# THE EFFECT OF FAT ON LIPID PROFILES IN THE ABDOMINALLY OBESE POSTMENOPAUSAL WOMAN

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## DEDICATION

With deepest appreciation and devotion, I wish to dedicate this work to the four most important people in my life: the Lord Jesus Christ, my mother, my father, and my fiance'. With the Lord as my guiding light, I have accomplished goals beyond my greatest expectations.

Without the tremendous understanding, loyal support, and guidance of my wonderful parents, Dr. and Mrs. Lessie James Broussard, this work could not have been completed. Their never ending love and support has always been evident in my life. Without them as exceptional role models and excellent examples, I would not be the person that I am today. Thank you, Mom and Dad, for providing me with the following gifts that no amount of money could even begin to purchase --- a strong foundation of faith, family, and individuality.

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ABSTRACT

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Thirty-three postmenopausal women with abdominal obesity participated in a weight reduction study. The purpose was to determine if a low-fat, reduced kcalorie diet would improve the lipid profile in abdominally obese postmenopausal women when compared to a reduced kcalorie normal-fat diet. Subjects were randomly assigned to either a control group or to one of two weight reduction (1200 kilocalorie) diets: one having a normal fat level and the other being low in fat, for six weeks. The low calorie, normal-fat diet contained 20% protein, 30-35% fat and 40-50% carbohydrate, whereas the low-fat diet contained 20% protein, 20-25% fat and 55-60% carbohydrate. Fasting serum total cholesterol, HDL-cholesterol and triglyceride were measured initially and at the end of 6 weeks. Weight loss regimens improved two parameters significantly ($p < 0.001$), by decreasing waist-to-hip ratio (WHR) and body mass index (BMI) ($p < 0.001$) in all experimental subjects. Total serum cholesterol decreased significantly ($p < 0.004$) in the group consuming the low-fat diet. However, cholesterol levels in the control group and

the group consuming the normal-fat diet did not change significantly. Triglyceride levels and HDL-cholesterol levels did not change significantly in any of the diet groups. Due to an increased caloric intake by the low-calorie, normal-fat diet group, data were reorganized into a post-hoc control diet group and a reduced-fat diet group. Statistical analysis of the reorganized data confirmed that significant ($p < 0.01$) decreases in body weight and BMI occurred in the reduced-fat diet group. A significant decrease ($p < 0.01$) in WHR occurred in both the post-hoc control diet group and the reduced-fat diet group. However, the reduced-fat diet group was the only group to experience any significant ($p < 0.01$) decrease in blood lipid concentrations. These findings suggest that an energy-restricted, reduced-fat diet may be necessary to successfully decrease cholesterol concentrations in abdominally obese postmenopausal women.

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# CHAPTER I

## INTRODUCTION

Obesity represents a major avoidable contribution to the costs of illnesses in the United States. Obesity is a major risk factor for many chronic diseases such as cardiovascular disease (CVD) and non-insulin dependent diabetes (NIDDM) (1). It also constitutes a greater risk for developing hypertension, hypertriglyceridemia, gall bladder disease, gout, and some types of cancers (2). Although it is accepted that obesity has an important role in the development of CVD, its exact definition in the causal sequence of events is still unclear (3). However, the American Heart Association has implicated nutritional factors in the etiology, pathology and treatment of CVD (4). Although level of energy balance is one of the main factors affecting body fat deposition, other factors include: gender, age, diet composition, level of physical activity, and a variety of social factors (5).

Obesity has traditionally been regarded simply as excess adipose tissue, with little attention being paid to

its anatomic location (6). However, recent studies suggest that morbidity and mortality are considerably affected by the regional distribution of body fat, particularly android or abdominal obesity (5). The android phenotype, as measured by the waist-to-hip ratio (WHR), in women (WHR > .80) and in men (WHR > 1.0), is associated with an increased risk for non-insulin dependent diabetes mellitus, heart disease, stroke and premature death (7-10). Android fat distribution, the location of fat in and around the abdominal area, has been related directly to an increase in the development of CVD as well as indirectly through lipid profiles associated with CVD risk whereas gynoid fat has not (11-12). Hypertriglyceridemia, decreased high-density-lipoprotein (HDL) cholesterol, increased low-density-lipoprotein (LDL) cholesterol, and increased very-low-density-lipoprotein (VLDL) are just a few changes in lipid profiles often evident in android obesity (9, 13).

Due to changes in metabolism and physical activity that occur with aging, lean body mass typically decreases while fat mass increases (14-15). Because of these changes the prevalence of overweight increases with advancing age until about age 50 for men and age 70 for women (16-17). According to Flegal et al. (18), the U.S. population is unlikely to get leaner any time in the near future. Overall, obesity is more prevalent among women than men and is more

common among non-Hispanic blacks and Mexican Americans than among non-Hispanic whites (19-20). These statistics have led the U.S. Department of Health and Human Services to prospectively target these special at risk groups through preventive education (17). The postmenopausal woman is one subgroup that is potentially at risk for the development of CVD (21).

Differences in regional fat metabolism exist and are sex dependent. Sex differences in regional fat metabolism may in part explain the well-documented sex differences associated with the incidence of cardiovascular risk (22). Upper body fat, independent of total body fat, has been shown to be related to increased blood pressure, glucose, insulin, and lipid concentrations in both men and women (23-26). However, mean total and LDL-cholesterol have been shown to be lower in premenopausal women and higher in postmenopausal women than men of comparable age (27). Women with android obesity are also at greater risk for the development of breast, ovarian and endometrial carcinomas as well as CVD and NIDDM (28-29).

The onset of menopause has been suggested to be a risk factor for cardiovascular disease in women (30). Menopause is also associated with the disappearance of the female characteristics common to adipose tissue deposition, which now allows fat to accumulate without regional preference

(31-33). This is believed to be caused by problems in the liver that deal with the clearance of triglycerides which in turn causes the accumulation of abdominal fat. Android fat distribution is greater in postmenopausal women than in premenopausal women and is greater still in men than in postmenopausal women, a pattern that mirrors the increasing incidence of CVD seen for these groups (34).

Studies have demonstrated that android body-fat distribution is associated with diabetes, where increased very-low-density-lipoprotein (VLDL) cholesterol, low-density-lipoprotein (LDL) cholesterol, insulin, and blood pressure are more prevalent (35-38). These factors are all known to be associated with an increased risk of developing CVD. In non-diabetics, android-body-fat distribution has also been reported to be linked directly with hyperinsulinemia, insulin resistance and adverse lipid profiles, metabolic factors that are all thought to increase the risk of CVD (12, 39-41). The changes in plasma lipoproteins that are observed after menopause are partially due to the reduction of the protective effect of endogenous estrogens (33, 42).

Changes in the hormonal activity are responsible for the enlargement of subcutaneous abdominal adipose cells which is thought to contribute to the increased flux of plasma free fatty acids (FFAs) in the portal and systemic

circulations (43-45). This increase results in a natural flow towards the liver thereby exposing the liver to extremely high concentrations of free fatty acids and in turn increasing hepatic secretions from the liver (7).

The knowledge of the effects of a low-fat diet on lipid profiles may be useful in understanding the physiologic effects of diets in postmenopausal abdominally obese women. Low-fat diets have demonstrated positive changes by improving lipid profiles which are commonly associated with abdominal obesity. Few diet intervention studies have been conducted on the effects of diet in postmenopausal abdominally obese women who are disease free (46-48).

## Purpose of the Study

The purpose of this study was to determine if a low-fat reduced kcalorie diet will improve the lipid profile in abdominally obese postmenopausal women when compared to a normal-fat diet. Specific objectives of the study were:

1. To design a normal-fat diet containing 1200 calories with approximately 20% protein, 30-35% fat, and 45-50% carbohydrate.

2. To design a low-fat diet containing 1200 calories with approximately 20% protein, 20-25% fat, and 55-60% carbohydrate.

3. To compare concentrations of serum total cholesterol, HDL-cholesterol and triglycerides of subjects with no intervention (the control group) and those on normal- and low-fat diets.

## Hypotheses

The null hypothesis of the study was there are no significant differences in metabolic lipid profiles of the three treatment groups (control, normal-fat diet, and low-fat diet) on all parameters over a six week period.

# CHAPTER II

## REVIEW OF LITERATURE

Despite attempts to change the public's consumption of excess calories and fat intake, obesity is steadily increasing in the United States. A conservative estimate of the economic costs attributable to obesity was $39.3 billion, or 5.5% of the costs of illness in 1986 (1). Obesity is associated with increased risk of NIDDM, hypertension, CVD, gallbladder disease and cholecystectomy, and colon and postmenopausal breast cancer (49-53).

Observations indicate that factors other than body weight may be involved in the expression of health risks associated with obesity (54). Many disease risk factors are related to body composition rather than body weight (55).

Individuals differ with respect to the location of fat (56). Men tend to have more abdominal fat, giving them the android or male pattern of fat distribution. Women, however, tend to have greater amounts of gluteal fat and thus have larger hip circumferences, giving them the gynoid or female pattern of fat distribution. The relative

preponderance of one pattern or the other may be expressed by the abdominal-gluteal, android-gynoid, as determined by the waist-to-hip circumference/ratio (WHR) (8).

Even though the android pattern of obesity is more common in males, it may also occur in females. In both men and women, upper body fat, independent of total body fat, has been shown to be related to blood pressure and to lipid, glucose, and insulin concentrations (2, 7, 24, 26, 57). Moreover, individuals with upper body obesity, as assessed by the WHR, are at greater risk for developing cardiovascular disease (58, 59), diabetes mellitus (60), and breast, ovarian, and endometrial carcinomas (28, 29).

The recognition of abdominal obesity or abdominal distribution of adipose tissue as a clinical entity associated statistically with increased risk of disease seems to be a significant advance in the field of obesity research (61). In the fifties, J. Vague reported the relationship between upper trunk android obesity and the prevalence of diabetes mellitus, lipid disturbances and hypertension (35). More recent studies (62-64) confirmed Vague's original findings.

The relationship between regional fat distribution and health has come clearly into focus within the past ten years. During this time span, several prospective

epidemiological studies strongly suggested that in addition to total body fat, the location of that fat plays an important role in the risks of cardiovascular disease and in the risks of developing diabetes, hypertension, gallbladder disease, stroke, and overall mortality (5).

A recent review by Emery et al. (65) of 73 studies showed a significant association between distribution of body fat to the abdominal region, especially intra-abdominal fat, and increased risk of cardiovascular disease, breast cancer, endometrial cancer, and overall mortality. Kissebah et al. (62), Depres et al. (66) and Lundgren et al. (67) showed that abdominal fat distribution or upper-body obesity was a better predictor of cardiovascular disease, diabetes, and metabolic disorders than was overall obesity. However, Bonithan-Kopp et al. (68) showed that body mass index (BMI), which reflects overall adiposity, was more strongly associated with blood lipids, except for triglycerides, than was WHR which reflects body-fat distribution. A subsequent study reported a positive association with both BMI and WHR and all blood lipids that were studied in 209 French women (69). The relations between diet, abdominal obesity and the occurrence of cardiovascular disease among women have been limited. This review of literature will address the metabolic consequences of abdominal obesity as well as

current uses of fat intake as a mode of possible diet therapy for the abdominally obese.

## Obesity and Cardiovascular Disease

The death rate attributable to cardiovascular disease (CVD) rose steadily through most of the twentieth century until the 1960's, when it then began to level off and start declining. Since 1968 the death rate (on an age adjusted basis) has been declining between two to three percent per year. From 1968 to 1985, the rate decreased 35%. Although reasons for the decrease are indistinct, some possible factors are believed to be: increased exercise, improved detection and treatment, better diets along with certain dietary supplements, and less smoking (70).

However, even considering past improvements, CVD still represents the single largest cause of death in the United States (71). The cost is enormous; over 12 million potential years of life are lost each year from these premature deaths. In terms of dollars and cents, it is estimated that the economy lost well over 59 billion dollars in productivity due to illness and premature deaths attributed to CVD (72).

The importance of body weight, body mass and other measurements of adiposity in the prediction of CVD has been

the subject of long-standing debate. Many studies (73-75) have shown that the incidence of certain types of CVD, particularly coronary heart disease and stroke, is greater in heavier persons.

Observations indicate that factors other then body weight may be involved in the expression of health risks associated with obesity (54). CVD risk factors relate to body compostion rather than body weight (55). Recent studies (11, 58, 59, 76) suggest that the distribution of body fat may be a stronger predictor of coronary heart disease (CHD) than the total amount of body fat. Distribution of body fat has been found to be an independent predictor of metabolic aberrations including cardiovascular morbidity and mortality (59).

Results of a study by Dennis and Goldberg (77) affirmed the deleterious effects of upper-body regional fat distribution in obese women on lipoprotein risk factors for cardiovascular disease. In a study by Bjorntorp (61), obesity was examined without the influence of body fat distribution, just as an increase of body fat mass, usually measured as body mass index (BMI). Similarly, distribution of fat was analyzed as a seperate factor, independent of obesity, as measured as the waist-over-hip circumference ratio (WHR). WHR was found to be positively associated with the incidence of myocardial infarction, stroke, and death

from all causes in both men and women, after adjusting for BMI. Recent studies indicate that an increased WHR, an indirect index of visceral fat distribution, may be particularly hazardous to health (59).

BMI and WHR, although convenient for epidemiological work, are by no means perfect measurements of either obesity or abdominal distribution of fat. WHR has a unique power as a predictor of various diseases and symptoms which is better than other more elaborate measurements. For example, all of the three strongest risk factors for CVD (hypertension, cholesterol, and smoking) have been found to be associated with the WHR independently of the BMI (61).

Studies currently available (11, 59, 76) consistently found a correlation between the WHR and show stronger correlations to other measurements of central obesity than BMI or CVD. Most of these studies concern middle-aged populations where CVD is a large part of the mortality. Therefore, it is not surprising that the WHR is also a strong predictor for premature deaths (61).

## Lipids and Lipid Metabolism

Obesity is a problem of nutrient imbalance whereas more foodstuffs are stored as fat than are used for energy and metabolism (78). When an individual consumes more food or energy than he or she expends, this additional energy is

stored in the form of fat. A gain of approximately 3,500 kilocalories of energy usually results in the storage of one pound of fat. The body's fat mass has a large storage capacity; and, therefore, fat supplies approximately two-thirds of the body's ongoing energy needs. Most fat is stored in adipocytes (fat cells) in the form of triglycerides: however, a small amount is also stored in muscle cells (3, 79).

Lipids, or fats, are generally defined as substances that are insoluble in water but soluble in organic solvents. Fat in foods occurs in greatest proportion as triacylglycerol (triglyceride). Other lipids in food are sterols, fat-soluble vitamins (A, D, E, and K), phosphoglycerides (phospholipids) and sphingolipids, and some waxes and other minor complex lipidic components (78). To comprehend the effect of fats on the human body, it is pertinent to understand their physical properties and nomenclature (3).

Due to the fact that lipids are not soluble in water, they have to be complexed with other materials in order to be transported by the blood. Absorbed lipids are therefore made water soluble by their incorporation into lipoproteins. Apoproteins, which are various proteins that are made by the liver and small intestines, are combined with lipids to form lipoproteins (3, 70, 78-79). The many different lipoproteins

are classified according to the following densities: very low density lipoprotein (VLDL); low density lipoprotein (LDL); and high density lipoprotein (HDL). LDL contains much more lipid than does HDL, in fact, there is so little fat and cholesterol carried by HDL that few cardiologists paid attention to it until Miller and Miller's historic paper in 1975. Miller and Miller (80) brought impressive epidemiological evidence together to demonstrate that an elevated HDL protected against CVD and that a reduced level of HDL was a risk factor. HDL seems to carry cholesterol from peripheral tissue back to the liver for degradation and excretion. This is an important mechanism due to the fact that ordinary cells cannot remove, degrade or excrete cholesterol and once cholesterol invades the arterial wall, the only way it can be removed is to have it transported back to the liver by HDL. HDL is currently recognized as a powerful protective factor with greater risk predictive value than serum cholesterol (3, 68, 76). Low density lipoprotein (LDL) transports lipids from the liver to peripheral tissues including the arterial wall and, because of this, has an opposite relationship to heart disease than does HDL. LDL correlates directly with CVD risk and appears to accelerate atherosclerosis. Serum cholesterol (the amount of cholesterol found in all lipoproteins) is usually elevated when LDL is elevated (70).

## Fat Distribution, Abdominal Adipocytes and Hyperlipidemia

A variety of determinants influence total body fat and its distribution. Factors affecting the percent body fat include age, gender, level of energy balance, composition of the diet, level of physical activity, and a variety of social factors including smoking and alcohol (5). The distribution of adipose tissue on and within the adult body has been associated with sex hormone levels, glucose and insulin metabolism, the type of lipid stored, the size and number of adipocytes and their metabolic activity (24).

As stated previously, the distribution of adipose tissue can range from central to peripheral. A central or android pattern of fat distribution is one in which there are greater amounts of adipose tissue on and within the trunk than the extremities (24). Many of the same factors including age, gender, total body fat, level of energy balance, adipose tissue lipoprotein lipase, and lipolytic activity also influence the amount of subcutaneous truncal-abdominal fat (5).

The regional distribution of body fat has considerable effects on mortality and morbidity. No matter what the index used for the measurement of the distribution of fat, there is a clear and robust association of increased abdominal or upper-body fat with overall mortality (5).

The enlargement of central, abdominal adipose tissue is closely associated with disease, while enlargement of peripheral adipose tissue does not show such associations to the same degree. Specifically, CVD, cerebrovascular disease, and NIDDM are closely associated with abdominal obesity. However, only varicose veins and joint problems have been found to be positively related to gluteal-femoral obesity. The latter form of obesity also carries an increased risk of development of NIDDM, although less marked than in the case of abdominal obesity (37).

Android or upper body obesity predicts higher concentrations of cholesterol, triglycerides and insulin and lower concentrations of HDL-cholesterol (65, 81-86). Other abberations may include: increased VLDL triglycerides and LDL- cholesterol, reduced HDL-cholesterol, and raised blood pressure (13). High concentrations of total cholesterol, triglycerides and LDL-cholesterol and low levels of HDL-cholesterol are significantly associated with the occurrence of CVD and increased risk of death (58, 43-45).

Among other factors, regional fat distribution is also related to steroid hormone concentrations. For example, in patients with Cushing's disease or hyperadrenocorticism there is an increased central fat deposition associated with an increased activity of lipoprotein lipase in the abdominal fat cells (5). Hormonal changes help to determine the

disturbances in lipid and lipoprotein metabolism observed in the abdominally obese. Changes in the hormonal activity are responsible for the enlargement of subcutaneous abdominal adipose cells which is thought to contribute to the increased flux of plasma free fatty acids (FFAs) in the portal and systemic circulations (43-45).

Intra-abdominal adipose tissues have specific metabolic characteristics. In particular, those tissues drained by the portal circulation have an extremely sensitive free fatty acid mobilizing system (87). When the central adipose tissue has a substantial mass, free fatty acid mobilization into the portal vein will be considerable (88). An increase in free fatty acids results in a natural flow towards the liver thereby exposing the liver to extremely high concentrations of free fatty acids. Exposure of the liver to high concentrations of FFAs is known to cause increased hepatic secretion of very low density lipoproteins (VLDL) (7). Recent studies have shown that the synthesis and secretion of VLDL are regulated by the availability of FFAs (89). Consequently, VLDL is secreted from the liver in proportion to elevated portal FFA levels, thus increasing the risk of increased concentrations of circulating low density lipoproteins (LDL) as well (7).

