AND WITHOUT AEROBIC EXERCISE ON BODY WEIGHT AND COMPOSITION, PLASMA VARIABLES AND NUTRIENT INTAKE IN OBESE BLACK WOMEN

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(ABSTRACT)

The effects of a multifaceted weight loss program on the body weight, body composition, plasma variables, and nutrient intakes of 50 obese black women were investigated. Subjects were between 20 and 51 years of age, with an average BMI of 34.5 (range = 23.6 - 57.3). Subjects attended nutrition education/behavior modification classes once per week for three months. Thirty-five of the women attended 80% or more of the classes (NU). Twenty-eight women attended 30% or more of the low-impact aerobic exercise classes that were offered three days per week, for six months. An average 2.2 kg weight loss was observed for the whole study group, as body fat fell, and lean body mass increased by 2.1% of total body weight. Slightly greater changes were observed in the group that exercised consistently, as well as those who regularly attended nutrition classes, but not in either of the groups which more sporadically attended exercise or nutrition classes. Significant reductions in plasma total cholesterol, HDL-Cholesterol, HDL_2-Cholesterol, and insulin were observed for

the group of 50 subjects. Significant reductions in plasma TC occurred in both the SE (attended between 30 to 70% of exercise sessions) and SN (< 80% attendance of nutrition classes) groups. Plasma LDL-Cholesterol followed the same pattern as plasma total cholesterol but there were no significant differences. Exercise appeared to mitigate decreases in plasma HDL-C. Significant reductions in total kcal, including CHO, protein, fat (including saturated, polyunsaturated, and monounsaturated fatty acids), dietary cholesterol, and sugar were noted for the study group. A weight-loss program which included diet, nutrition education, behavior modification, stress management and exercise was effective in producing favorable changes in body composition, plasma variables, and dietary components in obese black women over six months, with a high degree of variability in motivation and participation.

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INTRODUCTION

Despite the increasing awareness about health, nutrition, fitness, and weight loss (1), a 20 billion dollar-a-year diet and exercise industry (2), and a pervading prejudice against obesity in America (3), the American populace continues to become more overweight (4) (Figure 1). According to the most recent National Health and Nutrition Examination Survey (NHANES II, 1976-1980) fully 28% of the adult American populace, or 34 million people aged 25-74, were overweight (5), up from 25.4% in the period 1960-1962, and from 26.7% between 1971 and 1974 (4). Overweight was defined as a body mass index (BMI, kg/m^2) of 27.3 for women, and 27.8 for men (6). These cutoff points were based on the sex-specific 85^{th} percentiles of individuals in the 20-to-29 year old range in that survey. This age group was used because young adults are relatively lean, and an increase in body weight with age is generally due to fat accumulation (5). The BMI is commonly used to determine both overweight and obesity, because it is a convenient measure which has been correlated highly with body fat as determined by hydrodensitometry (7). The percentage of the population that was defined as severely overweight during this period was approximately 10%, or 12.4

Sex	Race and Period								
	Total*			White			Black		
	1960-1962	1971-1974	1976-1980	1960-1962	1971-1974	1976-1980	1960-1962	1971-1974	1976-1980
	Percent of population								
Both sexes	25.4	26.7	27.0	24.2	25.7	25.9	35.1	38.0	40.0
Males	23.1	24.8	25.3	23.3	24.8	25.2	22.5	27.0	30.0
Females	27.4	28.4	28.6	24.9	26.4	26.3	47.1	46.8	48.1

Figure 1. Overweight persons aged 25 to 74 years, by race and sex: United States, 1960-1962, 1971-1974, and 1976-1980. Overweight is defined as body mass index greater than or equal to 28 kg/m^2, and 35 $kg/m^{1.5}$ for men and women, respectively. *includes all other races not shown separately.

From: Gillum RF. Overweight and obesity in black women: a review of published data from the National Center for Health Statistics. Journal of the National Medical Association 1987;79: 865-71.

million individuals (6). This was greater than during 1971-74, in which 8.4 million people were severely overweight (6). For females, by age groups, the trends in the prevalence of severely overweight for three time periods between 1960 and 1980 can be seen in figure 2. Severely overweight in NHANES II was defined as a BMI equal to or greater than 32.3 and 31.1 for women, and men, respectively, which represented the sex-specific 95th percentiles of the 20-to-29 year old NHANES II population (5). Often, the population of severely overweight individuals is included in the group that is reported to be overweight. In this most recent national survey, NHANES II, the overweight condition was more prevalent in women (29.6% of females) than in men (26.3% of males) (5), and in blacks (40.0% of blacks) than in whites (25.9% of whites) (4). Black women accounted for substantially more of the higher prevalence in the black population than their male counterparts (48.1% of black females vs 30.0% of black males), and the discrepancies between black females and either sex of the white race were even greater (26.3% of white females, 25.2% of white males) (4) (Figure 1). This trend was true for three time periods from 1960 to 1980 (Figure 1). A closer look at the occurrence of overweight among black women revealed that such individuals living in rural and southern areas were more overweight than their urban, northern and western counterparts, and that overweight was inversely related to

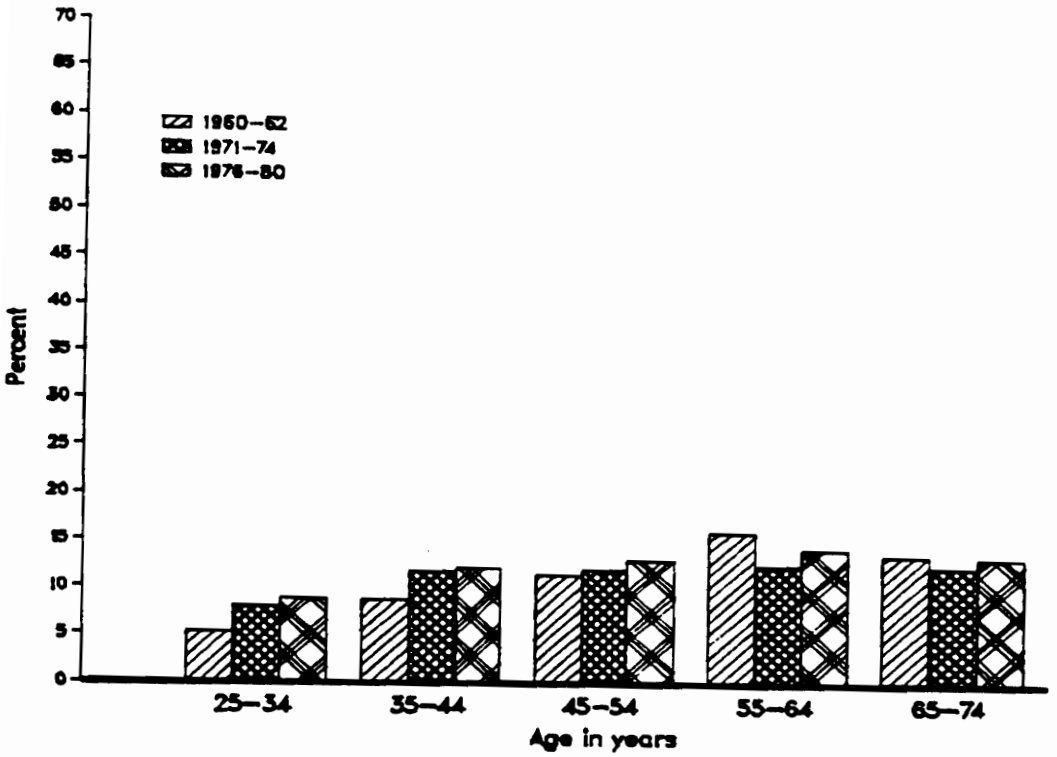
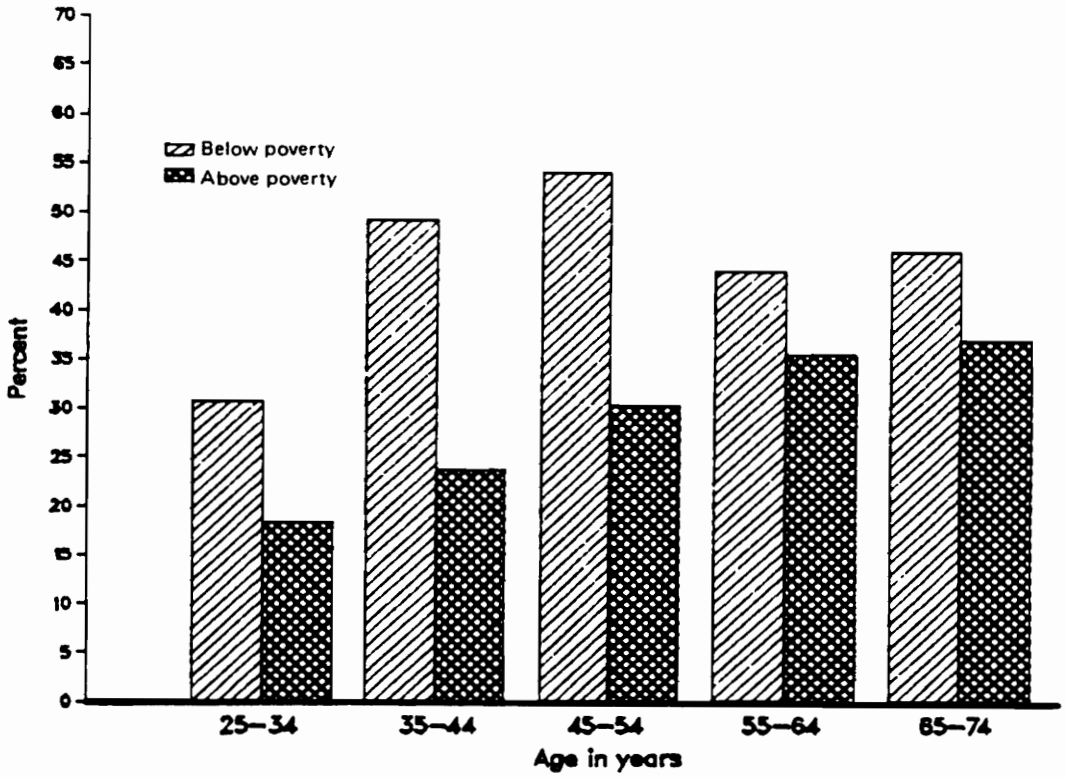


Figure 2. Percent of nonpregnant females severely overweight, by age: 1960-62, 1971-74, and 1976-80.

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).

family income (Figure 3) and education. These relationships were found to be stronger for black than for white women (4). Socioeconomic status has also been found by many researchers to have a strong inverse relationship with obesity in women (8). In addition, the stigmatization of obesity in women is recognized to be greater than that for men (9, 10, 11). Despite these statistics, there is a paucity of information relating to attempts to address the problem of obesity in low-income women (12) or low-income black women (13, 14).

The obese condition carries with it numerous associations with other health complications, and is considered a risk factor in coronary heart disease (CHD) (Figure 4). Hypercholesterolemia, a major risk factor in CHD, is 1.5 times more common in overweight versus normal-weight persons (6) (Figure 5). Being overweight has been positively associated with several risk factors for CHD, including total cholesterol (TC), (15, 16), and negatively with high-density lipoprotein cholesterol (HDL-C) (17, 18, 15, 16). The association between obesity and hypertension has been well-established for some time (19, 17, 20), and it has recently been estimated that the risk for hypertension is 2.9 times greater for overweight than non-overweight individuals (6) (Figure 5). Furthermore, for all age groups, there is a greater percentage of blacks than whites with hypertension, as well as for those below the poverty



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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).

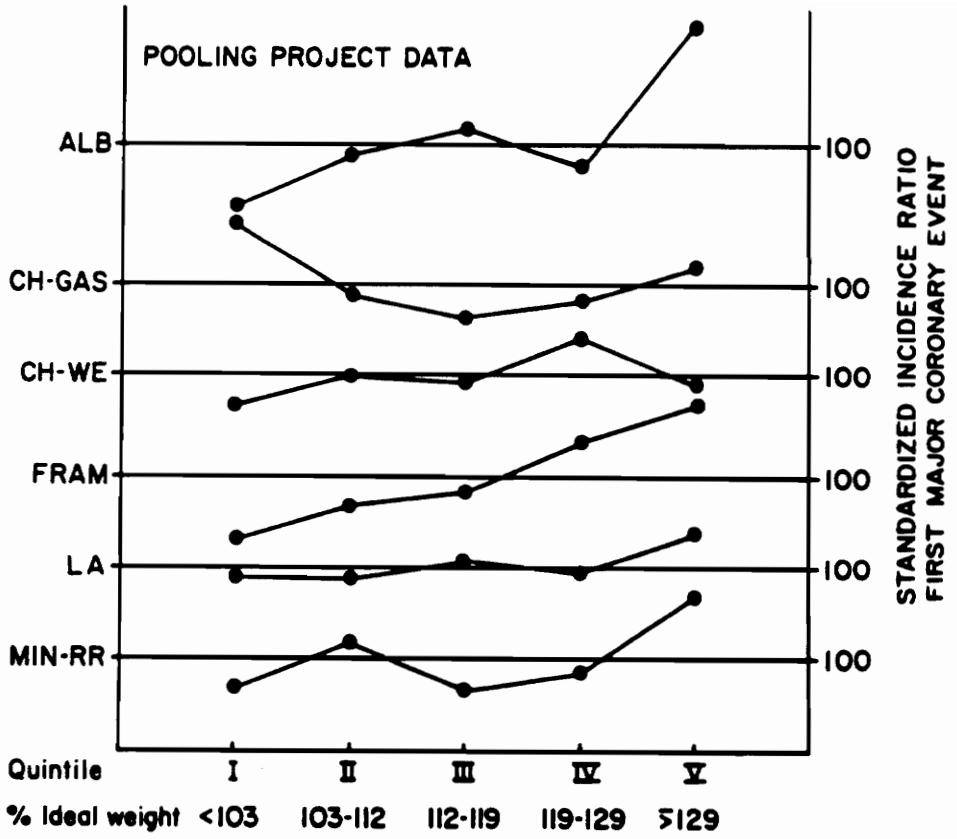

Figure 4. Standardized incidence ratio for first major coronary event by quintile of relative weight in six Pooling Project cohorts. ALB=Albany; CH-GAS=Chicago People's Gas Company; CH-WE=Chicago Western Electric; FRAM=Framingham; LA=Los Angeles Heart Study; MINN-RR= Minnesota Railroad Workers Study.

From: Barrett-Connor EL. Obesity, atherosclerosis, and coronary heart disease. Annals of Internal Medicine 1985; 103(6 pt 2):1010-19.

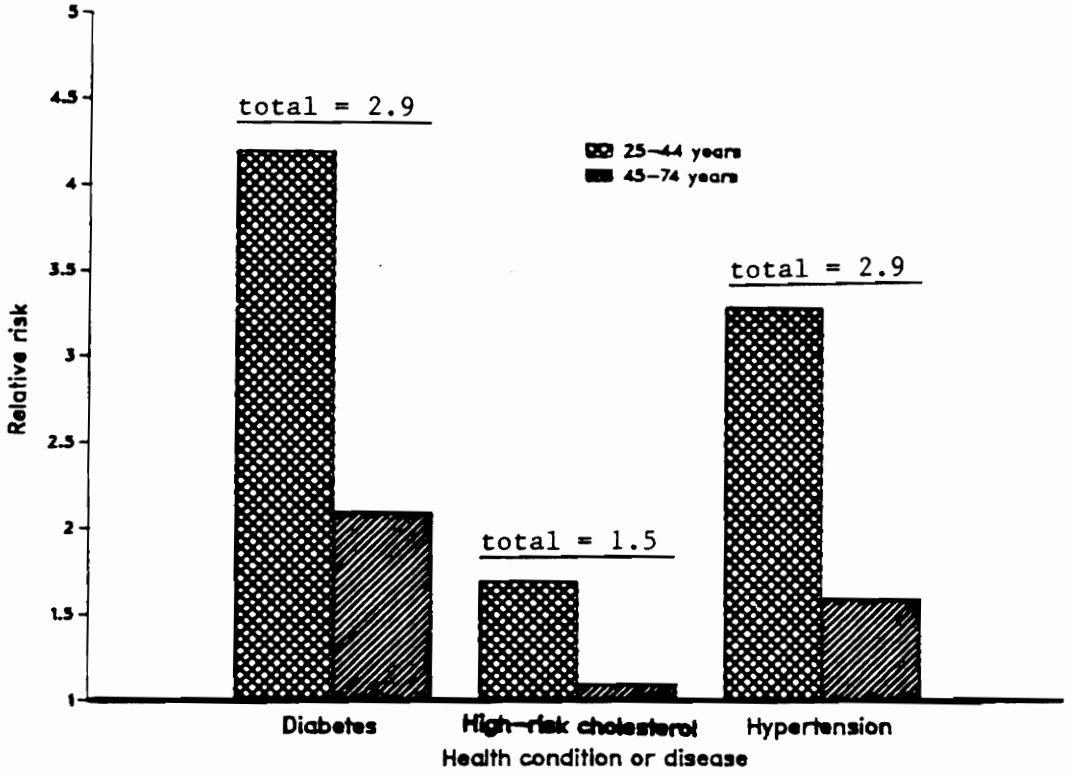

Figure 5. Relative risk of diabetes, high-risk serum cholesterol level, and hypertension for overweight persons (relative to not overweight persons), by age: 1976-80.

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).

level, as compared to those above it (5). Obesity has not only been associated with these risk factors for CHD, but has also been found to be an independent risk factor (21) for cardiovascular disease (CVD). The term CVD pertains to conditions affecting the heart and blood vessels and includes CHD (5). While CHD is the number one killer in both blacks and whites (5), it appears that the rate for black women is higher than that for white women (22), and that Virginia ranks among the top two quartiles among the states in terms of CHD mortality in blacks.(23).

The relative risk of diabetes in overweight adults has also been estimated to be 2.9 times that of non-overweight persons (6) (Figure 5), while being black, female or below poverty level are all associated with further risk (5). The incidence of various cancers increase with obesity (24), as do several other conditions such as ventilatory disorders, circulatory impairment, digestive diseases, various endocrine abnormalities, obstetric complications, and trauma to weight-bearing joints (25, 26). Therefore, a higher mortality ratio for obese individuals should not be surprising (27, 28, 29). That there is a curvilinear relationship between this mortality ratio and overweight and/or obesity suggests that lower levels of overweight or obesity may not be associated with the same degree of risk for various health complications (27) (Figure 6). Indeed, there is some evidence which suggests that this may be true

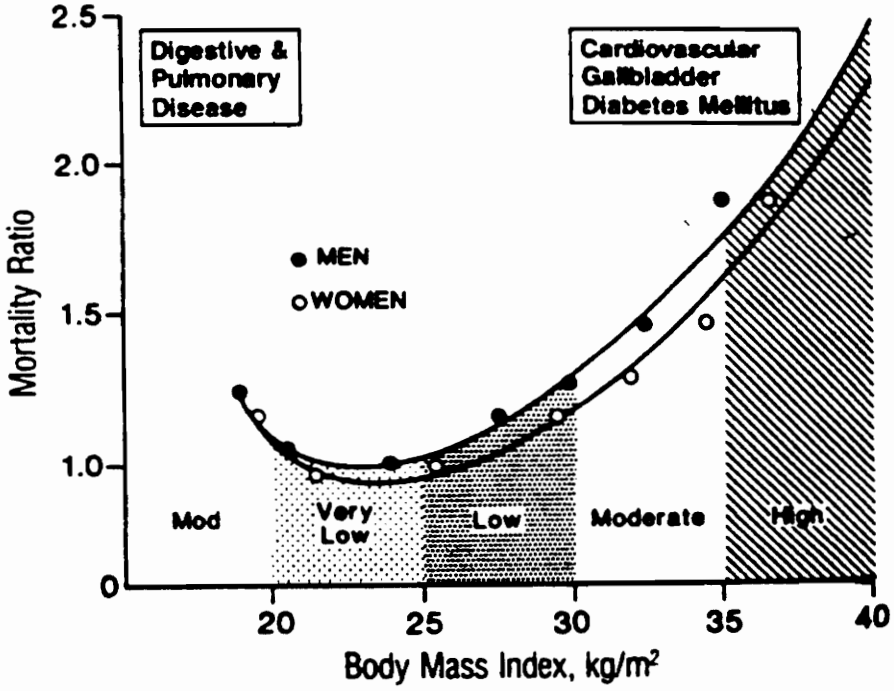

Figure 6. Relationship of body mass index to overall mortality risk. At a body mass index below 20 kg per m^2 and above 30 kg per m^2, there is an increase in relative mortality. The major causes for this increased mortality are listed, along with a division of body mass index groupings into various levels of risk.

From: Bray GA, Gray DS. Obesity. Part I-Pathogenesis. West J Med 1988;149:429-41.

(30, 25, 31, 32), and that weight loss can lead to improvements in many of these complications (33), including blood variables which are considered risks for CHD (34, 35, 36, 37).

Knowledge of etiological factors in obesity has increased significantly in the past several decades. We have progressed from a time when 'a personality disorder, causing one to overeat and be lazy' was believed to be the cause of all individuals being overweight (8). It is now recognized that there are a variety of causes of obesity, that some of these are interrelated, and that they probably have a genetic component (38). The idea that obese people eat more has been proven erroneous by numerous investigations (39, 40, 41, 42, 43), and data from the first NHANES indicated that black women consumed slightly fewer calories than white women, even when expressed per kilogram of body weight (44). Studies of activity patterns within this decade indicated that obese individuals may be generally less active than their normal-weight peers (45, 46, 38); however, since obese persons expend more energy for a given movement (47), and have a higher RMR (48) than non-obese persons, the question of total energy expenditure has remained unanswered.

Efforts aimed at treatment of the obese condition can be classified under one of five general headings: diet, behavior modification, exercise, pharmacotherapy and

surgery. Conservative treatments, (which are discussed more fully in a later section), initially focused on dietary restriction until behavior modification therapy began to emerge in the early 1970's. The use of exercise in weight management has become prevalent more recently (49). These therapies have been traditionally employed separately, with varying degrees of success. Dietary interventions were not found to result in substantial weight reductions in the majority of participants, and attrition rates were found to be high (50, 51). Behavior modification trials have also resulted in only moderate weight loss, but attrition rates have been considerably lower than other programs (52).

Exercise alone may not be very effective in the short-term reduction of body weight in obese individuals, (53); however, several studies have indicated that long-term results may be improved by including exercise in a weight control program (31, 54). Moreover, exercise may offset the diet-induced decrease in metabolic rate as well as preserve or increase lean body mass (53). Exercise can lead to decreased levels of insulin and triglycerides (TG), increased levels of plasma HDL-C, and improvements in glucose tolerance and insulin sensitivity (53). In addition, psychological benefits such as enhanced mood, self-image and self-esteem may occur with exercise, and this may serve to promote adherence to a program of weight control (55). These effects are in contrast to the adverse

psychological reactions sometimes seen in response to dieting (3). In recent years, these therapies have been combined in various ways. Programs emphasizing nutrition education to attain weight loss in conjunction with exercise have begun to incorporate a behavioral management component (56), while behavioral modification therapy has shifted its emphasis from an individual to a group format (57). Recently, numerous investigators who had reviewed various aspects of the different treatments of obesity recommended a multi-faceted approach (53, 56, 57, 58, 59, 60). In 1984 a panel organized under the auspices of the International Congress of Obesity stated that three components of therapy -diet, exercise, and psychological support- are 'interrelated, interdependent and mutually supportive, and thus should be prescribed together' (60). A more recent statment included the idea that nutrition education was important in the attainment of the dietary component of a comprehensive weight loss program (53). The results of several studies which have included nutrition education, behavior modification and exercise in comprehensive weight loss programs supported recommendations for their use with obese individuals (12, 13, 61). Limited data indicated that such therapies could be of use in black populations as well (13, 14).

REVIEW OF LITERATURE

Definition and Measurement of Obesity

The term overweight is defined as a body weight which is above a standard according to height, while the term obesity is defined as an excess of body fat. However, since convenient measures of body fat are often not available, and the correlation of body weight-for-height with body fat as determined by hydrodensitometry is rather high, (.7 to .8) (7), body weight-for-height is generally used to determine obesity. Overweight indicates an excess weight-for-height of 10-20% while the term obesity is often used to indicate an excess weight-for-height of 20% or more (53). Mild, moderate and morbid obesities are defined as 20-40%, 40-100% and more than 100% above desirable weight, respectively (53). The Metropolitan Life Insurance tables of 'desirable weight' have most commonly been used as the standard; however, the designations of 'small', 'medium', and 'large' frame sizes are not based on specific measurements, and thus allow some subjectivity with its use. The measure of weight-for-height that is most frequently used in the assessment of obesity or overweight is known as the Quetelet or body mass index (BMI). This has units of kilogram per

meter squared (kg/m^2). For women, overweight is defined as a BMI of 27.3 or greater, and for men, as 27.8 or greater (5). A BMI of 31.1 is considered obese for women, while for men this value is 32.3 (5). Between the ages of 20 and 50, the fat content in men approximately doubles, while in women it increases by about 50 %. Total body weight does not increase proportionally, however, reflecting a reduction in lean body mass.

Obesity has been classified according to at least three different schemes: 1) anatomic characteristics and regional distribution of adipose tissue 2) the age at onset 3) etiological factors (38). The scheme involving anatomic characteristics pertains primarily to the suggestion of distinguishing between those mainly with enlarged fat cells (hypertrophic obesity) and those mainly with an increased number of fat cells (hyperplastic obesity) (62). The regional distribution scheme of classification deals with comparing the amounts of fat on different areas of the body, based on the idea that different patterns are associated with a greater risk of complications (63). It was found that when fat was stored predominantly in the gluteal or femoral area (gynoid obesity) (most prevalent in females), there were fewer complications than when it was stored primarily in the abdominal area (android obesity, typical of fat deposition in males). The waist-to-hip ratio has been used as a measure of regional fat distribution and

associated risk (63). The classification of obesity by age-at-onset distinguishes between childhood-onset and adult-onset, and are usually synonomous with the classifications of hyperplastic and hypertrophic obesity, respectively. It is recognized that there may be different sets of complications depending on when obesity is acquired (38).

The human body can be discussed in terms of two components, the fat and fat-free masses. In men, adipose tissue accounts for approximately 28% of the body weight, while in women, this comprises roughly 40% of total weight (64).

Fat has a density of .9 g/ml and the fat-free mass for most individuals has a density of 1.10 g/ml; therefore the ratio of fat to fat-free mass can be estimated by determining the density of the whole body. This procedure is called hydrodensitometry, and is considered the most accurate method for the determination of body composition. However, because it is costly, cumbersome, and time consuming, it is impractical for routine use. More conventional methods used to estimate body fatness include skinfold measurements and body or limb circumferences. Accurate determination of fatness in obese individuals is difficult with these methods, due to natural variations in adipose tissue compressibility, and site-specific accumulation of adipose tissue (65, 66). The impracticality of hydrodensitometry in field situations illuminates the

need for a rapid, precise, and relatively inexpensive method to determine body composition. In the assessment of change in body composition, a method need not necessarily be accurate, but precision over time within the population is essential. One method which can be used to accomplish this, and which measures water content, is easy to use and provides reasonably accurate results, is the bioelectrical impedance analyzer (31).

Its use in the assessment of body composition rests on the finding by Pace and Rathburn that water is not stored in adipose tissue and that fat-free mass contains a rather constant percentage (73.2%) of water (67). Its two underlying principles are: that the conduction of an electrical impulse through a body is via the water and electrolytes contained in the fat-free component, while the fat acts as an insulator to this current; and that the impedance of a geometrical system is related to conductor length and configuration, its cross-sectional area, and signal frequency. Glassford has developed the technique so that both body density and lean body mass are utilized in the prediction of body fat. It is reported that this approach yields better results on both lean and obese subjects, which had previously been overestimated, and underestimated in fat content, respectively (68, 69, 70). This problem is reported to have been overcome by the development of regression equations from the particular

population of subjects under consideration (71, 72). In the further refinement of such formulas, the incorporation of weight into the regression equation has been found to improve the correlation of BIA-determined TBW with that determined by deuterium isotope dilution (73), while integration of age into the regression formula has enhanced correlations between per cent body fat predicted by BIA and that determined by hydrostatic weighing (72). Several investigators have found that the use of the variables: height, weight, gender and age in regression equations which predict body fat have yielded high correlations with values determined by each of the 'gold standard' methods, hydro-densitometry or deuterium isotope dilution (70, 71, 72, 74).

Several factors which may affect the outcome of the bioelectrical impedance analyzer have been identified; they are: the stage of the menstrual cycle, excessive sweating, breath-holding, improper electrode placement and body temperature changes (75). However, Hoffer, in 1969 (76), found a correlation of .91 between TBW determined by tritiated water and by impedance, for 34 patients with varying degrees of hydration. This correlation was improved to .93 when the subjects' height was included: (TBW vs ht^2/R) in the regression analysis.