In two separate studies, Dennis and Golberg (77) and Kissebah (45) compared two age and and weight-matched

subgroups of obese women with either android or gynoid fat distribution. Results from both studies showed that the women with android fat distribution developed higher triglyceride levels and had larger adipose cells than the gynoid group. The gynoid group presented with smaller abdominal fat cells. Based on the results from his study, Kissebah (45) proposed the following hypothesis: that large adipocytes from the abdominal region of android type women exhibited higher rates of lipolysis than the same cell location in gynoid obese women. The increased adipose cell size may indirectly cause a higher rate of lipolysis resulting in an increase of fatty acids into the circulation. The increased plasma free fatty acids might stimulate hepatic secretion of triglycerides which would lead to hypertriglyceridemia. Therefore, the presence of the hypertrophied abdominal fat cells in the android type of obesity could be a significant factor in their susceptibility to hypertriglyceridemia. Dennis and Goldberg (77) concluded that although weight loss improved CVD risk factors regardless of BMI or WHR, the magnitude of the increase in levels of plasma HDL-C and the decrease in levels of triglycerides in women with an upper-body fat distribution suggest that these women should be the primary target for nutrition and dietary intervention because they are likely to benefit the most in the prevention of CVD.

An increased VLDL production and a reduced catabolism of triglyceride-rich lipoprotein, the result of high plasma VLDL levels, may be associated with an increased exchange of VLDL-TG for cholesterol esters that have been derived from HDL-cholesterol and LDL-cholesterol (90). Depres et al. (90) stated that the previously mentioned exchange can eventually lead to a TG enrichment of LDL and HDL and can reduce the plasma HDL-cholesterol and the formation of small "dense" LDL particles which have been recognized as significant risk factors for the development of CVD.

Landin (91) examined the importance of obesity and regional fat distribution with regards to plasma lipid levels, blood pressure, glucose tolerance, and insulin. Landin found that plasma triglycerides were higher in the abdominally obese women than in the lean women. It was therefore concluded that in this study the amount of body fat by itself is more important than the WHR.

Disturbances in lipid metabolism have been found to be related to body fat distribution and abdominal obesity (64). Folsom et al. (28), Lanroth (40) and Chumlea et al. (92) all found increased lipolysis in the abdominal subcutaneous fat aggravating an accessible supply of free fatty acids into the area of the abdomen. In a study by Van Gaal et al. (93), variations in apolipoprotein concentrations were examined in male and female obese adults with both gynoid and android

obesity. Results confirmed that WHR was a significant indicator for most lipid profiles in men and women. According to Kissebah et al. (94), women with abdominal obesity had significantly higher fasting plasma triglyceride concentrations than those women with gynoid obesity. The individuals with lower body obesity had serum triglyceride concentrations that were within the range of the non-obese controls. Fujioka et al. (63) found that subjects with a high ratio of intra-abdominal visceral fat to subcutaneous fat (V/S Ratio) had significantly higher concentrations of fasting serum triglycerides and total cholesterol than did subjects with a lower ratio. The subjects with the higher ratio had serum triglycerides of 177 mg/dL and total cholesterol levels of 232 mg/dL as compared to subjects with lower ratios whose serum triglycerides were 106 mg/dL and total cholesterol levels were 189 mg/dL. In a study by Krotkiewski et al. (64), WHR was found to be a better predictor of increased triglyceride concentrations than was epigastric fat cell weight for both men and women. Zamboni et al. (95) compared body fat distribution and metabolic variables in both pre- and postmenopausal women. However in contrast with other investigators, the data gathered by Zamboni failed to show any significant difference between the pre- and postmenopausal groups with regard to WHR. Yet, blood cholesterol values were significantly higher in

postmenopausal women. Zamboni et al. concluded that visceral abdominal adipose tissue increased with age and that metabolic abnormalities are related more to these tissue areas than to actual body weight.

The following are normal ranges for the various blood lipid measurements: cholesterol: 140-200 mg/dl; HDL-cholesterol: 35-86 mg/dl; and triglyceride: 35-135 mg/dl (96).

## Weight Loss and Anthropometric Characteristics

The multitude and variety of eating disorders that afflict the United States' society stand as a striking testimony to the enormous problem that confronts the medical and public health communities today (97). However of all of the eating disorders that affect our society, obesity has become the one topic that is constantly being discussed. The definition of obesity has been and still remains a controversial issue (7). Also included in this controversial issue is the debate as to how obesity should be evaluated. There is also the lack of agreement on a classification system based on body fat and distribution especially regarding terminology that should be used (8).

Due to the fact that few laboratories have the facilities to use direct measurements of body fat and such

measurements can be very complex (7), several methods are now widely used to measure regional fat distribution indirectly (98). The most common methods include the body mass index (BMI) and the waist-to-hip circumference (WHR) (98). The BMI attempts to approximate to the logical variable, the total mass of body fat (7). WHR, however, has become a popular index for quantitatively describing adipose tissue distribution.

To determine WHR, the waist circumference is measured at the level of the umbilicus which is midway between the lowest rib margin and the iliac crest. The subject should be supine and breathing out normally. Hip circumference is the measure that yields the maximum diameter over the buttocks. WHR has been found to be equally as effective as more time-consuming and difficult procedures (43). Waist-to-hip ratios >.80 are generally considered indicative of a masculine or central distribution of fat. Men having WHR > 1.0 are considered to be abdominally obese. Women with WHR > .80 are significantly at increased risk for cardiovascular disease, NIDDM and hormone related cancers (28, 92, 95, 99). According to Johnston et al. (100), the WHR was found to be the best indirect predictor of upper body fat patterning.

Both genetic and environmental factors appear to contribute to upper body fat. Smoking, low amounts of exercise, and stress have been associated with upper body fat distribution (101-104). Upper body fat obesity is also strongly associated with body weight: previous studies show correlations between WHR and BMI (104-105). Several studies (66-67, 98) reported BMI, which is a reflection of overall adiposity, to be more strongly associated with blood pressure, blood lipids (except for triglycerides) than WHR, which is an indicator of body fat distribution.

In a study by Kopp-Bonithan et al. (69), longitudinal data were collected on changes in overall adiposity and regional fat distribution using WHR and BMI. No significant associations were found between changes in BMI or WHR, age, menopausal status, educational level or changes in sports activity. Results suggested that the pattern of body fat distribution and weight changes may be more important determinants of subsequent changes in WHR than were behavioral variables. The rates of changes in waist circumference were significantly more pronounced in android women than were in gynoid women, whereas the rates of changes in hip circumferences were approximately the same in both groups. Due to the fact that android women changed their waist circumference when they changed their BMI, a significant association between changes in BMI and changes

in WHR were found. Also, changes in BMI were positively associated with changes in triglyceride, total cholesterol and blood pressure whereas changes in WHR were associated with changes in triglyceride and diastolic blood pressure.

The measurement of central adipose tissue distribution poses multiple problems (24). However, circumference measurements are thought to be more reliable than skinfold thickness measurements (106). Skinfold thicknesses are direct measurements of skin and adipose tissue, but they can only measure variation in the distribution of subcutaneous adipose tissue. There is growing evidence that central fat distribution also involves increases in deep, intra-abdominal adipose tissue which are more active metabolically than subcutaneous adipose tissue. Indices of central fat pattern based on circumferences, such as the WHR, may capture some variation in deep abdominal adipose tissues, but circumferences are also influenced by variation in muscle and bone (24). According to Ley et al. (34), the significance of the concept of android and gynoid-body-fat distribution has been based on indirect measures. BMI is an index, not a direct measure, of body fat. In addition, the use of BMI has recently attracted some criticism (107-108). Garn et al. (108) suggested that BMI may be stature dependent over at least a part of the age range, affected by leg length, and may reflect both lean and fat tissue to a

comparable degree. In the study by Ley et al. (34), BMI in men reflected a greater lean tissue mass whereas the higher BMI in postmenopausal than in premenopausal women reflected a greater fat mass.

In a study by Lapidus et al. (59), waist-to-hip ratios are claimed to be better than measures of skinfold thickness especially as predictors of cardiovascular risk. However, according to Ley et al. (34), both types of measurements are indirect and include tissue other than fat. Because of this, they provide only a qualitative assessment of regional fat mass and distribution. Ley continues by stating that hip-girth measurements reflect not only fat but also known differences between male and female pelvic structures and therefore overestimates sex differences in fat distribution. The study by Ley et al. (34) confirmed that there was a significant difference in fat distribution in women after menopause and the pattern of distribution approaches that of men. Postmenopausal women had a 20% greater fat mass than premenopausal women. The proportion of android fat was significantly lower in premenopausal women (38.3%) than postmenopausal women (42.1%).However, Ley's results conflicted with those of Lanska et al. (6) who failed to show any change in fat distribution on postmenopausal women.

In Lanska's study, menopausal status was not found to be related to body-fat distribution. According to Ley, the contrast may be related to study design.

In a study by Landin (91), the importance of obesity and regional fat distribution on glucose tolerance, insulin, blood pressure, and plasma lipids was analyzed. Results showed that the amount of body fat is by itself far more important than the WHR. Plasma triglycerides were higher in obese women with abdominal fat distribution than in lean women.

The relationship between diet and disease seems positively correlated. Because of this, the National Academy of Sciences and the National Research Council (NAS/NRC) has reviewed the impact of dietary patterns in the etiology of diseases and assessed the potential for reducing their frequency and severity (109). Researchers (110) have recently proposed a relationship between the distribution of morbidity and mortality from cardiovascular diseases and the eating habits of populations of different geographical areas. According to the Seven Countries Study (110), the Mediterranean countries where CVD morbidity and mortality are low, have low serum cholesterol levels compared to Finland and/or the United States. The authentic Mediterranean diet of the 60s is defined as a diet high in cereal (more than 60%), excluding alcohol and low total fat

(less than 30%, with moderate amounts of fats, predominantly olive oil (representing more than 70% of total lipids). However, at the present time there is insufficient information to provide proper definition of the Mediterranean diet, either in terms of foods or in terms of defined chemical compounds. This definition does not consider the presence, amount, or interation of the many non-nutrients that are likely contained in a high proportion of plant foods in this region. Also, today's Mediterranean diet varies depending upon the region that is studied.

The Nationwide Food Consumption Survey, which observed dietary practices and changes in the United States between 1977 to 1987, recorded that intakes of high-fat beef and pork, whole milk and white bread decreased while intakes of low-fat beef, pork, poultry, fish, low-fat milk and whole-grain breads increased. From 1960 to 1980, reductions in serum cholesterol concentrations and fat intake were reported for adults aged 20-74 years of age (111). One possible cause in this decrease could be due to the past years' increase in public health awareness concerning nutrition and disease.

The public's health as a social movement has become a significant force in the U.S. (112). Of all of the health topics to be considered, the topic of weight loss is one

that is constantly being discussed and practiced. In 1984, the National Health Interview Survey (113) reported that 46% of women were dieting at the time of the survey, and girls are beginning to diet at younger ages than ever before (114). At any one time, approximately 20-24% men and 33-40% women are trying to lose weight while 28% in both groups are trying to maintain their weight (115). Even though weight loss practices have been occurring for many years, it is only recently that the behavioral, metabolic, and health consequences for women undergoing repeated bouts of weight loss and regain (weight cycling) are being explored (116). Improving self image, overall health and well-being as well as gaining acceptance are the primary objectives for weight reduction. Dietary and exercise changes are the most common strategies used (48).

## Dietary Fat Intake

There is great popular interest in nutrition particularly in relation to dietary fat because of implications for obesity, heart disease, and more recently, cancer. These concerns are accompanied by interest in the more complex aspects of cardiovascular disease, hypertension, immune system, aging, and cancer by health professionals. Current knowledge is reaching a state of

better understanding of all of these conditions and a consensus toward recommendations for dietary fat consumption (76). However, the appropriate diet composition to alleviate the common disturbances in lipid metabolism which are apparent in the abdominally obese has not yet been developed. Debate still exists on the definition of an optimal diet regimen for obese individuals (117). Not debatable is the fact that overconsumption of dietary fat increases an individual's susceptibility to cancer; fat and cholesterol may both contribute to heart and coronary artery disease (3). Epidemiological data suggest that there is a causal relationship between the consumption of dietary saturated fat and the eventual development of coronary heart disease (118-120). Due to the fact that such data does exist, the American Heart Association (AHA) recommends a dietary strategy of no more than 30% of total calories being derived from fat with no more than 10% of total calories being derived from saturated fats (121). However, even with the recommendations of the AHA and the data to confirm the advantages of decreasing fat intake, very few Americans are practicing this dietary strategy.

Food disappearance and diet studies have both produced the same finding: people today are eating approximately 40 to 50 percent of their kcalories as fat. This percentage is

even more than the amount eaten as fat at the turn of the century and is definitely much more than needed (3).

Even though there is an awareness concerning the reduction of fat intake, a controversy exists about which type of diet should be recommended to the general population for the optimalization of serum lipid levels (122). Brussaard et al. (123) reported the effects of diets differing in type and amount of dietary fat on serum lipoproteins in healthy volunteers over a 5-week period. HDL cholesterol (HDL-C) was lowered and total serum triglycerides and VLDL concentrations increased, when polyunsaturated fat was replaced by carbohydrates. However, Antonis and Bersohn (124) reported that the increase in serum triglycerides on carbohydrate-rich diets subsided after 3-4 months on this diet, and therefore both the changes in VLDL and HDL observed previously could have been temporary only.

As evident from the above statements, the composition of plasma lipids in man and animals is a reflection of the type and amount of dietary lipids consumed. Lipid metabolism is also endogenously controlled by pancreatic, pituitary, and adrenal hormones. Furthermore, the levels of these hormones appear to regulate lipid metabolism in different metabolic conditions such as obesity and diabetes (125). Changes in blood lipid patterns are often taken to reflect

alterations in the quantitative and qualitative nature of dietary fat (126).

However, most research involving changes in the dietary fat and the resultant effects on blood lipids has involved adult men at risk for heart disease due to elevated serum cholesterol. These studies led to the general recommendations that increasing the ratio of polyunsaturated to saturated fatty acids (P:S ratio) of dietary fat along with concomitant decrease in intake of total fat and cholesterol would decrease plasma cholesterol and thereby reduce the risk of heart disease (127).

## Exercise

In the complex and often uncertain world of obesity research, one fact can be stated with authority: exercise is associated with weight loss (128). Correlational studies (129-133) have consistently found that exercise is associated with successful weight loss and maintenance. Exercise is a factor also thought to improve lipid profiles and should be considered a major component of any long term cardiovascular fitness program (134). In actuality, exercise training may be a nonpharmacological method for favorably altering lipid profiles (135). Slow weight reduction and mild exercise has shown to raise HDL-C and lower LDL-C and triglyceride levels (134, 136).

Blumenthal et al. (135) examined the effects of aerobic exercise on lipid levels in premenopausal and postmenopausal women. Fifty healthy middle-aged women were randomly assigned to 12 weeks of either aerobic exercise or nonaerobic strength training. Pre- and post-menopausal women in both groups experienced slight reductions in high-density lipoprotein cholesterol and total cholesterol over the 12-week study period.

Johnson and Greenland (136) investigated the effects of dietary cholesterol intake changes on plasma lipids in normolipidemic healthy men that exercised regularly and consumed a moderately fat-restricted, low saturated fat diet. In this group, mean plasma triglycerides and high-density lipoproteins did not change significantly. Therefore, Johnson and Greenland suggested that restriction of dietary cholesterol may be justifiable even when other lifestyle and dietary measures are undertaken to minimize blood cholesterol levels.

Doshi et al. (137) tested a multidisciplinary nutrition education and fitness training program for its effectiveness in lowering lipid profiles of elderly clients. The program served 31 free-living, predominantly female, black elderly aged 56-88 years. Dietary, anthropometric, biochemical, and fitness assessments were performed before and after a ten-week, biweekly program. Significant decreases ($p < 0.05$)

were seen in waist circumference, total cholesterol, low-density lipoproteins, and total cholesterol to high-density-lipoprotein cholesterol ratio. These finding suggest that a ten-week, biweekly program can be effective in producing both significant, as well as favorable changes, in atherogenic lipids in elderly black subjects.

The response of plasma lipids to dietary fat intake in groups other than high-risk males remains unclear. Until recently, studies with women have been particularly scarce (126). It was observed during this review of literature that no study had examined the present fat intake and serum lipid levels of postmenopausal, abdominally obese women nor had any study investigated the effects of a general low-fat diet on blood lipid levels in this particular population. Due to the above reasons, dietary fat intake and the metabolic effects of this intake will be investigated in postmenopausal, abdominally obese women.

# CHAPTER III

# PROCEDURES

This study received the approval of the Human Subjects Review Committee at Texas Woman's University. A copy of the approval letter can be found in Appendix A. All subjects were asked to read either Consent Form 1, 2 or 3 (Appendix B), and an oral description of procedures was provided by the investigator. After reading the Consent Form, all subjects were asked to sign, and a copy was provided to them while an original was retained in each file.

## Subject Recruitment

During January of 1994, thirty-six postmenopausal women who met the study criteria were recruited through advertisement in the local newspaper of the Mid-county area of Jefferson County, Texas (Appendix C).

Respondents were screened by telephone regarding current weight, height, medications presently being taken, and preexisting disease states, and date since last menses. Respondents were screened by telephone for initial eligibility.

Each subject completed an extensive health

questionnaire (Appendix D) in order to assess medical histories. After reviewing the completed health questionnaire, appointments were scheduled for the second phase of the screening.

Inclusion criteria included the following: female, postmenopausal, ambulatory, a WHR > .80, BMI > 27 $kg/m^2$, no personal history of diabetes mellitus or cardiovascular disease, no hormone therapy and not taking any medications which might interfere with lipid metabolism. Once subjects were weighed and measured, BMI and WHR were calculated. Body mass index was calculated as: weight in kg divided by height in meters squared. Waist-to-hip ratio was calculated as: waist measurement divided by hip measurement.

During the second screening, the health questionnaires were intently reviewed with each subject by emphasizing laboratory values. If a physician's release was warranted prior to admission in the study, appropriate calls were made to a subject's personal physician and a written release was obtained. All possible risks and benefits were provided orally during the second screening sessions and written consent was obtained.

Attendance at weekly meetings was also addressed during the second phase of screening. Subjects were informed as to the importance of adhering to the specified dietary regimen,

preparation of detailed dietary records and dates scheduled during week 0 and 6 for blood sampling.

## Experimental Design

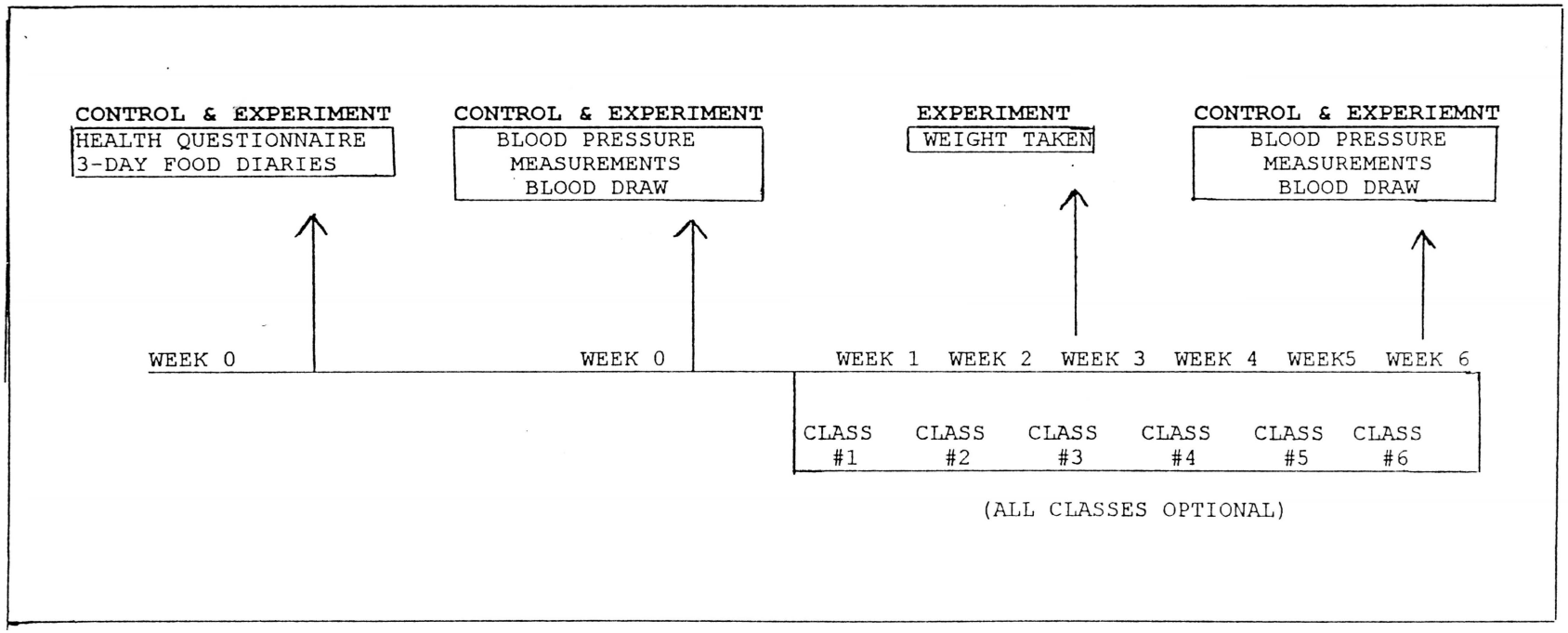
After subjects were recruited, each was randomly assigned into one of three diet groups during week 0 of the study. Refer to Table 1 for characteristics of study population for control, 30% and 20% fat diets. One group was not to have any intervention during the six-week period (control); the second group was to follow a 30% calories as fat diet and attend nutrition counseling sessions for six weeks; and the third group was to follow a 20% calories as fat diet and also attend nutrition counseling sessions for six weeks. The study design can be found in Figure 1.

## Body Measurement

Weight was assessed during week 0, week 3 and week 6 of the study. Body weight was measured in kg to the nearest 0.1 kg using a beam balance scale. Height, waist and hip circumferences were assessed using a flexible measuring tape. Height was measured by affixing an accurate tape measure to the wall. Subjects were measured to the nearest centimetre in bare feet with back to the wall. Waist measurements were made between the last rib and the iliac crest above the umbiliccus. Hip circumference was measured

Figure 1

**TIME FRAME FOR NUTRITION STUDY**



at the greatest girth of the hip area. Each potential subject was weighed and measured in light street clothes and without shoes.

## Diet Assessment

Documentation during the follow-up sessions was kept on all subjects regarding any physical complications, intolerances toward the diets or illnesses incurred during the study. Nutrient intakes were calculated by computer using the Minnesota Nutrition Data System (138). The weekly meetings also included nutritional counseling regarding the diet, purchase of food, and preparation as well as six nutrition and weight-loss sessions developed by the researcher from the following programs (Appendix E): "The LEARN Program for Weight Control (139), The American Heart Association "Heart at Work Program" (121) and Texas Woman's University Department of Nutrition "Weight Control Through Behavior Modification" (140).

Diets were designed for weight reduction for six weeks providing a minimum of 1200-1250 kcal or 5160 kJ. The diets were developed using the ADA exchange system (141) and the Minnesota diet system (138). The two test diets were distinguished by different percentages of fat from the diet. Experimental 1 (EX1) followed a diet consisting of 30%

calories from fat whereas Experimental 2 (EX2) followed a diet consisting of 20% calories from fat. Both experimental diets contained 1200 calories. However, EX1 contained an average of 20% protein, 30-35% fat, and 40-50% carbohydrate providing approximately 60 gms of protein, 44 gms of fat, and 135 gms of carbohydrate whereas EX2 contained an average of 20% protein, 20-25% fat, and 55-60% carbohydrate providing approximately 60 gms of protein, 30 gms of fat, and 171 gms of carbohydrate. Diet exchange foods (Appendix F) and food diaries (Appendix G) were provided. During week 3 and 6 of the study, subjects' compliance to the diet was verified using the Minnesota Diet System which expressed diet intake in percentages and grams of nutrients contained in those foods eaten over the week and the course of the study. Diet analysis was compiled over three days within each week to assess adherence to the experimental diets (see Appendix H for sample menus for both the normal- and low-fat diet). Ranges were devised that allowed for a +20% deviation from the established level of kcals, grams of carbohydrate, protein and fat.

## Biochemical Measurements

The laboratory at St. Mary Hospital in Port Arthur, Texas provided assistance and expertise with the investigator of this study. All assays were performed after

5 milliliters (5 ml) of fasting blood samples were taken. Two milliliters (2 ml) of serum was required for each analysis. Whole blood was collected and allowed to clot according to the tube manufacturer's (Ventroject) instructions. All samples stood at room temperature for half an hour before centrifuged for 10 minutes at 3000 revolutions per minute (rpm) to separate serum from whole blood cells. Serum samples were pour off into transporting tubes and then transported to the St. Mary Laboratory where they were refrigerated until the analysis occurred. Samples were collected at week 0 and week 6. Fasting blood samples were analyzed for serum total cholesterol, serum HDL-cholesterol and serum triglyceride. All samples were drawn in a sitting position after a 12-hour overnight fast.

Lipid Determination

The lipid profile for each subject included: total-cholesterol, HDL-cholesterol, and triglycerides. Two milliliters (2 ml) of fasting serum was transported in microtubes to St. Mary Laboratory at the end of each day during week 0 and week 6 of the drawings. Upon arrival, samples were prepared for coding which is required by the KODAK EKTACHEM (Kodak Clinical Diagnostics) procedure. Each subject sample was given an identification number using a bar code system that was typed in for each specimen at the

computer terminal as well as the test type required. The investigator performed all analytical procedures using the KODAK EKTACHEM under the supervision of Kathy McBride of St. Mary Laboratory. Serum total-cholesterol, HDL-cholesterol, and triglyceride analysis were determined using the KODAK EKTACHEM. Since serum samples were used, a minimum of 10 uL of sample into the tube was ususally necessary unless specified otherwise. All bar codes were typed in by the investigator using the KODAK EKTACHEM keyboard and terminal as well as samples pippeted into the test tubes specialized for the KODAK EKTACHEM carousel.

Determination of Total Cholesterol

The KODAK EKTACHEM Clinical Chemistry Slide is based on a modification of the method of Allain et al. (142). See Appendix I for discussion of principle. The KODAK EKTACHEM Clinical Chemistry Slide (CHOL) is a dry, multilayered analytical element coated on a clear polyester support. Each sample tube was placed in the KODAK EKTACHEM carousel with positive ID codes typed in by the investigator. The (CHOL) select button was pressed as well as the (RUN) button after loading the carousel. Sample size required was 10 uL per carousel tube and micropippeted by the investigator. Test temperature required was 37 C incubation. Wavelength specification for the spectrophotometer reading was 540 nm.

The total reaction time required was 5 minutes. The KODAK EKTACHEM procedure recommends specimens to be processed within 24-48 hours. Two controls and two standards were run before subject analysis began to assure quality control. Results of all subjects were available by the end of the day during the days of blood collection for analysis. The techniques for cholesterol determination for all KODAK EKTACHEM tests were outlined in the KODAK EKTACHEM Analyzer Operator's Manual (96). The normal range expected was: 140-240 mg/dl for each subject.

Determination of Triglyceride

The procedure outlined for triglyceride was the same as for cholesterol. The principle of the triglyceride reagent formulation, however, is based on a modification of the procedure by Spayd et al. (143). See Appendix I for discussion of principle. Test volume required was 10uL. Incubation temperature was 37 C. Wavelength setting was 540 nm. Total reaction time required was 5 minutes. Sample tubes with KODAK EKTACHEM codes were typed into terminal as well as test selection denoted. The (TRIG) button was pressed as well as the (RUN) button after loading the carousel. Two controls and two standards were run before specimens were placed on the carousel for a quality control check. The normal range is: 35-160 mg/dl.

Determination of HDL-Cholesterol

Serum is the specimen of choice for HDL-cholesterol determination. Serum was removed from the red cells as soon as clotting had occurred. All samples were analyzed within 48 hours for HDL-cholesterol at each drawing. The principle of HDL-cholesterol includes a solution of dextran sulfate which was added to the subject's sample to precipitate out the very-low-density lipoprotein-cholesterol (VLDL) and the low-density lipoprotein-cholesterol (LDLC) in the presence of magnesium sulfate. Centrifugation resulted in a supernatant which was enzymatically analyzed for the HDL-cholesterol remaining. See Appendix I for summary of reagents used. There were 2 controls and 2 standards run with each batch of subjects. Each subject specimen was coded as well as standards. Each sample was micropippeted into appropriate EKTACHEM tubes using 1 mL. Each sample then received 50 uL of dextran sulfate reagent as well as 100 uL of magnesium sulfate reagent. The tubes were then gently vortexed for 10-15 seconds. Samples were incubated for 5 minutes at room temperature. Test samples were then placed in the centifuge for 10 minutes at 3000 rpm at room temperature. Test tubes were then placed in the KODAK EKTACHEM carousel using 100 uL and setting the test selection on (HDLC) and (RUN). Wavelength was specified for 670 nm. Incubation temperature was 37 C. Five minutes

reaction time was required. Any patient sample greater then 130 mg/dl was repeated with dilution protocol, if necessary. This was necessary if the samples had incomplete removal of LDL and VLDL. Since the measurement of HDL-cholesterol is helpful in determining risk of coronary heart disease, the RISK RATIO can be calculated using the following equation: Total Chol/HDL-Cholesterol. The normal range of HDL-Cholesterol is 35-70 mg/dl.

## Statistical Analysis

Two approaches were taken to the analysis of the pre- and post-test group differences. Analysis of variance was performed for differences among diet groups, differences between pre- and post-testing, and the interaction of diet and testing. This analysis permitted observance of pre- and post-test change within and between groups.

Secondly, analysis of covariance was performed between diet groups on the post-test, using the corresponding pre-test as the covariate or control variable. This approach allowed the observance of group differences on the post-test means adjusted for group differences on the pre-test, or post-test means which would be expected if all groups were equal on the pre-test. The level of statistical significance was set at $p < .05$

# CHAPTER IV

# RESULTS

Abdominally obese, postmenopausal females were to consume either no specific (control) diet (N = 11); a 1200 kcal normal-fat (30% calories as fat) diet (N = 11); or a 1200 kcal low-fat (20% calories as fat) diet (N = 11) for a six week period. Changes in anthropometric and biochemical parameters are presented. Due to the fact that analysis of covariance showed no statistically significant differences for any of the parameters, data reported is by analysis of variance. All statistical values reported in the tables are means ($\pm$ Standard Deviation).

## Description of the Subjects

Thirty-six postmenopausal women with abdominal obesity were recruited from the Nederland, Texas community. Study subjects included 30 Caucasian, 2 Mexican-American, and 1 African-American. The thirty-six subjects were divided into three groups of 12 subjects.

After the first biochemical measurements, three of the thirty-six subjects initially recruited withdrew from the study during the fourth week. Reasons given by two of

subjects included interference with personal schedules. The third subject withdrew from the study due to an illness not related to the study. Anthropometric characteristics of the subjects assigned to one of the three groups are shown in Table 1.

Mean age and menopause years of subjects by groups is shown in Table 2. The mean age of subjects by groups is as follows: control group, 56 years (±7.6) of age; normal-fat group, 59 years (±4.2) of age; and low-fat group, 57 years (±10.2) of age. The average years of menopause for subjects in the control group was 9.2 years (±8.4) whereas the average years of menopause for subjects in the normal-fat and low-fat diet groups were 10.6 years (±4.6) and 9.7 years (±10.1), respectively. The majority of the subjects in each group reported on the questionnaire a history of familial diseases that could have increased their risk of cardiovascular disease. These diseases included: cardiovascular disease, cerebrovascular disease, high blood pressure, hypercholesterolemia, diabetes and obesity. At least 50% of the subjects in each group had some previous form of medical complication, with the most common complication being increased blood cholesterol.

Only three of the thirty-three subjects (9%) presently smoked with seven subjects having smoked in the past but at the time of the study were no longer smoking. Forty-five

percent of the subjects reported having a recent increase in body weight with 100% of the women reporting an appetite of either good or excellent.

Consumption of vitamin supplementation varied. Only 27% of subjects in the control group consumed a vitamin supplement whereas 73% of the women in the normal-fat diet group and 45% of the women in the low-fat diet group consumed vitamin supplementation. On the average, all subjects in each group consumed approximately three meals per day with ranges of 1-4. At least 80% of the subjects in each group reported consuming some form of snack food at least one time during a 24-hour period. Ninety percent of the subjects in each group also reported eating out at least one time during a one-week period with ranges of 0-5. The cafeteria was listed as the most common place to engage in food consumption outside of the home.

Before the study began, 45% of the control group engaged in some form of exercise; 73% of the normal-fat group exercised; and 36% of the low-fat group exercised. The most commonly reported form of exercise was walking; of the women who did exercise, the average number of days per week reported was three with ranges of 1-7. At the completion of the study, 45% of the control group engaged in some form of exercise, whereas 100% of both the normal-fat and low-fat diet group engaged in some form of aerobic activity.

## Anthropometric Measurements

Regardless of the diet consumed, changes in body weight and modifications in body composition were evident in experimental subjects over the entire six week period. For both experimental groups waist-to-hip ratio (WHR) (Figure 2) and body mass index (BMI) (Figure 3) were decreased ($p < 0.01$) for both parameters. However, the control group did not have a significant weight decrease. From Table 1, it is evident that a 2.3 kg ($\pm$17.3) decrease in weight was observed in those subjects consuming a 30% fat diet. A 3.3 kg ($\pm$12.2) weight loss occurred ($p < 0.01$) at the end of the six week period for subjects consuming a 20% fat diet. However, for the subjects in the control group consuming no specific diet, no significant weight change occurred. It should be noted that the subjects randomly assigned to the 30% fat diet exhibited higher weight measurements at the beginning of the study (Table 1). This was not intentional. Analysis of variance (ANOVA) weight data can be found in Table 3.

The BMI decreased significantly for the two experimental groups regardless of the diet consumed ($p < 0.01$). From Table 1 it is evident that a BMI decrease of 1.0 $kg/m^2$ ($\pm$6.9) occurred in subjects consuming the 30% fat diet. In subjects consuming the 20% fat diet, a BMI

Table 1

Anthropometric characteristics of postmenopausal abdominally obese females consuming no specific diet, normal-fat diet and low fat diet at week 0 and week 6 of study

| Anthropometric Characteristic | NO SPECIFIC DIET (N=11) | | | NORMAL-FAT DIET (N=11) | | | LOW-FAT DIET (N=11) | | |
|---|---|---|---|---|---|---|---|---|---|
| | Week 0 | Week 6 | P | Week 0 | Week 6 | P | Week 0 | Week 6 | P |
| | mean SD | mean SD | | mean SD | mean SD | | mean SD | mean SD | |
| Weight (kg) | 79.5±13.2 | 78.8±13.5 | 0.7 NS | 83.8±16.9 | 81.5±17.7 | 2.3 <.001 | 74.7±12.3 | 71.4±12.3 | 3.3 <.001 |
| BMI* ($kg/m^2$) | 31.1± 5.8 | 30.9± 6.0 | 0.2 NS | 32.1± 6.7 | 31.1± 7.0 | 1.0 <.001 | 29.3± 2.2 | 28.1± 2.3 | 1.2 <.001 |
| WHR** | 0.95±0.07 | 0.94±0.07 | .01<.001 | 0.94±0.07 | 0.93±0.07 | .01 <.001 | 0.96±0.5 | 0.94±0.5 | .02 <.001 |

*Body Mass Index
**Waist-to-Hip Ratio
Statistically Significant = $p < .05$
Statistical Method = Analysis of Variance

Table 2

Age and menopausal characteristics of postmenopausal abdominally obese females consuming no specific diet, normal-fat diet and low-fat diet

| Charcteristic | NO SPECIFIC DIET (N=11) | | NORMAL-FAT DIET (N=11) | | LOW-FAT DIET (N=11) | |
|---|---|---|---|---|---|---|
| | mean | SD | mean | SD | mean | SD |
| Age (years) | 56 | ±7.6 | 59 | ±4.2 | 57 | ±10.2 |
| Menopause (years) | 9.2 | ±8.4 | 10.6 | ±4.6 | 9.7 | ±10.1 |

FIGURE 2

MEAN WAIST-TO-HIP RATIO FOR 33
ABDOMINALLY OBESE POSTMENOPAUSAL WOMEN

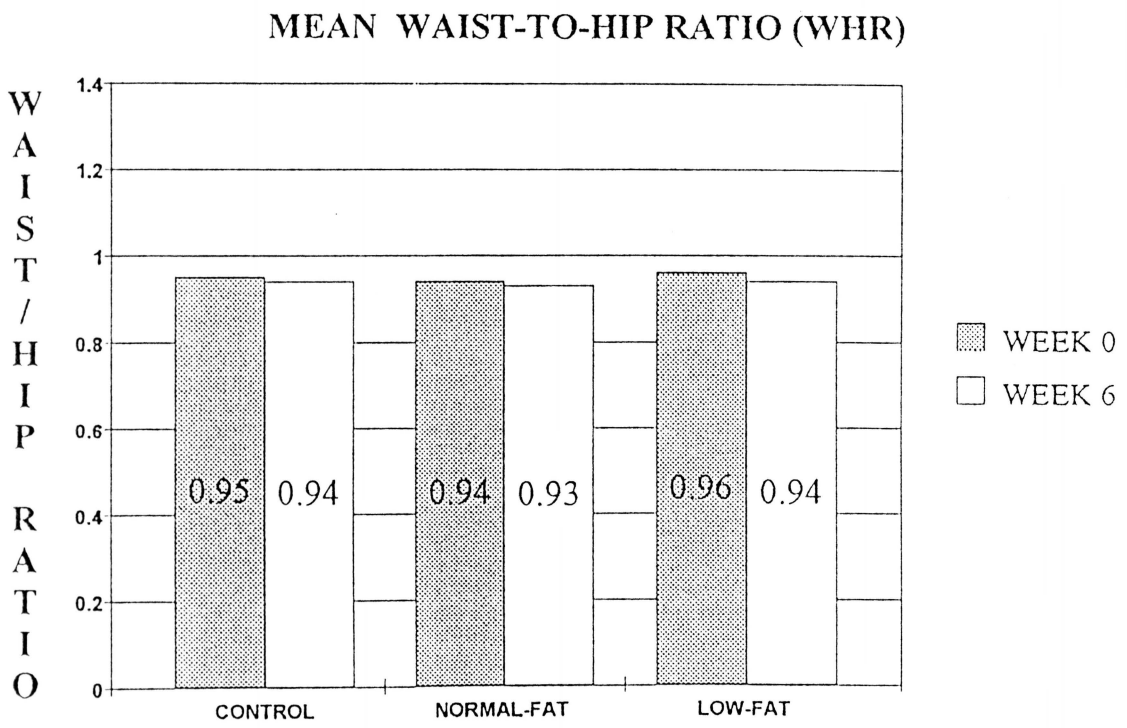


FIGURE 3

MEAN BODY MASS INDEX FOR 33
ABDOMINALLY OBESE POSTMENOPAUSAL WOMEN

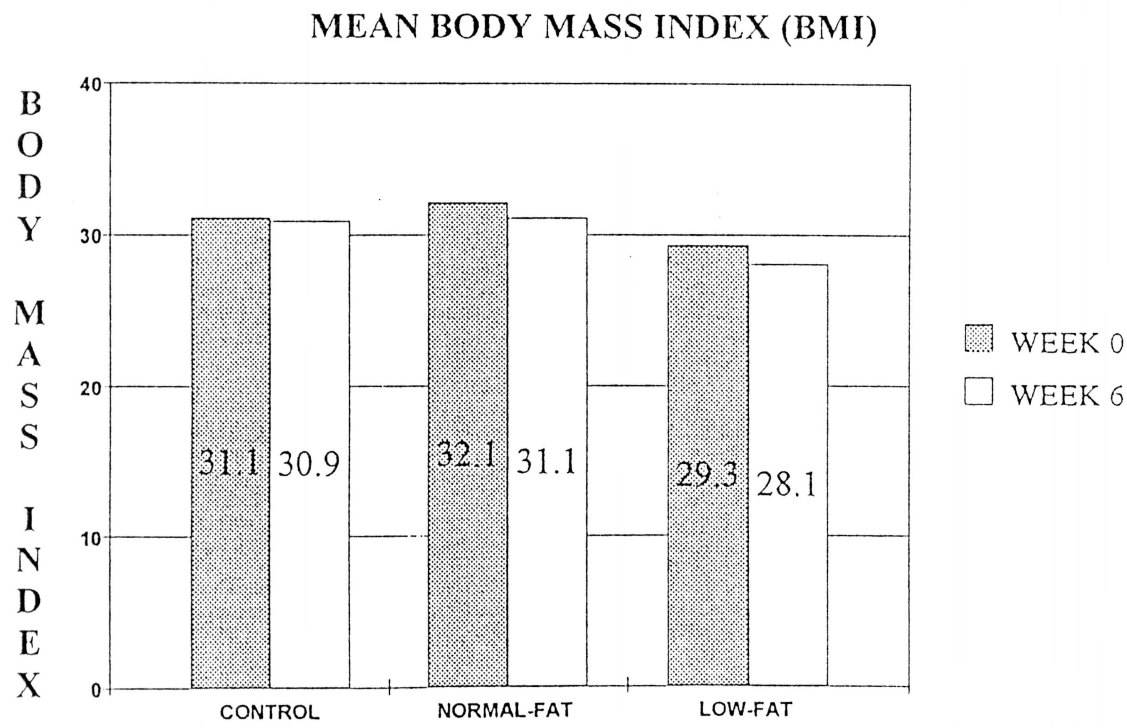


Table 3. ANOVA weight table for study subjects

| Source of Variance | SS | df | MS | F | F-Significance |
|---|---|---|---|---|---|
| Within Cells | 12485.00 | 30 | 416.87 | | |
| Group | 1035.93 | 2 | 517.96 | 1.24 | 0.303 |
| Within Cells | 40.02 | 30 | 1.33 | | |
| Time | 77.03 | 1 | 77.03 | 57.74 | 0.000* |
| Group by Time | 19.53 | 2 | 9.78 | 7.33 | 0.003* |

* Statistically Significant

decrease of 1.2 kg/m$^2$ ($\pm$2.3) occurred. However, BMI did not change significantly for subjects in the control group ($p > 0.05$) (Table 1). The ANOVA data for BMI can be found in Table 4. Declines in WHR were significant ($p < 0.01$) for all subjects in both the control group and the experimental groups (Table 1). The ANOVA data for WHR can be found in Table 5.