Another problem with the use of the bioelectrical impedance analyzer is that it has been found to underestimate body fat (68, 69, 70) as well as

overestimate TBW (70) in obese individuals. However, through the incorporation of height, weight, and gender into regression equations, high correlations between hydrodensitometrically- and BIA-determined LBM have been found. For example, Segal et al found this correlation to be .96 for a group of males and females who ranged from five to thirty-five per cent body fat (71). Lukaski et al (77) reported correlations of .98 and .95 for men and women, respectively, between densitometrically-determined fat-free mass and BIA-determined fat-free mass, in subjects ranging from four to forty-one per cent body fat. Kushner and Schoeller (73) found that equations incorporating ht^2/R, weight and sex used with the BIA method were effective in predicting deuterium-determined TBW (.98 and .95 for obese and non-obese female subjects, respectively). Segal et al (78) conducted a study which involved 1567 subjects, ranging from three to fifty-six per cent body fat, found that fatness-specific equations -which also incorporated height, resistance, weight and age- considerably improved previous correlations for both LBM and per cent fat determined by bioelectrical impedance with values determined by densitometry. Furthermore, in women, the correlation of lean body mass between the two methods was greater for the obese than the normal subjects (.95 vs. .91). Still another study has yielded a correlation of .98 between body fat determined by bioelectrical impedance analysis and that

determined by hydrodensitometry for 56 adult males and females (79).

In addition to accuracy, the precision, or reliability, of an instrument should be considered, especially where the intent is to assess change over time. Reliability with the BIA has been reported by Lawlor et al to be .991 and .996 for measurements made one and twenty-four hours apart, respectively (72), while values ranging from .990 to .999 for single and double measurements have been reported by other investigators (74, 80, 81). Estimates of variation for individual replicate measurements have been reported as 1.3% (with a range of .3 to 1.9%) for same-day testing, with week-to-week variability slightly larger (2.2%) (70, 73).

Therefore, if one is careful in the screening procedures to assure normal fluid balance, stage of the menstrual cycle and proper electrode placement, one should be comfortable in the use of the BIA in the assessment of changes in body composition in individuals within a population.

Etiology

The etiology of obesity is now recognized as a heterogenous disorder (29) for which genetic components may play important roles (82). The various factors which have been implicated in the etiology of obesity include alterations in

food intake as well as deviations in the components of energy expenditure: resting or basal metabolic rate (RMR or BMR), the thermic effects of food (TEF), physical activity, and possibly the thermic effects of exercise (TEE).

Regarding food intake in the obese, several authors, in their reviews of the literature, have reported that obese persons in fact consumed less calories on average than lean persons (31, 42, 55), and this had also been reported for infants and animals (41). There has been some concern, however, with the reliability of these estimates when based on self-reports, since at least two investigators found an under-reporting of caloric intake in obese individuals (83, 84). The fact that there was a wide variation in the ability of people (85, 86,) and animals (87) to gain weight, which has been found to be unrelated to body weight or fat content, duration of overfeeding or the amount or type of food consumed (86), has suggested that overconsumption may well be atypical for many obese individuals. Two other aspects of dietary intake which have received attention in the pathogenesis of obesity are the composition of the diet, mainly considering the amount or proportion of dietary fat and sugar, and the size and frequency of meals. Experiments in rodents have found that when the diet is high in fat or sugar, more calories are ingested than with regular diets, and obesity develops (88, 89). An inability to compensate for changes in caloric density has been noted in obese

persons, and was found to be of greater magnitude than in normal-weight counterparts (90). Recent evidence has indicated a positive correlation between fat intake, especially saturated fat, and BMI in middle-aged women (43).

It has been observed that obese individuals eat fewer (91) and larger (92) meals than non-obese persons. There are several implications for the development of obesity and its complications with this type of meal pattern. Bray (93) has demonstrated enzymatic changes which promote lipogenesis in women consuming a single large meal per day compared to those consuming several smaller meals. LeBlanc and Diamond (94) have demonstrated in dogs that the thermic effect of a single large meal as a percentage of total kcal was less than when an equal amount of food was divided into four smaller meals. Furthermore, the metabolism of glucose (93) and cholesterol (95) in humans have been shown to improve upon consumption of smaller, more frequent meals in humans.

Energy expenditure can be partitioned into the RMR, TEF, physical activity, and the TEE (31, 55). The basal, or resting metabolic rate (BMR or RMR) is the energy needed to maintain the body in physiological equilibrium in an unstressed state. This accounts for the majority of energy expedniture; estimates range form 60 to 70 (31), and to 75% (96) of the total. The total RMR of obese persons is typically higher than that of the non-obese, but this has generally been accounted for by the increased fat-free mass

of the obese (97, 98, 99). That a low RMR, on a per-kg of lean body weight basis, may be a factor in the development of obesity (100, 101), or at least for a subgroup of individuals (96), has been postulated. It was first documented 70 years ago that, following caloric restriction, there was a decline in RMR, and hence a limitation to weight loss (102). Since then, this has also been documented in non-obese individuals (97), but most notably in obese persons (97, 103, 104, 105, 106). Several researchers have found that the percentage decrease in RMR or 24-hour energy expenditure for obese subjects who have lost weight was greater than would be expected from the decrease in weight (97, 103, 104) or lean body mass (96, 106, 107). This reduction in RMR following weight loss has been observed to be sustained for up to six years (107).

The thermic effect of food, or thermic effect of a meal (TEF or TEM), is the increase in energy expenditure which occurs when nutrients are consumed. Part of this is due to the energy required for digestion, absorption and storage, and part is the loss of metabolizable energy as heat. There is a body of evidence (96, 108, 109, 110, 111) which indicates that, at least in some people, a lower thermogenic response to food may be a factor leading to increased storage of energy, and thus obesity. A lower TEF in post-obese persons compared to lean controls has been demonstrated in response to both a carbohydrate (111, 112,

113), and a mixed meal (114). A closer look at the occurrence of a blunted thermogenic response to food in the obese has shown that there is an inverse relationship with the degree of insulin resistance or glucose intolerance (55), suggesting a physiological mechanism for a lowered TEF.

Physical activity, as mentioned previously, has been observed to be lower in some obese persons compared to lean controls (38, 45, 46, 115, 116, 117). That this may be an important factor in the pathogenesis of obesity has been suggested by more than one author (38, 55). However, since an obese person must expend more energy for a given movement than a lean individual (55), and, as noted previously, the BMR of the obese is typically greater than that of their lean counterparts, comparisons of total energy expenditure are pertinent, but frought with difficulties.

Devlin and Horton (118) have shown that the increase in energy expenditure following exercise in lean individuals was not present in their obese counterparts, while evidence from another study indicated that exercise may have potentiated the thermic effect of food in lean, but not obese individuals (119).

There is now abundant evidence for the role of genetics in the etiology of obesity (31, 82). In evolutionary terms, obesity has been argued by several authors to be a result of what was once an adaptive set of mechanisms: the storage of

nutrients during brief times of food plentitude, the ability to subsist on food of poor nutritional quality, and a decrease in energy expenditure when food was scarce (55, 97, 120). Beyond the identification of a number of rare genetic forms of obesity (38), genetic factors have been implicated in all of the plausible etiological factors of obesity which have been discussed here (82).

Some of the first evidence that there may be a genetic component to obesity came from adoption and twin studies. Adoption studies have found a strong relationship between the weight of adoptees and their biological parents, and the lack of such a relatioship between adoptees and their adoptive parents (121, 122). This relationship has also been found to exist between adoptees and their biological siblings, but not between adoptees and their siblings by adoption (123). Furthermore, an adoption study which considered both full and half siblings of adoptees found that there was a stronger correlation between the BMI values of adoptees and their full biological siblings (124), than between adoptees and their biological half siblings.

Data from twin studies have provided further definitive information on the role of genetics in human obesity. The rationale is that identical twins (monozygotic) twins have the same genetic makeup, whereas fraternal (dizygotic) twins have only half of their gene pool in common. The largest such study thus far considered 1974 monozygotic and 2097

dizygotic male twins (125). When ranked by percent overweight, beginning with 15% overweight and grouped into increments of five per cent, up to 40% overweight, the concordance rates for all degrees of overweight were found to be twice as high for monozygotic as dizygotic twins. This greater concordance of obesity in monozygotic versus dizygotic twins has been supported by other studies (126, 127). An increase in correlation with an increase in genetic relatedness was shown for BMI in various pairs of relatives in a recent study (123). Twin studies have also indicated that there were strong intrapair correlations in the responses of: body weight, skinfolds, fat mass and body fat distribution to experimental overfeeding (120, 128). Such information has made possible the estimation of the heritability of various measures of obesity (8, 123), as well as obesity itself (8). Furthermore, there was evidence for a genetic contribution to the various components of energy expenditure implicated in the etiology of obesity. Genetic components have been recognized for: RMR (82, 100), TEE and TEM (82), as well as physical activity (129) and possibly some components of energy intake (130). Studies have also revealed genetic contributions to the responses in RMR, TEE and TEM to overfeeding (82, 128). Such information has led researchers to speculate on the precise mode of inheritance of obesity, or factors contributing to its development (120). Several models have been discussed (8).

It would appear that the current thinking regarding the interaction between the genetic and environmental contributions to obesity is that one inherits a certain predisposition to become overweight, and that the environment influences its manifestation (8, 82).

Given the range of phenomena which have been implicated to have a role in the pathogenesis of obesity, it is clear that obesity is of multifactorial origin. At present, the relative importance of the various mechanisms in the etiology of obesity is unclear. It is likely that the etiology, or etiologies, of obesity vary widely from individual to individual, so that treatment efforts should be of a multidimensional approach (at least until it may become possible to determine pathogenecity on an individual basis).

Treatment

The treatment of obesity now encompasses a variety of therapeutic approaches. Most methods can be classified under one of five general headings: diet, behavior modification, exercise, pharmacotherapy and surgery. Conservative treatments are those other than invasive methods such as surgical procedures, or more radical methods such as pharmacotherapy or very low-calorie diets which serve to take the locus of control away from the overweight

individual (56). The conservative treatments will be the focus here, although it is recognized that less conservative approaches may have their place in the treatment of certain obesities, or perhaps most appropriately, when combined with more conservative treatments.

As obesity was for a long while considered to be the result of overeating, initial treatment efforts logically focused on restricting caloric intake. Early reports by Stunkard et al, and Wing et al, indicated that such efforts had met with little success (50, 51). Evidence since then has indicated some success with balanced low-calorie diets (49, 131). However, several authors, in their reviews of the literature, have cited low overall weight losses and poor long-term adherence to be typical of such programs (52, 53, 132). Atkinson (133) has identified several difficulties with such diets: the abundance of highly palatable foods in the U.S., combined with the stimuli to eat produced by environmental cues, habits, and social pressures; the tendency of obese individuals to undergo mechanisms to maintain a higher body weight; and the typically high level of fat consumed which facilitates the ingestion of a high number of calories, while fat is calorically less costly to store. Perhaps these, as well as other dificulties, have contributed to the relatively low efficacy of most low-calorie diets.

During the past 20 years or more, behavioral

modification programs have become the most widely used therapies for the treatment of mild obesity (134). Following the concepts and techniques as originally put forth by Fester and colleagues (135), Stuart (136), in 1971, reported a study which demonstrated the efficacy of this method. The core procedures utilized in early behavior modification trials were self-monitoring and stimulus control (134), primarily as pertaining to eating behavior. Later, reinforcement techniques were frequently incorporated. Currently, behavioral management programs typically emphasize nutrition education, exercise, social support and cognitive restructuring in addition to the original three components (134). Such amalgamation has taken place gradually, incorporating measures from the various types of programs (56). The idea behind behavioral treatment of obesity is that of changing habits or behaviors. Originally this dealt almost exclusively with eating behaviors; today most programs apply behavioral principles to a variety of areas in order to affect a reduction in weight. Several authors have reported summary statements based on reviews of the literature regarding behavioral programs (49, 52, 137,). They have concluded that: there is a wide individual variation in weight loss; group results are consistently better than those for other treatments; weight loss is moderate, averaging one pound per week or 10-12 pounds for the duration of the program; there

is little additional weight lost after treatment; attrition rates are considerably less than other programs, (15% versus 25-75); and there are no negative side effects (49, 52, 137). Long-term weight maintenance has been reported to be better than other programs (49), and to be sustained for up to a year and a half following treatment (137). Since their inception, the short-term results of behavioral programs have improved (134). This was believed to be due to the comprehensiveness and refinement of programs, as well as the increase in their duration (134). Brownell and Jeffery (138) have compiled results from several studies, and the long-term results at one and two years look promising. Beyond this however, a trend toward weight regain was obvious. This trend was also evident in several other investigations (87, 139, 140). There has been some research into understanding and preventing relapse (141, 142, 143, 144), which is important as there is some indication that previous attempts at weight loss have a poor prognosis for subsequent weight loss (145), and that there can be detrimental effects of repeated weight loss and regain (146). It has been found that increasing treatment length and incorporating aerobic exercise may be of some value (134). Other approaches to behavior modification have included psychoanalysis, work-site programs, and various commmercial and noncommercial self-help programs (49). Attrition has been cited as a major drawback to these latter

two approaches, although programs have varied widely, and studies on their effectiveness is limited. An interesting twist to the work-site approach is the weight loss competition. In contrast to behavioral-based work-site programs, weight loss competitions have been found to have high participation rates, low attrition rates, and to result in moderate weight loss as well as improvements in morale and social functioning. They were also the most cost effective of various programs (147).

Physical activity has also been investigated as a treatment for obesity. Weight loss has been found to occur with exercise alone in obese individuals (148, 149, 150), although the opposite effect has also been observed (151). It may be that there is a certain level of frequency, intensity, or duration which is necessary to achieve an effect, while caloric intake is certainly an important factor (119). Overall, it would appear that exercise alone has produced only modest decreases in body weight (53), but has been frequently associated with beneficial changes in body composition (52). This has been especially true when combined with caloric restriction. Exercise has been shown to offset the diet-induced declines in resting metabolic rate (53, 59) and lean body mass (49).

The effects of the addition of physical activity to caloric restriction on body weight are not clear; greater weight loss (53) as well as no change in weight loss (31)

have been reported. However, when exercise was combined with behavior modification, Dahlkoetter and colleagues observed a greater weight loss than with either therapy alone (152). Exercise has been cited as the best predictor of success in a comprehensive weight loss program (54), and to be an important factor in long-term weight maintenance (49, 153). It has been found that exercise can increase the thermic effect of a meal, although this may be blunted in some obese individuals (59, 119). It has been reported that caloric compensation in response to exercise may be incomplete in obese persons, such that a negative energy balance results (154). Improvements in glucose tolerance and insulin sensitivity (155), as well as plasma TG and lipoprotein-cholesterol levels have often occurred in response to exercise (156, 157). Moreover, exercise may serve to promote a sense of well-being and self-esteem, which could be an important stimulus to adherence in a weight control program (55).

These various treatments for obestiy have traditionally been administered independently, and have been assessed based on their relative cost, risk, and short- and long-term efficacies (49). The amalgamation of nutrition education and aerobic exercise has proven to be a successful approach to weight loss (14, 158). Recently, several researchers have suggested that a comprehensive program, which incorporates nutrition education, behavior

modification and exercise is most likely to produce the most effective long-term control of weight (53, 57, 59). This has been substantiated by severel recent studies (12, 13, 61). Although information is limited, a few investigations have indicated that such conservative treatments can be of value in black populations (13, 14). Given the heterogenous nature of the pathogenesis of obesity, it would seem logical that the best treatment approach would be comprehensive in nature, encompassing nutrition education, behaviour modification, and aerobic exercise in a manner which promotes permanent changes in habits.

Walking and Aerobic Dance

There are several modes of endurance exercise which are typically recommended for obese individuals in a weight loss program, and Sheldahl has discussed some of these (159). The American College of Sports Medicine (ACSM) recommennds that when exercise is prescribed for the primary purpose of promoting weight loss, a minimum of 300 kcal be expended per session. Regarding the obese patient, the ACSM states that exercise prescription should emphasize caloric expenditure, and that intensity should be prescribed at approximately 50% of functional capacity, with the duration adjusted, dependent upon body weight, in order to elicit a caloric

deficit of 1750 calories, or .25 kg, per week (160).

Several studies have examined the impact of walking programs on cardiorespiratory and body composition parameters (148, 161, 162, 163). Leon et al (148) found that 16 weeks of vigorous walking for 90 minutes, five days per week on a treadmill, in which an average of 1100 kcals were expended per session, resulted in improvements in cardiovascular parameters, work capacity, and body composition, as well as HDL levels and the insulin-to-glucose ratio in obese sedentary men. Shoenfeld et al (162) found significant improvements in aerobic capacity in all three groups of subjects who walked for three or four weeks with loads of either: three kilograms, six kilograms; or a combination of the two, for 30 minutes per session, five days per week. Lewis and colleagues (164) in 1976 found that overweight women who participated in a walk/jog/calisthenic program and self-determined caloric restriction, significantly reduced their relative body fat and weight, while increasing their lean body mass. At least two investigations by Pollock and coworkers (161, 163) have shown that walking programs resulted improvements in: aerobic capacity, as measured by VO_2 max and resting heart rate; body composition, as measured by body weight and skinfold thickness; and working capacity as measured by mile walk time. Furthermore, the effects of a walking program on aerobic capacity and body composition has been compared to

those of running and cycling programs. Pollock (161) found similar improvements when frequency, intensity and duration were held constant.

Although beneficial effects can be realized with a walking program, exercise compliance must be achieved. Probably the greatest drawback to a program solely involving walking is that it can become boring to many individuals. This was epitomized by Kenrick, who stated that "...monotony was the biggest problem" with their treadmill protocol (165). Therefore, the need to recognize and counteract such boredom in an exercise program may be tantamount to participant continuation, since this is often cited as a factor in attrition from exercise programs (159). Alternative activities can be important elements in various fitness programs. Games are social, which can provide an opportunity for competition, comraderie and group interaction. They can also be effective in maintaining participant interest, improving flexibility and coordination, and in teaching activities and skills which can be enjoyed and perpetuated throughout life. Moody and coworkers noted that when potentially monotonous endurance activities are performed with a group, and individuals are allowed a choice of daily routines that 'the group participation motive and mutual moral support was an important element in exercise adherence', which was instrumental in the attainment of weight and body fat loss

for a group of overweight women (166). More recently, results obtained by the addition of a structured aerobic exercise program to a dietary weight loss program for obese women suggest that the exercise program served to increase group cohesiveness, as well as communication and information exchange among participants, which contributed to greater weight loss and nutrition class attendance for exercising individuals (167).

Aerobic dance is another form of exercise that may be recommended for overweight persons. Commonly referred to as 'aerobics', this was first introduced by Jacki Sorensen in 1972 as a means of improving cardiorespiratory endurance (168). A real advantage and appeal of this form of exercise lies in its diversity. It can be performed at the commonly designated intensities of low, medium and high, or any combination in between, as best suits the needs of the class or the individual. Additionally, participants can be taught to alter their movements to attain a prescribed intensity, which can be self-monitored by taking the heart rate. In 1986 it was estimated that 23 million people regularly engage in some form of aerobic dance, and that the lowest intensity form of aerobics was becoming increasingly popular (169). The designation as low-impact indicates that all movements are performed with at least one foot on the ground at all times is generally recommended for overweight, low-fit, or those individuals with skeletal problems, as it

decreases the chance of injuries.

The results of several other investigations have provided evidence that aerobic dance is capable of producing a training effect, as evidenced through increased cardiorespiratory capacities such as: VE max (l/min); VO_2 max (l/min), or (ml/kg/min); decreased submaximal or resting heart rate (170, 171, 172, 173, 174, 175, 176); and/or improvement in performance measured by maximal work capacity (171), or max work time (175, 177).

Investigation specifically into the physiological effects of low-impact aerobics indicates that it, too, can be an effective exercise modality. A study which compared the effects of aerobic dance at intensities of 65-70% of maximal heart rate vs. 80-90% of maximal heart rate suggested that those working at the lower intensity can burn fat as effectively as those working at the higher intensity, if the activity is performed long enough (169). And an unpublished study found that a ten-week program of low-impact aerobics produced a significant decline in resting heart rate, systolic and diastolic blood pressure, increased stroke volume, and decreased body fat in 15 women aged 15 to 34 (169).

The work of Cearly and Moffatt (172) suggested the existence of a threshold level for the frequency of aerobic exercise which must be attained before improvements in cardiorespiratory function become manifest. They found

increases in VO_2 max and VE max in the group of college students that participated in the two-hour aerobic dance program three times per week, but not in the group participating two times per week, both groups for ten weeks. Improvements in exercise tolerance, as measured by treadmill time to exhaustion, corresponded to the amounts of time spent in the aerobic dance program; the 3-day/wk group made greater improvements than the 2-day/wk group, which improved significantly more than the control group.

Most of these investigations have not yielded encouraging results regarding the effect of such aerobic dance programs on body weight (171-175, 177). However, neither did any of these studies consider the interaction of diet. Body composition changes during aerobic exercise programs have been less frequently evaluated than body weight. At least three studies have found no change in body composition (173-175) while two other studies resulted in reductions in body fat, as measured by changes in skinfold thicknesses (170, 176). An unpublished study found that a ten-week program of low-impact aerobics resulted in a decline of 2.5% bodyfat (169). The few studies which have investigated the interaction of dietary restriction with aerobic dance have reported beneficial changes in body composition. Zuti and Golding used equal energy deficits to compare the effects of either: exercise alone (one hour per day, five days per week for 16 weeks); dietary restriction

alone; or the combination of these two protocols (178). They found that although similar amounts of weight were lost among the three groups of subjects, that both of the exercising groups lost a greater amount of fat than the diet-only group, and that the exercisers had increases in lean body mass, whereas the diet-only group lost lean body mass, as determined by underwater weighing. The subjects were 25 women, aged 25 to 42, and were overweight by 20 to 40 pounds. Sikand et al (167) have examined the effects of a structured aerobic exercise program twice weekly for 16 weeks to the addition of a very-low-calorie diet in thirty obese women, aged 21 to 60. Both exercising and non-exercising groups lost significant amounts of weight and body fat. The exercisers lost a greater proportion of their weight in fat compared to the non-exercisers, although differences between groups were not significant. Significant differences were noted, however, in the latter half of the treatment period for weight loss, with the exercise group continuing to lose weight, while the non-exercising group exhibited a decline in the rate of weight lost.

These investigations support idea that aerobic dance, at least when in conjunction with dietary restriction, as well as a program of walking, could prove to be an acceptable means of decreasing weight and improving body composition and physical fitness in overweight or obese

individuals. Observations suggest that the social interaction and group structure of aerobic dance may be important elements in its effectiveness.

The effects of exercise-with-diet versus caloric restriction alone on body composition

In contrast to the relatively small amount of research into the effects of aerobic dance on cardiovascular or body composition parameters among participants, there has been more research conducted regarding such effects of more traditional forms of aerobic exercise. Exercise has been shown in a number of studies, to elict body weight, and particularly body fat, losses (179). However, some research suggests that exercise alone may not produce a significant decrease in total body weight, which is due to an increase in lean body mass concommitant to a decrease in body fat (150). Other investigations have found no change in body fat, but an increase in body weight (180, 181), due to an increase in lean tissue, following exercise programs.

The combination of exercise with dietary restriction has been found by more than one investigator to result in greater losses of both body fat and body weight. In 1985, Lennon and coworkers considered changes in body weight and per cent body fat resulting from either: self-selected, or prescribed exercise with diet, or diet alone in overweight

individuals (182). The regimen of prescribed exercise with diet resulted in the greatest decrements in body weight and per cent body weight, as well as per cent body fat. And in 1987, Wirth et al found that the addition of physical training to a very-low-calorie diet resulted in greater declines in: body weight, per cent body weight, body fat, and per cent body fat than a hypocaloric diet alone (183). However,the results of a study reported in 1984 suggest that the addition of exercise to dietary control enhances fat, but not total body weight loss (184). This apparent lack of effect of exercise on total body weight may be more appropriately evaluated by considering changes in lean body mass.

In 1985, Sopko and coworkers provided equal energy deficits, which were based on individual requirements, as well as fixed nutrient composition of the diet for all groups in their study of the effects of exercise, diet, or diet-with-exercise in 21 sedentary men (185). They found that although dietary weight loss produced the greatest decrements in total body weight, that exercise-with-diet produced the greatest decrements in per cent body fat, as determined by hydrostatic weighing. Neither the exercise-only nor the control group exhibited significant decrements in either body weight or body fat. Although it is not readily apparent from the authors' reported results, calculation reveals that the exercise with dietary regimen

further improved body composition by increasing lean body mass, whereas the diet-only group experienced a decrease of lean body mass.

In 1987, Belko and coworkers employed equal energy deficits and equal dietary compositions between diet-only and diet-with-exercise groups. They found that diet alone produced a greater weight loss, but that fat losses did not differ from the diet-with-exercise group, such that a greater proportion of weight loss was due to fat loss and less from the loss of lean tissue, in the exercising group (186). At least three other studies, published between 1985 and 1987, which compared the effects of diet-with-exercise vs. diet found that weight losses were similar between the two protocols, but that the addition of exercise improved the proportion of weight lost as fat, while either decreasing the proportion lost from fat-free mass (187, 188), or increasing the fat-free mass (189) in obese individuals.

The Effects of Exercise and/or Dietary Restriction on Serum Lipoprotein Cholesterol and Triglycerides

Obesity has been associated with increased levels of triglyceride (18, 190, 191, 192, 193), and total cholesterol (190, 194, 195), as well as decreased levels of HDL-C (18, 196); a profile that is condidered predictive of CHD (34,

197). Elevated total cholesterol (TC) levels have long been recognized as an increased risk for the development of atherosclerosis and subsequent coronary event (197, 198, 199, 200). However, the distinction has since been made between subfractions of cholesterol which are based upon various densities (201, 202). The four different classes which are recognized are termed: low-density lipoprotein cholesterol (LDL-C), very-low-density lipoprotein cholesterol (VLDL-C), intermediate-low-density lipoprotein cholesterol (IDL-C), and high-density lipoprotein cholesterol (HDL-C). Even more recently, two major classes of HDL have been identified, called HDL_2; and HDL_3 (203). HDL-C is believed to be involved in the transport mechanism of cholesterol from tissues, (including arterial walls), to the liver for degradation and excretion (204, 205, 206), while LDL and its precursor, VLDL, have been shown to be involved in the transport of cholesterol to the arterial intima (206), thus leading to the development of atherosclerotic plaque. Support for this concept comes from angiographic studies which have shown the severity of coronary artery disease to be positively associated with LDL-C concentration and negatively associated with HDL-C concentration (207, 208, 209, 210), as well as epidemiological data which reveal that populations with a lower incidence of coronary artery disease also have higher levels of HDL cholesterol and/or lower levels of LDL

cholesterol (34, 211, 212, 213, 214). Furthermore, several studies have found HDL-C to be a better predictor of CHD than LDL-C (34, 211, 212). The roles of HDL_2-C and HDL_3-C in the atherogenic process are not as well-established. It is believed that they undergo different metabolisms (215, 216), and that HDL_2 contributes a greater part of the variability in HDL (217).

Based on such evidence, it has been proposed that HDL and its HDL_2 subfraction should be used to predict the risk of CHD (210, 214), and it has therefore been suggested that increasing HDL levels (and/or decreasing LDL levels -such that the HDL/LDL ratio is increased,) rather than the non-specific reduction of total cholesterol may be the most appropriate approach in the prevention of athersclerosis and subsequent myocardial infarction (204, 212). In fact, measurements of angiographic changes made over a five-year period have indicated that changes in the ratios of HDL-C/TC and HDL-C/LDL-C were better predictors of angiographic dynamics than either variable alone (218). Furthermore, the American College of Sports Medicine (ASCM) recognizes a ratio of TC/HDL of greater than 5 to be a major coronary risk factor (160).

Like TC and LDL levels, triglyceride (TG) levels have also been found to be positively associated with coronary artery disease (CAD) (219, 220). Although the association has not typically been as strong or as consistent as for

those of cholesterol (211), studies have shown that 20-30% of individuals with CHD also have hypertriglyceridemia (221, 222, 223, 224). This would suggest that a high level of TG is associated with the atherogenic process, even if it is not an independent risk factor.

The effects of caloric restriction on serum lipoprotein cholesterol and triglycerides

The effects of caloric restriction alone on lipoprotein profiles have been widely studied. Improvements have frequently been observed in TC, LDL, and/or VLDL fractions in obese individuals following hypocaloric diets (225, 226, 227, 228, 229). Triglyceride levels have also been found to be decreased following caloric restriction (225, 226, 230), while the effects on HDL-C have been more variable: increases (226, 229, 231), decreases (225-227, 230, 232, 233), as well as no change (228, 234) have been observed in obese subjects. Studies which have conducted follow-up evaluations, however, have often found subsequent changes in the magnitude and/or direction of one or more lipid parameters (227, 230, 235, 236, 237), particularly HDL-C. HDL-C levels have been found to be increased above post-treatment levels at follow-up evaluations conducted between two and eight months after treatment (228, 230, 233). The frequency of this occurrence has led to the suggestion that

the mechanisms affecting lipoprotein levels, particularly HDL, may be different during active, versus stable weight loss (227, 232). Where measured, changes in the HDL_2 subfraction have been found to be primarily responsible for changes in HDL-C during diet-only protocols (238).

The effects of exercise on serum lipoprotein cholesterol and triglycerides

Substantial information also exists regarding the effects of exercise upon serum lipoprotein cholesterol and triglyceride. At least as early 1973, there was evidence which suggested that exercise could produce favorable changes in blood lipid profiles (239), and by 1981 it was generally believed that regular physical activity would reduce TC, LDL cholesterol, and TG, but elevate HDL cholesterol (156, 157, 240). In 1983, an analysis which considered the results of 66 training studies conducted over the 26 years prior to December 1981, was published (241). It was found that, across all types of subjects, treatments, sources, and research designs, exercising subjects underwent significant reductions in TC, TG, LDL, and the TC/HDL ratio, as well as a non-significant increase in HDL, while none of these changes for the control groups were significant. The analysis also revealed that initial levels of TC, TG, HDL, and the TC/HDL ratio were strongly correlated with their

respective changes following training.

Work since this analysis has leant further espousal to its results. Wood and coworkers, in 1985 (242), reported significant decreases in total cholesterol and LDL-C, and a significant increase in HDL-C among fourteen middle-aged, sedentary men who participated in a progressive running program over a two-year period.

The effects of physical exercise on the blood lipids of healthy young men were investigated by Danner and coworkers in 1984 (243). After seven months of training, significant decreases in TC and TG and significant increases in HDL-C were found in 15 oursmen compared to 21 sedentary controls who were matched for age, as well as smoking and drinking habits.

Studies which have compared individuals based on their habitual activity levels have found better better lipoprotein profiles, especially higher HDL levels, in those persons who exercise regularly (244, 245, 246, 247, 248, 249, 250, 251, 252). When measured, the HDL_2 subfraction has also been found to be elevated (253, 254, 255). Lower levels of LDL-C (249, 251, 252, 256), VLDL (252), TG (249, 251, 252) and TC (249) have also been found to be typical of routinely active individuals.

One study by Adner and Castelli found that although TC levels were similar in active and sedentary individuals, that the HDL-C level was significantly higher, and thus the

TC/HDL-C ratio significantly lower, in the active, compared to the sedentary group (244). Furthermore, levels of HDL-C, TC, and the HDL-C/TC ratio have been found to be correlated to the amount of activity, as measured by the number of miles run per week in a group of marathon runners (249). Another study found that when runners ceased running, there were significant decreases in both HDL and HDL_2, concomitant to a significant increase in LDL (257).

The effects of exercise with caloric restriction, versus caloric restriction alone on serum lipoprotein cholesterol and triglycerides

Several studies have compared the effects of exercise-with-diet versus diet alone on plasma variables. Saldivar et al (258), and Hill and coworkers (188) have found similar effects on lipid parameters resulting from caloric restriction, either with, or without exercise. Two other investigations have shown the addition of exercise to caloric restriction to be more effective in eliciting a beneficial lipoprotein profile than caloric restriction alone. Lampman et al (259) evaluated the effects of the addition of an exercise program to a hypocaloric diet in severely obese individuals for six months. The exercise program consisted of fast walking and slow jogging for 45 minutes, three times per week, at an intensity of 75% of

maximal heart rate; the hypocaloric diet was described only as consisting of 600 kcal per day. It was found that TC decreased significantly only in the-diet-with exercise group, while triglycerides were not observed to change significantly in either group. LDL and HDL were not measured. Thompson and colleagues (257) took the reverse of this approach by implementing exercise cessation for the diet-only group, rather than initiating exercise for the exercise-with-diet group. All subjects consumed a baseline diet composed of 15% protein, 32% fat, and 53% carbohydrate; all ran 16 km daily during this baseline period. For a ten day experimental period, the control group continued running and consumed the same isocaloric baseline diet. The other two groups consumed 20% fewer calories of this same diet and either continued running, or ceased their training regimen. Significant atherogenic changes in HDL-C and HDL_2 were found upon exercise cessation, while continued training combined with caloric restriction was found to result in significant beneficial changes in HDL-C, HDL_2, and LDL-C.

The effects of exercise with caloric restriction, versus caloric restriction alone, versus exercise alone, on serum lipoprotein cholesterol and triglycerides

Although a substantial amount of research has been conducted on the effects of diet alone or exercise alone,

and a fair amount has been conducted on the combination of exercise with diet vs diet alone, there have been few well-controlled studies which have concomitantly examined the independent and interrelated effects of exercise and diet on changes in lipoprotein patterns. One such study was reported by Sopko in 1985 (185). Twenty-one obese sedentary male subjects were divided into four groups: inactive and constant weight (control); exercise and constant weight; inactive and weight loss; and exercise with weight loss. All consumed the same dietary composition, fixed at 45% complex carbohydrates, 15% protein, and 40% fat (p/s ratio = .2). The exercise protocol, treadmill walking, was designed to produce an energy deficit of 3500 kcal per week for each individual, while dietary restriction was set at 3500 kcal per week below individual isocaloric requirements. It was found that 12 week periods of either diet or exercise were equally effective in inducing increments in HDL-C, but that there was an additive effect for the combination of the two protocols in obese men. This synergistic effect of diet and exercise was also observed for levels of the TC/HDL ratio which were significantly decreased in the exercise-with-weight-loss group compared to inactive-with-weight-loss or exercise-and-constant-weight groups. The slight increases seen in TC, LDL-C and TG seen both with exercise and with diet were not significantly different, but significant decrements occurred in the group which combined diet with

exercise.

In 1986, Hagan et al (260) examined the independent and interrelated effects of exercise and a dietary protocol. Forty-eight males and 48 females, who were 120-140% of ideal body weight, were randomly assigned to groups (N = 12) of either: diet-exercise (DE), diet (D), exercise (E), or sedentary control. The exercise regimen consisted of a walk/run program five days per week for 30 minutes per session, while the dietary regimen was a 1200 kcal per day diet. No significant differences in any of the plasma variables were found among women. However, in males, significant decrements in TG and TC occurred in those who were dieting while exercising compared to diet-only, exercise-only, and control groups. It should be noted, however, that because the three protocols were not based on equal energy deficits, that both male and female groups of (DE) lost a significantly greater amount of body weight and percentage of body fat than either respective (D) or (E) groups. These factors could have affected the observed changes in plasma variables.

The Effects of Dietary Restriction and/or Exercise on Glucose and Insulin Metabolism

Obesity, and glucose and insulin levels

The association between obesity and high plasma insulin levels has been recognized at least since 1963 (261), and is often concomitant to some degree of insulin insensitivity (262, 263,264). This decreased insulin sensitivity is manifest in several ways: decreased peripheral glucose uptake, excessive hepatic glucose production in the basal state, and/or inadequate suppression of hepatic glucose output by exogenous insulin (262). Such associations between overweight and alterations in carbohydrate metabolism have been emphasized by demonstrated improvements and decrements in glucose tolerance or insulin resistance with weight loss (265, 266) and regain (266), respectively. Weight loss, both by dieting alone (267), as well as exercise alone (148), has been shown to be associated with improvements in carbohydrate metabolism in obese individuals. Exercise, however, even in the absence of weight loss (268), or even concomitant to weight and body fat gain (151), has resulted in improvements in glucose tolerance and insulin sensitivity.

The effects of exercise in conjunction with dietary restriction on glucose and insulin metabolism

Since both physical training and various forms of food intake modifications have been documented to be instrumental in the enhancement of insulin sensitivity in obesity, it should be of interest whether the combination of these two treatments results in an even more pronounced improvement in insulin sensitivity. At least four groups of investigators have found this to be true. Schteingart et al, in 1983 (269) investigated the effects of physical training in conjunction with dietary weight loss compared to dietary weight loss with occupational therapy in 16 severely obese (150-300% of ideal body weight) subjects for six months. Exercise consisted of fast walking and jogging for 45 minutes, three times a week at 75% of maximal heart rate. Diet was a uniform 600 kcal per day diet for all subjects; dietary compostion was not detailed. All subjects also received psychotherapy. At six months, both groups showed an improved sensitivity to insulin, but there was a 41% greater increase in response to insulin in the diet-plus-exercise group. The change in insulin sensitivity was closely associated with the improvement in aerobic capacity, only in the diet-plus-exercise group.

Lampman et al, in 1980 (270), investigated the effects of the addition of exercise to a calorically restricted type

IV hyperlipoproteinemia diet, for nine weeks, in 27 middle-aged men with type IV hyperlipoproteinemia. The diet consisted of: 20% protein; 40% fat; and 40% carbohydrate, of which 70% was starch, and 30% sugars. The p/s ratio was 1:1, and dietary cholesterol less than 350 mg per day. Intake was adjusted individually in each group to maintain a weight loss of one pound per week. The exercise regimen was reported to be based on walking/jogging for 30 minutes per day, three days per week, at 85% of maximum heart rate. Aerobic capacity increased by 12% in the exercising group, and although this did not reach statistical significance, the increase in total performance time did, and was significantly greater than for the non-exericising group. Only the exercise-plus-diet group displayed a significant reduction in fasting plasma insulin values. There was a strong correlation between the decline in fasting insulin and increases in aerobic capacity, only in the physically trained group.

Two other investigations, conducted in 1981 and 1987, (183, 271) examined the effects of the addition of a very-low-calorie diet to regular exercise on glucose and insulin metabolism in obese persons. Krotkiewski et al found that glucose tolerance deteriorated in the diet-only group, but was unchanged in the group which combined diet with exercise. Wirth and coworkers reported that fasting insulin levels decreased with the duration of both treatments, but

that this was more pronounced in the exercising group.

Collectively, these studies suggest that a balanced, calorically-limited diet in conjunction with physical exercise, is a better approach than diet alone for the improvement of insulin and glucose levels, and insulin sensitivity, in obese as well as hypertriglyceridemic patients. The beneficial effects of these protocols have also been observed in diabetic individuals and would suggest that they could conceivably act as preventive measures in obese individuals, given that such individuals appear to be at an increased risk for developing diabetes.

Effects of Food Components on Serum Cholesterol, Lipoprotein Cholesterol, Glucose, and Insulin

The effects of dietary fat and cholesterol on serum cholesterol and lipoproteins

The individual effects of dietary fat and cholesterol upon serum cholesterol have been recognized for at least 30 years (272). In fact, numerous studies by various researchers in the 1960's led to the development of prediction equations for the change in serum cholesterol based upon the percentage changes in dietary saturated, monounsaturated and polyunsaturated fatty acids, as well as the (absolute) change in dietary cholesterol (273, 274).

The contribution of the monounsaturates had later been found to contribute little to the overall regression equation, and so this coefficient was consequently dropped. Such equations indicated that both dietary cholesterol and saturated fat increased serum cholesterol, but that dietary cholesterol was about three times more effective than saturated fats, while polyunsaturated fats decreased serum cholesterol, but were about three-fourths as effective as saturated fats. Subsequent research has generally supported these findings (275, 276, 277, 278, 279, 280, 281). However, monounsaturates have since been found to have a more beneficial effect than previously believed (282). A diet high in monounsaturated fats has been found to be at least as effective as a low-fat, high carbohydrate diet in lowering plasma cholesterol (283) and monounsaturated fats have been found to be as effective as polyunsaturated fatty acids (PUFA) in lowering LDL-cholesterol, while apparently reducing HDL-C less frequently than polyunsaturates (281). Investigation into the specific effects of monunsaturates on plasma cholesterol parameters has stemmed from concerns regarding the safety of polyunsaturated fats which have been shown to cause carcinogenesis in rats (284), and to alter the composition of cell membranes (285). They have also been implicated in the lowering of HDL levels (286, 287, 288, 289). Conversely, diets with a low p/s ratio (low in PUFA, high in saturated fat) have been associated with

higher TC levels, but also higher HDL levels (290, 291). In view of the currently accepted roles of HDL- and LDL-cholesterol in the etiology of CHD (34), such findings highlight the importance of distinguishing between the effects of dietary fats and cholesterol on specific parameters of cholesterol metabolism.

Observational studies of populations consuming traditionally low-fat diets (292, 293) as well as such diets which were administered experimentally (286, 294), have exhibited lower levels of total and LDL-cholesterol, as well as lower levels of HDL and the HDL_2/HDL_3 ratio.

A study by Hulley et al, in 1977, which was designed to assesss the effect of an intervention program aimed at lowering conventional CHD risk factors (295) through decreased fat and cholesterol intake, found that HDL rose slightly compared to the control group. Changes in neither LDL-C nor TC were reported.

Many studies have indicated that most of the rise in serum cholesterol upon dietary cholesterol supplementation is due to LDL-cholesterol (290, 296, 297, 298, 299,300, 301, 302, 303). HDL-C concentrations have been unaffected (290, 296, 299, 300, 302, 304) and increased (297, 301, 305) upon cholesterol supplementation, as well as decreased upon cessation of egg consumption (306).

These positive relationships between both dietary cholesterol and saturated fat with serum cholesterol have

been found to be predictive of the risk of dying from CHD. The Western Electric Study in Chicago followed 1900 men for 20 years. It was found that the risk of dying from CHD was significantly related to the diet score (the sum of dietary cholesterol plus saturated fat minus polyunsaturated fat) (307). Similar conclusions were drawn in 1985 by Kushi and coworkers from a 20-year study of 1000 men from three different cohorts (308). The correlations between dietary scores and death from CHD were found to remain significant even after adjustment for other CHD risk factors.

Polyunsaturated fats

Although it was generally accepted by 1965 (273) that polyunsaturated fats were capable of lowering total serum cholesterol concentrations in man (309, 310, 311, 312), less was known about their effects on the cholesterol specifically associated with lipoproteins of various densities. Since then, the majority of published research has supported the role of polyunsaturated fats in lowering both LDL-C and HDL-C in their decretional effect upon total serum cholesterol (279, 286, 289, 313, 314, 315, 316, 317), while changing from a high to a low p/s ratio diet has been observed to result in increases of both LDL-C and HDL-C (318).

However, more recently published work by Weisweiler et

al, and Judd et al, has suggested that at least under some circumstances, diets rich in polyunsaturated fats can result in lower total cholesterol and LDL-C levels, while HDL-C levels remain unaffected (319, 320). Moreover, a population study has found a positive correlation coefficient between the dietary p/s ratio and HDL-cholesterol levels (321).

Irrespective of individual changes in LDL- and HDL-cholesterol levels, an improvement in the LDL/HDL cholesterol ratio has been found by many investigators to be a major advantage to diets high in polyunsaturated fat (279, 289, 316, 318, 319, 320). Several researchers have reported that high p/s ratio diets resulted in significantly decreased TG levels (279, 286, 289, 319), while increased levels following diets with a low p/s ratio have also been reported (317, 318). Where HDL_2-cholesterol levels have been measured, polyunsaturated fat diets have been found to lower (286), or not affect initial levels (320).

Monounsaturated fats

Investigation into the consequences of substituting monounsaturated fat for polyunsaturated fats was was born out of an incidental finding: in 1970 Keys reported that in Mediterranean countries, despite a rather high (40% of total calories) average fat intake, plasma cholesterol levels and CHD rates were relatively low (322). The typical diet in

these countries is high in olive oil, which consists primarily (77%) of monounsaturated fatty acids (primarily oleic acid). From the research into the effects of monounsaturated fatty acids on plasma lipids has emerged a rather consistent pattern: monounsaturated fats lower total cholesterol and LDL-C at least as much as polyunsaturates, while they also increase or maintain HDL-C levels, or decrease them less than polyunsaturates (281, 317, 323). This has also been found to be true when monounsaturated fats were compared with low-fat, high-carbohydrate diets in normal volunteers (283, 324, 325), and in diabetics (326). Plasma glucose levels have been found to be significantly lower on an olive- versus a corn-oil or baseline diet (317). Triglyceride levels have been found to be improved (decreased) by olive vs corn-oil (323). Perhaps one explanation for their typical improvement of lipoprotein-cholesterol is related to cholesterol metabolism; a positive correlation has been reported between monounsaturated fatty acid intake and the fractional catabolic rate (FCR) of LDL-C (327).

Carbohydrates

Dietary carbohydrates are perhaps most recognized for their roles in glucose and insulin metabolism. At least as early as 1935, Himsworth showed that isocaloric reduction of

carbohydrate (CHO) impaired, while increased CHO improved, oral glucose tolerance (328). Evidence of improvements in oral glucose tolerance as well in insulin sensitivity have been reported with high CHO diets in normal (329, 330, 331), obese (331, 332, 333, 334, 335), and those individuals with impaired glucose metabolism (331, 332). Following such diets, fasting glucose as well as insulin levels have been reported to decrease in the face of an unchanged insulin response to oral glucose, thus suggesting increased tissue sensitivity to insulin (332).

As long ago as 1930, evidence was presented which indicated that dietary carbohydrates could attenuate the hyperlipemic effects of fats in the diet (336). When compared to usual diets, high-carbohydrate diets have been observed to have beneficial effects on total cholesterol levels in normal individuals (337), those with type III hyperlipoproteinemia (338), as well as those with maturity-onset diabetes (339).

Where LDL-C and HDL-C have been measured, both have been found to contribute to the decrease in total cholesterol upon application of a high CHO diet (283, 294, 337-339, 340). This decrease in HDL-C is purported to be one of two major arguments against a recommendation to greatly increase the CHO content of the diet (341). The other major argument against greatly increasing carbohydrates in the diet is their hypertriglyceridemic

effect (341). This has been established in numerous studies (283, 331, 337, 342, 343, 344, 345, 346, 347, 348, 349), and has been shown to occur in a range of subjects: obese, with and without impaired glucose tolerance (331); hyperlipoproteinemics (347, 349); individuals with varying degrees of atherosclerosis (344, 346); diabetics (346); as well as normal individuals (283, 331, 337, 342, 343, 345, 347-349).

Simple carbohydrates, particularly sucrose

Carbohydrates can be seperated on the basis of their abilities to undergo digestion and absorption. Simple carbohydrates, or sugars, have fewer chemical bonds, and are digested more easily and absorbed more rapidly than complex carbohydrates.

In 1978, Macdonald reported the effects of a high sucrose diet (6 g/kg/day) for 19 days in nine healthy young volunteers. Following the dietary period, total cholesterol and TGs were found to be significantly elevated, and HDL-C and the HDL-C/TC ratio significantly lower, than initial values (350).

In 1979 Reiser et al compared the effects of six weeks of two natural-food diets differing only in that 30% of the calories were from either sucrose or cooked starch (351). For the group of 19 subjects, TG and TC levels were

significantly higher on the sucrose diet than on the starch diet.

In 1983, the consequences of switching from a diet in which 25-30% of the calories came from sucrose, to a 10% sucrose diet was reported (352). It was found that serum TGs decreased by 33% during the lower-sucrose period. This effect was more pronounced in obese than non-obese subjects.

Simple, vs more complex CHOs, have also been shown to elicit greater insulin responses, which is beleived to stimulate VLDL-C synthesis, and thus result in increased TG levels (353). Fujita et al found that glucose, when combined with gelatin (.6 gm per kg bodyweight of each), resulted in much higher insulin levels than did an equivalent amount of gelatin alone (345).

Crapo and coworkers, in 1976, studied the effects of glucose, sucrose, and various starches on postprandial glucose and insulin responses in 19 normal volunteers. Among the findings were: a) that the ingestion of raw starch resulted in 35-65 per cent lower insulin response, and a 44% lower plasma glucose response than did either glucose or sucrose ingestion; b) that glucose and sucrose elicted equivalent glucose responses, but that sucrose elicited a 20% greater insulin response; c) that different cooked starches differed in their elicitation of glucose and insulin responses; d) and that glucose responses were (40-

60%) lower, while insulin responses gererally similar, when the CHO was given as a meal versus a drink -this being true for all CHOs examined (354). Thus, the form of a diet, as well as constituent dietary variables are likely to affect glycemic responses. The credence of such work is further borne out by numerous epidemiological surveys which have found negative associations between sucrose intake and HDL-cholesterol (355, 356, 357, 358), and in particular its HDL_2 mass (359), as well as positive associations between sucrose intake and LDL-cholesterol (355) as well as TG levels (358).

Fiber

The consumption of a fibrous-type diet had been recognized at least thirty-five years ago to be associated with lower serum lipid concentrations (360). It has been suggested that the low cholesterol levels (361) and rates of CHD in African populations may be due to their high intakes of complex carbohydrates and fiber (362, 363). Burkitt and Trowell have observed that in Western countries, grain and cereal consumption has declined markedly during the past two centuries, while atherosclerosis, diabetes, hypertension, and cancer have increased (364). Moreover, several epidemiological studies have found an inverse correlation between dietary fiber intake and CHD (308, 365, 366, 367, 368). This has included the measurement of CHD by mortality

rates (308, 366, 368), symptomatic CHD (365), as well as coronary lesion growth (367). Such findings have incited further investigation into the effects of complex-carbohydrate, fibre-supplemented diets.

In 1976 Albrink et al examined the effect of a high-CHO diet given as high fiber food in seven young adults (369). They were fed alternatively in random order isocaloric high- and low-fiber diets for one week each in a double cross-over study. Both diets consisted of 70% CHO, 15% fat and 15% protein. In the high-fiber diet, whole foods such as baked beans, rice, and wheat bread were provided. In the other, the CHO consisted of glucose and dextrins in a liquid formula. This diet produced significant increases in both TC and TG from baseline, while the high-fiber diet resulted in significant decreases.

The effects of dietary fiber supplementation to both a fat- and CHO-modified diet as well as a 'typical US diet' were assessed by Lewis et al in 1981 (370). A 'Western" reference diet (diet A = 46% of energy from CHO, 40% from fat, 14% from protein) was compared with: a fat-modified diet (diet B = 59% of energy from CHO, 27% from fat, 14% from protein); with a fiber-supplemented, fat-modified diet (diet C = 59% of energy from CHO, 27% from fat, 14% from protein, fiber in the form of fruits, vegetables, and cereal fiber); and with a fiber-supplemented 'Western' diet (diet D = 47% of energy from CHO, 40% from fat, 14% from protein).

Subjects were 12 healthy monks living in a confined dwelling. All diets were fed for five weeks and all participants consumed all four diets. Compared to diet B, both fiber-supplemented diets C and D produced greater decrements in TC and LDL-C. These fiber-supplemented diets also resulted in significant decreases in both serum TG and VLDL-TG, whereas diet B (containing only 20, vs an average in diets C and D of 51 g fiber/2500 kcal per day) produced no change in serum TG and a slight (4.8%) increase in VLDL-TG. The two fibre-supplemented diets also produced less deleterious effects on HDL-C and HDL_2 than did diet B.

Work with particular types of soluble fibers has shown some to be more effective than others in the improvement of lipoprotein parameters. Pectin, for example, is a soluble fiber found in high concentrations in citrus fruits, as well as other fruits and vegetables that has proven to be of particular beneficience. A review published in 1987 considered studies which examined the effects of pectin or pectin-containing foods on the metabolism of cholesterol, lipoproteins and carbohydrate (371). The summary of 18 studies stated that in 14 of them, there were significant decreases in total cholesterol, which ranged from four to thirty-one per cent. Two of the studies reported insignificant decreases of three and eight per cent, while two studies reported no change in serum cholesterol. In none of the (five) studies in which it was

considered, did a reduction in HDL-C occur. This would suggest that the pectin-induced decrement in TC is largely accounted for by declines in LDL- or VLDL-C. It was found that TG levels were unaffected by pectin, and while there were decreases in insulin responses, they were usually less marked. The analysis of these studies has led Reiser to conclude that there is some evidence that the cholesterol-lowering effect of pectin is dose-dependent, and that there is a greater effect in those with higher cholesterol levels.

Protein

The National Research Council's recommendation for dietary protein of .8 g per kg of body wieght has remained little changed since 1953 (372, 373, 374). Perhaps because we recognize a distinct role for protein, as that of forming the structural integrity of all body cells, as well as constituting hormones, enzymes, and antibodies for life's processes, inquiry into the specific effects of protein on aspects of obesity, CHD or other metabolic abnormalities has been a more latent area of investigation. Some research into the effects of protein has been conducted, however, at least as early as 1967 (375). Since World War I, when soybean use developed as an alternative source of protein and vegetable oil (376) epidemiological studies have shown animal protein in the diet to be positively, and plant

protein to be negatively correlated with: the percentage of atherosclerotic lesions present on the aorta and coronary arteries (377), mortality from CHD (378, 379, 380), as well as serum levels of: triglycerides, VLDL mass (381), and total cholesterol (293). Work by numerous researchers in the 1970's and '80's established with little uncertainty that soy protein, which was becoming the standard for vegetable protein, when substituted for, or compared to, diets in which the protein was primarily of animal origin, produced beneficial changes in TC, LDL-C, VLDL-C, HDL-C and TG (382, 383, 384, 385, 386, 387, 388, 389. 390.) or smaller decrements in HDL-C (390, 391) in healthy, hypercholesterolemic, and/or obese individuals.

Protein is recognized to potentiate insulin secretion, although generally less effectively than carbohydrates (392), and the effect in normocholesterolemic individuals is typically less than that in hypercholesterolemics or diabetics (393). The differing effects of various protein sources on insulin response observed by Gannon and coworkers (394) and the lower insulin values observed by Sanchez and colleagues (395) in response to arginine and lysine has suggested that individual amino acids may be important in the effect of a protein on insulin response. And since insulin and glucagon play such leading roles in cholesterol metabolism, the authors suggested that these two amino acids may be tantamount to a protein's effect on lipoprotein

cholesterol levels. Whatever the cause, there is sufficient evidence which suggests that plant protein affects total cholesterol and lipoprotein cholesterol levels differently than animal protein, and it appears that the effects of soy protein are most marked.

Consideration of the data amassed in the past four decades suggests that a wise dietary approach to a healthy lipoprotein profile is one which restricts the daily intake of cholesterol and sataurated fats, while replacing, or partially replacing, these with complex carbohydrates and fibrous foods. The American Heart Association recommends a diet with a limit of 300 mg of cholesterol per day, and a limit of 30% of calories as fat, in which not more than 10% are in the form of saturated fats and at least 10% are polyunsaturated fats. In view of some of the deleterious effects of polyunsaturated fats, perhaps a greater proportion of monounsaturated fats should be advised. While maintaining one's weight in the ideal range, such a diet is, to the best of scientific research thus far, the most ideal one to maintain acceptable levels of TC, the ratio of LDL- to HDL-cholesterol, TG, glucose and most likely the levels of VLDL-C, HDL_2-C, HDL_3, and insulin.

Project Objectives

1. To assess the effects of a multifaceted weight loss program on body weight and composition, plasma variables, and nutrient intake in obese black women for six months. The weight loss program encompassed:

   a. 1200 kcal exchange diet
   b. nutrition education
   c. behavior modification
   d. aerobic exercise for able subjects

2. To determine the effects of the addition of aerobic exercise to a program that included a 1200 Kcal exchange diet, nutrition education, and behavior modification on:

   a. Body Composition
      i) weight
      ii) % body fat
      iii) % lean body mass
      iv) % body water

   b. Plasma Variables
      i) TC
      ii) LDL-C
      iii) HDL-C

iv) HDL_2-C

v) TC/HDL-C

vi) TG

vii) Insulin

c. Nutrient Intake

3. To compare the effect of the level of compliance to nutrition education/behavior modification classes on body composition, plasma variables, and nutrient intake.

4. To evaluate the effects of the level of exercise participation on body composition, plasma variables and nutrient intake.

5. To identify meaningful correlations among changes in body composition, plasma variables, and nutrient intake following a comprehensive weight loss program in obese black female subjects.

6. To identify meaningful correlations between changes in body composition, plasma variables, and nutrient intake components resulting from different levels of exercise participation, and different levels of compliance to nutrition education/behavior modification classes.

7. To determine whether changes resulting from a comprehensive weight loss program were dependent on initial levels of selected body composition, plasma, and nutrient variables.

8. To determine, for different levels of exercise participation and nutrition education/behavior modification compliance, whether changes in selected variables were dependent on their initial levels.

9. To subjectively observe characteristics, attributes or other factors which may be helpful in the planning or implementation of future weight loss programs for obese black women.

JOURNAL ARTICLE

The effects of a nutrition program with and without aerobic exercise on body weight and composition, plasma variables and nutrient intake in obese black women.

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Footline: Metabolic effects of weight reduction.

Abstract

The effects of a multifaceted weight loss program on the body weight, body composition, plasma variables, and nutrient intakes of 50 obese black women were investigated. Subjects were between 20 and 51 years of age, with an average BMI of 34.5 (range = 23.6 - 57.3). Subjects attended nutrition education/behavior modification classes once per week for three months. Thirty-five of the women attended 80% or more of the classes (NU). Twenty-eight women attended 30% or more of the low-impact aerobic exercise classes that were offered three days per week, for six months. An average 2.2 kg weight loss was observed for the whole study group, as body fat fell, and lean body mass increased by 2.1% of total body weight. Slightly greater changes were observed in the group that exercised consistently, as well as those who regularly attended nutrition classes, but not in either of the groups which more sporadically attended exercise or nutrition classes. Significant reductions in plasma total cholesterol, HDL-Cholesterol, HDL_2-Cholesterol, and insulin were observed for the group of 50 subjects. Significant reductions in plasma TC occurred in both the SE (attended between 30 to 70% of exercise sessions) and SN (< 80% attendance of nutrition classes) groups. Plasma LDL-Cholesterol followed the same pattern as plasma total cholesterol but there were no significant differences. Exercise appeared to mitigate

decreases in plasma HDL-C. Significant reductions in total kcal, including CHO, protein, fat (including saturated, polyunsaturated, and monounsaturated fatty acids), dietary cholesterol, and sugar were noted for the study group. A weight-loss program which included diet, nutrition education, behavior modification, stress management and exercise was effective in producing favorable changes in body composition, plasma variables, and dietary components in obese black women over six months, with a high degree of variability in motivation and participation.