## Lipoprotein Biochemical Analyses: Cholesterol, Triglyceride, and HDL-cholesterol

Lipid values for each subject can be found in Appendix J. Blood lipid levels did not differ significantly using analysis of covariance. However, when using analysis of variance total cholesterol was significantly reduced ($p < 0.01$) at the end of week 6 in subjects consuming the 20% fat diet. No significant change in total serum cholesterol occurred in subjects consuming no specific diet or the 30% fat diet. Values for serum cholesterol can be found in Table 6. Mean total serum cholesterol concentrations over time are shown in Figure 4.

The means for serum triglyceride concentrations are shown in Table 6 as well as Figure 5. Serum triglyceride concentrations in the group consuming the 30% fat diet decreased over 6 weeks but not significantly. Unfortunately, concentrations in both the control group and the group

Table 4. ANOVA body mass index table for study subjects

| Source of Variance | SS | df | MS | F | F-Significance |
|---|---|---|---|---|---|
| Within Cells | 1726.28 | 30 | 57.54 | | |
| Group | 103.45 | 2 | 51.73 | 0.90 | 0.418 |
| Within Cells | 5.51 | 30 | 0.18 | | |
| Time | 10.09 | 1 | 10.09 | 54.95 | 0.000* |
| Group by Time | 3.33 | 2 | 1.65 | 9.01 | 0.001* |

* Statistically Significant

Table 5. ANOVA waist-to-hip ratio table for study subjects

| Source of Variance | SS | df | MS | F | F-Significance |
|---|---|---|---|---|---|
| Within Cells | 0.24 | 30 | 0.01 | | |
| Group | 0.00 | 2 | 0.00 | 0.20 | 0.822 |
| Within Cells | 0.00 | 30 | 0.00 | | |
| Time | 0.00 | 1 | 0.00 | 14.52 | 0.001* |
| Group by Time | 0.00 | 2 | 0.00 | 0.54 | 0.588 |

* Statistically Significant

consuming the 20% fat diet increased non-significantly over the six-week period (See Table 6). Subjects assigned to the control group presented with higher levels initially (161.3 mg/dL versus 130.7 mg/dL and 147.2 mg/dL). Mean serum triglyceride concentrations over time are shown in Figure 5.

Although not significant, serum HDL-cholesterol concentrations in the group consuming the 30% fat diet increased over the six-week period (See Table 6). No significant change in HDL-cholesterol was observed in subjects who consumed no specific diet or the 20% fat diet. See Figure 6 for mean serum HDL-cholesterol concentrations over time and Table 6 for average concentrations.

The mean ratios of total cholesterol/HDL-cholesterol at the beginning of the study were as follows: control group, 5.04; normal-fat diet group, 4.69; and the low-fat diet group, 4.43. Mean ratios at the end of the six-week diet period resulted in the following measurements: control group, 4.74; normal-fat diet group, 4.44; and the low-fat diet group, 4.10. (See Table 6).

## Diet Assessment

Mean dietary consumption by individual groups can be found in Table 7. Nutrient consumption for each subject can be found in Appendix K. The average daily total caloric intake (kilocalories) for the experimental group consuming

Table 6

Serum lipid parameters of postmenopausal, abdominally obese females consuming no specific diet, normal-fat diet and low-fat diet at week 0 and week 6 of study

| Lipid Parameter mg/dL | NO SPECIFIC DIET (N=11) Week 0 | Week 6 | Δ | P | NORMAL-FAT DIET (N=11) Week 0 | Week 6 | Δ | P | LOW-FAT DIET (N=11) Week 0 | Week 6 | Δ | P |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | mean SD | mean SD | | | mean SD | mean SD | | | mean SD | mean SD | | |
| Total chol* | 230.8±51.0 (5.95) R:157-316 | 218.6±47.8 (5.65) R:129-289 | -12.2 | NS | 196.6±23.0 (5.06) R:168-228 | 201.9±19.2 (5.22) R:129-248 | +5.3 | NS | 228.7±50.3 (5.89) R:158-284 | 208.0±47.9 (5.38) R:145-269 | -20.7 | <.004 |
| TGY** | 161.4±55.1 (4.17) R:75-233 | 178.4±62.8 (4.61) R:87-272 | +17 | NS | 130.7±46.2 (3.38) R:48-194 | 118.0±31.4 (3.05) R:71-180 | -12.7 | NS | 147.2±94.4 (3.81) R:59-341 | 154.0±81.1 (3.98) R:58-292 | +6.8 | NS |
| HDL-chol*** | 52.3±14.9 (1.35) R:33-76 | 48.5±14.0 (1.25) R:35-75 | -3.8 | NS | 44.9±10.4 (1.16) R:30-64 | 46.2± 6.0 (1.19) R:36-55 | +1.3 | NS | 57.8±23.7 (1.49) R:38-122 | 52.3± 9.5 (1.35) R:42-90 | -5.5 | NS |
| Total/ HDL-c**** | 5.04±1.8 (0.13) R:2.7-7.3 | 4.74±1.4 (0.12) R:2.5-6.7 | -0.3 | NS | 4.69±1.1 (0.12) R:2.7-5.0 | 4.44±0.67 (0.13) R:3.3-5.4 | -0.25 | NS | 4.43±1.8 (0.11) R:2.1-7.3 | 4.1±1.1 (0.11) R:2.6-6.2 | 0.33 | NS |

Statistically Significant = $p < .05$
*Chol = Cholesterol
**TGY = Triglyceride
***HDL-chol = High-density-lipoprotein cholesterol
****Total/HDL-c = Total cholesterol to HDL-cholesterol ratio
(SI Units) = mmol/L
R = Range
Statistical Method = Analysis of Variance (ANOVA)

FIGURE 4

MEAN TOTAL SERUM CHOLESTEROL CONCENTRATIONS
IN 33 ABDOMINALLY OBESE POSTMENOPAUSAL WOMEN

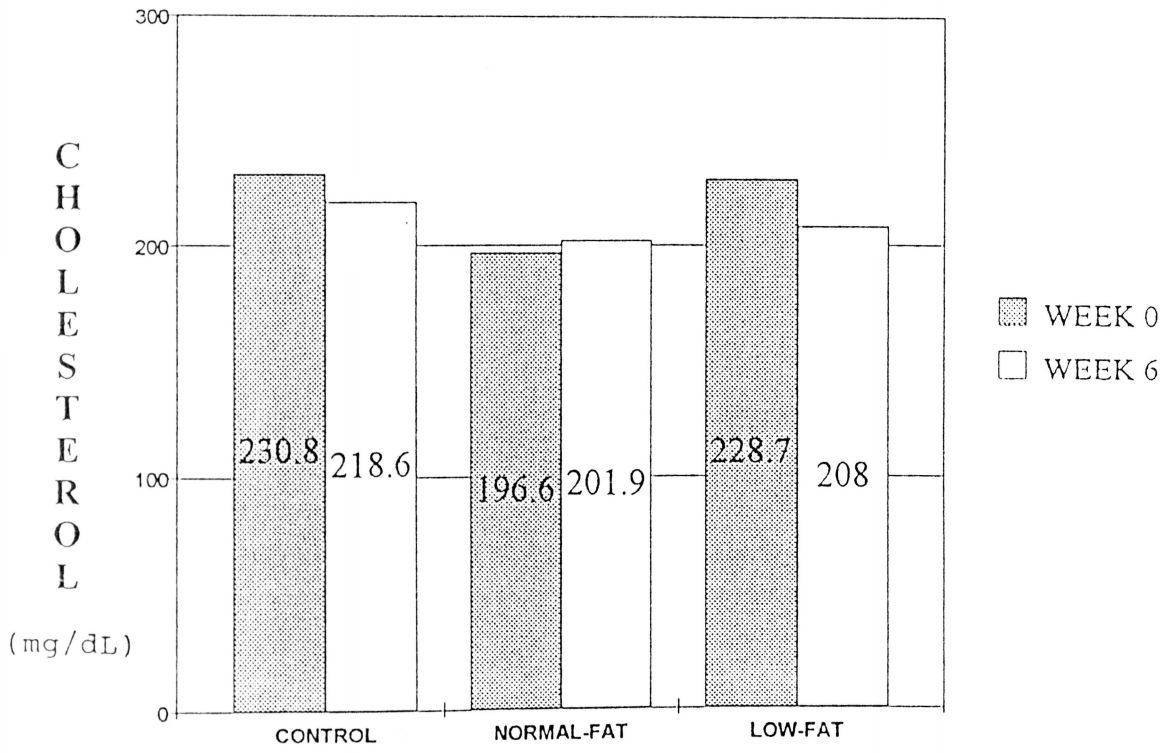


FIGURE 5

MEAN SERUM TRIGLYCERIDE CONCENTRATIONS IN 33 ABDOMINALLY OBESE POSTMENOPAUSAL WOMEN

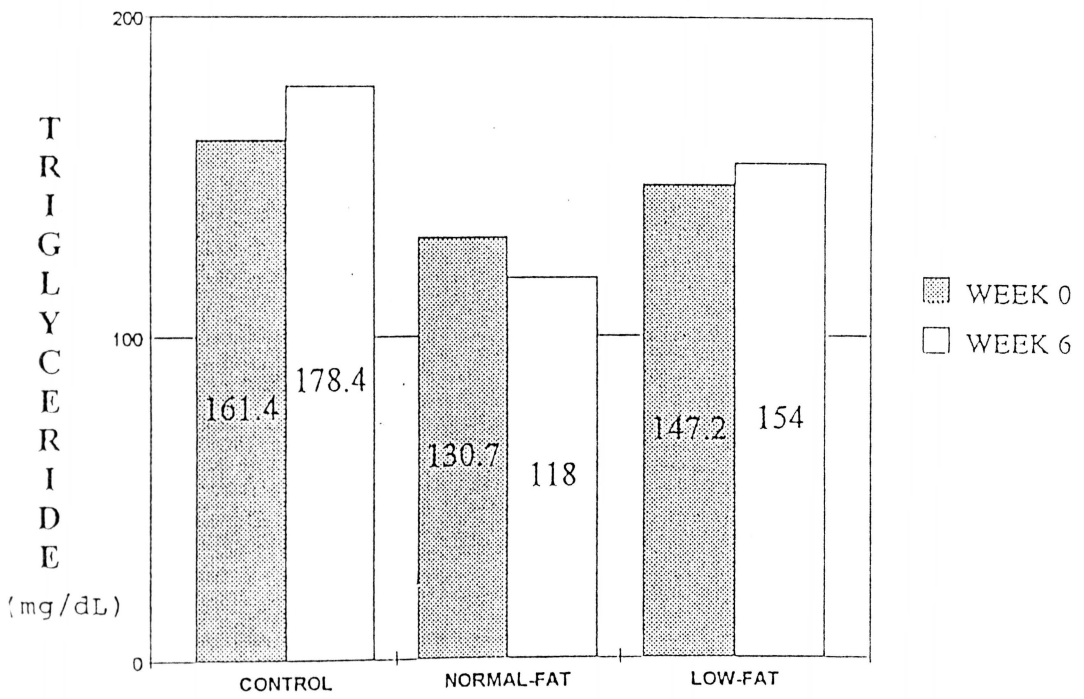
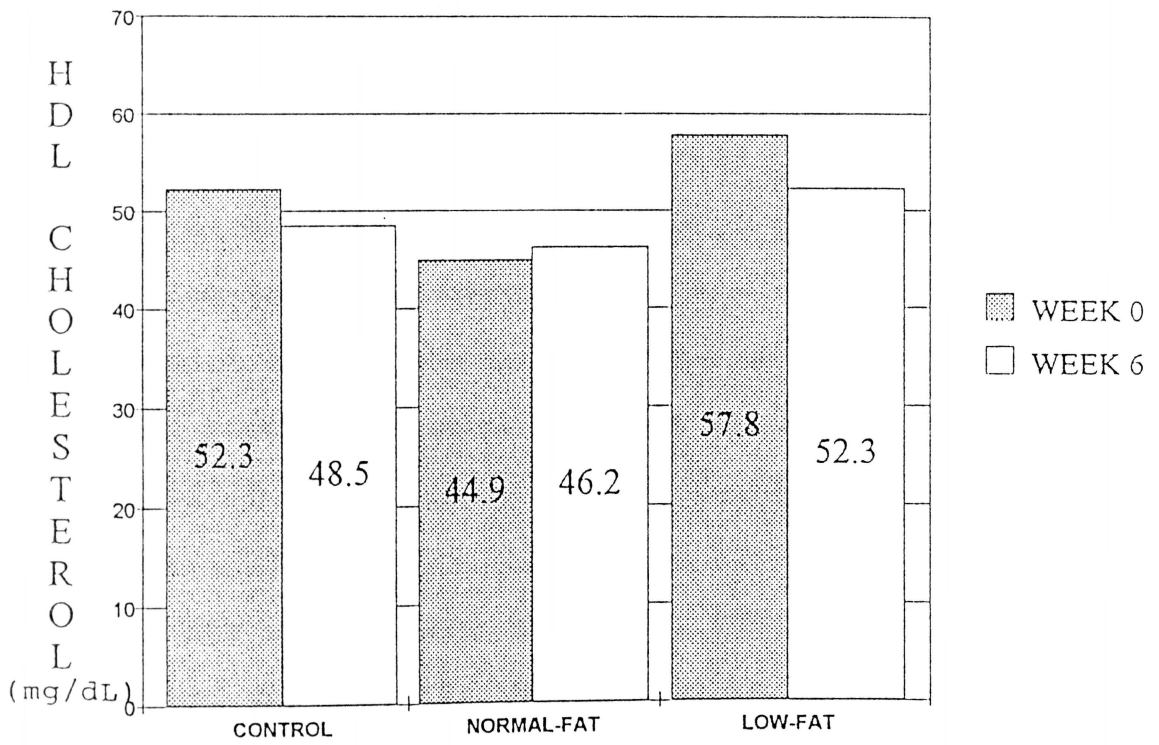


FIGURE 6

MEAN HDL-CHOLESTEROL CONCENTRATIONS IN 33 ABDOMINALLY OBESE POSTMENOPAUSAL WOMEN



the normal-fat diet was 1746 kcal/day (±257). The average intake of carbohydrate in grams for the normal-fat group was 217 g (±31) with a range of 145-253 (g). Contribution to the overall calories was 49%. The average intake of protein in grams for this group was 83 g (±12) with a range of 53-97 (g). Contribution to the overall calories per day was 19%. Fat contributed an average of only 63 g (±13) per day with a range of 42-82. Total percentage contributed was 32%. The average intake of dietary cholesterol for the normal-fat diet group was 236 mg (±77) .

The average daily caloric intake (kilocalories) for the experimental group consuming the low-fat diet was 1465 (±102) kcal/day. The average intake of carbohydrate in grams for the low-fat group was 189 g with a range of 150-211 (g). Contribution to the overall calories was 52%. The average intake of protein in grams for this group was 79 (±7) g with a range of 70-91 (g). Contribution to the overall calories per day was 21%. Fat contributed an average of only 45 g (±7) per day with a range of 34-56. Total percentage contributed was 27%. The average intake of dietary cholesterol for the low-fat diet group was 201 mg (±51). Subjects in both experimental groups consumed their dietary exchanges in three small meals and two snacks per day.

Diet analysis of the control group showed that the average daily total caloric intake was 1871 kcal/day (±315).

The average intake of carbohydrate in grams for this group was 218 g (±31) with a range of 154-264 (g). Contribution to the overall calories per day was 16%. Fat contributed an average of 78 g (±23) per day with a range of 49-117 (g). Total percentage contributed was 37%. The average intake of dietary cholesterol for the control group was 248 mg (±17).

Both the low-fat and the normal-fat diets were analyzed for intakes of carbohydrate, protein, fat, vitamin A, vitamin C, calcium, thiamin, riboflavin, niacin, and iron. Nutrient intakes were compared with the 1989 Recommended Dietary Allowances (RDAs) (144). Because the RDAs are not requirements but instead allowances, adequate intake is usually achieved at intake at least two-thirds of the allowance. Because of this, 66% of the RDA was considered nutritionally adequate for this study. Analyses of the low-fat diet and the normal-fat diet showed that both were nutritionally adequate and provided at least two-thirds of the RDA. Analysis of the diet assessments revealed that the majority of the subjects consumed over 100% of the RDA.

Originally, analyses of the diets were to occur every two weeks. However, once the study began a mid-point dietary analysis and a concluding dietary analysis were decided upon. Ranges were devised that allowed for a ± 20 deviation from the established levels of kcals, grams of carbohydrate, protein and fat. If the established range was met, subjects

were considered to have complied to the experimental diet. Mean caloric intake over the six-week period for the two experimental groups can be found in Figure 7. Mean percentage of calories from fat for the two experimental groups can be found in Figure 8. Experimental subject's attendance for the 6 weekly class sessions can be found in Appendix L.

Subjects participating in the study were to decrease normal caloric intake to a minimum of no less than 1200 kcals/day. However, due to the fact that some subjects in the normal-fat diet group had an increase in caloric consumption over the six-week period, the data from the three dietary groups were reorganized into two post-hoc groups based upon what could be considered clinically significant decreases in dietary intake. (See Appendix M for Reorganized Data Grouping). Data from the normal-fat diet group were re-established into either a post-hoc control diet group or a reduced-fat diet group based upon dietary intake criteria.