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

Introduction

Despite an increasing awareness about health, nutrition, and fitness ([1]), the American populace has continued to become more overweight ([2]). That the obese condition carries with it numerous other health complications has been recognized. Obesity has been identified as an independent risk factor for coronary heart disease (CHD) ([3]), and has also been associated with other CHD risk factors. The condition has been positively associated with total cholesterol (TC) ([4], [5]), and negatively with high-density lipoprotein cholesterol (HDL-C) (4, 5, [6], [7]). It has been estimated that both the risk for hypertension ([8]), and the risk for diabetes (8) are 2.9 times greater for overweight versus non-overweight individuals. Additionally, the incidence of several cancers ([9]), as well as other ventilatory, circulatory, digestive, and endocrinological disorders ([10], [11]) increase with obesity. Not surprisingly, then, a higher mortality ratio among obese individuals has been observed ([12], [13], [14]).

In the United States, the prevalence of obesity in black women is higher than for either sex of either the black or white race (2). This prevalence in black women has been found to be inversely related to income and education (2). As determined by the second National Health and

Nutrition Examination Survey ([15]), the obese black woman is also at a greater risk for several of the health complications associated with obesity. Diabetes and hypertension are more prevalent in blacks, as well as those below the poverty level, while being female is a further risk for diabetes. There is also evidence which suggests that the rate of CHD in black women is greater than for white women ([16]).

Despite these statistics, there is a paucity of information related to attempts to address the obese condition, particularly in black females. Among the white population, treatment has typically focused on caloric restriction, behavior modification and exercise. Applied separately, there has been some success with each of these methods. However, various drawbacks to these individual methods has led several investigators to recommend a multi-faceted approach to a weight-loss program ([17], [18], [19], [20], [21], [22]). In 1984, a panel organized by the International Congress of Obesity stated that: 'diet, exercise and psychological support are three components of therapy which are interrelated, interdependent, and mutually supportive, and thus should be prescribed together (22) '. The results of several studies which have used such a combined approach support recommendations for their use with obese individuals ([23], [24], [25]). Limited data further suggest that this type of treatment approach can be of use in black ([26]), and black

female populations (24). This article reports the effects of the integration of diet, nutrition education, behavior modification, and exercise on weight loss and body composition, plasma variables, and nutrient intakes in 50 healthy, obese, black women, aged 20-51 years.

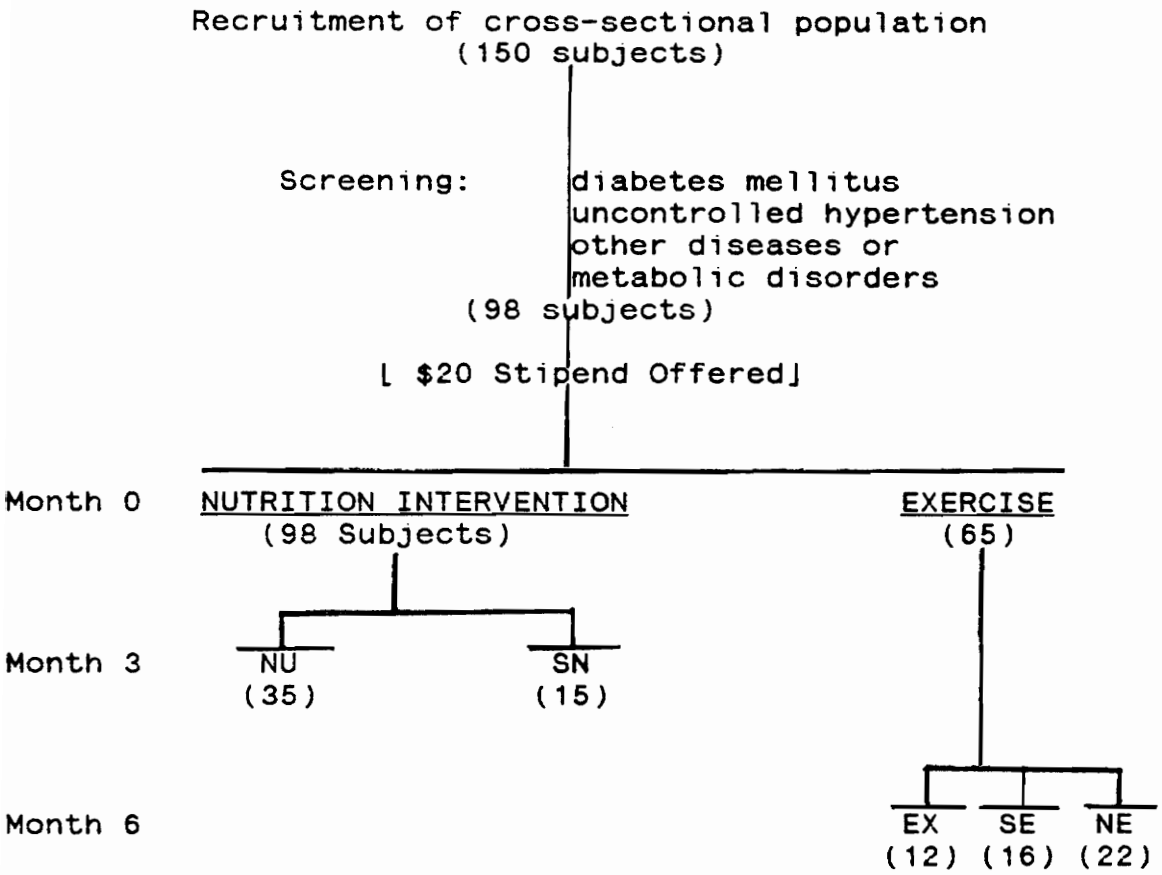
METHODS

Subjects

Through notices and pamphlets posted throughout the campus of Virginia State University, local public bulletin boards, as well as word-of-mouth, 150 black women between the ages of 20 and 51 years, at least 20% above the desirable weight for age according to the Metropolitan Life Insurance Co. tables, and who were considered sedentary according to a questionnaire, were identified. Those with diabetes mellitus, uncontrolled hypertension, or other identified diseases or metabolic disorders were excluded from this cross-sectional population. Potential subjects were interviewed regarding their personal motivation and committment towards weight loss and completion of the intervention program. Ninety-eight women who met all criteria were identified and began the intervention program (Figure 7). A 20 dollar stipend was offered as compensation for successful completion of all data collection.

Nutritional Education Program

Comprehensive nutrition education sessions were held once a week, for approximately two hours each. Four different times were offered each week to accommodate the



NU = Attended 80% or more of nutrition classes

SN = " less than 80% "

EX = Participated in 70% or more of exercise sessions

SE = " 30 to 69% "

NE = " less than 30% "

FIGURE 7. EXPERIMENTAL PROCEDURE

Table 1

Characteristics of 50 Obese Black Female Subjects

	Mean ± SEM	Range
Age in years	36.5 ± 1.1	22 - 51
Height in cm	161.6 ± .82	148.5 - 179.1
Weight in kg	90.0 ± 2.3	60.8 - 144.6
BMI (kg/m2)	34.5 ± .91	23.6 - 57.3
Years of Formal Education	14.1 ± .4	5 - 20
Total Family Income (per year)	28,025 ± 3348	4,500 - 163,000
Total Family Income ÷ Number in Family	11,131 ± 1039	1,182 - 40,750

varied schedules of the participants. The duration of this intervention was 11 weeks (Figure 7). These sessions encompassed nutrition education, behavior modification of eating habits, as well as discussions of stress management. The basic material used for the instruction of these classes, except for the section on stress management, was from the Virginia Cooperative Extension Service (VCES), in the publication titled: Diet, Exercise, and Behavior Modification (DEB) Program ([27]), which was designed specifically for overweight women. However, additional information from various sources was provided as deemed appropriate, or where interest was expressed by participants.

Nutrition education provided information on dietary nutrients and their functions, the concept of energy balance, and the caloric content of foods. Subjects were asked to choose their desired rate of weight loss, not to exceed two pounds per week, and to select foods which were grouped into six categories based on their nutrient composition and caloric values, such that the individually-determined caloric intakes be maintained concomitant to a proper nutrient balance. If subjects acknowledged excessive intakes or strong desires for particular foods, it was not advised that they eliminate these items from their diets entirely, but moderation and individual control of the diet was emphasized. Fasting, fad diets, and very-low-calorie

diets were discouraged. Information was provided regarding fast foods, prepared foods and dining out, as well as their associations with behavior modification. Identification of sources of fat in the diet, and various means of reducing dietary fat were emphasized.

Behavior modification involved recognition of inappropriate eating behaviors, cues which triggered such responses, and methods to counteract this phenomenon. Stress management dealt with daily life stresses, how to recognize stress and its associated outcomes, as well as methods to avert such stress. Additionally, individual counseling and/or support groups were encouraged for all subjects following the termination of the more structured classes.

Exercise Program

Pending a physicians' approval, subjects were allowed to self-select for the exercise regimen, but were told that they would have to adhere to a minimum of two exercise sessions per week, or would not be allowed to continue in the exercise program. This resulted in an initial enrollment of 65 out of 98 persons in the exercise program (Figure 7). Exercise classes were held three times per week, for an hour each session, at the YMCA of Southside Virginia, located in Petersburg, Virginia. The first ten

minutes of each session was designated for walking; subjects walked in the indoor gymnasium, a room, or on an indoor or outdoor track. Subjects were encouraged to maintain a strong pace, and conversation was encouraged to reduce boredom and develop interest and peer support. The exercise sessions were conducted by YMCA staff, and were designated as low-impact aerobics. As subjects improved their stamina, sessions were increased in tempo, and/or number or degree of movements. Each session included five to ten minutes of warm-up, 20-25 minutes of exercise, and at least ten minutes of cool-down activities, which included various stretching and calisthenics. Subjects were initially encouraged to work at 60 to 70% of their age-adjusted maximum heart rate. After two weeks, this was increased to 65 to 75%, and three weeks after this, subjects were encouraged to work at a range of 70 to 85% of maximum heart rate. A chart indicating the suggested target heart rates was kept visible during exercise classes until the subjects became familiar with their particular target range. Individuals were taught to take their own pulse, for ten-second counts. During each session, pulses were taken after 10 minutes of walking, after warm-up activities, several times during the routine, and again at cool-down. These were recorded next to each subjects' target range, so that exercise leaders could immediately suggest appropriate alterations of activities, if necessary, in order to achieve an appropriate exercise

heart rate. The pulse rate as assessed by the subject was verified periodically throughout the six month training program by an exercise leader. Individuals consistently worked at or above their designated target heart rates, except in cases where it was recommended that a particular individual not over-exert herself, such as those few with high blood pressure, or several recovering from surgeries which were unrelated to the intervention program.

As a measure of fitness, recovery heart rates were taken at the start of the exercise program in January, and again at the conclusion of the program in June. One-and two-minute recovery heart rates were taken by the subjects themselves following 10 minutes of walking for which they were instructed to walk as far as they could at a strong pace, for the full 10 minutes.

Those subjects who did not participate in the exercise protocol offered in this study (the non-exercising group) attended one or two additional one-hour sessions during the intervention period. The benefits of regular exercise were explained, printed information was distributed, exercise demonstrations were given, and instructions provided for an at-home aerobic/stretching program to be followed in conjunction with a complimentary cassette tape. The ease and benefits of various methods for increasing daily living energy expenditures were also provided. Walking was proposed as a most convenient mode of exercise, and the

formation of groups for this purpose was encouraged. An exercise/activity questionnaire was completed by these individuals so that levels of unsupervised exercise could be assessed.

Diet Analysis

Diet analysis was performed both at the beginning and end of the study, coinciding with blood sampling. At each time, two 24-hour recalls were obtained; one at the doctor's office, and another within two weeks before or after this (approximately 90% were obtained prior to blood samplings). These were then coded and analyzed using the computer data bank maintained by the Nutritional Analysis System (Department of Experimental Statistics, Louisiana State University, Baton Rouge, LA), and average values for the two recalls, both at the beginning and at the end of the intervention period for each subject, were obtained. The recall which was obtained at the doctor's office included part of a 14-hour fast in preparation for the blood drawing. Therefore, nutrient intake values were likely to be lower than might otherwise be expected, but the changes between pre- and post-treatment should not have been affected.

Plasma Lipids, Glucose and Insulin

Blood samples were drawn by a registered nurse, and a doctor performed a general physical exam for each subject, both at the start of the study period, and again at the end of the six-month treatment period. A 15 ml venous blood sample for each subject was drawn into vacutainers containing EDTA, and held on ice until centrifuged at 3000 rpm for 20 min. Analysis of triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), HDL_2-C, glucose, and insulin were performed on the plasma. Plasma TG were determined according to the Stanbio Enzymatic Triglycerides Procedure No. 2000 (Stanbio Laboratory Inc., San Antonio, TX), which follows the enzymatic method of Wahlefeld ([28]).

Plasma TC was determined by a colorimetric reaction which is based on the classical Liebermann-Burchard reaction involving the C^5-C^6 double bond in the sterol molecule ([29]). Plasma HDL-C was determined in the supernatant after precipitation of non-HDL apo-B associated lipoproteins by a heparin-manganese-chloride solution ([30]). Plasma HDL_2-C values were determined by dextran sulfate (15,000 MW) precipitation of HDL_2-C ([31]), which allowed quantitation of HDL_3-C in the supernate. Plasma HDL_3-C was then subtracted from the plasma total HDL-C concentration to obtain the value for HDL_2-C. Plasma LDL-C values were calculated

according to the equation: LDL-C = TC - (HDL-C + TG/5) ([32]). Plasma glucose was determined using the Stanbio Direct Glucose Procedure No. 0270 (Stanbio Laboratory, Inc., San Antonio, TX). This is a direct quantitative colorimetric procedure based on the works of previous investigators ([33], [34]). In this procedure, O-toluidine is the reactive reagent which condenses with the aldehyde group of glucose to form a colored mixture of glycosamine and the corresponding Schiff base ([35]). Plasma insulin values were obtained by using a radioimmunoassay analysis (Diagnostic Products Corporation, Los Angeles, CA).

Body Weight and Composition

Body weight were determined at the beginning of the study, and at the end of the six-month treatment period, using a kg scale. Body composition was determined by bioelectrical impedance, using the BES 200Z model (Bioelectrical Sciences, La Jolla, CA). This relatively new method of body composition analysis has the advantage of being a rapid and non-invasive technique. Bioelectrical impedance is based on the electrical conductivity of water; since the lean body mass contains the majority of the body's water (73.2%), a current will flow through this portion of the body tissues. Adipose tissue, however, is a poor conductor, and acts as an insulator. An individual's

height, weight, gender and impedance reading are utilized in an equation, developed by E. J. Glassford, of Biolectrical Sciences, which yields body composition values. Since variations in body hydration affect body composition analysis by bioimpedance, subjects were asked to refrain from alcohol for 12 hours prior to analysis, and to maintain normal intakes of fluids for 24 hours before analysis. Subjects were not tested if their fluid intake was abnormal or they were within two days of menstruation; in these few circumstances individuals were asked to return at a later date. To obtain the impedance reading, subjects were in a supine position on a non-conductive surface of a cot, with arms and legs positioned so that no contact existed between the limbs and/or body; electrodes were placed according to instruction: one each at the proper sites on a toe, ankle, finger and wrist. Bioimpedance determinations were performed a month after the start of the intervention period (as the BES 200Z was not obtained by the research group until then), and again following the six-month intervention. Two- and three-month values for these individuals have been reported elsewhere ([36]).

Statistical Analysis

Pre-treatment mean values were statistically analyzed for differences by Duncan's Multiple Range Test, with the

significance level set at alpha = .05. Differences between pre- and post-treatment least-squares mean values within groups, as well as differences in post-treatment least-squares mean values between nutrition, or among exercise groups were analyzed by t-test, using alpha levels of .05, .01, and .001. To obtain the critical p-values, alpha levels were divided by the number of comparisons made, which maintained the overall experiment-wise error rate at each alpha level. This is the conservative Scheffe method, which is recommended when sample sizes are unequal. Additional analyses of recovery heart rates included: 1) analysis of the differences in one- and two-minute recovery heart rates between pre- and post-treatment for all exercising groups, by paired t-test 2) analysis of the differences between one- and two-minute recovery heart rates at both pre- and post-treatment, for all groups of exercising subjects, by paired t-test 3) a t-test of whether one-minute recovery heart rates, at both pre- and post-treatment, for all groups of exercising subjects, are equal to the immediate-post-exercise heart rate. Correlations for all variables were determined by Pearson Correlation Coefficients, with statistical significance reported for $p < .05$, $p < .01$, and $p < .001$.

RESULTS

Nutrition Education/Behavior Modification Class Attendance

Subjects were divided into two groups based on their attendance at the nutrition education/behavior modification classes. Thirty-five subjects who attended 80% or more of the sessions were placed in the group designated as Nutrition (NU). The value of 80% was subjectively chosen in an effort to evaluate differences in body composition, plasma variables, and nutrient intake components between those who consistently attended nutrition classes, and those who attended less consistently. The remaining 15 subjects attended less than 80% of the nutrition classes, and fell into the group designated as Some Nutrition (SN) (Figure 7).

Exercise Participation

Subjects were divided into three different exercise groups, based on the number of exercise sessions in which they participated. This was done in order to evaluate the effects of the amount of exercise on body composition, plasma variables, and nutrient intake components. Twelve subjects participated in 70% or more of the aerobic exercise

sessions over the six-month period, and as a group were referred to as the Exercise group (EX). Those 16 subjects who participated in more than 30%, but less than 70% of the exercise sessions were placed in the group referred to as Some Exercise (SE). The 22 subjects who either chose not to attend exercise sessions at all, or who failed to attend at least 30% of the exercise sessions were designated as the No Exercise group (NE) (Figure 7).

Body Weight and Composition

All Subjects

Values for body weight, per cent body fat (% BF), per cent lean body mass (% LBM), and per cent body water for 50 subjects at pre- and post-treatment are shown in Table 2. Overall, there were beneficial changes in all variables; that is, a significant decrease in body weight (2.1 kg) and BF (2.2%), with increases in LBM (2.2%) and per cent body water (1.8%). Weight changes within the group of 50 subjects ranged from a loss of 16.7 kg to a gain of 3.9 kg. Thirty-six subjects lost an average of 3.8 kg; 14 subjects gained an average of 1.6 kg. The maximum individual decrease in % BF was 11.3, the maximum increase was 4.9%. The decline in body weight for the group as a whole was significantly correlated with the change in % BF ($r = .35$, $p < .01$), and similarily, negatively correlated with % LBM (r

Table 2

Body Weight and Composition of All Subjects, and Divided into Nutrition and Exercise Participation at Pre- and Post Treatment

LS Means ± SEM

		% Body Fat	Body Fat (kg)	% Lean Body Mass	Lean Body Mass (kg)	Body Weight (kg)	% Body Water
All Subjects,	pre	39.8 ± .38	34.9	60.1 ± .38	52.7	87.7 ± .44	39.1 ± .30
(N=50)	post	37.6 ± .38*	32.2	62.3 ± .38*	53.3	85.6 ± .44#	40.9 ± .30*
Nutrition,	pre	38.9 ± .40	35.0	61.0 ± .40	55.0	90.1 ± .46	39.8 ± .32
(N = 35)	post	35.5 ± .40^{s*}	30.8	64.4 ± .40^{s*}	55.9	86.8 ±.46^{m*}	42.6 ±.32^{s*}
Some Nutrition,	pre	40.8 ± .65	34.8	59.1 ± .65	50.4	85.3 ± .76	38.4 ± .52
(N=15)	post	39.8 ± .65^{t}	33.6	60.1 ± .65^{t}	50.7	84.4 ± .76^{n}	39.2 ± .52^{t}
Exercise,	pre	38.4 ± .77	32.4	61.4 ± .77	51.9	84.5 ± .90	40.0 ±.62
(N = 12)	post	35.6 ± .77$^{gm\Delta}$	28.7	64.3 ± .77$^{gm\Delta}$	51.8	80.6 ± .90$^{t\Delta}$	42.4 ±.62$^{g\Delta}$
Some Exercise,	pre	40.4 ± .67	33.6	59.4 ± .67	49.4	83.2 ± .78	38.5 ± .53
(N = 16)	post	38.6 ± .67^{h}	31.8	61.3 ± .67^{h}	50.4	82.3 ± .78^{t}	40.0 ± .53^{h}
No Exercise,	pre	40.6 ± .51	38.7	59.3 ± .51	56.6	95.4 ± .60	38.7 ± .41
(N = 22	post	38.7 ± .51$^{n\Delta}$	36.3	61.2 ± .51$^{n\Delta}$	57.5	93.9 ± .60^{s}	40.2 ±.41$^{h\Delta}$

Superscripts for post treatment values indicate that there was a significant change for that group from pre- to post treatment, where the level of significance is as follows: Δ = .05 # = .01 * = .001. Between nutrition groups or among exercise groups, values within columns with different superscripts are significantly different from each other, where the level of significance is as follows: g-l = .05, m-r = .01, s-w = .001. Values with different superscripts that represent two different levels of significance are not significantly different from each other. Based on raw mean values, there were no significant differences between nutrition groups, or among exercise groups for any pretreatment values. Subjects were divided into groups based on the percentage of their attendance: NU ≥ 80% nutrition classes, SN < 80%; EX ≥ 70% exercise sessions, SE 30 - 69%, NE ≤30%.

= -.35, $p < .01$). Body weight change also correlated negatively with the change in % body water ($r = -.32$, $p < .05$). For all groups, due to the principles of the bioelectrical impedance analyzer and the nature of body composition, there was a correlation of -1.0 between % BF and % LBM, as well as a high correlation ($r > .96$) between % body water and % LBM, with the same corresponding negative correlation between % body water and % BF.

Nutrition Groups

There were no differences between Nutrition groups for body weight or composition at pre- treatment (see Appendix I). Body weight and composition of subjects divided into nutrition groups can be seen in Table 2. The beneficial changes seen in body weight and body composition variables for the group as a whole were reflected only in the group that attended nutrition classes 80% or more of the time (NU). Those who attended nutrition classes less than 80% of the time (SN) showed no significant change in any of these four variables. Values for all four variables were significantly different between the groups at post-treatment. The NU group lost a significant per cent of their original body weight (3.5%, $p = .0001$), while the group that received Some Nutrition (SN) lost a non-significant 1.2%. Within the NU group, there was a positive correlation between the change in body weight and the change

in % BF of r = .33 ($p < .05$).

Exercise Groups

Pre- and post-treatment LS Mean values for body weight and composition among groups of subjects based on exercise attendance can be seen in Table 2. There were no significant differences in pre-treatment mean levels among groups (see Appendix I). Only the EX group displayed a significant decrease in body weight between pre- and post-treatment (-3.9 kg, $p < .05$). The group of subjects who participated in Some Exercise (SE) showed no significant change from pre- to post-treatment in any variable. Both of the EX and NE groups underwent significant ($p < .05$) declines in % BF (2.8 and 1.9 kg, respectively) and corresponding increases in % LBM, as well as increases in % body water (2.4 and 1.5, respectively). At post-treatment, there were significant differences between the EX group and both of the SE and NE groups for all three body composition variables; the EX group had less fat, correspondingly more lean, and had a higher percentage of body water than either of the SE or the NE groups. The body weight of the NE group at post-treatment was significantly ($p < .001$) higher than both the EX and SE groups.

For the EX group, there was a significant positive correlation of change in body weight with change in % BF ($r = .69$, $p < .05$), and a corresponding negative correlation of

change in body weight with change in % LBM and % body water ($r = -.69$, $p < .05$ and $r = -.66$, $p < .05$, respectively). The change in body weight for the EX group was the only significant correlation with pre-treatment levels ($r = .65$, $p < .05$). For neither the SE or the NE group were there any significant correlations among body composition variables, or their pretreatment levels.

Plasma Total Cholesterol, Lipoprotein Cholesterol, Triglyceride and Insulin

All Subjects

The effects of combined treatments on plasma parameters in all 50 subjects are presented in Table 3. There were declines (some non-significant) in all variables, excepting the TC/HDL-C ratio, in which a slight (non-significant) increase occurred, which was due to a greater proportional decrease in plasma HDL-C (10.1%) than plasma TC (7.2%). The post-treatment values of plasma TC and plasma HDL-C were significantly different from their respective pre-treatment values at the $p < .01$, and $p < .001$ levels, respectively. Significant ($p < .001$) decrements in plasma insulin and HDL_2-C also occurred, while no significant change was observed in plasma LDL-C. The decline in plasma TG approached significance ($p = .0581$).

For the group of 50 subjects, there were significant

Table 3

Plasma Lipids, Lipoprotein Cholesterol, Triglycerides and Insulin of All Subjects, and Divided into Nutrition and Exercise Participation at Pre- and Post Treatment

LS Means ± SEM

		TC (mg/dl)	LDL-C (mg/dl)	HDL-C (mg/dl)	HDL_{2}-C (mg/dl)	TG (mg/dl)	Ins (uI U/ml)	TC/HDL-C
All Subjects,	pre	205.0 ± 3.4	136.8 ± 3.7	49.4 ± .65	26.3 ± .92	93.8 ± 5.5	23.4 ± 1.2	4.38 ± .11
(N = 50)	post	190.2 ± 3.4$^{\#}$	130.8 ± 3.8	44.4 ± .66*	21.6 ± .88*	78.4 ± 5.6	17.4 ± 1.0*	4.48 ± .11
Nutrition,	pre	201.6 ± 3.6	131.8 ± 3.9	49.2 ± .68	25.1 ± .94	102.8 ± 5.8	24.1 ± 1.2	4.34 ± .12
(N =35)	post	194.5 ± 3.6	133.0 ± 4.2	46.3 ± .70$^{m\Delta}$	23.9 ± .94g	83.0 ± 6.2$^{\Delta}$	19.3 ± 1.1$^{\#}$	4.40 ± .12
Some Nutrition,	pre	208.3 ± 5.8	141.8 ± 6.4	49.6 ± 1.1	27.6 ± 1.6	84.7 ± 9.4	22.6 ± 2.2	4.42 ± .19
(N = 15)	post	185.8 ± 5.8$^{\Delta}$	128.5 ± 6.4	42.5 ± 1.1^{n*}	19.3 ± 1.5^{h*}	73.9 ± 9.4	15.5 ± 1.8$^{\Delta}$	4.56 ± .19
Exercise,	pre	199.2 ± 6.9	134.1 ± 7.6	47.3 ± 1.3	25.9 ± 1.8	88.9 ± 11.1	18.9 ±2.1^{g}	4.48 ±.23
(N = 12)	post	185.5 ± 6.9	127.3 ± 7.8	45.5 ± 1.3	23.3 ± 1.8	69.5 ± 11.5	12.7 ± 2.1Δ	4.22 ± .23
Some Exercise,	pre	219.0 ± 6.0	147.6 ± 6.6	53.0 ± 1.1	27.8 ± 1.6	91.9 ± 9.6	18.7 ± 2.3	4.37 ± .20
(N = 16)	post	190.4 ± 6.0$^{\#}$	130.7 ± 6.7	45.5 ± 1.2*	20.9 ± 1.6$^{\Delta}$	75.7 ± 9.9	17.0 ± 1.9	4.42 ± .20
No Exercise,	pre	196.7 ± 4.6	128.7 ± 5.0	47.8 ± .88	25.3 ± 1.4	100.5 ± 7.4	32.4 ± 1.9^{h}	4.29 ± .15
(N = 22)	post	194.7 ± 4.6	134.4 ± 5.0	42.3 ± .88*	20.5 ± 1.2$^{\Delta}$	90.1 ± 7.4	22.5 ± 1.4*	4.79 ± .15

Superscripts for post treatment values indicate that there was a significant change for that group from pre- to post treatment, where the level of significance is as follows: Δ = .05, # = .01, * = .001. Between nutrition groups, or among exercise groups, values within columns with different superscripts are significantly different from each other, where the level of significance is as follows: g-h = .05, m-n = .01. Pretreatment values were analyzed for differences based on raw mean values. Subjects were divided into groups based on the percentage of their attendance: NU ≥ 80% nurtrition classes, SN < 80%; EX ≥ 70% exercise sessions, SE 30 - 69%, NE < 30%.

correlations between the change in plasma TC and the changes in plasma HDL-C ($r = .29$, $p < .05$), plasma LDL-C ($r = .94$, $p = .0001$), and the TC/HDL-C ratio ($r = .62$, $p = .0001$). The change in plasma HDL-C corresponded to the change in its HDL_2-C component ($r = .41$, $p < .01$) and to the change in the TC/HDL-C ratio ($r = -.45$, $p < .01$). There were also significant correlations between the change in plasma TG and the change in plasma LDL-C ($r = -.46$, $p < .001$), as well as between the change in plasma LDL-C and the change in the TC/HDL-C ratio ($r = .75$, $p = .0001$).

There were significant correlations between the initial (pre-treatment) levels of TG and lipoprotein variables and their own subsequent (absolute) changes. These correlations ranged from .43 for the TC/HDL-C ratio to .84 for TG. The correlation ($r = .30$) between pre-treatment insulin levels and subsequent (absolute) change in insulin approached significance ($p = .0505$).

Nutrition Groups

Changes in plasma variables between nutrition groups are shown in Table 3. There were no differences between the groups at pre-treatment for any variables as determined by an analysis of variance (ANOVA) using raw means (see appendix K). There was a significant decline in plasma TC for the SN group, while no change occurred in the NU group. No changes in LDL-C were observed for either group.

Both nutrition groups exhibited significant differences between their pre- and post-treatment plasma HDL-C values. However, this was more pronounced in the SN group, which experienced an absolute decline of 7.1 mg/dl ($p < .001$), compared to an absolute decline of only 2.8 mg/dl ($p < .01$) in the NU group. The post-treatment values of plasma HDL-C (Table 3) were significantly different between nutrition groups ($p < .01$). A similar trend was observed for plasma HDL_2-C.

No changes occurred in the TC/HDL-C ratio for either group. Both the NU group and the SN group displayed declines in fasting plasma insulin ($p < .01$ and $p < .05$, respectively). Only the NU group underwent a significant ($p < .05$) decline in plasma TG.

For the NU group, correlations among plasma variables, and between plasma variables and body weight or composition were assessed. There were significant correlations between the change in plasma TC and the changes in both plasma LDL-C ($r = .93$, $p < .001$), and the TC/HDL-C ratio ($r = .66$). The change in plasma LDL-C was significantly correlated with the changes in both the TC/HDL-C ratio ($r = .79$) and plasma TG ($r = -.50$). The change in the TC/HDL-C ratio was significantly correlated with the change in the plasma HDL-C ($r = -.50$), the change in plasma TG ($r = -.38$).

Pre-treatment levels of all plasma variables, except insulin, were significantly correlated with their respective

absolute changes within the NU group .

Within the SN group, which was similar to the NU group, there were significant, positive correlations between the change in plasma TC and the changes in both the plasma LDL-C ($r = .96$, $p < .001$), and the TC/HDL-C ratio ($r = .70$, $p < .01$), but a negative correlation with the change in insulin ($r = -.61$, $p < .05$). The change in plasma LDL-C correlated significantly with the change in the TC/HDL-C ratio ($r = .79$, $p < .001$). The change in body weight for this group corresponded to the change in TG ($r = .56$, $p < .05$).

Within the SN group, initial levels of plasma HDL-C, TG, and insulin were significaty correlated with their absolute ($r = .79$, $r = .53$, $r = .83$, respectively) changes. Unlike the NU group, in the SN group initial levels of plasma TC, LDL, and the TC/HDL-C ratio were not significantly correlated with their subsequent absolute changes. The correlation between initial plasma HDL_2-C levels and the subsequent absolute change approached significance ($r = .54$, $p = .0562$).

Exercise Groups

Plasma lipid changes among exercise groups are presented in Table 3. As determined by a t-test which used raw means, there were no differences in pre-treatment values among exercise groups for any of the variables except plasma insulin; the raw mean value of 31.5 uIU/ml for the NE group

was significantly ($p < .05$) higher than the raw mean value of 18.7 uIU/ml for the EX group (see Appendix K). All groups experienced declines in plasma TC, but this was significant only for the SE group (-28.6 mg/dl, $p < .01$). No significant changes were seen in plasma LDL-C for any group, although both groups who exercised more than 30% of the time exhibited declines (NS) in plasma LDL-C, while the NE subjects showed a slight, but non-significant increase.

All three exercise groups experienced declines in plasma HDL-C and its subfraction, plasma HDL_2-C; however, these decreases were more pronounced and were significant for the SE and NE groups, but not for the EX group. Although there were no significant changes in the TC/HDL-C ratio for any group, close examination of the data revealed that the EX group was the only group to undergo a non-significant reduction in the TC/HDL-C ratio, while the other two exercise groups showed increases in this ratio, which approached significance ($p < .10$) for the NE group.

All three exercise groups showed non-significant declines in plasma TG from pre- to post-treatment, but apparently due to a large SEM, none were significant. The NE group was the only group in which a significant ($p < .001$) drop in the fasting plasma insulin value occurred. Since insulin values at pre-treatment for the EX and NE groups were significantly different at the $p < .05$ level (see Appendix K), absolute changes were considered. The

absolute decreases in plasma insulin levels for both the EX (6.2 +/- 3.0) and the NE (9.9 +/- 2.4) groups were significant at the $p < .05$ and $p < .001$ levels, respectively. No significant change occurred for the SE group. None of these (absolute) decreases were significantly different among groups.

As with the NU and SN groups, within the EX group there were significant correlations between the change in plasma TC and the changes in both plasma LDL-C ($r = .99$, $p < .001$), and the TC/HDL-C ratio ($r = .90$), as well as between the change in plasma LDL-C and the TC/HDL-C ratio ($r = .94$). There were no significant correlations between body composition and plasma variables for the EX group.

Initial levels of plasma TC, LDL-C, HDL_2-C, and the TC/HDL-C ratio were all significantly correlated with their respective changes ($r = .60$, $r = .70$, $r = .63$, $r = .75$, respectively).

For the SE group, the change in plasma TC was significantly correlated with changes in plasma LDL-C ($r = .92$) and the TC/HDL-C ratio ($r = .54$). The change in the LDL-C was significantly correlated with the TC/HDL-C ratio ($r = .70$) and with plasma TG ($r = -.57$). The change in plasma HDL-C was significantly correlated with the change in the TC/HDL-C ratio ($r = -.56$). Correlations between changes in % BF (as well as opposite correlations for % LBM) and both plasma TC ($r = -.48$) and HDL_2-C ($r = -.53$) approached

significance ($p < .059$).

Pre-treatment levels of five of the seven blood variables were significantly correlated with their respective changes and correlations approached significance ($p < .10$) for plasma HDL_2-C and LDL-C for the SE group.

For the NE group the change in plasma TC was significantly correlated with changes in plasma LDL-C ($r = .89$), the TC/HDL-C ratio ($r = .48$) and HDL-C ($r = .49$). The change in plasma LDL-C was significantly correlated with the change in the TC/HDL-C ratio ($r = .67$) and the change in plasma TG ($r = -.72$). Changes in plasma HDL-C and HDL_2-C were significantly correlated ($r = .47$), which might be expected, but was not seen in any of the other groups. Correlations between body weight or composition and plasma variables included a significant correlation between the change in body weight and the change in plasma TG ($r = .43$).

Initial levels of the plasma variables HDL-C, HDL_2-C, and TG were correlated with their subsequent change ($r = .81$, $r = .87$, $r = .91$, respectively) in the NE group.

Nutrient Intakes

All Subjects

Pre- and post-treatment daily nutrient intakes for 50 subjects are presented in Table 4. There was a significant decline of 580 total kcals consumed by all

Table 4

Energy and Nutrient Intakes of All Subjects, and Divided into Nutrition and Exercise Participation at Pre- and Post-Treatment
LS Means ± SEM

		Total Kcal	Carbohydrate (g)	Protein (g)	Total Fat (g)	Saturated Fat (g)	Polyunsat Fat (g)
All Subjects,	pre	1969.1 ± 68.0	214.6 ± 9.4	72.8 ± 3.0	89.0 ± 3.8	28.9 ± 1.4	18.9 ± 1.4
	post	1389.4 ± 68.0*	155.8 ± 9.4*	57.8 ± 3.0*	59.2 ± 3.8*	18.2 ± 1.4*	13.3 ± 1.4$^{\#}$
Nutrition,	pre	1900.5 ± 71.0	210.6 ± 9.8	69.0 ± 3.1	86.8 ± 4.0	28.8 ± 1.5	19.3 ± 1.4
	post	1340.4 ± 71.0*	159.0 ± 9.8$^{\#}$	53.0 ± 3.1$^{\#}$	54.9 ± 4.0*	16.6 ± 1.5*	12.0 ± 1.4$^{\#}$
Some Nutrition,	pre	2037.7 ± 115.9	218.6 ± 16.1	76.6 ± 5.1	91.2 ± 6.4	29.0 ± 2.4	18.5 ± 2.3
	post	1438.4 ± 115.9$^{\#}$	152.6 ± 16.1$^{\Delta}$	62.6 ± 5.1	63.4 ± 6.4$^{\#}$	19.8 ± 2.4$^{\Delta}$	14.5± 2.3
Exercise,	pre	1880.5 ± 137.7	194.7 ± 19.1	67.9 ± 6.1	92.1 ± 7.7	29.4 ± 2.9	24.2 ± 2.8^{g}
	post	1290.4 ± 137.7$^{\Delta}$	137.6 ± 19.1	60.2 ± 6.1	54.8 ± 7.7$^{\#}$	16.3 ± 2.9$^{\#}$	14.7 ± 2.8
Some Exercise,	pre	1868.6 ± 119.3	202.8 ± 16.6	73.1 ± 5.3	85.6 ± 6.6	27.7 ± 2.5	15.8 ± 2.4^{h}
	post	1390.7 ± 119.3$^{\Delta}$	143.2 ± 16.6$^{\Delta}$	54.9 ± 5.3	64.2 ± 6.6	20.2 ± 2.5	13.0 ± 2.4
No Exercise,	pre	2158.2 ± 91.6	246.3 ± 12.7	77.4 ± 4.0	89.4 ± 5.1	29.7 ± 1.9	16.7 ± 1.8^{h}
	post	1487.1 ± 91.6*	186.6 ± 12.7$^{\#}$	58.3 ± 4.0$^{\#}$	58.6 ± 5.1*	18.0 ± 1.9*	12.1 ± 1.8

Superscripts for post treatment values indicate that there was a significant change for that group from pre- to post treatment, where the level of significance is as follows: $\Delta = p < .05$, $\# = p < .01$, $* = .001$. Between nutrition groups or among exercise groups, values within columns with different superscripts are significantly different from each other, where the level of significance is as follows: g-l $= p < .05$, m-r $= p < .01$, s-w $= p < .001$. Pretreatment values were analyzed for differences based on raw mean values. Subjects were divided into groups based on the percentage of their attendance: NU ≥ 80% nutrition classes, SN < 80%; EX ≥ 70% exercise sessions, SE 30 - 69%, NE ≤ 30%.

Table 4 *(Continued)*

Energy and Nutrient Intakes of All Subjects, and Divided into Nutrition and Exercise Groups at Pre- and Post-Treatment
LS Means ± SEM

		Monounsaturated Fat (g)	P/S ratio	M/S ratio	Dietary Fiber (g)	Cholesterol (mg)
All Subjects,	pre	32.6 ± 1.6	.705 ± .075	1.15 ± .05	3.41 ± .28	325.2 ± 18.8
	post	20.6 ± 1.6*	.812 ± .075	1.13 ± .05	2.99 ± .28	270.2 ± 18.8$^{\Delta}$
Nutrition,	pre	29.7 ± 1.7	.701 ± .078	1.05 ± .05	3.39 ± .30	269.2 ± 19.6^{g}
	post	19.0 ± 1.7*	.762 ± .078	1.16 ± .05	2.66 ± .30	231.4 ± 19.6
Some Nutrition,	pre	35.6 ± 2.7	.709 ± .128	1.26 ± .08	3.43 ± .48	381.2 ± 32.0^{h}
	post	22.3 ± 2.7$^{\#}$	.863 ± .128	1.11 ± .08	3.32 ± .48	308.9 ± 32.0
Exercise,	pre	31.3 ± 3.3	.861 ± .151	1.08 ± .10	2.48 ± .58	338.2 ± 38.0
	post	18.5 ± 3.3$^{\Delta}$	.980 ± .151	1.16 ± .10	3.51 ± .58	311.4 ± 38.0
Some Exercise,	pre	36.6 ± 2.8	.628 ± .131	1.33 ± .09	3.62 ± .50	319.4 ± 32.9
	post	22.6 ± 2.8$^{\#}$	.785 ± .131	1.10 ± .09	2.28 ± .50	258.2 ± 32.9
No Exercise,	pre	29.9 ± 2.2	.626 ± .101	1.05 ± .07	4.13 ± .38	318.0 ± 25.3
	post	20.9 ± 2.2$^{\Delta}$	.671 ± .101	1.15 ± .07	3.19 ± .38	240.8 ± 25.3

Superscripts for post treatment values indicate that there was a significant change for that group from pre- to post treatment, where the level of significance is as follows: Δ = $p < .05$, # = $p < .01$, * = $p < .001$. Between nutrition groups or among exercise groups, values within columns with different superscripts are significantly different from each other, where the level of significance is as follows: g-l = $p < .05$, m-r = $p < .01$, s-w = $p < .001$. Pretreatment values were analyzed for differences based on raw mean values. Subjects were divided into groups based on the percentage of their attendance: NU ≥ 80% nutrition classes, SN < 80%; EX ≥ 70% exercise sessions, SE 30 - 69%, NE ≤ 30%.

Table 4 *(Continued)*

Energy and Nutrient Intakes of All Subjects, and Divided into Nutrition and Exercise Groups at Pre- and Post-Treatment
LS Means ± SEM

		Sugar (g)	Sucrose (g)	Pectin (g)	% of kcal as Carbohydrate	% of kcal as Protein	% of kcal as Fat
All Subjects,	pre	103.9 ± 6.6	77.8 ± 5.8	1.79 ± .43	43.4 ± 1.4	15.0 ± .6	41.2 ± 1.2
	post	82.4 ± 6.6$^{\Delta}$	64.0 ± 5.8	1.40 ± .43	44.5 ± 1.4	17.1 ± .6$^{\Delta}$	38.3 ± 1.2
Nutrition,	pre	105.9 ± 6.9	78.1 ± 6.1	1.45 ± .45	44.3 ± 1.5	14.7 ± .6	41.0 ± 1.2
	post	80.0 ± 6.9$^{\Delta}$	61.1 ± 6.1	1.25 ± .45	46.6 ± 1.5	16.6 ± .6	36.9 ± 1.2$^{\Delta}$
Some Nutrition,	pre	102.0 ± 11.2	77.4 ± 10.0	2.12 ± .74	42.4 ± 2.4	15.2 ± 1.0	41.4 ± 2.0
	post	84.8 ± 11.2	66.9 ± 10.0	1.54 ± .74	42.4 ± 2.4	17.5 ± 1.0	39.7 ± 2.0
Exercise,	pre	97.2 ± 13.3	77.8 ±11.8	1.22 ± .88	40.6 ± 2.8	14.6 ± 1.1	45.0 ± 2.4
	post	74.9 ± 13.3	57.9 ±11.8	2.16 ± .88	42.3 ± 2.8	18.9 ± 1.1$^{\Delta}$	38.2 ± 2.4
Some Exercise,	pre	89.0 ± 11.5	66.6 ± 10.3	1.04 ± .76	43.8 ± 2.5	15.7 ± 1.0	40.8 ± 2.0
	post	72.0 ± 11.5	55.1 ± 10.3	0.98 ± .76	41.5 ± 2.5^{h}	15.9 ± 1.0	41.2 ± 2.0
No Exercise,	pre	125.6 ± 8.8	88.9 ± 7.9	3.11 ± .58	45.7 ± 1.9	14.6 ± 0.8	38.0 ± 1.6
	post	100.4 ± 8.8	79.1 ± 7.9	1.06 ± .58	49.6 ± 1.9^{g}	16.3 ± 0.8	35.4 ± 1.6

Superscripts for post treatment values indicate that there was a significant change for that group from pre- to post treatment, where the level of significance is as follows: $\Delta = p < .05$, # = $p < .01$, * = $p < .001$. Between nutrition groups or among exercise groups, values within columns with different superscripts are significantly different from each other, $p < .05$. Pretreatment values were analyzed for differences based on raw mean values. Subjects were divided into groups based on the percentage of their attendance: NU ≥ 80% nutrition classes, SN < 80%; EX ≥ 70% exercise sessions, SE 30-69%, NE ≤ 30%.

subjects. Individual changes ranged from a decrease of 2166 kcal, to an increase of 462 kcal consumed per day. The overall drop in caloric consumption was accompanied by significant decreases in grams of carbohydrate, protein and total fat. The change in total fat included significantly lowered intakes of saturated, polyunsaturated and monounsaturated fatty acids. Daily intakes of cholesterol and sugar also decreased significantly, by 55 mg and 21.5 g, respectively ($p < .05$), while the percentage of kcal as protein in the diet increased 2.1% ($p < .05$).

Nutrition Groups

Nutrient intakes before and after treatment for both Nutrition groups are shown in table 4. Both the NU and SN groups significantly ($p < .001$, $p < .01$, respectively) reduced their total caloric intake; for both groups this encompassed significant reductions in carbohydrate and total fat, and for the NU group, a significant ($p < .05$) drop in the percentage of kcal derived from fat, as well as a significant decline in the grams of protein consumed ($p < .01$). For both groups, the decrease in total fat intake included significant declines in both saturated and monounsaturated fats; for the NU group, polyunsaturated fat intake dropped as well. There was no change for either group in: dietary fiber, pectin, sucrose or the P/S or M/S ratios. There was a significant ($p < .05$) decrease in sugar

intake for the NU group, while the SN group displayed no significant change.

The pre-treatment cholesterol intake level of the SN group was significantly ($p < .05$) higher than that of the NU group (see Appendix L). This was the only nutrient for which there was a significant difference in initial values between nutrition groups. For neither group were the post-treatment values significantly different from their respective pre-treatment values; neither were the absolute changes significantly different from zero for either group.

Exercise Groups

The effects of different exercise class attendance on nutrient intakes are presented in Table 4. At pre-treatment, the intake of polyunsaturated fats was significantly ($p < .05$) higher for the EX group than both the SE and NE groups (see Appendix L). No other intake variables were significantly different among exercise groups at pre-treatment.

All three exercise groups exhibited significant declines in total kcal consumed. However, the macronutrients which also underwent significant decreases in consumption varied among the groups. The EX group exhibited a significant ($p < .05$) decrease only in total fat. The SE group showed a significant ($p < .05$) reduction in carbohydrate consumption, while total fat and protein intake

approached a significant ($p < .10$) decrease. The NE group displayed a significant drop in total fat ($p < .001$) as well as both carbohydrate and protein ($p < .01$).

All exercise groups significantly reduced their intakes of monounsaturated fats, while significant reductions in saturated fat intake occurred for both the EX and NE groups, but not for the SE group. For the EX group, there was also a significant ($p < .05$) increase in the percentage of kcals as protein in the diet.

The absolute decline in polyunsaturated fat intake for the EX group (-9.5 +/- 4.0 g) was significantly ($p < .05$) different from zero.

Correlations Between Changes in Energy Intake and Nutrients

Correlations between the change in kcal consumed and the changes in other nutrient components were assessed. For the whole study group, as well as for all nutrition and exercise groups, this included significant correlations for all three macronutrients: total fat ($r = .76$ to $.90$), CHO ($r = .68$ to $.93$) and protein ($r = .61$ to $.86$), as well as saturated fat ($r = .44$ to $.86$). The significant correlation with polyunsaturated fat observed for the whole group ($r = .31$) was reflected only in the NU group ($r = .58$). The significant correlation with monounsaturated fatty acids ($r = .58$) in the study group was reflected in all except the SN

group (r = .56 to .76). The significant correlation with the change in fiber and/or that observed for pectin in the study group (r = .43 and .38, respectively) were evident in the SN, SE, and NE groups (r = .44 to .73). The significant, but rather low, correlation with the change in sugar intake seen in the group of 50 subjects (r = .39) was evident only in the NU group (r = .83), while significant correlations existed for the change in sucrose intake within the EX and SE groups (r = .70 and .65, respectively).

Correlations Between Changes in Body Weight or Composition and Nutrients

For the study group, there were significant correlations between the changes in body weight or composition and the changes in kcal, CHO, sugar, sucrose and % of kcal as fat; however, they tended to be low (r = .30 to .39). In contrast, for both the SN and EX groups, significant correlations between the change in body weight and the changes in total kcal (r = .53 and .67, respectively), CHO (r = .74 and .80, respectively) and sugar (r = .64 and .69, respectively) were higher. Other significant correlations with the changes in body weight included, within the SN group: total fat (r = .53) and pectin (r = .66) and within the EX group, fiber (r = .66). Within the NU group, correlations between the change in % BF

and the changes in all types of fat: total, % of kcal as fat, saturated, polyunsaturated, and monounsaturated fatty acids were low ($r < .48$) and negative, which is not easily explained, because both % BF and total fat intake decreased. The positive correlation between the change in % BF and the change in sugar intake for this group was positive, but also low ($r = .35$), while that for the EX group was higher ($r = .57$), but only approached significance ($p = .0543$).

Correlations Between Changes in Plasma Variables and Nutrients

There were significant correlations between changes in nutrient intake components and changes in plasma variables for the group of 50 subjects. However, these also tended to be low ($r = .28$ to $.36$) and inconsistent. For both the SN and NE groups, however, there were higher, significant, negative correlations between changes in plasma TC, LDL-C and HDL-C with nutrient components. For plasma TC and LDL-C among these groups, these significant negative correlations with nutrient components included: total kcal ($r = -.52$), CHO ($r = -.48$ to $-.58$), polyunsaturated fats ($r = -.42$) and the P/S ratio ($r = -.53$), dietary cholesterol ($r = -.45$), fiber ($r = -.52$), and pectin ($r = -.51$ to $-.77$). For the SN group, the change in HDL-C was associated with the change in the percentage of kcal derived from fat ($r = -$

.56).

Within the SE group, the change in TG correlated with the change in both polyunsaturated fats (r = -.64) and the P/S ratio (r = -.58).

For the whole group, there were significant correlations between the change in insulin and the changes in: total kcal, total fat, saturated fat, monounsaturated fat, protein, dietary cholesterol, and % CHO (r = .51, .51, .48, .45, .43, .35 and -.42, respectively). Except for dietary cholesterol in the NE group, these correlations were all reflected in the NU and NE groups. The correlation between the change in insulin and the change in total fat was present in all groups (r = .45 to .67). Additionally, the SN group exhibited significant correlations between the change in insulin and the changes in both total kcal and total fat (r = .61 and .67, respectively), while the EX group also showed a significant correlation with the change in monounsaturated fats (r = .70).

Of the 17 nutrient variables considered, only total kcal and total fat were assessed regarding the degree that the initial level might affect subsequent changes. For all groups of subjects, pre-treatment levels of both of these variables were significantly ($p < .05$) correlated with their subsequent changes (r = .54 to .90). Interestingly, for total kcal, this was lowest in the NU group, and for total fat, lowest in the EX group, indicating that in these

groups, more than in other groups, it was not necessarily those who had the highest intake of kcal or fat that subsequently reduced their intake most dramatically. This suggests that there was some other factor involved. Possibly the level of motivation was more important for individuals in these two groups.

Recovery Heart Rates

One- and two-minute recovery heart rates were calculated as percentages of an individuals' immediate-post-exercise (IPE) heart rate, following a ten-minute walk, at both pre- and post-treatment. These are presented in Table 5 for each exercising group (the EX and SE groups), as well as all exercising subjects (both exercise groups combined). There were no significant differences between any pre-treatment value and its respective post-treatment value for any group. There were no significant differences between exercise groups for either pre- or post-treatment values.

Presented in Table 5 are the results of a test of the hypothesis that one-minute recovery heart rates are equal to 100%; that is, equal to the IPE heart rate. At pre-treatment, the one-minute recovery heart rate for the SE group was not different from its IPE heart rate; the one-minute recovery heart rate of the EX group at pre-

Table 5

One- and Two-minute Recovery Heart Rates, as a Percentage of the Immediate Post-exercise Heart Rate, at Pre- and Post Treatment for all Exercising Subjects, and Divided into Exercise Groups

Mean ± SEM

	Pre			Post		
	One-Minute Rate	Two-Minute Rate	difference	One-Minute Rate	Two-Minute Rate	difference
All Exercising Subjects	89.5 ± 3.6[b] (28)	83.3 ± 5.9 (25)	5.9 ± 3.4[a] (24)	82.9 ± 2.2[c] (19)	75.8 ± 2.2 (19)	7.2 ± 1.8[c] (19)
Exercise	82.0 ± 4.6[b] (10)	68.8 ± 5.5 (9)	11.8 ± 5.3[a] (9)	86.6 ± 2.0[c] (10)	78.4 ± 2.3 (10)	8.2 ± 2.4[b] (10)
Some Exercise	95.4 ± 5.7 (15)	94.6 ± 9.7 (13)	1.0 ± 4.8 (13)	78.9 ± 3.8[c] (9)	72.9 ± 3.8 (9)	6.0 ± 2.8[a] (9)

Values of one-minute recovery heart rates with superscripts are significantly different from 100% of the respective immediate post-exercise heart rate; values of differences with superscripts are significantly different from zero, where the level of significance is indicated as follows: a: $p \leq .05$, b: $p \leq .01$, c: $p \leq .001$. Numbers in parentheses indicate the number of data values used in the calculation above it.

treatment was significantly different from its IPE heart rate at the $p < .01$ level. At post-treatment, both exercise groups exhibited one-minute recovery heart rates which were significantly different ($p < .001$) from their respective IPE heart rates.

The combined group of all exercising subjects exhibited significant differences between their one-minute heart rates and IPE heart rates at both pre- and post-treatment.

A test of the hypothesis that differences between one- and two-minute recovery heart rates at both pre- and post-treatment are equal to zero is also shown in Table 5. Both the combined group of all exercising subjects and the EX group exhibited significant differences between one- and two-minute rates at both pre- and post-treatment. There was no significant difference between the one- and two-minute rates for the SE group at pre-treatment, but there was a significant ($p < .05$) difference at post-treatment.

Differences in both one-minute and two-minute recovery heart rates between pre- and post-treatment for exercise groups and all exercising subjects are presented in Table 6. As a combined group, all exercising subjects experienced a significant ($p < .05$) decline in the one-minute recovery heart rate, when expressed as a percentage of the IPE heart rate; the two-minute rate, expressed in the same manner, did not change. The EX group did not exhibit a significant change in either the one-minute or the two-minute recovery

Table 6

Differences in One- and Two-Minute Recovery Heart Rates, as a Percentage of Immediate Post-exercise Heart Rate, between Pre- and Post Treatment for all subjects, and divided into exercise groups

Means ± SEM

	One-Minute Rate	Two- Minute Rate
All Exercising Subjects	-7.9 ± 4.5[a] (18)	-4.8 ± 5.8 (16)
Exercise	1.5 ± 4.8 (9)	8.9 ± 8.0 [d] (8)
Some Exercise	-17.3 ± 6.5 [a] (9)	-18.5 ± 5.1 [b,d] (8)

Negative signs in front of values indicate a decline in recovery heart rate from pre- to post treatment; other values represent an increase. Values with superscripts a or b represent a significant change in rate between pre- and post treatment , where the level of significance is indicated as follows: a: p = .05 b: p = .01 Between exercise groups , significant differences exist between values with the same letters as superscripts, where the level of significance is designated as follows: c: p = .10 d: p = .05. Differences between one- and two-minute rates were not evaluated, and the group of all exercising subjects was analyzed seperately. Numbers in parentheses indicate the number of data values used in the calculation above it.

heart rate 2 between pre- and post-treatment. The SE group experienced significant decreases in both the one-minute ($p < .05$) and two-minute ($p < .01$) recovery heart rates. The change between pre- and post-treatment in the two-minute recovery heart rate for the SE group was significantly larger than the non-significant change for the EX group.

DISCUSSION

The amalgamation of nutrition education and aerobic exercise has proven to be a successful approach to weight loss (26, 37). Several researchers have suggested that a comprehensive program, which incorporates diet with nutrition education, behavior modification, and exercise is most likely to produce the most effective long-term control of weight (19, 20). In this study, all 50 subjects lost an average of 2.1 kg ($p < .01$) which resulted from a loss of 2.7 kg ($p < .001$) of body fat (BF) and a gain of .6 kg ($p < .001$) lean body mass (LBM).

It has been reported that behavior modification, in conjunction with nutrition education in a weight loss program will produce moderate weight loss, averaging one pound per week, or 10-12 pounds for the duration of a program (38-40). Those women who attended more than 80% of the classes (NU) lost an average of 3.3 kg, or 7.3 pounds (lb) ($p < .001$), which was significantly ($p < .01$) more than the .9 kg (2 lb) weight loss (NS) by those who attended less than 80% of the nutrition sessions (SN) (Table 2). However, even the 3.3 kg weight loss by the more consistent NU group was considerably less than the average loss of one pound per week as observed with other behavior modification programs (38-40). It should be noted, however, that the nutrition

education/behavior modification intervention in this study was for a three month period, and the post-treatment weights presented here were taken three months after the last scheduled nutrition session. A trend toward regaining weight has been cited as a drawback to behavior modification programs, although this had not been reported until one to two years post-treatment (41).