Data were re-established into the post-hoc control diet group if kilocalories/day and percentage of calories from fat exceeded 1500 kcals/day and 35% calories from fat, respectively. Data were re-established into the reduced-fat diet group if kilocalories/day and percentage of calories from fat did not exceed 1500 kcals/day and 35% calories from

fat, respectively. The following results reflect the re-organized data.

Analysis of variance was computed on the reorganized data. Anthropometric values for the reorganized groups can be found in Table 8. The reduced-fat diet group experienced significant ($p < 0.01$) decreases in weight (3.4 kg $\pm$ 11.2) and BMI (1.4 kg/m$^2$ $\pm$ 4.4) over the six-week study period when compared to the post-hoc control diet group. However, both the post-hoc control diet group and the reduced-fat diet group experienced a significant decrease ($p < 0.01$) in WHR. Blood lipid concentrations for the reorganzied groups can be found in Table 9. The reduced-fat diet group experienced a significant decrease (20.5 mg/dl $\pm$ 46.1) ($p < 0.01$) in total cholesterol concentrations over the six-week period. Subjects in the post-hoc control diet group did not experience a significant change in total cholesterol concentrations. No significant difference in triglyceride concentrations or HDL-cholesterol concentrations was found for either groups.

Table 7

Mean dietary consumptions of postmenopausal abdominally obese females consuming no specific diet, normal-fat diet and low-fat diet at week 0, week 3, and week 6 of study

| Nutrient Intake | NO SPECIFIC DIET (N=11) | | NORMAL-FAT DIET (N=11) | | LOW-FAT DIET (N=11) | |
|---|---|---|---|---|---|---|
| | mean | SD | mean | SD | mean | SD |
| Caloric Intake (kcal/day) | | | | | | |
| Week 0 | 1774 | ±379 | 1861 | ±607 | 1692 | ±287 |
| Week 3 | ---- | ---- | 1591 | ±220 | 1256 | ±115 |
| Week 6 | 1968 | ±594 | 1786 | ±283 | 1447 | ±134 |
| Average | 1871 | ±315 | 1746 | ±257 | 1465 | ±102 |
| Carbohydrate (grams) | | | | | | |
| Week 0 | 206 | ±49 | 206 | ±77 | 188 | ±38 |
| Week 3 | --- | --- | 218 | ±34 | 175 | ±23 |
| Week 6 | 230 | ±56 | 229 | ±41 | 205 | ±21 |
| Average | 218 | ±31 | 217 | ±31 | 189 | ±17 |
| Protein (grams) | | | | | | |
| Week 0 | 71 | ±15 | 67 | ±28 | 81 | ±21 |
| Week 3 | -- | --- | 82 | ±9 | 76 | ±8 |
| Week 6 | 78 | ±18 | 86 | ±12 | 84 | ±5 |
| Average | 74 | ±13 | 83 | ±12 | 79 | ±7 |
| Fat (grams) | | | | | | |
| Week 0 | 73 | ±28 | 80 | ±31 | 68 | ±20 |
| Week 3 | -- | --- | 48 | ±12 | 32 | ±5 |
| Week 6 | 85 | ±41 | 60 | ±11 | 37 | ±8 |
| Average | 78 | ±23 | 63 | ±13 | 45 | ±7 |
| Cholesterol (milligrams) | | | | | | |
| Week 0 | 232 | ±103 | 343 | ±260 | 274 | ±133 |
| Week 3 | 225 | ±97 | 165 | ±31 | 168 | ±38 |
| Week 6 | 257 | ±117 | 201 | ±61 | 162 | ±46 |
| Average | 248 | ±17 | 236 | ±77 | 201 | ±51 |

FIGURE 7

MEAN DAILY CALORIC INTAKE FOR 22
ABDOMINALLY OBESE POSTMENOPAUSAL WOMEN

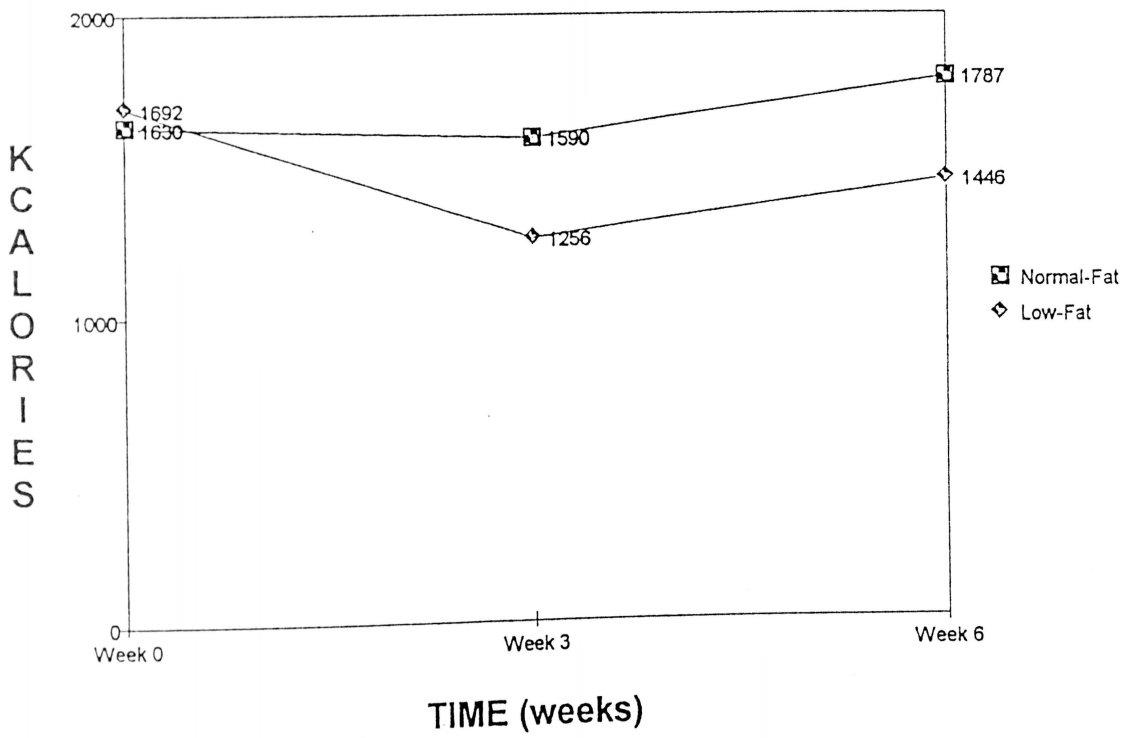


FIGURE 8

MEAN PERCENT CALORIES FROM FAT FOR 22 ABDOMINALLY OBESE POSTMENOPAUSAL WOMEN

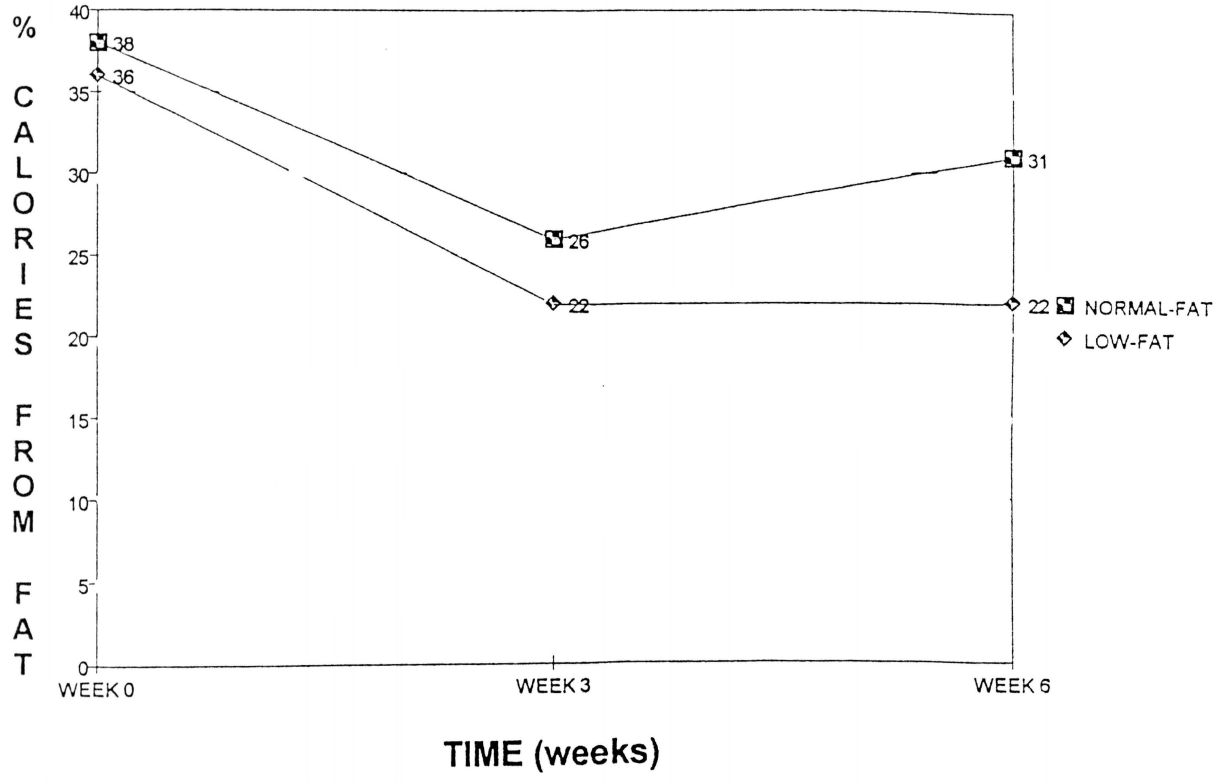


Table 8

Anthropometric characteristics of postmenopausal abdominally obese females in post-hoc control diet group and reduced-fat diet group at week 0 and week 6 of study

| Anthropometric Characteristic | POST-HOC CONTROL (N=20) | | | | REDUCED-FAT (N=13) | | | |
|---|---|---|---|---|---|---|---|---|
| | Week 0 | Week 6 | Δ | p | Week 0 | Week 6 | Δ | p |
| | mean SD | mean SD | | | mean SD | mean SD | | |
| Weight (kg) | 82.1±15.7 | 80.7±16.0 | -1.4 | NS | 75.2±11.3 | 71.8±11.2 | -3.4 | <.01 |
| BMI* ($kg/m^2$) | 32.8±5.7 | 32.3±6.0 | -0.5 | NS | 30.1±6.2 | 28.7±2.5 | -1.4 | <.01 |
| WHR** | 0.95±.07 | 0.94±.07 | -0.01 | <.01 | 0.96±.04 | 0.95±.05 | -0.01 | <.01 |

* Body Mass Index
**Waist-to-Hip Ratio
Statistically Significant = $p < .05$
Statistical Method = Analysis of Variance (ANOVA)

Table 9

Serum lipid parameters of postmenopausal abdominally obese females in post-hoc control diet group and reduced-fat diet group at week 0 and week 6 of study

| Lipid Parameter mg/dl | POST-HOC CONTROL (N=20) Week 0 | Week 6 | Δ | p | REDUCED-FAT (N=13) Week 0 | Week 6 | Δ | p |
|---|---|---|---|---|---|---|---|---|
| | mean SD | Mean SD | | | mean SD | mean SD | | |
| Total chol* | 214.6±43.9 (5.5) | 212.7±37.5 (5.5) | -1.9 | NS | 225.1±47.6 (5.8) | 204.6±44.6 (5.3) | -20.5 | <.01 |
| TGY** | 151.1±51.5 (3.9) | 153.9±56.9 (4.0) | +2.8 | NS | 139.3±88.8 (3.6) | 144.4±78.1 (3.7) | +5.1 | NS |
| HDL-chol*** | 48.7±12.7 (1.3) | 47.4±10.8 (1.2) | -1.3 | NS | 56.2±23.0 (1.5) | 51.4±9.6 (1.3) | -4.8 | NS |
| LDL-chol**** | 128±54 (3.3) | 132±44 (3.4) | +4.0 | NS | 140±36 (3.6) | 125±33 (3.2) | -15 | <.01 |

Statistically Significant = $p < .05$
*Chol = Cholesterol
**TGY = Triglyceride
***HDL-chol = High-density-lipoprotein cholesterol
****LDL-chol = Low-denisty-lipoprotein cholesterol
(SI Units) = mmol/L
Statistical Method = Analysis of Variance (ANOVA)

# CHAPTER V

# DISCUSSION

The overall goal of most diet intervention and weight loss studies is to decrease risk factors associated with various diseases. Substantial weight loss in the obese individual, regardless of adipose location, could be expected to improve both the quality and the quantity of life (45). The purpose of this study was to determine if a low-fat, reduced kcalorie diet would improve the lipid profile in 11 postmenopausal abdominally obese women when compared to a normal-fat diet. This is the only study we know of that investigates the effects of the lipid profile by modifying the amount of dietary fat consumed in abdominally obese postmenopausal women.

At the completion of this study, all subjects in both experimental groups (n = 22) experienced significant reductions in body weight, WHR and BMI regardless of the diet consumed. However even though subjects experienced statistically significant anthropometric decreases (such as WHR and BMI), these decreases were not considered physiologically significant due to the fact that subjects were still abdominally obese at the end of the six-week

period. As is the case in other studies, energy restriction is commonly listed as a factor of weight loss. Weinsier et al. (145) reported an average weight loss of 13 kg in obese postmenopausal women consuming an energy-restricted diet. Phinney et al. (146), who assessed serum polyunsaturated fatty acids in 12 obese women, documented a mean weight loss of 17 kg over a 3-5 month period in subjects consuming a very-low-calorie diet. Weight loss with an accompanying decrease in specific body composition parameters (WHR and BMI) has also been documented in other studies (54, 147, 148, 149).

Postmenopausal women with abdominal obesity are normally considered at increased risk for the development of cardiovascular disease. Since hyperlipidemia in the abdominally obese has been confirmed by other researchers (11-12, 35-41), blood lipid tests were utilized in the present study to assess serum concentrations. However unlike the previously mentioned studies, subjects participating in this dietary study had considerably healthy blood lipid levels when taking into account their excess weight and high body mass index.

When considering their age and gender, the majority of subjects in this study population were within normal range (140-240 mg/dl) for total cholesterol. Before the study began, 11 subjects presented with desirable cholesterol

levels of less than 200 mg/dL; 11 subjects presented with borderline high-risk levels of 200-239 mg/dL; and 11 subjects presented with high-risk levels of greater than 240 mg/dL. At the end of the six-week period, 16 subjects presented with levels less than 200 mg/dL; 9 subjects presented with levels of 200-239 mg/dL; and 8 subjects presented with levels greater than 240 mg/dL.

According to the original data, subjects consuming the low-fat diet experienced a significant ($p < .01$) reduction in total serum cholesterol at the end of the sixth week as compared to subjects consuming no specific diet and the normal-fat diet. These results were unexpected since studies previously cited have shown that a reduction of total dietary fat does not lead to any significant fall in total serum cholesterol. However when taking weight loss into consideration, it was surprising that only the experimental group consuming the low-fat diet encountered a decrease in total serum cholesterol. Similar weight loss studies (150-152) have demonstrated that weight loss favorably changes lipid concentrations.

Even though both experimental groups experienced significant ($p < 0.01$) weight loss during the six week study, only the group consuming the low-fat diet demonstrated a decrease in total serum cholesterol.

Consequently, the group consuming the normal-fat diet experienced a slight increase in serum cholesterol levels.

Conflicting evidence exists between the association of weight loss and changes on serum lipid concentrations. Studies previously cited have shown that weight reduction favorably changes lipid concentations. However, other studies (153-155) have found either no improvement or deterioration in concentrations. According to Weinsier et al. (145), failure to obtain a consistent pattern of responses to weight reduction could be due to the lack of control of variables that may have an impact on lipid concentrations during weight reduction. These variables would include such areas as energy restriction, changes in diet composition, exercise, and biologic variability of the study subjects. Due to the fact that subjects participating in this study were a free-living population, variables such as those listed by Weinsier (145) could, in part, be responsible for the inconsistent patterns of weight loss on blood lipid levels.

However, when the original data were redistributed into either the post-hoc control diet group or the reduced-fat diet group, only the reduced-fat diet group experienced a significant ($p < 0.01$) weight loss and a significant ($p < 0.01$) decrease in total serum cholesterol. This finding corroborates those of Sedgwick et al. (150), Valenta and

Elias (151), and Stevenson et al. (152) who reported favorable changes in lipid concentrations with the occurrence of weight loss.

A trend in decreasing serum triglyceride levels was noted at the end of the six-week period in the group consuming the normal-fat diet. However, a trend in increasing serum triglyceride levels was beginning to occur at the end of the six-week period in both the control group and the group consuming the low-fat diet. The increase in serum triglyceride concentration in the control group was an unexpected occurrence due to the fact that concentrations in this group, which had no dietary or behavior modification intervention, were expected to remain relatively constant. However, dietary analyses for this particular group revealed an increase in both caloric intake and carbohydrate intake over the six-week period which could, in part, explain the unexpected increase. However, serum triglyceride results of the low-fat diet group in this particular study were not unexpected due to the fact that similar studies (122-123, 126) have reported the same results.

Brussard et al. (122-123) conducted a series of controlled-diet studies with high-, moderate-, and low-fat diets of conventional foods. Results indicated that the influence of a low-fat, high-carbohydrate diet on serum lipoproteins such as triglycerides was less favorable than

that of moderate- or high-fat diets rich in polyunsaturated fatty acids. Subjects participating in the low-fat diet group in this study experienced similar increases in serum triglyceride concentrations at the completion of the study.

Jones et al. (126) conducted a study in which 31 premenopausal women were randomized into one of two diet groups: one diet with a P:S ratio of 1.0 and one diet with a P:S ratio of 0.3. Both groups were fed a high-fat diet (40% of energy from fat) for four menstrual cycles followed by a low-fat diet (20% of energy from fat) for the same time interval. Plasma triglycerides increased in both groups on the low-fat diet.

Elevated triglyceride levels typically observed in the abdominally obese represent an indirect risk for CVD as they reflect disturbances in plasma lipid transport such as a reduced catabolism of triglyceride rich lipoproteins and a greater lipid exchange among lipoproteins leading to triglyceride enrichment of LDL and HDL. This condition is associated with higher concentrations of LDL-cholesterol and low HDL-cholesterol levels (7, 63, 92).

Studies by Hollenbeck et al. (156-157) demonstrated that the isocaloric substitution of carbohydrate for fat in the diets of individuals with normal carbohydrate and lipid metabolism resulted in significant increase in total fasting plasma triglyceride and a significant reduction in HDL-

cholesterol. There was also not an improvement in total plasma cholesterol concentrations. Postprandial triglyceride concentrations were significantly elevated after the low-fat diet. Hollenbeck and Coulston (156) also reported a significant increase in fasting triglyceride concentrations in a group on nondiabetic individuals with endogenous hypertriglyceridemia when carbohydrate was substituted for fat in the diet. Total cholesterol did not significantly decrease. Grundy (158) and Grundy et al. (159) reported similar findings when complex carbohydrate or monounsaturated fat in the diets of individuals with normal lipoprotein and carbohydrate metabolism were studied. In their original study (158), significant increases in total plasma cholesterol and triglyceride concentrations were documented after a diet of complex carbohydrate vs. saturated fat was fed. HDL-cholesterol was significantly decreased. In a second study (159) a similar population experienced decreased HDL-cholesterol without any significant change in total cholesterol.

Our results of HDL are in agreement with Hollenbeck et al. (156-157), Grundy (158), and Grundy et al. (159) who reported decreased levels of HDL-cholesterol when dietary fat was restricted and replaced with carbohydrate. In our particular dietary study, HDL-cholesterol decreased in both the control group and the group consuming the low-fat diet.

HDL-cholesterol increased in the group consuming the normal-fat diet. However, none of these changes was statistically significant. HDL-cholesterol appears to be affected by the typical 50-60% of carbohydrate recommended for general dietary intake. Our study confirms this statement. The normal-fat diet group, which consumed dietary intakes containing 49% carbohydrate, experienced a trend toward increasing HDL-cholesterol levels. However the low-fat diet group, which consumed dietary intakes containing 52% carbohydrate, experienced a trend of decreasing HDL-cholesterol concentrations. This raises a concern since the new Food Pyramid (USDA) encourages a generous 60% of dietary intake to come from complex carbohydrate sources. This may not be a realistic or safe recommendation for the postmenopausal women or our aging population (48).

The effect of dietary fat on HDL-cholesterol has been intensively studied, primarily due to the empirical association between low HDL-cholesterol concentrations and the increased risk for coronary heart disease. In populations with low intakes of fat, plasma HDL concentrations are low, whereas in societies where intake of saturated fat is high, HDL concentrations tend to be high (160). Soler et al. (26, 161) examined the metabolic characteristics of postmenopausal women and their risk for various diseases. Abdominal adiposity was negatively and

significantly correlated with plasma HDL. Depres at al. (66, 162) noted a relationship between android obesity, HDL-lipoprotein cholesterol, serum triglycerides and obesity. This study included male subjects who were assessed by anthropometric measurements which compared skinfold of the trunk to the skinfold of the extremeties (T/E). BMI was also determined. Abdominal skinfold to the T/E ratio was significantly associated with serum lipids and high density lipoprotein cholesterol (HDL-cholesterol). Subcutaneous fat was also related to serum triglycerides, total cholesterol and HDL-cholesterol. Both distribution and amount of subcutaneous fat were concluded to be important predictors of serum lipid and HDL-cholesterol. The association between abdominal fat and serum HDL-cholesterol was independent of total adiposity.

Doshi et al. (137) tested a multidisciplinary nutrition and fitness program for its effectiveness in lowering lipid profiles of elderly clients through dietary modification and exercise. In the program, which served 31 free-living, predominantly female, black elderly aged 56-88 years, dietary anthropometric, biochemical and fitness assessments were performed before and after a 10-week, biweekly program. Significant decreases ($p < 0.05$) were seen in waist circumference, total cholesterol, low-density lipoprotein, and the total cholesterol/HDL-cholesterol ratio.

The effects of abdominal obesity in 20 obese and 20 lean postmenopausal women were examined by Landin et al. (91). Plasma lipid concentrations were measured for each subject. Investigators concluded that obesity was more closely associated with the metabolic abnormalities and that WHR enhanced the effects of obesity on metabolism. Obese women with abdominal adiposity had significantly higher triglycerides and cholesterol when compared to their lean counterparts.

Problems exist for the postmenopausal woman having hypertriglyceridemia regardless of abdominal adiposity. Zamboni et al. (95) confirmed evidence of delayed plasma clearance of intestinal lipoproteins, especially triglycerides, by isotope-labeled triglycerides. This appears to be more of problem in women than men. Reduced removal of triglyceride may actually be the problem. Lipoprotein lipase (LPL), the enzyme responsible for the removal of triglyceride, decreases with the aging process which, in turn, prolongs lipemia following a meal. Therefore, if the LPL receptor declines with age, then the clearance of VLDL, which are derived from the endogenous synthesis of triglyceride, will be impaired (163).

Kirscht and Rosenstock (164) have found patients' reported compliance with weight loss programs to be associated with weight loss. Dietary compliance in other

studies previously cited is considered relatively high. Dietary compliance by subjects participating in this study was also extremely high with a majority of the subjects decreasing their normal caloric intake to a minimum of no less than 1200 kcals/day. However because a small portion of subjects in the normal-fat diet group had an increase in caloric consumption over the six-week period, the data from the three original dietary groups were reorganized. The reorganized data were based upon dietary intake criteria and consisted of two post-hoc groups: a post-hoc control diet group and a reduced-fat diet group.

Although the present study did not measure reasons for the increased caloric consumption by the normal-fat diet group, several factors should be considered. First, an extreme caloric restriction for women of this stature may be unrealistic. Women in this particular study group were above normal range for both weight and body mass index. A diet plan with limited kcalories may not have allowed a sufficient amount of energy to sustain these women throughout normal, daily activity. This may have been one particular factor that caused subjects in the normal-fat diet group to increase their caloric consumption. Subjects in the low-fat diet group may have had more of a success in caloric reduction due to the fact that their particular diet

plan was extremely different from their normal, usual intakes and because of this, they had to accomplish more of a behavior modification change than did the women in the normal-fat diet group. Secondly, an increase in caloric consumption by the normal-fat diet group could have been the result of pressure from other family members to return to "normal" familial eating habits. Perhaps the normal-fat diet was perceived by the subject as similar to their regular dietary plan with few modifications being needed. Because of the limited number of modifications, subjects could have possibly returned to their normal eating habits without being fully aware of their actions. This could have resulted in the calorie increase experienced by this particular group.

Finally, self-reported dietary intake data were used as the basis for determination of compliance for each group of subjects. Because of this, the researcher relied solely and primarily on the information given by each participating subject. Variations in dietary intakes could have been the result of inaccurate dietary record information reported by the subjects.

However due to the caloric increase by the normal-fat diet group that was believed to have occurred, the data were re-evaluated and divided into two groups: a post-hoc control diet group and a reduced-fat diet group, based upon dietary

intake. Once the data had been reorganized, subjects in the reduced-fat diet group (n = 13) experienced significant reductions in body weight, WHR, and BMI. Subjects in the post-hoc control diet group (n = 20) only experienced a significant decrease in WHR. Subjects in the reduced-fat diet group demonstrated a significant decrease only in serum cholesterol. No other significant changes in serum lipid concentrations were observed.

Because there is evidence that low-fat, high-carbohydrate diets may contribute to an increase in some lipoproteins and other abberations of the abdominally obese, the Dietary Guidelines and the recent Food Pyramid (USDA) nay need to be reassessed as far as including general recommendations for this subsample of the population (48).

In conclusion, the caloric restriction most likely contributed to the overall decreases in the lipid parameters that were affected. Anthropometric changes typically decrease in weight reduction studies over a six-week period so these changes were not surprising. The blood lipid effects may have been more evident if the subjects had been placed in a metabolic ward setting for more consistent control over food intake. However, our interest was to study the effects of the diets in a free living sample. Promotion of a 30%-fat diet for decreasing serum cholesterol levels may need to be evaluated due to the fact that only a lower-

fat diet (such as a 20%-fat diet) caused significant decreases in blood cholesterol levels. Low kilocalorie diets may need to be re-evaluated for the obese individual due to the fact that extremely low kilocalorie diets, regardless of their nutritional adequacy, may not meet the energy levels that are needed for extremely large individuals. Techniques and suggestions for the improvement of compliance to basic dietary modifications may need to be developed in order to help individuals successfully attain healthy anthropometric measurements and blood lipid parameters. Failure to note a significant difference in all metabolic parameters may be a result of a small sample size (165).

# CHAPTER VI

## SUMMARY & CONCLUSIONS

The overall objective of this study was to determine if a low-fat, reduced kcalorie diet would improve the lipid profile in abdominally obese postmenopausal women when compared to no specific diet and a normal-fat diet. Thirty-three subjects completed the full six week protocol. Subjects were monitored at week 3 and week 6 for dietary compliance. A group behavior modification and nutritional counseling program was also provided. The diets began after week 0 fasting biochemical parameters were taken. Subjects were randomly assigned to one of three treatment groups: no specific diet; a 1200-kilocalorie normal-fat (30% fat) diet; and 1200-kilocalorie low-fat (20% fat) diet. Each subject served as her own control. Biochemical parameters measured in a fasting state included: serum total cholesterol, serum triglyceride and serum HDL-cholesterol. Anthropometric measurements taken at two assessment periods included: height, weight, waist circumference, hip circumference. BMI and WHR were calculated. Subjects were asked to complete a

weekly diet record. Three days were analyzed for all nutrients yet total calories, percentage of calories consumed as protein, fat and carbohydrate were reported.

Regardless of the experimental diet consumed, changes in body weight and modifications in body composition were evident in all subjects consuming either the normal-fat diet or the low-fat diet over the six week experimental period. Significant ($p < 0.01$) decreases were evident in both WHR and BMI over the six week period in all subjects in both experimental diet groups. No significant differences were observed in weight nor BMI for the group consuming no specific diet.

Total cholesterol was significantly reduced ($p < 0.01$) at the end of week 6 in subjects consuming the low-fat (20% fat) diet when compared to subjects consuming the normal-fat (30% fat) diet and no specific diet (control) which were not significant. In the group consuming the 30% fat diet, serum triglyceride levels decreased at the end of 6 weeks but not significantly. Triglyceride levels increased in both the control group and the group consuming the 20%, yet not significantly.

HDL-cholesterol levels in the group consuming the 30% fat diet increased over the six-week period, but not significantly. HDL-cholesterol levels decreased in both the control group and the group consuming the 20% fat diet.

Total caloric intake of the group consuming the 30% fat diet was 1746 kcal/day. The average intake of carbohydrate was 49%. The average intake of protein was 19%. The average intake of fat was 32%. Total caloric intake of the group consuming the 20% fat diet was 1465 kcal/day. The average intake of carbohydrate was 52%. The average intake of protein was 21%. The average intake of fat was 27%.

Due to the fact that the normal-fat diet group's caloric intake increased instead of decreased, data from this particular group was re-evaluated and recatagorized into either the post-hoc control group (more than 1500 kcal/day and 35% calories from fat) or the reduced-fat group (less than 1500 kcal/day and 35% calories from fat).

Statistical analysis of the reorganized data confirmed that significant ($p < 0.01$) decreases in body weight and BMI occurred in the reduced-fat diet group. A significant ($p < 0.01$) decrease in WHR occurred in both the normal-fat diet group and the reduced-fat diet group. The reduced-fat diet group was the only group to experience any significant ($p < 0.01$) decreases in blood lipid concentrations. Cholesterol levels were the only blood lipids levels resulting in any significant changes. Triglyceride and HDL-cholesterol concentrations showed no significant changes over the six-week period.

The original data confirmed that although the normal-fat diet group's caloric intake was increasing during the six-week period, weight, waist-to-hip ratio, and body-mass-index for this group decreased during this time frame. The decrease in the anthropometric measurements coinciding with an increase in calories could have resulted from the increased physical activity during the study from participants in this group. From the beginning of the behavior modification classes, exercise was strongly emphasized and participants were taught the benefits of increasing physical activity. Discussions in class confirmed that 100% of the participants in both experimental groups had increased their physical activity to levels far beyond those at the beginning of the study.

The null hypothesis that there would be no significant difference in blood cholesterol concentrations between subjects in the control group versus those on the normal-fat diet versus those on the low-fat diet is rejected. Subjects consuming the 20%, low-fat diet displayed decreases in total serum cholesterol significantly ($p < 0.01$) at week 6. The null hypothesis that there would be no difference in serum triglyceride concentrations between all subjects in various groups is accepted. Trends toward a decrease in triglycerides for subjects consuming the 30%, normal-fat diet were beginning to occur at the end of week 6.

Unfortunately, trends were beginning to show an increase at the end of the six-week period in both the control group and the group consuming the 20% fat diet. The null hypothesis that there would be no difference in HDL-cholesterol concentrations between all subjects in various groups is accepted. Although trends were resulting in an HDL-cholesterol level increase in the group consuming the 30% fat diet, the increase was not yet significant at the end of week 6.

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# APPENDIX A

# APPROVAL LETTER

TEXAS WOMAN'S
UNIVERSITY
DENTON / DALLAS / HOUSTON

HUMAN SUBJECTS REVIEW COMMITTEE

OFFICE OF
RESEARCH AND
GRANTS ADMINISTRATION
P.O. Box 22939
Denton, TX 76204-0939
Phone: 817/898-3375

January 19, 1994

Jamie Broussard
P.O. Box 75
Nederland, Tx 77627

Dear Jamie Broussard: Social Security #: 464-31-1700

Your study entitled "The Effect of Fat on Lipid Profiles in the Abdominally Obese Postmenopausal Woman" has been reviewed by a committee of the Human Subjects Review Committee and appears to meet our requirements in regard to protection of individuals' rights.

Be reminded that both the University and the Department of Health and Human Services (HHS) regulations typically require that agency approval letters and signatures indicating informed consent be obtained from all human subjects in your study. These are to be filed with the Human Subjects Review Committee. Any exception to this requirement is noted below. Furthermore, according to HHS regulations, another review by the Committee is required if your project changes.

Special provisions pertaining to your study are noted below:

___ The filing of signatures of subjects with the Human Subjects Review Committee is not required.

___ Other:

_X_ No special provisions apply.

Sincerely,

Chairman
Human Subjects Review Committee

cc: Graduate School
Dr. Betty Alford, Nutrition and Food Sciences
Dr. Dorice Czajka-Narins, Nutrition and Food Sciences

# APPENDIX B

# CONSENT FORMS

Informed Consent (Group 1)

TEXAS WOMAN'S UNIVERSITY
SUBJECT CONSENT TO PARTICIPATE IN RESEARCH

TITLE OF STUDY: THE EFFECT OF FAT ON LIPID PROFILES IN THE ABDOMINALLY OBESE POSTMENOPAUSAL WOMAN

| INVESTIGATORS: | OFFICE PHONE NUMBER: |
|---|---|
| 1. Dr. Betty Alford | 817-898-2636 |
| 2. Jamie Broussard | 409-722-7390 |
| 3. Office of Research & Grants | 817-898-3375 |

1. A. The study that will be conducted involves research in the area of nutrition.
   B. The study, which will be conducted in order to determine if fat in the diet affects the lipid profiles in the abdominally obese postmenopausal woman, will last six weeks.
   C. Once I have completed the health questionnaire and it has been reviewed by the investigator, I understand that the following measurements will be taken before the beginning of the study:
   Blood Pressure: Taken upright, seated position;
   Height: Measured by fastening a tape measure to the wall. Measured to the nearest centimeter in bare feet with back to the wall;
   Weight: Measured to the nearest 0.1 kg using a beam-balance scale;
   Waist-to-Hip Circumference: Measured using a flexible tape measure around the waist and hip area. Weights and measures taken in light street clothes and without shoes.
   I also understand that the study requires that I give 5 milliliters of blood which is equal to approximately two tablespoons. This blood will be used so that the researcher may measure my Serum Total Cholesterol and Triglycerides. I will then have the above measurements taken again in order to complete the study.

2. The potential risks that may occur to me as a result of this study are:
   A. Fainting during the blood draw
   B. Infection or bruising due to blood draw
   **NOTE**: The investigator will take extensive actions to insure the safety of each and every participant. The following actions will be taken:
   A. All blood samples will be drawn while in a seated position. A Registered Nurse will be available to assist in any medical emergency. After my blood has been drawn, if necessary, juice will be available for consumption.

B. Due to modern biochemical safety and sterilization, the chance of infection due to the blood draw is extremely small. A Registered Medical Technologist will be performing each procedure with sterile latex rubber gloves and sterile, disposable needles and syringes. Also, extensive caution will be taken on the part of the Registered Medical Technologist to prevent bruising.

3. I understand that there may be no direct benefits as a result from my participation in this study.

4. No alternative procedures available.

5. Infomration that I have given will be in complete and total confidentiality. The information that is obtained will be placed into a sealed file to which only the reseracher will have access. The data from this study will be stored only until the final documentation is completed and once this has occurred the data gathering material and codes will be destroyed.

6. I understand that no medical service or compensation is provided to subjects by the university as a result of injury from partcipation in research.

7. If any quesions about the research and/or the research subject's rights, or in the event of a research-related injury to myself, I understand that the Investigators listed above should be contacted.

8. An offer to answer all of my questions regaerding the study has been made and I have been given a copy of the dated and signed consent form. If alternative procedures are more advantageous to me, they have been explained. A description of the possible attendant discomfort and risks reasonable to expec have been discussed with me. I understand that I may terminate my participation in the study at any time.

* If you have any concerns about the way this research has been conducted, contact Texas Woman's University Office of Research and Grants Adminisration.

** It has been explained to me that because I may be following a specific diet I may choose to consult with my physician in oredr to get his/her approval.

_____ _____

Signature of Participant Date

Informed Consent (Group 2)

TEXAS WOMAN'S UNIVERSITY
SUBJECT CONSENT TO PARTICIPATE IN RESEARCH

TITLE OF STUDY: THE EFFECT OF FAT ON LIPID PROFILES IN THE ABDOMINALLY OBESE POSTMENOPAUSAL WOMAN

| INVESTIGATORS: | OFFICE PHONE NUMBER: |
|---|---|
| 1. Dr. Betty Alford | 817-898-2636 |
| 2. Jamie Broussard | 409-722-7390 |
| 3. Office of Research & Grants | 817-898-3375 |

1. A. The study that will be conducted involves research in the area of nutrition.
   B. The study, which will be conducted in order to determine if fat in the diet affects the lipid profiles in the abdominally obese postmenopausal woman, will last six weeks.
   C. Once I have completed the health questionnaire and it has been reviewed by the investigator, I understand that the following measurements will be taken before the beginning of the study:
   Blood Pressure: Taken upright, seated position;
   Height: Measured by fastening a tape measure to the wall. Measured to the nearest centimeter in bare feet with back to the wall;
   Weight: Measured to the nearest 0.1 kg using a beam-balance scale;
   Waist-to-Hip Circumference: Measured using a flexible tape measure around the waist and hip area. Weights and measures taken in light street clothes and without shoes.
   I also understand that the study requires that I give 5 milliliters of blood which is equal to approximately two tablespoons. This blood will be used so that the researcher may measure my Serum Total Cholesterol and Triglycerides. I will then have the above measurements taken again in order to complete the study.

2. The potential risks that may occur to me as a result of this study are:
   A. Fainting during the blood draw
   B. Infection or bruising due to blood draw
   **NOTE**: The investigator will take extensive actions to insure the safety of each and every participant. The following actions will be taken:
   A. I understand that I will be monitored on a weekly basis to determine if any intolerances or complications occur due to the precribed diet. If such circumstances arise, I will be immediately removed from the study and further medical attention will be sought.

B. All blood samples will be drawn while in a seated position. A Registered Nurse will be available to assist in any medical emergency. After my blood has been drawn, if necessary, juice will be available for consumption.
C. Due to modern biochemical safety and sterilization, the chance of infection due to the blood draw is extremely small. A Registered Medical Technologist will be performing each procedure with sterile latex rubber gloves and sterile, disposable needles and syringes. Also, extensive caution will be taken on the part of the Registered Medical Technologist to prevent bruising.

3. Benefits that may occur as a result of participation in this stauy are:
A. Weight loss
B. Improved blood lipid profile
C. Decreased blood pressure
D. Decrease risks related to heart disease

4. No alternative procedures available.

5. Information that I have given will be in complete and total confidentiality. The information that is obtained will be placed into a sealed file to which only the reseracher will have access. The data from this study will be stored only until the final documentation is completed and once this has occurred the data gathering material and codes will be destroyed.

6. I understand that no medical service or compensation is provided to subjects by the university as a result of injury from partcipation in research.

7. If any quesions about the research and/or the research subject's rights, or in the event of a research-related injury to myself, I understand that the Investigators listed above should be contacted.

8. An offer to answer all of my questions regaerding the study has been made and I have been given a copy of the dated and signed consent form. If alternative procedures are more advantageous to me, they have been explained. A description of the possible attendant discomfort and risks reasonable to expec have been discussed with me. I understand that I may terminate my participation in the study at any time.

* If you have any concerns about the way this research has been conducted, contact Texas Woman's University Office of Research and Grants Adminisration.