The use of bioelectrical impedance analysis in the estimation of body composition in obese subjects has previously been critized due to a tendency to overestimate TBW, and thus underestimate body fat, in such individuals (42-44). However, through the use of regression equations incorporating height, weight, and gender, strong correlations between BIA-determined body composition parameters, and those determined both by hydrodensitometry and deuterium-isotope dilution have been repoerted. In 1986, Lukaski et al reported a correlation of .95 between the fat-free mass determined by BIA and that determined by hydrodensitometry in women who ranged from four to forty-one per cent body fat (45). Kushner and Schoeller (46), found a correlation of .98 for TBW determined by BIA and that determined by deuterium-isotope dilution in obese female subjects. Two years later, in a study which involved 1567 subjects who ranged from three to fifty-six per cent body fat, Segal and coworkers (47) found that the

correlation between LBM as determined by BIA and that determined by densitometry was greater for the obese than the normal subjects (.95 vs. .91).

As mentioned previously, another problem that has been identified in the use of bioelectric impedance in the analysis of body composition is the state of hydration of the subject (48). Hydration can bee affected by body temperature changes and excessive sweating. Although Hoffer (49) has reported a correlation of .91 between TBW determined by tritiated water and by impedance in patients with varying degrees of hydration, the environmental conditions of this study should be kept in mind as one considers the subsequent changes in body composition. It is possible that the hotter, more humid weather in June (post treatment) versus January (pre-treatment) in this study had an effect on water retention in these obese subjects. This could cause an overestimation of TBW and thus LBM, and an underestimation of body fat at post treatment.

Like the changes in body weight, changes in body composition also appeared to be favorably affected by more consistent attendance to the nutrition education/behavior modification classes. Those who attended 80% or more of the nutrition classes (NU) decreased their % BF, and increased their LBM by 3.4% ($P < .001$). These changes in body composition, when expressed as total fat and lean weight

changes, represented a loss of 4.2 kg of body fat, and a gain of .9 kg of LBM. Those who were less consistent in their attendance (SN) experienced only a 1% decrease in BF, with a similar increase in % LBM, which represented a loss of only 1.2 kg of body fat and a gain of .3 kg of LBM. Other studies of behavior modification/weight loss programs have not typically considered the effect of the frequency of attendance on body weight or body composition changes, although Bray and Gray have identified both the frequency and regularity of attendance (to a nutritional weight loss program) as possible indicators for determining those who are likely to succeed (39). Whether this is an effect of the nutrition classes, or perhaps a reflection of greater motivation to lose weight by those who attended a greater number of sessions is unclear.

Several investigators (Kennrick et al, Lennon et al, Wirth et al) found that the addition of exercise to dietary restriction improved the amount of weight lost by dietary means alone (50-52). Sikand (53) found that the addition of exercise to a dietary weight loss program for obese women improved group cohesiveness, communication, and information exchange among participants which contributed to greater weight loss and nutrition class attendance. In the present study, those who participated most frequently in exercise, (EX) (> 70% of the time), lost more weight (3.9

kg, $p < .01$) (-3.7 kg fat, -.1 kg LBM) than both those who participated less consistently (SE) (1.0 kg, NS) (-1.8 kg fat, +1.0 kg LBM) and those who did not exercise or attended less than 30% of the sessions (NE) (1.4 kg, NS) (-2.4 kg fat, +.9 kg LBM) (Table 2). As with the nutrition classes, the degree that a greater weight loss was due to the role of exercise, versus perhaps a greater level of motivation toward weight loss or greater fitness, can only be speculated upon in the present study.

Although some investigators have found the addition of exercise to a dietary weight loss program not to produce any further weight loss than that by dietary means alone, it has been consistently shown to enhance the loss of fat weight, while preserving, or increasing lean body mass (54-59). In the present study, both the EX and NE groups experienced decreases in their percentages of body fat (2.8 and 1.9, respectively, $p < .05$, or -3.7 and -2.4 kg of body fat, respectively) and corresponding increases in their percentages of lean body mass (2.8 and 1.9, respectively). Interestingly, this increase in lean body mass was mirrored by a comparable increase in absolute lean body mass only for the NE group (+.9 kg lbm); the EX group remained essentially unchanged (-.1 kg lbm). There were no significant changes in % body fat or % lean body mass (1.8%) for those who less consistently exercised (SE group). However, like the NE

group, there was a similar increase in absolute lbm (+1.0 kg), and a smaller decline in body fat weight (-1.8 kg). This is unlike the study by Zuti and Golding (60) in which the effects of exercise, dietary restriction or their combination on body composition were compared. These researchers found that both groups of exercising subjects lost a greater amount of fat weight than the diet-only group, and that exercisers had increases in lean body mass, while the diet-only group lost lbm. It is unclear why the NE group in this study experienced an increase in lbm, while the EX group experienced no change (or a slight decrease) in this parameter. The effects on body weight and body fat in the EX group in this study are similar to the findings of Lennon and coworkers (51) that the regimen of prescribed exercise-with-diet resulted in greater decrements in: body weight, % body weight, body fat and % body fat than either regimens of self-selected exercise-with-diet or diet alone in overweight individuals.

Thus, in this study it would appear that, when compared both to less consistent, and no exercise, consistent exercise was an important element in overall weight loss, and enhanced the amount lost as fat, while preserving -although not increasing, LBM. This is supported by a stronger correlation of the change in body weight with the change in % BF for the EX group ($r = .69$, $p < .05$), than

for the group as a whole ($r = .35$, $p < .05$), a correlation that was not present in any other group.

Interestingly, however, less consistent exercise (SE) had less of an effect on body weight and fat loss than dietary means alone (NE). This suggests that consistent exercise may play a valuable role in reducing fat and body weight. However, the effects of caloric restriction and less consistent exercise on body weight and composition are not as readily apparent.

Plasma Variables

Obesity has been associated with a poor plasma lipid profile (4, 7, 61). In particular, elevated levels of plasma total cholesterol (TC) and triglycerides (TG) (5, 61-63), and reduced levels of plasma high density lipoprotein cholesterol (HDL-C) (4-7, 64) have been observed in obese individuals. Such a lipoprotein-cholesterol pattern is considered predictive of the risk of CHD (65). High levels of plasma LDL-C have also been associated with both a higher incidence (65-67) and an increased severity (68-70) of CHD. Some research has suggested that low levels of plasma HDL-C may be the best predictor of subsequent CHD (65-67). The subjects in the present study, however, had mean lipoprotein-cholesterol levels which were within normal ranges.

Caloric restriction and resulting weight loss have often been found to lower plasma TC (slightly), and LDL-C (more so) in obese individuals (71-75), while the effect on plasma HDL-C has been more variable (72, 76). In the present study, there was no effect on plasma TC or LDL-C despite a 3.3 kg weight loss, for those in the NU group (Table 3). The significant decline in plasma HDL-C (2.8 mg/dl, $p < .05$) for this group would not be atypical; several researchers have reported decreased plasma HDL-C levels following a hypocaloric diet (71, 73, 77, 78) in obese subjects, which has occurred in conjunction with decreases (71, 79), as well as with no change in plasma TC levels (78). This depression of plasma HDL-C levels has been reported to be a temporary phenomenon, increasing above baseline between two and eight months after treatment (74, 78, 79).

Interestingly, for those in the present study who attended nutrition classes less than 80% of the time (SN) and lost only .9 kg of body weight, there was a significant decline in plasma TC (22.5 mg/dl) (Table 3), which was accompanied by a significant decline in plasma HDL-C (7.1 mg/dl $p < .001$). This decline in plasma HDL-C was concomitant to a significant ($p < .001$) decline in the plasma HDL_2-C component as well. Schwartz (80) has found that changes in plasma HDL_2-C are primarily responsible for

changes in plasma HDL-C. Post-treatment values of plasma HDL-C and HDL_2-C for the SN group were significantly lower than for the group who consistently attended nutrition classes (42.5 vs 46.3 mg/dl, and 19.3 vs 23.9 mg/dl), suggesting that the greater decrease in plasma TC resulted in a greater decrease in plasma HDL-C.

Exercise, whether it resulted in weight loss or not, has been found to decrease levels of plasma TC (slightly) and LDL-C (81), and increase plasma HDL-C (82-84), as well as the ratio of TC to HDL-C (81). Among exercise groups in this study, only those who inconsistently participated in exercise (SE) experienced a significant ($p < .01$) decrease in plasma TC (Table 3). This was interesting, as correlations have been found between the amount of exercise and subsequent lipid changes (85-88). However, the recovery heart rate analyses suggested that there was a greater conditioning effect in the SE group, which was probably due to their apparently lower initial level of fitness. Tran et al (81) have found that a lower initial level of cardiovascular fitness (VO_2 max), was associated with larger decreases in lipids and lipoproteins in exercising subjects. Furthermore, correlations have been found between initial levels of lipoprotein-cholesterol levels and subsequent changes (81). While there were no initial differences among exercise groups based on raw mean values of plasma

lipoprotein-cholesterol, the plasma TC value of 219 mg/dl for the SE group was 19.8 mg/dl higher than that of the EX group, and 22.3 mg/dl higher than that of the NE group. Furthermore, there were strong correlations in both the EX and the SE groups between initial plasma TC levels and subsequent change (r = .60 and .52, respectively). These factors may partially explain the significant decline seen only in the SE group. Therefore, one might expect the changes in lipoprotein-cholesterol variables to be more pronounced in this group.

Schwartz (80) has suggested that exercise has its primary effects on lipoprotein-cholesterol through the plasma HDL-C mediated reverse transport system. In his review of 66 training studies, Tran (81) found that exercising subjects experienced a non-significant increase in plasma HDL-C. That a larger increase did not occur may be related to the subsequent finding that larger increases in HDL-C were associated both with more hours of training and lower training intensities. Although the more consistent participation in exercise (EX) in this study did not result in increased plasma HDL-C levels, it did appear to mitigate significant declines in plasma HDL-C and HDL_2-C seen in both groups that exercised less (Table 3). The trends in plasma HDL-C within all exercise groups were accompanied by similar changes in plasma HDL_2-C, as Schwartz

(80) has previously observed. Although exercise has been shown to decrease the ratio of plasma TC to HDL-C, in subjects who exercised only, as well as those who combined exercising with caloric restriction (58, 89), there were no significant changes in the plasma TC/HDL-C ratio for any group in this study.

Plasma TG levels have been found to be significantly related to BMI (90) and other measures of overweight or body fat distribution (91). They have also been associated with coronary artery disease (92, 93). Caloric restriction has been shown to result in lowered plasma TG levels in obese individuals (71, 72, 78). In this study, the group that most consistently attended nutrition classes experienced a decrease in plasma TG of 19.8 mg/dl ($p < .05$). This was similar to the finding by Schwartz (80) that plasma TG decreased in a group of subjects who were dieting only. However, all groups of subjects in this study did significantly reduce their caloric intake, yet there were no significant changes in plasma TG for any other groups, although there was a consistent trend to lower plasma TG for all groups.

Although exercise training has been found to result in lowered plasma TG levels (81), exercise in this study had no significant effect on levels of plasma TG. Similar to the results of this study, Lampman et al (94), and Hill et al

(58) both found no change in plasma TG levels in obese individuals who reduced their caloric intake and either exercised or remained sedentary. Perhaps the initial level of plasma TG (normal levels for all groups) and the greater variability of plasma TG levels were limiting factors within which the effects of nutrition or exercise would not be statistically significant.

The association between obesity and high plasma insulin has been recognized (95), and high plasma insulin values have been found in one study to be independently predictive of CHD (96). Weight loss, through caloric restriction (97) has been associated with improvements in carbohydrate metabolism in obese individuals. In the present study, both nutrition groups (NU and SN) experienced significant reductions (4.8 and 7.1 uIU/ml, respectively) in fasting plasma insulin (Table 3). In the SN group, this occurred despite the lack of a significant loss of weight (Table 2). For both groups, however, there were significant correlations between the changes in insulin and changes in total kcal, total fat and saturated fat, suggesting the importance of specific dietary changes on plasma insulin.

Weight loss through exercise has also been associated with improvements in carbohydrate metabolism in obese individuals (98). Furthermore, physical training which did not result in weight loss in obese subjects has previously

been reported to result in decreased insulin values (99). In this study, both the EX and NE groups experienced significant declines in fasting insulin, while the decrease for the SE group was not significant. However, Lampman (100) has suggested that exercise may have differing effects on insulin, dependent on the degree of glucose impairment and/or obesity. Indeed, the initial insulin value of the SE group was lower that any other group. However, it was similar to the EX group, which did undergo a significant decline in fasting insulin, suggesting that more consistent exercise may be necessary to reduce normal levels of insulin.

For all groups, there were significant correlations between the change in plasma TC and the change in plasma LDL-C ($r = .89$ to $.99$) as would be expected. For the group of 50 subjects, there was a significant correlation ($r = .41$, $p < .01$) between the change in plasma HDL-C and the change in plasma HDL_2-C. Although this correlation is relatively low it still supported the finding by Schwartz (80) that changes in plasma HDL_2-C account for the majority of variability in plasma HDL-C levels .

Nutrient Intake

All groups of subjects in this study were able to achieve significant ($p < .05$) reductions in total caloric

intake. All groups except the SE group, that inconsistently attended exercise, significantly ($p < .05$) reduced their intake of fat and saturated fat. There were no changes in the M/S or P/S ratios. The study group as a whole ($n = 50$) significantly ($p < .05$) decreased its intake of both dietary cholesterol and sugar, while the sugar intake of the NU group also declined significantly ($p < .05$).

For the whole study group, there were significant negative correlations for both CHO and pectin with the plasma TC/HDL-C ratio ($r = -.33$, $r = -.35$, respectively). For both nutrition groups (NU and SN), as well as the NE group, there were significant negative correlations (ranging from $r = -.34$ to $r = -.77$) between the changes in CHO or pectin and either the TC/HDL-C ratio or LDL-C. High-carbohydrate diets have previously been found to improve plasma TC levels in normal (101), hyperlipoproteinemic (102), and (maturity-onset) diabetic subjects (103). Decreases in plasma TC are accompanied by decrements in both plasma LDL-C and HDL-C for individuals on high-carbohydrate diets (101-106). Pectin, a soluble fiber, has been found by numerous investigators to result in lower plasma TC levels, while no reduction in plasma HDL-C was reported (107).

Although decrements in plasma TC and LDL-C have frequently been reported in obese individuals following a hypocaloric diet (71-75), in this study there were low, but

significant negative correlations between total kcal consumed and both plasma TC and LDL-C for the whole study group, and for both nutrition groups (r = -.35 to -.52). The positive association found in the present study between total kcal consumed and fasting plasma insulin (r = .51) for the whole group, as well as for the NU (r = .47), SN (r = .61), and NE (r = .57) groups, has been reported by others (108-110).

In this study, significant correlations which existed for all subjects between dietary fat intakes and plasma variables were relatively low (r = -.28 to -.34), and were inconsistent, thus extrapolation and interpretation were difficult. Total fat intake was found to be positively correlated in this study, for all 50 subjects, to fasting plasma insulin (r = .51). Similar correlations, ranging from r = .45 to r = .67, existed for other groups of subjects. Protein was also significantly and positively correlated to insulin for the whole group (r = .43) as well as the NE group (r = .82). Protein had previously been found to be insulinogenic (111). The negative correlation observed between the change in the percentage of CHO in the diet and fasting plasma insulin (r = -.42) for the study group was in agreement with data published by Himsworth at least 55 years ago (112).

That hypertriglyceridemia often occurs in conjunction

with high CHO diets has been cited as a major drawback to their use (113). In the present study, for the group as a whole, the correlation between the change in the percentage of kcal from CHO in the diet and the change in plasma TG ($r = .36$, $p < .05$) was relatively low, but present. Although there was no significant correlation, the decrease in plasma TG could perhaps be partially due to the significant drop in sugar intake (103) (23.5 g, $p < .05$).

Previous researchers have found various measures of overweight to be positively correlated with plasma TC, TG, LDL-C and insulin, and negatively with plasma HDL-C, HDL_2-C, and the HDL-C/TC ratio (5, 91, 114, 115). For both the SN and the NE groups in this study, there were significant correlations ($r = .56$, $r = .43$, respectively) between the change in body weight and the change in plasma TG. For none of the other groups were there any significant correlations between changes in body composition and changes in plasma variables. Perhaps the lack of correlation between changes in such variables in this study suggests that the static correlations observed by others may not be a cause-and-effect relationship, or that such associations are not stable during active weight loss.

Both dietary fat and dietary sugar, especially sucrose, have been implicated as having a causal role in the development and/or maintenance of obesity, at least in rats

(116, 117). For all groups except the SE and NE groups, there existed a significant positive relationship (ranging from $r = .32$ to $r = .69$, $p < .05$) between the change in body weight and the change in sugar and/or sucrose intake. The NU group, also had a significant correlation ($r = .35$) between the change in % BF and the change in sugar consumed, while the EX group approached significance ($r = .57$, $p < .06$). This supported previous work which found a high-sugar diet to lead to increased to body fat (117). There were, however, negative correlations ($r = -.32$ and $-.48$, respectively) between the change in % BF and the change in the per cent of calories derived from fat, for both the total group ($n = 50$) and the NU group, which cannot be explained.

While comparisons and contrasts have been made between the changes in nutrient intake components observed in the present study and those observed in other studies, it should be kept in mind that since subjects here were on ad lib diets, the correlations must be viewed with caution. That is, since nutrient composition of the diet was based on dietary recall, observed dietary changes may not accurately reflect long-term dietary patterns. Certainly, cause-and-effect relationships between variables cannot not be assumed. Additionally, other feeding studies, to which the results of the present study were compared, were controlled

for at least some other variables such as exercise, diet, weight loss, fat loss and dietary constituents. As the population in the present study was a free-living one, it was difficult or impossible to control such confounding variables.

Recovery Heart Rates

Exercise, performed at sufficient frequency, duration, and intensity, is likely to result in improvements in cardiovascular parameters, as well as work capacity. Several investigators (90, 118, 119) have shown walking to be an effective modality for the achievement of such physical improvements. Aerobic dance has also been proven to be capable of producing beneficial changes in work capacity, ventilatory exchange, as well as resting and exercising heart rates (40, 120-128). In the present study, there were no significant differences among recovery heart rates, expressed as a percentage of the immediate post-exercise (IPE) heart rates, among groups (Table 5). More detailed analysis, however, showed that the one-minute recovery heart rate of the EX group at pre-treatment was significantly different from its respective IPE heart rate, but that of the SE group was not (Table 5). This suggested that there was some degree of self-selection; that is, that those who were more fit (had a lower one-minute recovery

heart rate) were also those who attended exercise most frequently. At post-treatment, however, the one-minute recovery heart rates of both groups were significantly lower than their respective IPE values, indicating that exercise had a beneficial effect for the SE group, while it served to maintain the fitness of the EX group.

This effect of exercise on recovery heart rates between groups is also evident through the analysis of differences between heart rates one and two at both pre- and post-treatment (also Table 5). Again, for the EX group, the two-minute recovery heart rate was significantly lower than the one-minute recovery rate at pre-treatment, but this was not true for the SE group. At post-treatment, the two-minute rate was significantly lower than the one-minute rate, for both exercise groups. This again indicated a beneficial effect of exercise on recovery heart rates for the apparently less-fit SE group, while maintaining cardiovascular status in the EX group.

This apparent conditioning effect is further evident in Table 6. Those in the SE group, who appeared to be less fit initially, experienced significant declines in both one- and two-minute recovery heart rates between pre- and post-treatment (17.3% and 18.5% of IPE, respectively). The EX group, however, experienced (non-significant) increases in both heart rates, indicating no change in cardiovascular

conditioning. Moreover, the slight increase in the two-minute heart rate for the EX group was significantly different from the significant decline for the SE group (Table 6).

These results suggest that even moderate exercise may elicit more pronounced improvements in those who are less fit initially, while consistent exercise serves to maintain cardiovascular functioning in more fit individuals. Perhaps this greater conditioning effect in the SE group was a factor in the significant derease in plasma TC observed in the SE, but not the EX group. However, in considering these changes in recovery heart rate values, the possible role of differing environmental conditions should be kept in mind. Pre-treatment values were collected in January, but post-treatment at the end of June, in south-central Virginia. The greater heat at post treatment may have resulted in higher IPE heart rates, and the humidity in less evaporative cooling, and thus higher recovery heart rates at post-treatment. Indeed, non-significant increases in both one- and two-minute recovery heart rates were evident in the EX group. Moreover, the heat and humidity as well as the duration of the study may have had an effect on the motivation and effort of the participants during the post-treatment 10-minute walk, resulting in a lower IPE heart rate than otherwise might be achieved, but likely a higher

recovery heart rate.

This study has provided evidence that participation in a nutrition education/behavior modification program aimed at weight loss can produce modest weight loss. The addition of aerobic exercise on a consistent basis may also aid in the loss of both body weight and body fat, while preserving LBM. Additionally, exercise may enhance changes in lipoprotein patterns by mitigating declines in plasma HDL-C and HDL_2-C. General participation in nutrition education/behavior modification classes proved to be an effective modality to elicit beneficial changes in nutrient intakes in obese black women, even when measured three months after the end of the nutrition intervention program. However, it would appear that more consistent attendance was necessary to effect significant declines in dietary sugar and the percentage of calories derived from fat, as the declines in these variables were significant for those who attended more than 80% of the nutrition classes, but not for those who attended less than this.

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Summary and Conclusions

This study was conducted in order to evaluate the effects of a multi-factorial weight loss program on body weight and composition, plasma lipoprotein-cholesterol, triglycerides, insulin, and nutrient intake in obese black women. Fifty healthy females, between 20 and 51 years of age, who were at least 20% above the desirable weight-for-age based on the Metropolitan Life Insurance Co. tables, and who were considered sedentary according to a questionnaire participated in the study. Thirty-five subjects met once per week for two hours each, and attended 80% or more of the nutrition classes (NU group) which emphasized caloric reduction through nutrition education, behavior modification, and stress management. The fifteen subjects who participated in less than 80% of these classes (SN group) were considered as a separate group for comparison. The duration of this aspect of the program was 11 weeks. The low-impact aerobic exercise program, however, continued beyond this, and lasted a full six months. Twelve subjects participated consistently in these supervised exercise sessions, and attended more than 70% of the one-hour, thrice-per-week classes (EX group). Sixteen subjects participated inconsistently (30% to 70% of the sessions) (SE group), and 22 subjects participated in less than 30%, or did not attend exercise at all (NE group). Body weight and

composition were determined both at the beginning and the end of the six-month intervention period, as were plasma lipoprotein-cholesterol, plasma TG and plasma insulin. Two twenty-four hour dietary recalls were used to determine energy and nutrient intake both at the start and end of the six-month treatment period.

For all subjects (n = 50), there was an average weight loss of 2.1 kg, which was accompanied by a decrease in percent body fat (%BF) (2.2%), and an increase in percent lean body mass (% LBM) (2.2%). Among nutrition groups, these changes were accounted for primarily by the NU group; those subjects in the SN group experienced no significant change in either body weight or composition. Among exercise groups, the beneficial changes in % BF and % LBM observed for the whole group were accounted for by the EX and NE groups. A significant decrease in body weight occurred only in the EX group. Subjects in the SE group experienced no significant change in either body weight or body composition.

Post-treatment values of % BF and % LBM for the EX group were significantly lower and higher, respectively, than the corresponding values for either the SE or NE groups. This corresponded to the greater changes observed in these variables for the EX group [-2.8% BF, +2.8% LBM, which resulted in 3.7 kg BF lost and left the total LBM essentially unchanged (-.1 kg)], compared to either the SE

group (-1.8% BF, +1.9% LBM, or 1.8 kg BF lost and 1.0 kg LBM gained), or the NE group (-1.9% BF, +1.9% LBM, or 2.4 kg BF lost and .9 kg LBM gained).

For the total group of 50 subjects, there was a significant positive correlation between the change in body weight and the change in body fat ($r = .35$). There was a similar correlation among those who consistently attended nutrition classes (NU) ($r = .33$, $p < .05$), while this correlation was much stronger for those who consistently participated in exercise (EX) ($r = .69$).

Significant reductions in plasma TC, HDL-C, HDL_2-C and insulin were observed for all subjects. Within nutrition groups, there was a significant decrease in plasma TC (22.5 mg/dl) for the SN group, which was accompanied by significant declines in both plasma HDL-C (7.1 mg/dl) and HDL_2-C (8.3 mg/dl). The NU group experienced no significant decline in plasma TC, yet there was a significant decline in plasma HDL-C (2.9 mg/dl). However, post-treatment values of both plasma HDL-C and HDL_2-C for the NU group (46.3 and 23.9 mg/dl, respectively) were significantly higher than those for the SE group (42.5 and 19.3 mg/dl, respectively). Both groups experienced significant declines in fasting plasma insulin, while plasma TG values decreased significantly only in the NU group.

Within exercise groups, only the SE group experienced a significant decline in plasma TC (18.6 mg/dl). Significant

declines in both plasma HDL-C and HDL_2-C were observed in both the SE and NE groups, while no significant change in either of these variables occurred in the group that exercised consistently. This suggested that more consistent exercise may preserve plasma HDL-C and HDL_2-C levels, although this may be concomitant to the absence of a decrease in plasma TC. Similarily, no significant changes occurred in the TC/HDL-C ratio for any exercise group, yet the EX group was the only one for which a (non-significant) decrease occurred, while the levels for the SE and NE groups went up (NS). No signficant changes were seen for any exercise group in LDL-C, suggesting that during exercise, more of the variability in plasma TC is due to changes in the plasma HDL-C subfraction. However, both groups of exercising subjects experienced non-significant declines in LDL-C, while the NE group underwent a (non-significant) increase in this parameter. The declines in TG were not significant for any exercise group. The pre-treatment insulin value for the NE group was significantly higher than that for the EX group, which likely was related to the higher initial body weight in the NE group. Significant (absolute) declines in fasting plasma insulin occurred for both the EX and NE groups, while no change occurred for the SE group.

For the group of 50 subjects, as well as the No Exercise group, there was a significant correlation between

the changes in plasma HDL-C and its HDL_2-C component. There were also signficant correlations for initial levels of various lipoprotein-cholesterol, TG, and/or insulin with their respective subsequent changes, for all groups of subjects. For both the SN group, and the NE group, there was a significant correlation between the change in body weight and the change in TG ($r = .56$, $r = .43$, respectively).

For all subjects, a significant reduction of 580 kcal per day was reported. This was accompanied by significant declines in all three macronutrients; carbohydrate, protein, and total fat as well as all three types of fat: saturated, polyunsaturated, and monounsaturated. Significant declines in dietary cholesterol and sugar, as well as a significant increase in the percentage of calories derived from protein were also reported. Compared to the SN group, the NU group at pre-treatment consumed significantly less dietary cholesterol (112.0 mg) per day, and significantly reduced their intake of dietary sugar and the percentage of calories derived from fat. Compared to both the EX and NE groups, the SE group did not significantly reduce its intake of either total or saturated fat.

Significant correlations between changes in nutrient intake components and changes in body composition for the group of 50 subjects included a rather weak relationship of $r = .32$ for dietary fat and body fat. The changes in

caloric intake, sugar, and sucrose were all significantly correlated with the change in body weight ($r = .30, .36$, and $.32$, respectively).

Correlations ($p < .05$) between the change in nutrient intake components and the change in plasma variables included negative correlations for both carbohydrate and pectin with the TC/HDL-C ratio ($r = -.33, -.35$, respectively), and a positive correlation for the per cent of kcal as carbohydrate with plasma TG ($r = .36$). There were positive correlations between the change in total kcal and the change in plasma insulin ($r = .51$), and between the change in dietary saturated fat and the change in plasma insulin ($r = .48$). Negative correlations existed for both the changes in polyunsaturated fat and the P/S ratio with the change in plasma TC ($r = -.29$ and $-.30$, respectively).

Many obese persons have made several attempts at weight loss. The majority of programs for this purpose have typically focused on one, or perhaps two, treatment methods. Such programs have often been concomitant to high attrition rates and minimal-to-moderate weight losses, while the regaining of weight has been a major drawback to their use. In this study, 98 subjects began the intervention program; 65 of these began the exercise sessions. 50 subjects completed the six-month intervention program; 28 of these subjects had participated in more than 30% of the exercise sessions, while only 12 had participated in more than 70%.

Thus, the overall attrition rate was nearly 50%, and the exercise attrition rate 57%. These values are within the ranges reported by previous investigators.