** It has been explained to me that because I may be following a specific diet I may choose to consult with my physician in order to get his/her approval.

_____ _____

Signature of Participant Date

Informed Consent (Group 3)

TEXAS WOMAN'S UNIVERSITY
SUBJECT CONSENT TO PARTICIPATE IN RESEARCH

TITLE OF STUDY: THE EFFECT OF FAT ON LIPID PROFILES IN THE ABDOMINALLY OBESE POSTMENOPAUSAL WOMAN

| INVESTIGATORS: | OFFICE PHONE NUMBER: |
|---|---|
| 1. Dr. Betty Alford | 817-898-2636 |
| 2. Jamie Broussard | 409-722-7390 |
| 3. Office of Research & Grants | 817-898-3375 |

1. A. The study that will be conducted involves research in the area of nutrition.
   B. The study, which will be conducted in order to determine if fat in the diet affects the lipid profiles in the abdominally obese postmenopausal woman, will last six weeks.
   C. Once I have completed the health questionnaire and it has been reviewed by the investigator, I understand that the following measurements will be taken before the beginning of the study:
   Blood Pressure: Taken upright, seated position;
   Height: Measured by fastening a tape measure to the wall. Measured to the nearest centimeter in bare feet with back to the wall;
   Weight: Measured to the nearest 0.1 kg using a beam-balance scale;
   Waist-to-Hip Circumference: Measured using a flexible tape measure around the waist and hip area. Weights and measures taken in light street clothes and without shoes.
   I also understand that the study requires that I give 5 milliliters of blood which is equal to approximately two tablespoons. This blood will be used so that the researcher may measure my Serum Total Cholesterol and Triglycerides. I will then have the above measurements taken again in order to complete the study.

2. The potential risks that may occur to me as a result of this study are:
   A. Fainting during the blood draw
   B. Infection or bruising due to blood draw
   **NOTE**: The investigator will take extensive actions to insure the safety of each and every participant. The following actions will be taken:
   A. I understand that I will be monitored on a weekly basis to determine if any intolerances or complications occur due to the precribed diet. If such circumstances arise, I will be immediately removed from the study and further medical attention will be sought.

B. All blood samples will be drawn while in a seated position. A Registered Nurse will be available to assist in any medical emergency. After my blood has been drawn, if necessary, juice will be available for consumption.
C. Due to modern biochemical safety and sterilization, the chance of infection due to the blood draw is extremely small. A Registered Medical Technologist will be performing each procedure with sterile latex rubber gloves and sterile, disposable needles and syringes. Also, extensive caution will be taken on the part of the Registered Medical Technologist to prevent bruising.

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A. Weight loss
B. Improved blood lipid profile
C. Decreased blood pressure
D. Decrease risks related to heart disease

4. No alternative procedures available.

5. Information that I have given will be in complete and total confidentiality. The information that is obtained will be placed into a sealed file to which only the reseracher will have access. The data from this study will be stored only until the final documentation is completed and once this has occurred the data gathering material and codes will be destroyed.

6. I understand that no medical service or compensation is provided to subjects by the university as a result of injury from partcipation in research.

7. If any quesions about the research and/or the research subject's rights, or in the event of a research-related injury to myself, I understand that the Investigators listed above should be contacted.

8. An offer to answer all of my questions regaerding the study has been made and I have been given a copy of the dated and signed consent form. If alternative procedures are more advantageous to me, they have been explained. A description of the possible attendant discomfort and risks reasonable to expec have been discussed with me. I understand that I may terminate my participation in the study at any time.

* If you have any concerns about the way this research has been conducted, contact Texas Woman's University Office of Research and Grants Adminisration.

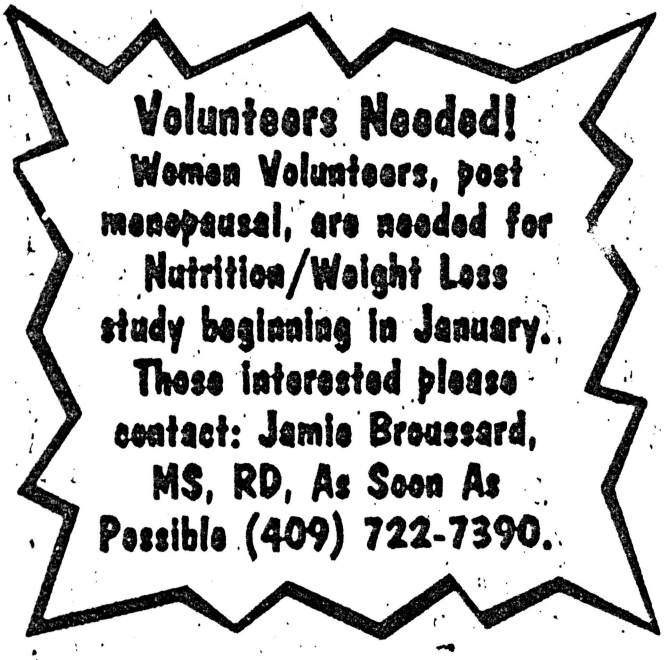
** It has been explained to me that because I may be following a specific diet I may choose to consult with my physician in order to get his/her approval.

_____ _____

Signature of Participant Date

# APPENDIX C

# RECRUITMENT ADVERTISEMENT


Volunteers Needed!
Women Volunteers, post menopausal, are needed for Nutrition/Weight Loss study beginning in January. Those interested please contact: Jamie Broussard, MS, RD, As Soon As Possible (409) 722-7390.

# APPENDIX D

# HEALTH QUESTIONNAIRE

HEALTH QUESTIONNAIRE

All information is private and confidential.

PLEASE PRINT

GENERAL INFORMATION

NAME: _____
(Last) (First) (Middle)

ADDRESS: _____ _____
(Number and Street) (City and State)

_____ _____ (  ) _____
(County) (Zip Code) (Home Phone)

AGE: _____

MARITAL STATUS:
1. Single _____
2. Married _____
3. Divorced _____
4. Widowed _____
5. Separated _____

RACE/ETHNIC ORIGIN:
1. Caucasian _____
2. African-American_____
3. Mexican-American_____
4. Oriental_____
5. Other _____

# MEDICAL HISTORY

Please place a check mark ( ) if answer is "YES"
Have you had or do you have any of the following:

_____ 1. High Blood Pressure

_____ 2. Heart Disease

_____ 3. Elevated Cholesterol

_____ 4. Elevated Triglycerides

_____ 5. Diabetes

_____ 6. Liver Disease

_____ 7. Kidney Disease

_____ 8. Ulcer or Gastrointestinal Problems

___ 9. Cancer (type:_____)

___ 10. Gall Bladder Disease

___ 11. Do you have any other medical problems not previously mentioned? Please explain: _____
_____

12. What prescribed or self-prescribed medications are you taking now?

| MEDICATIONS | DOSAGE | FREQUENCY/DAY |
|---|---|---|
| _____ | _____ | _____ |
| _____ | _____ | _____ |
| _____ | _____ | _____ |
| _____ | _____ | _____ |

13. In what year did you complete menopause? _____

## FAMILY MEDICAL HISTORY

FATHER: Alive _____ Deceased _____
Age Now____ Age at Death____
Cause of Death _____

MOTHER: Alive _____ Deceased _____
Age Now____ Age at Death____
Cause of Death _____

BROTHERS and SISTERS:

| AGE | SEX | HEALTH PROBLEM(S) | IF DECEASED, CAUSE OF DEATH |
|---|---|---|---|
| _____ | _____ | _____ | _____ |
| _____ | _____ | _____ | _____ |
| _____ | _____ | _____ | _____ |
| _____ | _____ | _____ | _____ |

FAMILIAL DISEASES: Have your parents, grandparents, sisters or brothers, aunts or uncles, or your children developed any of the following? Exclude cousins, relatives by marriage or adoption, and half relatives.

Please place a check mark ( ) if "YES" and write on corresponding lines.

| | | FAMILY RELATION | AGE OF ONSET |
|---|---|---|---|
| _____ | Heart attacks under age 50 | _____ | _____ |
| _____ | Heart attacks between 50 & 70 | _____ | _____ |
| _____ | Heart attacks at 70 or over | _____ | _____ |
| _____ | Congenital heart disease (heart defects present at birth) | _____ | _____ |
| _____ | Heart surgery or coronary bypass surgery under 50 | _____ | _____ |
| _____ | Heart surgery or coronary bypass surgery between 50 & 70 | _____ | _____ |
| _____ | Heart surgery or coronary bypass surgery over 70 | _____ | _____ |
| _____ | Strokes under age 50 | _____ | _____ |
| _____ | Strokes 50 or over | _____ | _____ |
| _____ | High blood pressure | _____ | _____ |
| _____ | Elevated cholesterol | _____ | _____ |
| _____ | Diabetes | _____ | _____ |
| _____ | Obesity (20 or more lbs. overweight) | _____ | _____ |

## CORONARY RISK FACTORS

SMOKING

1. Do you currently smoke?
   Yes _____ (If yes, how many cigarettes per day? _____
   When did you start? Year 19 ___

   No _____ (If no, go to question 2).

2. Have you smoked in the past but do not smoke now?
   Yes _____ (If yes, how many cigarettes per day? _____
   When did you start? Year 19 __
   When did you stop? Year 19 __

WEIGHT RECORD:

1. What do you consider a good weight for yourself?
   _____ lbs.

2. Present weight: _____lbs.

   Maximum weight: _____lbs. (age _____)

3. Has your weight recently changed?
   _____ No
   _____ Gained
   _____ Lost

   How much? _____
   Over what period of time? _____

GENERAL EATING PRACTICES:

1. Is your appetite generally:
   _____ Poor
   _____ Good
   _____ Excellent

2. List any food allergies or intolerances: _____
   _____

3. Describe a specific diet or dietary restrictions (if any)
   you follow: _____
   _____

4. List any vitamin/mineral/dietary supplements (& amounts) you use. _____
_____

5. How many meals do you eat daily? _____

6. If you omit a meal, please specify which one _____ and why: _____

7. How many times do you snack daily?
_____ 0
_____ 1-2
_____ 3-5
_____ over 5

8. How many meals on the average do you eat out weekly?_____

9. Where do you eat out most frequently?
_____ Restaurants
_____ Cafeteria
_____ Fast Food Places
_____ Other (Specify:_____)

10. Is your eating pace generally:
_____ Fast
_____ Slow
_____ Moderate

EXERCISE:

Do you regularly engage in any of the following activites?
Please place a check mark ( )if "YES"
_____ Walk, run, jog
_____ Bicycling
_____ Swimming
_____ Golf
_____ Aerobics, calisthenics or weight lifting
_____ Tennis
_____ Handball, raquetball, etc

How often do you execise per week? _____

Is your exercise program consistent from month to month?
_____ Yes
_____ No

Are you satisfied with your current exercise program?
_____ Yes
_____ No If no, state what type of changes you would like to make: _____

# APPENDIX E

# LESSON PLANS

## LESSON 1

### COURSE INTRODUCTION

1. Greet class participants and provide telephone number.

2. Course description
   * Six weekly classes
   * Explanation of class schedule (meeting time)

3. Expectations of Participants
   Nutrition and Exercise Assessments of Participants

### LECTURE

1. Introduction to Behavior Modification and Weight Control

2. Introduction to Record Keeping and Portion Sizes
   Detailed Explanation of Keeping Adequate Food Diaries

3. Presentation of Diet Plan and Exchange Lists

### CLASS ACTIVITY

* Food Record on Overhead with a Detailed Explanation of how to label and categorize all entries.

## LESSON 2

### QUESTIONS

1. Questions dealing with previous week's contents

2. Questions dealing with Food Records or Exchanges

### LECTURE

1. General Nutrition Introduction

   This section based on the Food Guide Pyramid

   Participants will be shown a poster of Food Guide Pyramid

   and lesson will be designed around educating them on the basics of this tool

2. Reading Labels: Understanding the Basics

3. Fad Diets vs. Balanced Diets

4. Exercise Introduction

### CLASS ACTIVITY

* Participants will examine grocery labels and make comments on the different ingredients on the labels.

### CLASS ASSIGNMENT

* Participants will begin some type of exercise program

## LESSON 3

**QUESTIONS**

1. Questions dealing with previous week's contents

2. Questions concerning new exercise program.

**LECTURE**

1. "Fat is Where It's At": A detailed and focused look at fat in the diet.

2. Calculating Percent of Fat in Food and in the Diet

3. Sources of Fat

4. Healthy Substitutions

5. Cholesterol

**CLASS ACTIVITY**

* Participants will take labels they have brought from home and calculate percent of calories from fat using these labels

* Participants will conduct class discussion dealing with ways to make healthy substitutions

## LESSON 4

QUESTIONS

1. Questions dealing with previous week's contents

2. Questions concerning class activities (labels and substitutions).

LECTURE

1. Exercise: Making It Fun for Fitness

2. 25 Reasons to Exercise

3. Aerobic vs. Anaerobic Exercise

4. Selecting an Aerobic Activity

5. Determining Safe and Healthy Heart Rates

CLASS ACTIVITY

* Participants will calculate their safe heart rate

## LESSON 5

**QUESTIONS**

1. Questions dealing with previous week's contents

**LECTURE**

1. A Focus on Behavior Modification
   a. Self-Monitoring
   b. Portion Control
   c. Eating Situations
   d. Plate Size

2. Techniques and Hints for Eating Out

3. Packing a Nutritious "Brown Bag" Lunch

4. Eating on the Run

**CLASS ACTIVITY**

* Particpants will bring menus from their favorite restaurants and practice ordering the "low-fat" way.

LESSON 6

QUESTIONS
1. Questions dealing with previous week's contents

LECTURE
1. Program Review

2. Pulling It All Together: A Look Back at the High Points of the Program

3. Backsliding: How to Successfully Deal With a Crisis

4. Tips for a Successful Weight Loss Future

CLASS ACTIVITY
* Participants will complete a class evaluation form

# APPENDIX F

# DIET EXCHANGE FOODS

# DIET EXCHANGE FOODS

## STARCHY VEGETABLES
(1/2 cup)
Beans
Peas
Corn
Potatoes
Soups (1 cup)

Recommended: Baked, broiled, roasted potatoes and corn. Beans prepared without meat fat, soups with clear broth base.
Not Recommended: Fried or creamed or "au gratin" style potatoes, "chunky" or stew-like soups, beans seasoned with bacon or pork.

## BREADS
Bread (1 slice)
Bun (1/2)
Roll (1 small)
Tortilla (1 6")
Muffin (1 small)
Pancake (2)
Taco Shell (2)
Waffle (1)

## CEREALS
Bran Cereal (1/3-1/2 cup)
Puffed Cereal (11/2 cup)
Cooked Cereal (1/2 cup cooked)
Grits (1/2 cup cooked)

## GRAINS & PASTAS
Rice (1/2 cup cooked)
Pasta (1/2 cup cooked)
Egg Noodles (1/2 cup)

## CRACKERS & SNACKS
Animal crackers (8)
Graham Crackers (2 squares)
Popcorn (3 cups)
Pretzels (3/4 oz)
Saltines (6)

Recommended: Any plain bread, cracker, or cereal, especially those made with whole grains or added bran.
Not Recommended: "Buttered" rolls or breads, sweet rolls and pastries, doughnuts, croissants.

## LOWFAT DAIRY
Skim or 1/2% Milk (1 cup)
2% Lowfat Milk (3/4 cup)
Lowfat Buttermilk (1 cup)
Lowfat Yogurt (1/2 cup)
Lowfat Cottage Cheese (1 1/2 cup)
Lowfat Cheese (3/4 oz)

## VEGETABLES
Basically all vegetables (See Note)

Recommended: Raw or cooked (steamed, boiled, sauteed or stewed) Alone or added to salad and main dishes.
Not Recommended: Breaded, fried vegetables, such as onion rings, fried mushrooms, etc.
Vegetables seasoned with bacon fat or salt pork or vegetables in cream or cheese sauces.

FRUITS

Basically all fruits (See Note)

Recommended: Fresh, frozen, dried or canned in water or juice.
Not Recommended: Fruits packed in syrup or sweetened.
Fruit "drinks" or punch which contain sweeteners.

MEAT, FISH, POULTRY AND PROTEIN

(1 oz of the following)
Beef (lean)
Lamb (lean)
Pork (lean)
Poultry
Fish (fresh frozen)
Canned Tuna or Salmon (1/4 cup)
Sardines (3 whole)
Shellfish (2 oz)
Wild Game (1 oz cooked)
Luncheon Meats (1 oz)
Eggs (1 large)
Beans (3 oz)

Recommended: Broiled, baked, steamed, charcoal grilled, or sauteed meats.
Not Recommended: Breaded, fried,or "stuffed" dishes.
Heavily suaced dishes, or fatty cuts of meat.
Avoid most sausages and cold cuts.

FATS

(1 teaspoon of the following)
Oil (corn, safflower, olive, etc)
Margarine
Mayonaisse
Salad Dressing
Olives (10 small)
Bacon (1 slice, crisp)
Nuts (portion sizes vary)

# APPENDIX G

# FOOD DIARY

FOOD DIARY

WEEK _____

DAY _____

| TIME | FOOD EATEN | AMOUNT | HOW PREPARED |
|---|---|---|---|
| | | | |

Remember: Did you add salt, sugar, sauces?
Record exact amount (ex: 1 oz)

# APPENDIX H

# SAMPLE MENUS

NUTRITION STUDY DIET

DIET A
EX1 - 30% FAT

Your recommended daily intake is as follows:

6 servings of meat
2 servings of dairy
3 servings of bread,cereal or starchy vegetable
2 servings of fruit
4 servings of fat
2 servings of non-starchy vegetable

A sample 1200 calorie daily meal plan would be as follows:

**BREAKFAST:**

| | |
|---|---|
| 1 | servings of fruit |
| 1 | servings of bread, cereal |
| 1 | servings of lowfat dairy products |
| 1 | servings of fat |

**A.M. SNACK:**

1 serving of non-starchy vegetable

**LUNCH:**

| | |
|---|---|
| 2 | ounces of meat |
| 1 | servings of bread, cereal or starchy vegetable |
| | servings of non-starchy vegetable |
| | servings of fruit |
| | servings of low-fat dairy products |
| 1 | servings of fat |

**P.M. SNACK:**

1 piece of fresh fruit

**DINNER:**

| | |
|---|---|
| 4 | ounces of meat |
| 1 | serving of bread, cereal or starchy vegetable |
| 1 | servings of non-starchy vegetable |
| | servings of fruit |
| | servings of low-fat dairy products |
| 2 | servings of fat |

**AFTER-DINNER SNACK:**

1 serving of low-fat dairy (example: milk or yogurt)

REMEMBER: No more than two eggs per week.

NUTRITION STUDY DIET

DIET B
EX2 - 20% FAT

Your recommended daily intake is as follows:

6 servings of meat
2 servings of dairy
4 servings of bread, cereal or starchy vegetable
2 servings of fruit
2 servings of fat
2 servings of non-starchy vegetable

A sample 1200 calorie daily meal plan would be as follows:

**BREAKFAST:**

| | |
|---|---|
| 1 | servings of fruit |
| 1 | servings of bread, cereal |
| 1 | servings of lowfat dairy products |
| 1 | servings of fat |

**A.M. SNACK:**

5-6 crackers, with jam or jelly

**LUNCH:**

| | |
|---|---|
| 2 | ounces of meat |
| 1 | servings of bread, cereal or starchy vegetable |
| 1 | servings of non-starchy vegetable |
| | servings of fruit |
| | servings of low-fat dairy products |
| | servings of fat |

**P.M. SNACK:**

1 piece of fresh fruit

**DINNER:**

| | |
|---|---|
| 4 | ounces of meat |
| 1 | serving of bread, cereal or starchy vegetable |
| 1 | servings of non-starchy vegetable |
| | servings of fruit |
| | servings of low-fat dairy products |
| 1 | servings of fat |

**AFTER-DINNER SNACK:**

1 serving of low-fat dairy (example: milk or yogurt)

REMEMBER: No more than two eggs per week.