Few reports of weight loss programs for obese individuals have considered the degree of individual participation on weight loss outcomes. Fewer have considered the level of motivation or psychosocial characteristics of the subjects and how these relate to the level of participation. Furthermore, psychosocial, ethnic, or cultural characteristics which may impinge upon weight loss have frequently not been considered. In this study, although there were no statistically significant differences among pre-treatment body weight mean values, the pre-treatment weight of the NE group was considerably higher than either the EX or the SE group (differences of 10.9 and 12.2 kg, respectively). Furthermore, the pre-treatment insulin value of the NE group (32.4 uIU/ml) was significantly higher than that of the EX group (18.9 uIU/ml), while the value for the SE group (18.7 uIU/ml) was similar to that of the EX group. This suggested that some degree of self-selection may be have been operable in this study. That is, the heaviest subjects, which were those with the highest insulin levels, and therefore those most in need of exercise for the promotion of decreased insulin and body weight values, were the same subjects who failed to attend 30% or more of exercise sessions. Perhaps the larger

size of these women contributed to a greater negative self-image, and thus the decision not to participate in organized exercise. As was observed with several subjects who did not complete the study, a larger body size presented a physical hindrance to movements required for land-based exercise. For some of these same women who also lacked personal transportation, their physical size presented a further hindrance to the use of public transportation.

Although it was not measured, it appeared that there were differences in motivation among groups of subjects. Those who consistently attended exercise and/or nutrition classes appeared outwardly motivated. Those who attended exercise or nutrition less consistently seemed not to be as strongly motivated. There was an expectation among these latter individuals that the program would accomplish their weight loss goals, without their having to work too hard themselves.

A factor in this study which appeared to impinge upon an individual's attitude and motivation, and thus the subsequent success or failure of weight loss, was the degree of support from family or friends. Several women who experienced particularly successful weight loss outcomes had become friends, and frequently talked and provided encouragement to each other during the exercise sessions, and exchanged information gained during nutrition classes. Several women reported making beneficial changes in the

diets of their families, and that the support of their families or spouses helped them to maintain suggested dietary changes. On the other hand, some women reported a lack of acceptance by their families to recommended dietary changes, while a few women reported rather strong negative attitudes by their husbands to their weight loss efforts. Single women sometimes reported that eating alone, as well as their busy schedules, often contributed to unhealthy food choices.

This study was conducted in an area of the state known as Southside, Virginia. The pork industry has long been a large part of the economy of this region, and many subjects reported that pork and pork products have been an integral part of their diet for generations. Thus, a high intake of fat and saturated fat was typical for many subjects, while efforts to decrease these levels in the diet were reported to be difficult for many subjects.

Although difficult to measure or control, attempts should be made in future weight loss programs to identify attitudes, customs, and social or psychological factors, which may be specific within an ethnically or culturally defined population, that may impinge upon an individual's motivation, attitude, and/or subsequent success in a comprehensive weight loss program.

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APPENDICES

Appendix A

Individual Pretreatment Body Weight and Composition For 50 Obese Black Women

Subject	Body Weight (Kg)	% Fat	% Lean	% Water
201	113.9	47.4	52.5	33.2
206	104.9	40.6	59.3	38.1
209	78.0	31.5	68.4	45.9
218	111.6	43.0	56.9	36.3
219	92.1	32.9	67.0	44.6
222	75.6	32.9	67.0	44.6
230	91.0	48.0	51.9	34.9
242	75.4	33.5	66.4	44.1
248	116.0	45.1	54.8	34.8
252	84.2	41.1	58.8	37.8
256	68.4	35.2	64.7	42.6
302	82.5	37.3	62.6	40.9
303	76.0	40.1	59.8	38.6
307	95.2	53.2	46.7	29.6
308	72.7	37.3	62.6	40.9
312	98.1	46.2	53.7	34.1
319	77.1	43.0	56.9	36.3
322	92.8	40.1	59.8	38.5
336	102.6	43.9	56.0	35.6
347	69.4	35.6	64.3	42.2
352	77.7	29.0	70.9	48.2
354	99.4	35.5	64.4	42.4
355	96.4	49.8	50.1	31.6
358	60.8	38.9	61.0	39.5
361	76.3	40.9	59.0	37.9
363	117.4	56.1	43.8	27.9
364	85.9	37.1	62.8	41.0
366	81.6	42.1	57.8	37.0
402	88.3	37.7	62.2	40.5
404	103.4	30.4	69.5	46.9
405	69.2	37.7	62.2	40.5
406	99.4	44.1	55.8	35.5
407	76.8	42.6	57.3	36.6
408	66.6	35.2	64.7	42.6
409	106.6	43.5	56.4	36.0
410	76.5	32.4	67.5	45.1
413	94.7	38.1	61.8	40.2
414	77.5	28.8	71.1	48.4
416	144.6	42.2	57.7	36.9

Appendix A *(Continued)*

Individual Pretreatment Body Weight and Composition
For 50 Obese Black Women

Subject	Body Weight (Kg)	% Fat	% Lean	% Water
417	80.2	33.8	66.1	43.8
421	87.7	38.9	61.0	39.5
422	106.8	36.9	63.0	41.2
425	108.8	45.0	54.9	34.9
428	89.2	50.4	49.5	31.3
429	102.5	36.9	63.0	41.2
434	106.7	46.3	53.6	34.0
435	65.0	37.2	62.7	40.9
437	75.1	30.5	69.4	46.8
440	102.0	38.3	61.6	40.0
444	80.5	39.1	60.8	39.4

Appendix B

Individual Post-treatment Body Weight and Composition For 50 Obese Black Women

Subject	Body Weight (Kg)	% Fat	% Lean	% Water
201	99.7	39.9	60.0	38.7
206	103.0	36.3	63.6	41.7
209	76.6	30.7	69.2	46.6
218	114.7	42.6	57.3	36.6
219	84.0	31.7	68.2	45.7
222	70.4	30.7	69.2	46.6
230	83.2	40.7	59.2	38.1
242	70.6	27.6	72.3	49.5
248	112.5	45.4	54.5	34.6
252	85.4	35.1	64.8	42.7
256	68.1	37.5	62.4	40.7
302	79.0	32.9	67.0	44.6
303	69.4	37.1	62.8	41.0
307	94.6	58.1	41.8	26.9
308	73.2	26.0	73.9	51.1
312	101.7	45.5	54.4	34.5
319	75.2	36.9	63.0	41.2
322	93.1	34.4	65.5	43.3
336	100.5	41.3	58.6	37.6
347	65.0	33.0	66.9	44.5
352	78.0	29.7	70.2	47.6
354	100.6	29.9	70.0	47.4
355	100.3	50.0	49.9	31.5
358	60.2	37.6	62.3	40.6
361	72.3	36.3	63.6	41.7
363	117.2	51.2	48.7	30.7
364	86.8	40.2	59.7	38.5
366	83.8	35.6	64.1	42.1
402	88.0	31.9	68.0	45.5
404	100.6	32.5	67.4	45.0
405	67.6	30.3	69.6	47.0
406	102.5	44.0	55.9	35.6
407	76.0	45.2	54.7	34.7
408	61.5	32.4	67.5	45.1
409	105.7	44.7	55.2	35.1
410	73.5	32.7	67.2	44.8
413	90.5	36.4	63.5	41.6
414	79.1	27.5	72.4	49.6
416	141.9	41.5	58.4	37.5

Appendix B (*Continued*)

Individual Post-treatment Body Weight and Composition
For 50 Obese Black Women

Subject	Body Weight (Kg)	% Fat	% Lean	% Water
417	77.2	30.3	69.6	47.0
421	82.1	36.6	63.3	41.4
422	90.1	28.9	71.0	48.3
425	107.0	43.0	56.9	36.3
428	89.6	47.3	52.6	33.3
429	98.1	35.9	64.0	42.0
434	96.7	39.2	60.7	39.3
435	84.2	36.2	63.7	41.8
437	75.8	29.3	70.6	47.9
440	99.3	36.7	63.2	41.3
444	79.9	36.0	61.9	40.2

Appendix C

One- and Two-Minute Recovery Heart Rates as a Percentage of Immediate Post-Exercise Heart Rate at Pre- and Post-Treatment for Exercising Subjects

	PreTreatment		Post-Treatment	
Subject	One-Minute Rate	Two-Minute Rate	One-Minute Rate	Two-Minute Rate
201	67	56	82	86
209	64	95	100	67
222	87	91	89	78
242	83	77	81	76
252	92	84	79	75
256	93	86	77	69
302	125	100	72	67
319	156	200	-	-
336	76	76	-	-
347	78	68	96	67
355	106	106	86	68
358	80	70	75	67
361	67	37	91	86
366	90	80	59	52
402	107	-	86	82
407	81	-	-	-
408	95	95	-	-
410	90	70	-	-
414	100	59	93	67
416	67	67	-	-
421	-	-	86	76
422	95	-	90	85
425	94	78	79	74
428	-	-	-	-
429	82	73	79	71
435	56	67	-	-
444	100	114	74	87

Appendix D

Individual PreTreatment Values of Total Cholesterol (mg/dl), Lipoprotein Cholesterol (mg/dl), Triglyceride (mg/dl), and Insulin (ulU/ml) for 50 Subjects

Subject	TC	TG	LDL-c	HDL-c	HDL_2	TC/HDL-c	INS
201	160	77	96.6	46	22	3.3	32.5
206	166	77	102.6	48	29	3.4	40.9
209	249	77	159.6	74	47	3.4	19.6
218	128	57	82.6	34	13	3.8	47.4
219	165	155	97.0	37	20	4.4	-
222	187	68	121.4	52	21	3.6	12.7
230	203	184	107.2	59	40	3.4	28.4
242	326	122	260.6	41	12	8.0	12.8
248	232	93	164.4	49	28	4.7	23.9
252	233	202	133.6	59	22	3.9	23.9
256	217	102	147.6	49	31	4.4	19.0
302	283	145	226.0	28	17	10.1	31.6
303	202	430	81.0	35	5	5.8	36.8
307	199	103	137.4	41	27	4.8	26.3
308	210	87	136.6	56	39	3.8	11.7
312	243	84	177.2	49	27	5.0	14.6
319	176	58	114.4	50	26	3.5	10.6
322	226	64	164.2	49	-	4.6	-
336	144	57	84.6	48	25	3.0	10.1
347	175	69	108.2	53	33	3.3	17.2
352	180	53	105.4	64	-	2.8	23.2
354	166	65	112.0	41	23	4.0	21.2
355	269	62	172.6	84	42	3.2	-
358	192	124	128.2	39	21	4.9	-
361	122	39	66.2	48	33	2.5	11.5
363	195	68	134.4	47	27	4.1	47.5
364	205	130	155.0	24	9	8.5	18.9
366	247	153	171.4	45	30	5.5	41.5
402	176	69	95.2	67	25	2.6	25.7
404	151	117	82.6	45	30	3.4	22.8
405	287	154	207.2	49	22	5.8	31.1
406	382	130	297.0	59	23	6.5	-
407	236	83	167.4	52	26	4.5	6.6
408	166	39	110.2	48	30	3.4	7.0
409	161	106	105.8	34	4	4.7	46.8
410	171	74	103.2	53	29	3.2	14.4
413	170	56	107.8	51	29	3.3	28.6

Appendix D (*Continued*)

Individual PreTreatment Values of Total Cholesterol (mg/dl), Lipoprotein Cholesterol (mg/dl), Triglyceride (mg/dl), and Insulin (ulU/ml) for 50 Subjects

Subject	TC	TG	LDL-c	HDL-c	HDL_2	TC/HDL-c	INS
414	197	55	121.0	65	26	3.0	16.4
416	191	69	135.2	42	28	4.5	33.1
417	158	88	97.4	43	22	3.7	19.9
421	190	60	114.0	64	38	3.0	18.8
422	192	121	123.8	44	25	4.4	12.0
425	230	127	173.6	31	20	7.4	27.3
428	230	42	168.6	53	19	4.3	13.5
429	204	188	125.4	41	24	5.0	25.5
434	131	68	82.4	35	23	3.7	98.7
435	165	71	98.8	52	15	3.2	13.3
437	194	37	104.6	82	36	2.4	15.4
440	188	62	127.6	48	29	3.9	24.8
444	275	112	199.6	53	-	5.2	-

Appendix E

Individual Post-Treatment Values of Total Cholesterol (mg/dl), Lipoprotein Cholesterol (mg/dl), Triglyceride (mg/dl), and Insulin (ulU/ml) for 50 Subjects

Subject	TC	TG	LDL-c	HDL-c	HDL_2	TC/HDL-c	INS
201	208	72	147.6	46	22	4.5	20.1
206	151	55	97.0	43	22	3.5	18.6
209	209	48	143.4	56	30	3.7	13.1
218	155	79	112.2	27	17	5.7	31.0
219	218	121	161.8	32	11	6.8	32.0
222	164	-	-	50	17	3.3	15.8
230	187	114	115.2	49	30	3.8	12.7
242	223	86	169.4	36	18	6.2	10.4
248	203	142	128.6	46	20	4.4	9.3
252	220	100	145.0	55	28	4.0	37.8
256	189	71	122.8	52	29	3.6	7.7
302	229	120	173.0	32	16	7.2	12.8
303	251	177	184.6	31	16	8.1	28.4
307	171	104	113.2	37	17	4.6	22.2
308	198	85	132.0	49	23	4.0	14.6
312	262	87	204.6	40	14	6.6	13.0
319	170	47	113.6	47	24	3.6	17.7
322	207	65	147.0	47	24	4.4	25.7
336	138	62	78.6	47	28	2.9	4.7
347	182	47	119.6	53	31	3.4	5.7
352	153	55	92.0	50	30	3.1	16.4
354	162	65	111.0	38	17	4.3	14.7
355	227	-	-	-	-	-	-
358	176	75	130.0	31	6	5.7	19.7
361	113	28	65.4	42	21	2.7	9.7
363	182	82	120.6	45	22	4.0	19.8
364	240	141	176.8	35	19	6.8	13.6
366	264	81	203.8	44	21	6.0	32.9
402	148	59	83.2	53	27	2.8	12.9
404	132	48	85.4	37	19	3.6	16.1
405	306	160	224.0	50	26	6.1	46.3
406	364	141	290.8	45	15	8.1	15.0
407	167	98	110.4	37	16	4.5	16.3
408	144	45	80.0	55	28	2.6	18.7
409	166	115	109.0	34	16	4.9	36.7
410	147	56	89.8	46	25	3.2	13.6
413	195	66	129.8	52	24	3.8	14.2

Appendix E (*Continued*)

Individual Post-Treatment Values of Total Cholesterol (mg/dl), Lipoprotein Cholesterol (mg/dl), Triglyceride (mg/dl), and Insulin (uIU/ml) for 50 Subjects

Subject	TC	TG	LDL-c	HDL-c	HDL_2	TC/HDL-c	INS
414	173	60	101.0	60	38	2.9	15.3
416	162	107	114.6	46	21	4.0	24.8
417	143	103	80.4	42	27	3.4	21.1
421	215	61	148.8	54	25	4.0	7.3
422	164	58	135.4	37	23	5.0	5.0
425	199	102	145.6	33	12	6.0	13.0
428	207	58	150.4	45	14	4.6	10.4
429	212	157	149.6	31	18	6.8	12.8
434	157	68	106.4	37	17	4.2	102.3
435	181	51	118.8	52	34	3.5	11.4
437	156	32	88.6	61	29	2.6	10.3
440	193	69	130.2	49	22	3.9	14.2
444	211	105	142.0	48	28	4.4	21.7

Appendix F

Individual PreTreatment Nutrient Intakes for 50 Subjects

Subject	Total Kcal (g)	Carbohydrate (g)	Protein (g)	Total Fat (g)	Saturated Fat (g)	Polyunsaturated Fat (g)
201	2460	276.1	81.1	115.8	32.1	40.4
206	2859	298.0	107.5	138.6	53.8	17.7
209	3273	344.2	132.4	153.8	58.0	20.1
218	1552	161.2	57.5	75.7	30.2	11.2
219	3254	458.0	100.1	131.1	14.7	22.0
222	1764	190.7	77.5	76.7	26.7	19.6
230	1248	151.0	54.0	47.2	18.0	8.0
242	1237	73.3	69.0	73.5	24.3	21.4
248	2041	205.4	73.5	104.0	40.4	19.9
252	1513	235.6	39.4	42.3	16.4	5.0
256	1302	103.9	42.7	80.8	23.8	26.5
302	1638	184.5	54.7	76.6	28.5	14.9
303	2003	281.5	71.2	70.6	31.0	14.7
307	1740	207.2	89.3	72.8	23.1	8.1
308	1324	169.6	49.4	54.2	19.0	13.9
312	1833	159.2	56.4	114.2	43.7	14.7
319	1514	171.2	50.2	63.8	13.4	8.1
322	1377	135.8	52.3	69.8	25.4	12.9
336	2038	218.2	69.1	100.4	34.8	19.7
347	2457	221.8	89.1	101.1	30.3	19.6
352	2560	347.5	96.8	89.8	36.6	11.6
354	2218	297.5	79.4	79.6	27.6	13.0
355	1777	166.5	86.4	84.9	25.9	24.1
358	1353	156.5	55.8	58.3	15.9	18.4
361	2023	194.7	81.8	103.0	32.0	25.6

Appendix F (*Continued*)

Individual PreTreatment Nutrient Intakes for 50 Subjects

Subject	Total Kcal (g)	Carbohydrate (g)	Protein (g)	Total Fat (g)	Saturated Fat (g)	Polyunsaturated Fat (g)
363	3620	270.5	113.2	128.1	47.7	17.4
364	2362	247.2	64.7	128.0	32.6	36.4
366	1696	174.5	67.7	84.1	30.4	10.2
402	1438	143.2	76.7	61.5	16.4	9.9
404	2352	306.0	77.8	94.3	36.7	19.6
405	1634	208.6	39.8	75.4	27.5	9.2
406	2192	196.0	77.4	68.3	24.7	11.0
407	1316	140.3	50.1	61.9	15.7	7.8
408	2039	219.6	75.5	98.7	28.5	24.8
409	1937	193.3	90.7	91.4	28.4	29.3
410	2046	271.0	26.1	88.2	26.7	29.7
413	2062	204.5	99.0	94.7	25.1	24.3
414	1930	191.8	79.8	95.5	29.8	27.8
416	2191	217.1	88.1	112.0	33.3	24.7
417	1689	226.6	57.2	65.3	27.4	14.4
421	1363	162.6	52.0	56.5	12.6	20.7
422	2243	318.2	79.0	75.2	34.1	13.8
425	2222	251.2	70.3	107.9	35.3	26.3
428	1782	186.1	87.7	76.0	25.8	12.0
429	2047	155.1	75.8	125.3	40.3	17.9
434	2070	276.3	59.5	83.0	31.3	12.6
435	2120	212.4	70.4	113.0	37.9	27.6
437	2370	351.2	79.8	75.2	20.5	23.8
440	1767	156.6	61.4	93.2	25.9	23.9
444	1737	230.5	66.8	66.4	23.1	14.5

Appendix F (*Continued*)

Individual PreTreatment Nutrient Intakes for 50 Subjects

Subject	Monounsaturated Fat (g)	P/S Ratio	M/S Ratio	Fiber (g)	Cholesterol (mg)
201	37.1	1.26	1.16	3.3	248
206	52.3	0.33	0.97	2.4	499
209	59.4	0.35	1.02	6.7	404
218	29.0	0.37	0.96	2.5	178
219	28.2	1.50	1.92	9.8	458
222	23.1	0.73	0.86	2.1	259
230	17.6	0.44	0.98	0.7	168
242	22.9	0.88	0.94	1.2	395
248	37.4	0.49	0.92	1.6	253
252	18.2	0.30	1.11	1.1	112
256	25.7	1.11	1.08	1.0	265
302	28.9	0.52	1.01	1.2	184
303	19.4	0.47	0.62	4.4	320
307	21.6	0.35	0.94	4.8	380
308	15.1	0.73	0.79	5.8	261
312	40.6	0.34	0.93	5.1	226
319	16.9	0.60	1.26	1.4	158
322	27.1	0.51	1.07	1.4	235
336	34.9	0.57	1.00	2.4	272
347	32.0	0.65	1.06	3.5	220
352	35.8	0.32	0.98	1.5	433
354	29.1	0.47	1.05	4.0	290
355	28.7	0.93	1.11	2.2	401
358	15.0	1.16	0.94	5.0	431
361	37.7	0.80	1.18	2.5	547

Appendix F (*Continued*)

Individual PreTreatment Nutrient Intakes for 50 Subjects

Subject	Monounsaturated Fat (g)	P/S Ratio	M/S Ratio	Fiber (g)	Cholesterol (mg)
363	45.5	0.36	0.95	4.7	508
364	37.2	1.12	1.14	10.4	336
366	31.8	0.34	1.05	5.8	186
402	16.9	0.60	1.03	1.4	261
404	31.4	0.53	0.86	9.8	212
405	29.4	0.33	1.07	3.4	159
406	28.1	0.44	1.14	2.7	196
407	14.8	0.50	0.94	1.3	228
408	39.1	0.87	1.37	4.7	220
409	27.3	1.03	0.96	2.3	472
410	26.5	1.11	0.99	3.2	259
413	38.4	0.97	1.53	2.7	538
414	31.3	0.93	1.05	2.5	240
416	38.2	0.74	1.15	4.4	366
417	15.6	0.53	0.57	6.0	171
421	20.2	1.64	1.60	1.8	185
422	21.5	0.40	0.63	3.6	268
425	38.5	0.74	1.09	3.3	495
428	84.4	0.46	3.27	3.7	446
429	57.4	0.44	1.42	1.5	478
434	27.3	0.40	0.87	1.9	373
435	40.9	0.73	1.08	2.9	192
437	20.8	1.16	1.01	3.5	258
440	36.7	0.92	1.42	4.5	155
444	21.2	0.63	0.92	7.4	243

Appendix F (*Continued*)

Individual PreTreatment Nutrient Intakes for 50 Subjects

Subject	Sugar (g)	Sucrose (g)	Pectin (g)	% Carbohydrate	% Protein	% Fat
201	136.5	127.2	0.2	44.9	13.2	41.4
206	186.7	126.3	1.3	41.7	15.0	43.6
209	151.0	136.7	0.6	42.1	16.2	42.3
218	87.5	72.4	0.5	41.5	14.8	43.9
219	227.5	57.8	24.4	56.3	12.3	36.3
222	88.6	68.9	0.0	43.2	17.6	39.1
230	61.6	54.7	0.2	48.4	17.3	34.0
242	28.6	15.0	0.3	23.7	22.3	53.5
248	96.1	35.9	0.2	40.2	14.4	45.8
252	174.6	72.1	0.5	62.3	10.4	25.2
256	30.7	17.3	0.0	31.9	13.1	55.8
302	105.7	97.9	0.1	45.0	13.4	42.1
303	143.5	104.7	4.8	56.2	14.2	31.7
307	110.6	81.5	4.3	47.6	20.5	37.6
308	87.4	65.4	1.0	51.2	14.9	36.8
312	31.0	13.0	9.7	34.7	12.3	56.1
319	69.6	49.1	0.1	45.2	13.3	37.9
322	58.4	30.3	0.2	39.4	15.2	45.6
336	110.9	85.9	0.7	42.8	13.6	44.3
347	73.7	40.6	4.5	36.1	14.5	37.0
352	237.5	222.1	0.3	54.3	15.1	31.6
354	137.9	105.7	1.5	53.6	14.3	32.3
355	69.7	47.8	1.8	37.5	19.4	43.0
358	43.0	25.9	2.3	46.3	16.5	38.8
361	97.8	85.7	0.9	38.5	16.2	45.8

Appendix F (*Continued*)

Individual PreTreatment Nutrient Intakes for 50 Subjects

Subject	Sugar (g)	Sucrose (g)	Pectin (g)	% Carbohydrate	% Protein	% Fat
363	92.1	60.1	1.4	29.9	12.5	31.8
364	102.7	42.7	2.3	41.9	11.0	48.8
366	73.8	44.2	1.3	41.2	16.0	44.6
402	31.5	13.1	0.5	39.8	21.3	38.5
404	134.1	108.0	0.5	52.0	13.2	36.1
405	141.2	115.5	3.2	51.1	9.7	41.5
406	85.4	52.3	3.3	35.8	14.1	28.0
407	46.4	30.2	0.7	42.6	15.2	42.3
408	111.8	82.2	1.1	43.1	14.8	43.6
409	112.5	88.7	0.5	39.9	18.7	42.5
410	133.2	115.5	0.4	53.0	5.1	38.8
413	110.0	86.1	1.3	39.7	19.2	41.3
414	104.6	82.6	2.1	39.8	16.5	44.5
416	90.6	80.9	0.6	39.6	16.1	46.0
417	149.9	134.4	0.9	53.7	13.5	34.8
421	72.2	63.6	0.5	47.7	15.3	37.3
422	197.0	157.8	1.8	56.7	14.1	30.2
425	133.9	109.3	2.1	45.2	12.6	43.7
428	88.5	74.7	0.5	41.8	19.7	38.4
429	63.5	44.7	0.2	30.3	14.8	55.1
434	191.0	176.0	0.7	53.4	11.5	36.1
435	120.8	92.0	0.4	40.1	13.3	48.0
437	191.3	179.8	0.4	59.3	13.5	28.6
440	63.2	23.1	1.2	35.4	13.9	47.5
444	114.0	63.7	5.3	53.1	15.4	43.2

Appendix G

Individual Post-Treatment Nutrient Intakes for 50 Subjects

Subject	Total Kcal (g)	Carbohydrate (g)	Protein (g)	Total Fat (g)	Saturated Fat (g)	Polyunsaturated Fat (g)
201	702	62.3	38.1	33.6	11.9	2.8
206	1826	259.3	63.7	62.3	22.7	4.6
209	1467	169.6	36.5	73.3	15.4	31.6
218	1102	168.0	28.4	37.2	10.2	3.1
219	1088	97.3	56.9	55.0	10.3	20.7
222	1111	133.9	33.8	50.2	19.5	9.9
230	542	66.8	30.7	17.4	7.6	2.3
242	1207	93.4	82.9	54.7	15.9	17.2
248	1894	230.6	52.0	87.9	16.4	32.1
252	1975	195.6	63.8	90.1	23.0	34.6
256	1350	103.3	70.8	57.4	13.0	23.9
302	1556	203.4	59.2	57.9	11.5	7.7
303	1396	138.2	50.3	71.6	27.9	7.7
307	1185	133.7	54.0	49.2	20.4	7.7
308	1559	129.8	60.3	89.1	30.9	12.3
312	597	57.8	43.5	22.2	6.0	4.3
319	1064	121.1	38.2	47.1	15.1	12.7
322	1198	155.1	41.3	47.7	15.3	12.5
336	1246	68.9	38.9	57.4	21.9	7.9
347	1446	163.8	54.0	68.3	18.0	22.9
352	2226	295.0	84.0	82.8	23.2	14.0
354	1951	262.8	43.7	85.0	23.3	27.9
355	1344	184.3	47.4	47.1	14.9	10.9
358	1075	127.9	48.6	42.8	11.2	12.6
361	1276	109.6	55.3	70.3	21.7	18.5

Appendix G (*Continued*)

Individual Post-Treatment Nutrient Intakes for 50 Subjects

Subject	Total Kcal (g)	Carbohydrate (g)	Protein (g)	Total Fat (g)	Saturated Fat (g)	Polyunsaturated Fat (g)
363	1574	213.8	50.6	59.4	19.1	15.9
364	1367	162.1	54.2	54.3	11.8	9.4
366	749	74.8	29.6	38.6	13.3	6.1
402	810	63.0	40.7	43.3	16.0	7.4
404	1885	286.2	72.4	53.2	16.3	9.8
405	1908	270.6	45.7	77.8	20.0	16.4
406	2009	238.6	72.5	87.5	33.3	7.9
407	1722	185.7	74.6	78.0	31.5	6.5
408	1801	216.3	56.3	80.1	20.8	7.2
409	1950	243.6	72.6	80.3	24.6	13.1
410	1354	169.7	41.5	57.3	14.4	14.4
413	1361	221.7	52.3	31.8	14.3	4.3
414	1230	142.1	39.6	54.2	22.2	5.5
416	1027	141.4	49.1	28.9	8.6	2.2
417	1490	166.0	84.4	55.9	14.5	23.0
421	881	128.3	48.4	21.0	6.2	4.2
422	988	121.0	46.8	36.7	8.0	16.8
425	1477	176.9	85.6	51.1	18.3	9.1
428	1623	95.4	79.7	91.4	36.2	5.5
429	1405	130.0	64.6	69.2	25.4	9.2
434	1929	266.9	92.6	59.4	20.9	7.8
435	1445	191.2	59.5	53.0	12.7	14.4
437	1424	164.6	71.3	54.9	12.9	17.0
440	1232	169.0	56.4	40.7	13.6	7.5
444	1384	132.2	70.7	66.4	17.7	22.5

Appendix G (*Continued*)

Individual Post-Treatment Nutrient Intakes for 50 Subjects

Subject	Monounsaturated Fat (g)	P/S Ratio	M/S Ratio	Fiber (g)	Cholesterol (mg)
201	14.9	0.24	1.25	1.4	145
206	23.7	0.20	1.04	3.8	454
209	20.8	2.05	1.35	1.7	201
218	12.2	0.30	1.20	1.9	77
219	15.9	0.65	1.30	4.8	97
222	14.6	0.51	0.75	1.1	287
230	5.6	0.30	0.74	1.1	72
242	17.5	1.08	1.10	0.8	471
248	23.3	1.96	1.42	2.7	171
252	26.0	1.50	1.13	1.2	483
256	16.2	1.84	1.25	1.0	497
302	11.5	0.67	1.00	1.1	174
303	34.2	0.28	1.22	1.1	167
307	17.8	0.38	0.87	2.9	172
308	30.9	0.40	1.00	3.7	164
312	8.9	0.72	1.48	2.2	114
319	14.9	0.84	0.99	0.3	600
322	13.9	0.82	0.91	3.4	172
336	22.2	0.36	1.01	0.9	148
347	20.3	1.27	1.13	5.9	366
352	32.8	0.60	1.41	3.5	373
354	26.0	1.20	1.12	3.4	173
355	17.9	0.73	1.20	0.5	244
358	7.7	1.12	0.69	3.6	420
361	22.6	0.85	1.04	4.4	434

Appendix G (*Continued*)

Individual Post-Treatment Nutrient Intakes for 50 Subjects

Subject	Monounsaturated Fat (g)	P/S Ratio	M/S Ratio	Fiber (g)	Cholesterol (mg)
363	20.2	0.83	1.06	1.8	477
364	14.6	0.80	1.24	2.7	330
366	17.3	0.46	1.30	2.8	212
402	17.1	0.46	1.07	1.1	167
404	24.4	0.60	1.50	3.7	227
405	33.6	0.82	1.68	5.9	71
406	40.4	0.24	1.21	2.8	239
407	32.8	0.21	1.04	4.2	227
408	21.8	0.35	1.05	4.1	216
409	35.8	0.53	1.46	4.3	175
410	15.6	1.00	1.08	1.9	114
413	9.2	0.30	0.64	3.2	227
414	19.3	0.25	0.87	2.8	125
416	10.3	0.26	1.20	1.4	126
417	14.1	1.59	0.97	2.5	460
421	8.1	0.68	1.31	2.0	126
422	9.0	2.10	1.12	1.9	221
425	19.5	0.50	1.06	6.6	280
428	43.8	0.15	1.21	2.0	256
429	30.6	0.36	1.20	1.9	276
434	25.3	0.37	1.21	5.5	385
435	20.8	1.13	1.64	5.4	161
437	16.0	1.32	1.24	3.7	369
440	13.1	0.55	0.96	6.0	166
444	21.3	1.27	1.20	3.1	125

Appendix G (*Continued*)

Individual Post-Treatment Nutrient Intakes for 50 Subjects

Subject	Sugar (g)	Sucrose (g)	Pectin (g)	% Carbohydrate	% Protein	% Fat
201	29.7	80.9	2.1	35.5	21.7	43.1
206	103.5	80.9	2.1	56.8	14.0	30.7
209	98.6	73.9	1.0	46.2	10.0	45.0
218	121.8	106.6	0.7	61.0	10.3	30.4
219	53.1	35.9	1.8	35.8	20.9	45.5
222	92.5	62.9	0.1	48.2	12.2	40.7
230	36.1	15.8	1.3	49.3	22.6	28.9
242	46.7	33.3	0.2	31.0	27.5	40.8
248	151.6	136.5	1.0	48.7	11.0	41.8
252	101.1	92.6	0.2	39.6	12.9	41.0
256	76.4	69.0	0.5	30.6	21.0	38.3
302	112.5	94.5	0.1	52.3	15.2	33.5
303	85.1	68.5	1.0	39.6	14.4	46.2
307	55.7	32.7	0.4	45.1	18.2	37.4
308	7.6	4.7	0.3	33.3	15.5	51.4
312	22.7	16.5	0.9	38.7	29.1	33.5
319	82.8	76.5	0.1	45.5	14.4	39.8
322	62.0	56.5	0.3	51.8	13.8	35.8
336	23.1	13.9	0.3	22.1	12.5	41.5
347	72.0	38.6	3.2	45.3	14.9	42.5
352	173.6	154.4	0.3	53.0	15.1	33.5
354	117.4	104.6	0.2	53.9	9.0	39.2
355	120.9	99.4	0.2	54.8	14.1	31.5
358	49.6	20.4	1.8	47.6	18.1	35.8
361	53.8	32.3	2.7	34.4	17.3	49.6

Appendix G (*Continued*)

Individual Post-Treatment Nutrient Intakes for 50 Subjects

Subject	Sugar (g)	Sucrose (g)	Pectin (g)	% Carbohydrate	% Protein	% Fat
363	147.7	122.9	0.3	54.3	12.8	34.0
364	64.0	53.1	0.6	47.4	15.8	35.7
366	57.5	30.2	0.9	39.9	15.8	46.4
402	19.3	14.5	0.5	31.1	20.1	48.1
404	184.0	132.7	1.8	60.7	15.4	25.4
405	144.6	120.4	2.3	56.7	9.6	36.7
406	177.4	148.3	0.9	47.5	14.4	39.2
407	114.9	95.6	3.7	43.1	17.3	40.8
408	126.2	90.7	0.2	48.0	12.5	40.0
409	92.1	44.6	2.6	50.0	14.9	37.1
410	64.9	60.8	0.0	50.1	12.3	38.1
413	126.4	110.8	0.1	65.2	15.4	21.0
414	70.0	44.2	2.0	46.2	12.9	39.6
416	48.2	35.7	0.4	55.1	19.1	25.3
417	67.7	67.5	0.9	44.6	22.6	33.8
421	78.3	55.2	3.2	58.2	22.0	21.4
422	83.1	49.7	1.7	49.0	18.9	33.4
425	85.7	73.3	3.8	47.9	23.2	31.1
428	34.2	32.1	0.0	23.5	19.6	50.7
429	34.2	23.1	0.5	37.0	18.4	44.3
434	159.1	132.4	0.2	55.3	19.2	27.7
435	83.2	32.6	3.0	52.9	16.5	33.0
437	73.7	38.8	0.7	46.2	20.0	34.7
440	79.1	27.0	7.2	54.9	18.3	29.7
444	44.8	24.6	0.5	38.2	20.4	43.2

Appendix H

Individual Height, and Exercise and Nutrition Group Designation for 50 Subjects

Subject	Height (cm)	Exercise Group	Nutrition Group
201	163.9	1	1
206	179.1	3	1
209	163.2	2	2
218	167.3	3	2
219	161.6	3	2
222	165.9	1	1
230	167.0	3	1
242	162.3	1	1
248	157.7	3	1
252	155.4	2	1
256	157.7	1	2
302	162.5	2	1
303	157.1	3	1
307	148.5	3	2
308	163.3	3	1
312	163.5	3	1
319	169.4	2	1
322	157.8	3	2
336	175.2	2	1
347	154.7	1	1
352	159.8	3	2
354	165.2	3	1
355	157.7	2	1
358	160.4	2	2
361	170.3	1	2
363	154.9	3	2
364	161.1	3	1
366	156.9	2	1
402	158.1	2	1
404	158.2	3	1
405	148.5	1	1
406	158.9	3	2
407	161.0	2	2
408	158.2	2	1
409	156.9	3	1
410	153.6	2	1
413	157.4	3	1
414	165.3	1	1
416	158.9	2	1

Appendix H (*Continued*)

Individual Height, and Exercise and Nutrition Group Designation for 50 Subjects

Subject	Height (cm)	Exercise Group	Nutrition Group
417	159.9	3	1
421	162.3	1	1
422	167.0	1	1
425	167.2	1	2
428	164.0	2	2
429	163.4	2	1
434	169.2	3	1
435	160.3	1	1
437	159.1	3	2
440	167.0	3	1
444	164.1	2	1

1 = Exercise (70%+ attendance) or Nutrition (80%+ attendance); 2 = Some Exercise (30-70% attendance) or Some Nutrition (less than 80% attendance), 3 = No Exercise (less than 30% attendance).

Appendix I

Pre-Treatment Mean Values of Body Weight and Composition for All Subjects, and Divided into Nutrition and Exercise Groups

	Body Weight (KG)	% Fat	% Lean	% Body Water
All Subjects	90.0	39.7	60.2	39.2
Nutrition	90.9	39.1	60.8	39.6
Some Nutrition	88.0	40.9	59.0	38.4
Exercise	84.5	37.5	62.4	40.8
Some Exercise	86.8	40.2	59.6	38.6
No Exercise	95.4	40.4	59.5	38.8

Between Nutrition groups or among Exercise groups, no significant differences exist at the $p = .05$ level.

Appendix J

Pre-Treatment One- and Two-Minute Mean Recovery Heart Rate Values, as a Percentage of Immediate Post-Exercise Heart Rate for All Exercising Subjects, and Divided into Exercise Groups

MEANS

	One-Minute Rate	Two-Minute Rate
All Exercising Subjects	90.1	84.0
Exercise	82.0	68.8
Some Exercise	95.4	94.6

No significant differences exist between Exercise groups at the $p = .05$ level.

Appendix K

Pre-Treatment Mean Values of Total Cholesterol (mg/dl), Lipoprotein Cholesterol (mg/dl), Triglyceride (mg/dl), and Insulin (ulU/ml) for All Subjects, and divided into Nutrition and Exercise Groups

	TC	LDL	HDL	HDL_2-C	TG	Ins	TC/HDL
All Subjects	202.9	133.9	49.2	25.4	98.6	24.7	4.35
Nutrition	200.0	130.4	46.6	25.0	104.9	25.1	4.35
Some Nutrition	209.7	142.2	50.6	26.4	84.1	23.4	4.34
Exercise	204.0	136.6	49.7	24.8	88.8	18.7^a	4.33
Some Exercise	215.1	143.4	52.2	27.4	97.1	20.2	4.40
No Exercise	193.4	125.6	46.8	24.2	105.2	31.5^b	4.32

Between Nutrition Groups or Among Exercise Groups, values with different letters as superscripts are significantly different form each other at the p = .05 level.

Appendix L

Pre-Treatment Nutrient Intake Mean Values for All Subjects, and divided into Nutrition and Exercise Groups

	Total Kcal	Carbohydrate (g)	Protein (g)	Total Fat (g)	Saturated Fat (g)	Polyunsaturated Fat (g)
All Subjects	1971.7	218.4	72.1	88.0	28.9	18.4
Nutrition	1904.2	211.9	69.0	87.0	29.0	19.0
Some Nutrition	2129.1	233.6	79.1	90.2	28.6	17.0
Exercise	1896.3	200.4	69.4	89.5	28.9	23.2[a]
Some Exercise	1837.4	200.9	68.9	84.6	27.1	16.4[b]
No Exercise	2110.5	240.9	75.8	89.5	30.2	17.3[b]

Between Nutrition Groups or among Exercise Groups, values with different letters as superscripts are significantly different from each other at the p = .05 level.

Appendix L (*Continued*)

Pre-Treatment Nutrient Intake Mean Values for All Subjects, and divided into Nutrition and Exercise Groups

	Monounsaturated Fat (g)	P/S Ratio	M/S Ratio	Fiber (g)	Cholesterol (mg)
All Subjects	31.1	.682	1.10	3 54	300.8
Nutrition	29.8	.685	1.04	3 50	273.7[a]
Some Nutrition	34.1	.675	1.23	3 63	364.1[b]
Exercise	30.0	.850	1.07	2 59	289.4
Some Exercise	33.3	.632	1.22	3.34	290.6
No Exercise	30 1	.626	1.03	4 20	314.6

Between Nutrition Groups or among Exercise Groups, values with different letters as superscripts are signficantly different at the p = .05 level.

Appendix L *(Continued)*

Pre-Treatment Nutrient Intake Mean Values for All Subjects, and divided into Nutrition and Exercise Groups

	Sugar (g)	Sucrose (g)	Pectin (g)	% Carbohydrate	% Protein	% Fat
All Subjects	108.0	79.2	1.87	44.2	14.8	40.5
Nutrition	106.3	77.9	1.48	44.5	14.7	41.0
Some Nutrition	112.1	82.4	2.79	43.5	15.2	39.4
Exercise	102.1	81.3	1.33	41.6	14.9	43.2
Some Exercise	92.4	66.5	1.04	44.1	15.1	41.0
Exercise	122.6	87.3	2.77	45.7	14.6	38.7

No significant differences exist between Nutrition Groups or among Exercise Groups, at the $p < .05$ level.

APPENDIX M

OBESITY IN BLACK WOMEN FORM NO. 1

SUBJECT NO._____
DATE_____YEAR_____

SOCIO-DEMOGRAPHIC INFORMATION

1. AGE_____ =_____
(RECORDED TO NEAREST MONTH) MONTH, DAY, YEAR

2. EDUCATION_____
(YEARS OF FORMAL EDUCATION COMPLETED)

3. IS SUBJECT:
(1) MARRIED; (2) WIDOWED (3) DIVORCED; (4) SEPERATED; (5) NEVER MARRIED

4. TOTAL FAMILY INCOME (INCLUDE WAGES/SALARY, WELFARE PAYMENTS, CHILD SUPPORT, SOCIAL SECURITY PAYMENTS) PER MONTH_____ PER YEAR_____

5. OTHER THAN MONETARY INCOME, DO YOU RECEIVE

(a) WIC_____
(b) FOOD STAMPS_____
(c) GIFTS (FRIENDS/RELATIVES)_____

6. HOW MANY PEOPLE DOES THIS INCOME SUPPORT?_____

7. WHO LIVES IN THE HOUSEHOLD WITH YOU? (INDICATE BY NUMBER)

(a) HUSBAND_____
(b) PARENTS OR GRANDPARENTS_____
(c) CHILDREN_____
(d) RELATED FAMILY YOUNGER_____, OLDER THAN YOU_____
(e) FRIENDS_____

8. IS SUBJECT EMPLOYED? YES_____ NO_____

9. IF EMPLOYED, IS SUBJECT EMPLOYED
(1) FULL-TIME
(2) PART-TIME

FORM NO. 1 (CONTINUED)

10. IF EMPLOYED, WHAT IS SUBJECT'S OCCUPATION? (CIRCLE ONE YOU DO MOST)

(1) PROFESSIONAL
(2) PROPRIETOR
(3) BUSINESS
(4) WHITE COLLAR
(5) BLUE COLLAR
(6) SERVICE
(7) FARM
(8) OTHER

11. LOCATION OF SUBJECT'S RESIDENCE

(1) SMALL CITY
(2) OUTSIDE OF CITY
(3) RURAL

12. SUBJECT BECAME OVERWEIGHT DURING:

(1) CHILDHOOD_____
(2) TEENAGE_____
(3) DURING PREGNANCY AND AFTER_____
(4) LATER TIME_____

13. DOES SUBJECT HAVE ANY OVERWEIGHT FAMILY MEMBERS?

RELATIONSHIP	AGE	HEAVY	VERY HEAVY

APPENDIX N

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? NAME_____HOW LONG?_____ YES__ NO__

8. ARE YOU TAKING ANY PRESCRIPTION MEDICINE, GENERIC OR BRAND NAME MEDICATION FOR WEIGHT LOSS? NAME_____DOSAGE_____HOW LONG?_____ YES__ NO__

9. DO YOU SMOKE? HOW MUCH/WEEK?_____ YES__ NO__

10. DO YOU CONSUME ALCOHOLIC DRINKS? YES__ NO__

    HOW MUCH/WEEK?_____

11. DO YOU HAVE HIGH BLOOD PRESSURE? YES__ NO__

APPENDIX N (Continued)

FORM NO. 2 (Continued)

12. DO YOU HAVE DIABETES? YES__ NO__

13. AMONG THE SUBJECT'S BROTHERS, SISTERS, PARENTS, AUNTS, UNCLES, 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 O

PHYSICAL EXAMINATION/MEDICAL APPROVAL, FORM NO. 11
OBESITY IN BLACK WOMEN
NAME OF SUBJECT_____SUBJECT NO._____
DATE_____

1. BODY TEMPERATURE_____

2. BLOOD PRESSURE SYSTOLIC_____ DIASTOLIC_____

3. FINDINGS OF EXAMINATION:

_____

_____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 aerobics, 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 limitatons_____

_____

APPENDIX P

ANTHROPOMETRIC MEASUREMENTS, FORM NO. 3

OBESITY IN BLACK WOMEN

SUBJECT NO._____

DATE_____YEAR_____

*ASK SUBJECT TO REMOVE SHOES AND ALL HEAVY OUTER GARMENTS.

1. BLOOD PRESSURE (1)_____ (1)_____
SYSTOLIC DIASTOLIC

(3 READINGS) (2)_____ (2)_____
SYSTOLIC DIASTOLIC

(3)_____ (3)_____
SYSTOLIC DIASTOLIC

AVERAGE OF 3 _____ _____
SYSTOLIC DIASTOLIC

2. ARM CIRCUMFERENCE_____CM

3. TRICEPS SKINFOLD_____MM

4. SUBSCAPULOR SKINFOLD_____MM

5. HEIGHT_____CM

6. BODY FRAME: SMALL_____MEDIUM_____LARGE_____

7. WEIGHT_____LBS. / 2.2 = _____KG

8. BODY WEIGHT_____KG = (WT - CLOTHING WT_____)

CHECK THE CLOTHING ITEMS WORN WHEN SUBJECT WAS WEIGHED IN ORDER TO OBTAIN CLOTHING ESTIMATE. CALCULATE AND RECORD IN #7.

CLOTHING LIST:

BRA: NATURAL (25 G) PADDED (40 G)

PANTIES: NYLON (18 G) COTTON (20 G)

SLIP: FULL (110 G) HALF (80 G)

SOCKS: FOOTLETS (30 G) SHORT SOCKS (35 G)
KNEE SOCKS (50 G)

APPENDIX P (Continued)

ANTHROPOMETRIC MEASUREMENTS, FORM NO. 3

OBESITY IN BLACK WOMEN

SUBJECT NO._____

DATE_____YEAR_____

CLOTHING LIST CONTINUED:

SHEER HOSE: KNEE LENGTH (25 G) PANTY HOSE (60 G)

GIRDLE: PANTY-TYPE KNEELENGTH LONG-LINE

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 WEIGHT SIMILAR ITEMS

APPENDIX Q

BIOCHEMICAL DATA, FORM NO. 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_____

APPENDIX R

BIOIMPEDANCE QUESTIONNAIRE

SUBJECT'S NAME:_____ NO.:_____

1. Is your menstrual period regular? Yes_____ No_____

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 or 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 S

PHYSICAL ACTIVITY FORM, FORM #5
(Modified from S-150)

Subject No._____
Date _____
Year _____

THE FOLLOWING SET OF INFORMATION NEEDS TO BE ASKED FOR EACH ACTIVITY IN WHICH THE SUBJECT HAS PARTICIPATED DURING THE PAST YEAR.

(YEARLY BASIS) (TOTAL HOURS)
1. YEARLY (TOTAL HOURS)
2. MONTHLY (THROUGHOUT THE 12 MONTHS)
3. WEEKLY (THROUGHOUT THE 12 MONTHS)

INTENSITY

1. LIGHT (NO SWEATING, NOT TIRING)
2. MODERATE (SWEATING, SOMEWHAT TIRING)
3. VIGOUROUS (PROFUSE SWEATING, TIRING TO EXHAUSTING)

****

1. BICYCLING YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

2. VIGOROUS DANCING YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

FORM #5 (Continued)

3. SWIMMING

YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

4. WALKING

YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

5. TENNIS

YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

6. RUNNING/JOGGING

YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

7. BOWLING

YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

FORM #5 (Continued)

BOWLING (Con't) INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

8. FARMING/GARDENING YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

9. OTHER (JOB RELATED) YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

10. OTHER (HOUSEHOLD TASKS)
YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

11. OTHER, (PLEASE SPECIFY)
YEARLY BASIS (TOTAL HOURS)_____

OF DAYS/WEEK _____

MINUTES _____

INTENSITY _____

ACTIVITY CONVERTED TO TOTAL HOURS _____

APPENDIX T

VSU Obesity Study

NAME
DATE
ACTIVITY SCORE

EXERCISE ACTIVITY LEVELS

1. Dancing(aerobic)
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 6.4

2. Bicycling
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 5.0

3. Swimming
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 4.0

4. Stretches
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 4.5

5. Golfing
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 5.0

6. Walking (circle)
   Outdoor:
   a. slowly
   b. moderate
   c. rapidly
   d. upstairs
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 5.0

7. Tennis
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 7.0

8. Running
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 14.0

9. Jogging
   Frequency
   # of months
   # of days/week
   minutes
   intensity
   activity factor 9.0

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

APPENDIX T (Continued)

10.	Skating, Ice	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ activity factor 7.0
11.	Skating, Roller	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ activity factor 7.0
12.	Bowling	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ activity factor 3.0
13.	Calisthenics	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ actvity factor 4.5
14.	Farming or Gardening	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ activity factor 3.7
15.	Snowskiing	Frequency_____ #of months_____ # of days/week_____	minutes_____ intensity_____ activity factor 8.0
16.	Housework (Standup work)	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ activity factor 3.7
17.	Volleyball	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ activity factor 5.5
18.	Other (Please specify)	Frequency_____ # of months_____ # of days/week_____	minutes_____ intensity_____ activity factor_____

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

APPENDIX U

ACTIVITY RECORD
VSU OBESITY STUDY
NAME:_____

Please record any activity you do, for the days listed, along with the number of minutes per activity and an intensity factor of either: 1) light, 2) mild, 3) moderate, 4) vigorous; or any other intensity indications (ie: heartrate, sweating or not) -Thank You!!

Monday

Tuesday

Wednesday

Thursday

Friday

Saturday

Sunday

APPENDIX V

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO._____

DATE OF RECALL_____YEAR_____

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

2. DID YOU EAT BREAKFAST YESTERDAY? YES_____NO_____

3. WHERE DID YOU EAT THE FOOD IF YOU ATE SOMETHING FOR BREAKFAST?
   (CIRCLE ONE)

   1. HOME

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

   3. RESTAURANT, CAFETERIA

   4. OTHER_____

4. WHAT DID YOU EAT AND/OR DRINK FOR BREAKFAST YESTERDAY? TIME_____

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

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_____

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

APPENDIX V (Continued)

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO._____

DATE OF RECALL_____YEAR_____

9. HOW MANY DAYS OF EACH WEEK DO YOU USUALLY EAT OR DRINK SOMETHING FOR LUNCH?
(CIRCLE ONE) 0 1 2 3 4 5 6 7

10. DID YOU EAT LUNCH YESTERDAY? YES_____NO_____

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

1. HOME

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

3. RESTAURANT, CAFETERIA

4. OTHER_____

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

APPENDIX V (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_____

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

APPENDIX V (Continued)

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO._____

DATE OF RECALL_____YEAR_____

17. HOW MANY DAYS OF EACH WEEK DO YOU USUALLY EAT AN EVENING MEAL?
(CIRCLE ONE) 0 1 2 3 4 5 6 7

18. DID YOU EAT AN EVENING MEAL YESTERDAY? YES_____NO_____

19. WHERE DID YOU EAT THE FOOD IF YOU ATE AN EVENING MEAL? (CIRCLE ONE)

1. HOME
2. FAST FOOD TYPE, GROCERY, VENDING MACHINE, SNACK BAR
3. RESTAURANT, CAFETERIA
4. OTHER_____

20. WHAT DID YOU EAT AND/OR DRINK FOR AN EVENING MEAL YESTERDAY?
TIME_____

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

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO._____

DATE OF RECALL_____YEAR_____

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

22. DID YOU EAT A BEDTIME SNACK YESTERDAY? YES_____NO_____

23. WHERE DID YOU EAT THE FOOD IF YOU ATE A BEDTIME SNACK? (CIRCLE ONE)

1. HOME
2. FAST FOOD TYPE, GROCERY, VENDING MACHINE, SNACK BAR
3. RESTAURANT, CAFETERIA
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

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO._____

DATE OF RECALL_____YEAR_____

25. HOW MANY DAYS OF EACH WEEK DO YOU USUALLY EAT PRE-BREAKFAST?
(CIRCLE ONE) 0 1 2 3 4 5 6 7

26. DID YOU EAT PRE-BREAKFAST YESTERDAY? YES_____NO_____

27. WHERE DID YOU EAT THE FOOD IF YOU ATE SOMETHING FOR PRE-BREAKFAST?
(CIRCLE ONE)

1. HOME
2. FAST FOOD TYPE, GROCERY, VENDING MACHINE, SNACK BAR
3. RESTAURANT, CAFETERIA
4. OTHER_____

28. WHAT DID YOU EAT AND/OR DRINK PRIOR TO BREAKFAST YESTERDAY?

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

FOOD RECALL #2, FORM NO. 9

OBESITY IN BLACK WOMEN

SUBJECT NO._____

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
2. MOTHER
3. FATHER
4. SELF
5. GIRLFRIEND(S)
6. BOYFRIEND(S)
7. MEDIA

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

APPENDIX V (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

APPENDIX W

OBESITY IN BLACK WOMEN
DIETARY RECORD

NAME:_____DATE:_____

What did you eat and/or drink for the entire day? Please record everything that you ate in the 24-hour period.

TIME	PLACE	FOOD/BEVERAGES	QUANTITIES	PREPARATION METHOD AND/OR INGREDIENTS

APPENDIX X

BEHAVIORAL AND EATING PATTERNS
OBESITY IN BLACK WOMEN
FORM #7

SUBJECT NO._____
DATE_____

This questionaire is desinged to study behavioral and eating patterns. The best answer to these questions is what you feel is true of yourself. Check one box per question.

QUESTIONS	ALWAYS 5	OFTEN 4	SOMETIME 3	SELDOM 2	NEVER 1
1. I eat when I'm tense or anxious.					
2. I eat well-balanced meals.					
3. I go on eating binges.					
4. I eat when I'm bored.					
5. I'm a compulsive eater.					
6. I overeat on special occasions (Thanksgiving, Christmas, picnics, etc).					
7. My eating habits are influenced by my moods.					
8. Controlling my eating habits is a struggle.					
9. I give up self-control while eating.					
10. I'm in control of my eating habits.					
11. I eat when I'm depressed					
12. I eat when I'm unhappy					

FORM #7 (Continued)

QUESTIONS	ALWAYS 5	OFTEN 4	SOMETIME 3	SELDOM 2	NEVER 1
13. I eat when I'm lonely.					
14. I sneak food.					
15. I overeat the food I really like.					
16. I vomit after I stuff myself.					
17. I cause myself to vomit after overeating.					
18. I take laxatives after overeating.					
19. I'm self-conscious about my eating habits.					
20. I have good eating habits.					
21. I eat to dull or block out painful emotions.					
22. I eat when I'm afraid or nervous.					
23. I eat when I'm shy.					
24. My eating habits control my life.					
25. Food controls my life.					
26. I eat when I'm feeling guilty.					
27. I eat when I'm sad.					
28. I sneak food when nobody will know.					
29. I take diuretics (water pills) after overeating.					

FORM #7 (Continued)

QUESTIONS	ALWAYS 5	OFTEN 4	SOMETIME 3	SELDOM 2	NEVER 1
30. I go on diets.					
31. I go on eating sprees.					
32. I overeat a little at most meals.					
33. I eat when I'm worried					
34. I feel powerless to control my eating habits.					
35. I organize my life around eating.					
36. I eat when I'm angry with others.					
37. I overeat on occasions when others are also overeating.					
38. I go on eating sprees during which I feel I have little or no control.					
39. I think about my eating patterns.					
40. I eat a slight excess of food on a regular and predictable basis.					
41. Other people have expressed concern about my eating habits.					
42. My eating patterns are in opposition to what I believe is right.					
43. When I overeat, it is usually because I cooked too much and do not want to waste food.					

FORM NO. 7 (Continued)

QUESTIONS	ALWAYS 5	OFTEN 4	SOMETIME 3	SELDOM 2	NEVER 1
44. I overeat to set a good example for my children so they will not be wasteful.					
45. I overeat because I love "soul" food so much.					
46. I overeat because my parents taught me to eat everything on my plate.					
47. I overeat because I remember times when I did not have anything to eat.					
48. Most times when I overeat, I am unaware that I am overeating.					
49. The reason I overeat is because I enjoy my own cooking.					
50. I am self-conscious about eating in public.					
51. I have an eating problem.					
52. I feel guilty when I eat too much.					
53. I'm a compulsive eater					
54. I feel more in control of my life when I am not eating.					
55. I feel more in control of my life when I'm eating					
56. I am satified with my eating habits.					

FORM NO. 7 (Continued)

QUESTIONS	ALWAYS 5	OFTEN 4	SOMETIME 3	SELDOM 2	NEVER 1
57. My eating habits indicate I lack self-control.					
58. I get pleasure just thinking about eating.					
59. My eating habits are primarily influenced by my environment.					
60. My eating habits are primarily influenced by my heredity (passed from parent to offspring).					

Permission to use this questionnaire has been granted by Susan Crockett, M.S., R.D. and John Littrell, Ed.D.

VITA

Katherine Williamson was born in Norfolk, VA in 1963, and graduated from a Fairfax County VA high school in 1981. In 1985 she graduated from Virginia Polytechnic Institute and State University with a B.S. in Animal Science and a minor in Biology, pursuant to a career in veterinary medicine. After a redirection of career goals, she began taking courses in the department of Human Nutrition and Foods in the fall of 1986. She has integrated various aspects of exercise physiology into her graduate studies, in both academic and research efforts. She is currently working as the Aquatics Director at the Hensel Eckman YMCA in Pulaski, VA, where she is able to utilize her skills in both nutrition and exercise physiology.