# APPENDIX I

# SUMMARIES AND PRINCIPLES OF BIOCHEMICAL MEASUREMENTS

## SUMMARY AND PRINCIPLE OF THE ENZYMATIC DETERMINATION OF CHOLESTEROL

The KODAK EKTACHEM Clinical Chemistry Slide is a dry, muiltilayered analytic element coated on a clear polyester support. The method is based on a modification of the method of Allain et al. (  ). A 10 uL drop of sample is deposited on the slide and the isotropically porous speading layer evenly distributes the sample. The surfactant in the spreading layer aids in dissociating the cholesterol and cholesterol esters from lipoprotein complexes present in the sample. Hydrolysis of the cholesterol ester hydrolase and the cholesterol is then oxidized in the presence of cholesterol oxidase to form cholestenone and hydrogen peroxide. Hydrogen peroxide oxidizes a triarylimidazole leuco dye in the presence of peroxidase to generate a colored dye. The density of the dye formed is proportional to the cholesterol concentration present in the sample and is measured by reflectance spectrophotometry.

The following is the slide ingredients:
Cholesterol oxidase
Cholesterol ester hydrolase
Peroxidase
2-(3,5-Dimethoxy-4-hydroxyphenol)-
4,5-bis(4-dimethyaminophenyl)imidazole

## SUMMARY AND PRINCIPLE OF THE ENZYMATIC DETERMINATION OF TRIGLYCERIDE

The KODAK EKTACHEM Clinical Chemistry Slide is a dry, muiltilayered analytic element coated on a clear polyester support. The analysis is based on a modification of the method by Spayd et al. (  ). A 10 uL drop of sample is deposited on the slide and is evely distributed by the spreading layer. The surfactant in the spreading layer aids in dissociating the triglycerides from lipoprotein complexes present in the sample. The triglyceride molecules are then hydrolyzed by lipase to yield glycerol and fatty acids. Glycerol diffuses through the scavenger layer to the reagent layer, where it is phosphorylated by glycerol kinase in the presence of adenosine triphophate (ATP) and magnesium chlorise. In the presence of L-a-glycerol-phosphate oxidase, L-a-glycerophosphate is then oxidized to dihydroxyacetone phosphate and hydrogen peroxide. The final reaction involves the oxidation of triarylimidazole leuco dye to hydrogen peroxide, catalyzed by peroxidase, to produce a dye. The density of the dye formed is proportional to the triglyceride concentration present in the sample and is measured by reflectance spectrophotometry.

The following is the slide ingredients:
Lipase
Peroxidase
Ascorbate oxidase
Glycerol kinase
L-a-glycerophosphate oxidase
2-(3,5-Dimethoxy-4-hydroxyphenyl-
4,5-bis(4-dimethylaminophenyl)imidazole
Adenosine triphophosphate

SUMMARY OF HDL-CHOLESTEROL REAGENTS USED IN ANALYSIS

The Reagents required for the HDL-cholesterol determination include the following:

A. Dextran Sulfate Reagent. This reagent is stored at room temperature and is stable until expiration marked on the vials. The Dextran Sulfate Reagent contains the following active ingredient and the approximate concentration of the component:

| Reangent | Concentration |
|---|---|
| 500,000 MW dextran sulfate | 20 g/L |

B. Magnesium Sulfate Reagent. This reagent is stored at room temperature and stable until expiration marked on the vials. The Magnesium Sulfate Reagent contains the following active ingredient and the approximate concentration of the component:

| Reagent | Concentration |
|---|---|
| Magnesium sulfate | 1.1 mmol/L |

# APPENDIX J

## INDIVIDUAL SUBJECT DATA FOR BLOOD LIPID VALUES

CONTROL DIET GROUP
BLOOD LIPID LEVELS FOR WEEK 0 AND WEEK 6

| | WEEK 0 | | | | | WEEK 6 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| ID# | CHOL | TRIG | HDL-C | LDL-C | CHOL/HDL-C | CHOL | TRIG | HDL-C | LDL-C | CHOL/HDL-C |
| #10 | 158 | 73 | 55 | 88 | 2.9 | 152 | 89 | 58 | 76 | 2.6 |
| #12 | 259 | 210 | 41 | 176 | 6.3 | 224 | 158 | 47 | 145 | 4.8 |
| #14 | 299 | 221 | 47 | 208 | 6.4 | 256 | 226 | 55 | 156 | 4.7 |
| #15 | 240 | 221 | 39 | 157 | 6.2 | 226 | 272 | 36 | 136 | 6.3 |
| #22 | 316 | 223 | 43 | 229 | 7.3 | 289 | 234 | 43 | 199 | 6.7 |
| #26 | 262 | 131 | 51 | 185 | 5.1 | 249 | 95 | 49 | 181 | 5.1 |
| #27 | 162 | 114 | 33 | 106 | 4.9 | 142 | 135 | 35 | 80 | 4.1 |
| #30 | 207 | 184 | 70 | 98 | 3.0 | 223 | 215 | 72 | 108 | 3.1 |
| #32 | 157 | 75 | 59 | 83 | 2.7 | 129 | 87 | 36 | 76 | 3.6 |
| #33 | 203 | 113 | 76 | 104 | 2.7 | 191 | 127 | 75 | 91 | 2.5 |
| #36 | 204 | 113 | 73 | 105 | 2.8 | 238 | 165 | 49 | 156 | 4.9 |

CHOL = CHOLESTEROL (mg/dl)
TRIG = TRIGLYCERIDE (mg/dl)
HDL-C = HIGH-DENSITY-LIPOPROTEIN CHOLESTEROL (mg/dl)
LDL-C = LOW-DENSITY-LIPOPROTEIN CHOLESTEROL (mg/dl)
CHOL/HDL-C = CHOLESTEROL/HIGH-DENSITY-LIPOPROTEIN CHOLESTEROL

NORMAL-FAT DIET GROUP
BLOOD LIPID LEVELS FOR WEEK 0 AND WEEK 6

| | WEEK 0 | | | | | WEEK 6 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| ID# | CHOL | TRIG | HDL-C | LDL-C | CHOL/HDL-C | CHOL | TRIG | HDL-C | LDL-C | CHOL/HDL-C |
| #2 | 183 | 119 | 31 | 128 | 5.9 | 191 | 112 | 38 | 131 | 5.02 |
| #3 | 227 | 73 | 64 | 148 | 3.5 | 181 | 71 | 55 | 112 | 3.3 |
| #4 | 172 | 196 | 35 | 98 | 4.9 | 202 | 101 | 41 | 141 | 4.9 |
| #5 | 203 | 148 | 36 | 137 | 5.6 | 194 | 140 | 37 | 129 | 5.2 |
| #8 | 188 | 77 | 50 | 123 | 3.8 | 183 | 90 | 46 | 119 | 4.0 |
| #13 | 225 | 138 | 48 | 149 | 4.7 | 224 | 158 | 47 | 145 | 4.8 |
| #18 | 195 | 94 | 48 | 158 | 4.1 | 198 | 108 | 53 | 123 | 3.7 |
| #21 | 205 | 178 | 37 | 132 | 5.5 | 194 | 106 | 48 | 125 | 4.0 |
| #24 | 169 | 79 | 57 | 96 | 2.7 | 199 | 130 | 53 | 120 | 3.8 |
| #34 | 203 | 113 | 76 | 104 | 2.7 | 207 | 102 | 44 | 143 | 4.7 |
| #35 | 228 | 142 | 51 | 149 | 4.5 | 248 | 180 | 46 | 166 | 5.4 |

CHOL = CHOLESTEROL (mg/dl)
TRIG = TRIGLYCERIDE (mg/dl)
HDL-C = HIGH-DENSITY-LIPOPROTEIN CHOLESTEROL (mg/dl)
LDL-C = LOW-DENSITY-LIPOPROTEIN CHOLESTEROL (mg/dl)
CHOL/HDL-C = CHOLESTEROL/HIGH-DENSITY-LIPOPROTEIN CHOLESTEROL

LOW-FAT DIET GROUP
BLOOD LIPID LEVELS FOR WEEK 0 AND WEEK 6

| | WEEK 0 | | | | | WEEK 6 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| ID # | CHOL | TRIG | HDL-C | LDL-C | CHOL/HDL-C | CHOL | TRIG | HDL-C | LDL-C | CHOL/HDL-C |
| #1 | 271 | 253 | 38 | 182 | 7.1 | 257 | 262 | 42 | 163 | 6.1 |
| #6 | 284 | 136 | 72 | 185 | 3.9 | 269 | 141 | 71 | 170 | 3.8 |
| #7 | 160 | 59 | 50 | 98 | 3.2 | 145 | 81 | 45 | 84 | 3.2 |
| #9 | 158 | 73 | 55 | 88 | 2.9 | 152 | 89 | 58 | 76 | 2.6 |
| #17 | 278 | 341 | 38 | 172 | 7.3 | 248 | 292 | 47 | 143 | 5.3 |
| #19 | 245 | 76 | 55 | 175 | 4.5 | 235 | 90 | 58 | 159 | 4.1 |
| #20 | 273 | 258 | 38 | 183 | 7.2 | 259 | 264 | 42 | 164 | 6.2 |
| #23 | 256 | 138 | 122 | 106 | 2.1 | 183 | 122 | 65 | 94 | 2.8 |
| #25 | 158 | 67 | 57 | 88 | 2.8 | 147 | 85 | 51 | 79 | 2.9 |
| #28 | 212 | 118 | 52 | 136 | 4.1 | 207 | 170 | 47 | 126 | 4.4 |
| #31 | 221 | 100 | 59 | 142 | 3.7 | 186 | 98 | 49 | 117 | 3.8 |

CHOL = CHOLESTEROL (mg/dl)
TRIG = TROGLYCERIDE (mg/dl)
HDL-C = HIGH-DENSITY-LIPOPROTEIN CHOLESTEROL (mg/dl)
LDL-C = LOW-DENSITY-LIPOPROTEIN CHOLESTEROL (mg/dl)
CHOL/HDL-C = CHOLESTEROL/HIGH-DENSITY-LIPOPROTEIN CHOLESTEROL

# APPENDIX K

# INDIVIDUAL SUBJECT DATA FOR NUTRIENTS

CONTROL GROUP
INTAKE FOR WEEK 0 AND WEEK 6

| ID# | WEEK 0 | | | | | WEEK 6 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | kCAL | FAT | PRO | CHO | %FAT | kCAL | FAT | PRO | CHO | %FAT |
| #10 | 2119.22 | 111.26 | 58.60 | 235.98 | 47% | 1493.92 | 66.59 | 56.96 | 168.34 | 40% |
| #12 | 1951.06 | 68.99 | 68.64 | 266.82 | 32% | 1659.31 | 89.59 | 57.66 | 163.58 | 49% |
| #14 | 2013.46 | 78.23 | 85.00 | 167.70 | 35% | 1740.55 | 47.69 | 75.51 | 260.53 | 25% |
| #15 | 1550.23 | 72.99 | 91.13 | 145.77 | 42% | 1321.49 | 53.99 | 53.35 | 162.76 | 37% |
| #22 | 1047.83 | 37.49 | 32.74 | 150.56 | 32% | 2149.24 | 89.14 | 67.83 | 277.02 | 37% |
| #26 | 1923.10 | 92.20 | 75.74 | 204.18 | 43% | 2065.44 | 73.09 | 87.89 | 239.12 | 32% |
| #37 | 1967.16 | 87.49 | 74.87 | 210.29 | 40% | 1916.95 | 80.02 | 72.03 | 229.85 | 38% |
| #30 | 1610.48 | 27.82 | 69.86 | 281.70 | 15% | 2004.49 | 78.62 | 87.20 | 246.82 | 35% |
| #32 | 2434.67 | 121.06 | 78.04 | 268.40 | 5% | 2328.23 | 112.13 | 99.65 | 234.48 | 43% |
| #33 | 1628.87 | 58.57 | 82.57 | 195.30 | 32% | 1395.64 | 39.57 | 75.98 | 191.81 | 26% |
| #36 | 1270.50 | 45.81 | 61.66 | 142.84 | 32% | 3574.93 | 189.44 | 119.52 | 359.94 | 47% |

kCAL = kCALORIES
FAT = FAT (g)
PRO = PROTEIN (g)
CHO = CARBOHYDRATE (g)
% FAT = % FAT

NORMAL-FAT DIET GROUP

INTAKE FOR WEEK 0, 3 AND 6

| | WEEK 0 | | | | | WEEK 3 | | | | | WEEK 6 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | CAL | FAT | PRO | CHO | %FAT | CAL | FAT | PRO | CHO | %FAT | CAL | FAT | PRO | CHO | %FAT |
| #2 | 733.07 | 33.00 | 30.29 | 80.35 | 40% | 1396.39 | 43.27 | 76.02 | 181.74 | 27% | 1424.07 | 50.08 | 52.30 | 172.28 | 31% |
| #3 | 2381.21 | 109.45 | 130.24 | 220.22 | 41% | 1223.48 | 40.40 | 76.14 | 145.75 | 29% | 1615.15 | 42.43 | 84.13 | 240.74 | 24% |
| #4 | 1114.57 | 45.83 | 50.44 | 128.48 | 37% | 1508.67 | 40.16 | 81.99 | 219.99 | 23% | 2023.02 | 72.35 | 89.11 | 269.09 | 32% |
| #5 | 2538.03 | 128.00 | 98.53 | 255.11 | 45% | 1819.91 | 62.74 | 86.66 | 235.43 | 31% | 1639.23 | 53.96 | 88.57 | 209.80 | 30% |
| #8 | 1397.13 | 74.74 | 77.25 | 104.31 | 48% | 1747.62 | 44.30 | 81.47 | 267.93 | 22% | 1980.23 | 76.22 | 87.71 | 249.48 | 34% |
| #13 | 2325.88 | 100.42 | 88.54 | 274.39 | 38% | 1941.92 | 74.45 | 97.01 | 229.51 | 34% | 1273.21 | 58.38 | 86.08 | 254.94 | 40% |
| #18 | 2373.43 | 67.99 | 98.40 | 328.26 | 25% | 1721.08 | 48.75 | 77.95 | 253.11 | 25% | 2166.03 | 76.04 | 96.46 | 134.30 | 31% |
| #21 | 2000.20 | 78.33 | 78.86 | 254.20 | 35% | 1826.79 | 56.56 | 60.34 | 266.31 | 28% | 1655.92 | 52.36 | 87.68 | 216.90 | 28% |
| #24 | 1460.67 | 52.28 | 65.75 | 170.47 | 32% | 1454.57 | 35.96 | 89.20 | 208.45 | 22% | 2203.64 | 69.89 | 100.27 | 260.26 | 31% |
| #34 | 2586.60 | 126.52 | 86.62 | 277.53 | 43% | 1499.41 | 49.25 | 88.77 | 184.08 | 30% | 1927.18 | 59.62 | 92.82 | 269.82 | 28% |
| #35 | 1559.50 | 66.83 | 71.24 | 171.99 | 38% | 1356.92 | 28.71 | 84.51 | 203.14 | 19% | 1743.50 | 53.32 | 84.51 | 240.75 | 28% |

kCAL = kCALORIES
FAT = FAT (g)
PRO = PROTEIN (g)
CHO = CARBOHYDRATE (g)
% FAT = % FAT

LOW-FAT DIET GROUP
INTAKE FOR WEEK 0, 3 AND 6

| | WEEK 0 | | | | | WEEK 3 | | | | | WEEK 6 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | CAL | FAT | PRO | CHO | %FAT | CAL | FAT | PRO | CHO | %FAT | CAL | FAT | PRO | CHO | %FAT |
| #1 | 1090.4 | 57.92 | 46.50 | 77.35 | 48% | 1414.23 | 38.58 | 85.08 | 201.29 | 24% | 1379.5 | 44.19 | 82.74 | 170.19 | 28% |
| #6 | 1556.93 | 50.85 | 64.32 | 209.25 | 30% | 1278.84 | 30.63 | 71.16 | 190.09 | 22% | 1477.28 | 34.9 | 91.59 | 206.75 | 21% |
| #7 | 1664.81 | 55.27 | 63.20 | 221.17 | 30% | 1261.77 | 27.09 | 81.80 | 197.59 | 19% | 1191.79 | 26.07 | 77.84 | 173.83 | 20% |
| #9 | 1300.41 | 32.61 | 65.50 | 182.96 | 23% | 1038.76 | 22.51 | 58.62 | 168.88 | 20% | 1624.00 | 46.23 | 85.06 | 227.40 | 25% |
| #17 | 1699.11 | 65.35 | 85.47 | 198.31 | 35% | 1111.92 | 34.06 | 76.48 | 128.70 | 27% | 1414.85 | 29.34 | 80.46 | 216.83 | 19% |
| #19 | 1693.81 | 67.39 | 56.78 | 219.41 | 36% | 1391.19 | 33.28 | 85.21 | 192.30 | 21% | 1613.11 | 43.54 | 91.67 | 222.41 | 24% |
| #20 | 1871.95 | 67.81 | 68.42 | 189.29 | 33% | 1205.00 | 32.10 | 77.36 | 163.98 | 23% | 1631.86 | 47.73 | 82.85 | 231.41 | 26% |
| #23 | 1988.35 | 99.81 | 106.41 | 171.03 | 45% | 1252.63 | 37.54 | 76.81 | 158.99 | 26% | 1338.84 | 30.89 | 88.53 | 190.46 | 20% |
| #25 | 1823.2 | 74.14 | 110.46 | 182.41 | 37% | 1315.76 | 27.63 | 73.67 | 164.06 | 19% | 1484.81 | 33.95 | 83.39 | 225.58 | 20% |
| #28 | 2171.81 | 103.16 | 102.03 | 211.82 | 43% | 1158.62 | 33.85 | 67.57 | 151.92 | 26% | 1305.52 | 30.38 | 78.86 | 188.39 | 20% |
| #31 | 1756.70 | 73.1 | 75.80 | 203.02 | 37% | 1391.03 | 30.00 | 81.12 | 203.92 | 19% | 1451.28 | 38.17 | 84.79 | 203.41 | 23% |

kCAL = kCALORIES
FAT = FAT (g)
PRO = PROTEIN (g)
CHO = CARBOHYDRATE (g)
% FAT = % FAT

# APPENDIX L

## EXPERIMENTAL SUBJECTS' RECORD OF ATTENDANCE

# Record of Attendance

| Subject Number | Week 1 | Week 2 | Week 3 | Week 4 | Week 5 | Week 6 |
|---|---|---|---|---|---|---|
| 1 | | | A | | | |
| 2 | | | | | | |
| 3 | | A | | A | | |
| 4 | | | | | | A |
| 5 | A | | | | | |
| 6 | | | A | | A | |
| 7 | | | | | | |
| 8 | | | | A | | |
| 9 | | | | | | |
| 13 | | | | | | |
| 16 | | A | | | | |
| 17 | | | | | | |
| 18 | | | | | | |
| 19 | | | A | | | |
| 20 | | | | | | |
| 21 | | | | | | |
| 23 | | A | | | | |
| 24 | | | | | | |
| 25 | A | | | | | |
| 31 | | | A | | | |
| 34 | | | | | | |
| 35 | | | | | | |

A = Absent

# APPENDIX M

# REORGANIZED DATA GROUPING

REORGANIZED DATA GROUPING

| POST-HOC CONTROL GROUP ID # | REDUCED-FAT GROUP ID # |
|---|---|
| #4 | #1 |
| #5 | #2 |
| #8 | #3 |
| #10 | #6 |
| #12 | #7 |
| #13 | #9 |
| #14 | #17 |
| #15 | #19 |
| #18 | #20 |
| #21 | #23 |
| #22 | #25 |
| #24 | #28 |
| #26 | #31 |
| #27 | |
| #30 | |
| #32 | |
| #33 | |
| #34 | |
| #35 | |
| #36 | |