

SWEARINGIN, BRENDA., Ph.D. The Comparison of the Effects of Lifestyle Activity and Structured Cardiovascular Exercise on Obesity-Related Risk Factors of African-American Women Ages 22-55. (2008)

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The purpose of this study was to examine the effects of lifestyle activity modification (LA) and structured exercise (Cardio) on obesity-related factors in sedentary African American women. Subjects were randomized to a control group, a Cardio group, or LA group for a twelve week intervention. The study examined the intervention effects on physical activity patterns, blood lipids, glucose & insulin response, blood pressure, cardiovascular fitness and body composition.

Analysis of variance (ANOVA) for pre-post differences in the dependent variables revealed significant baseline differences between groups for LDL cholesterol only. Thus, pre-post treatment differences for LDL were assessed using an ANCOVA model, with baseline LDL as the covariate. Significant pre-post effects were observed for cardiorespiratory fitness ($p = 0.024$), physical activity level ($p=0.000$), total cholesterol ($p=0.006$), and HDL cholesterol ($p=0.017$). Significant pre-post by treatment condition interaction effects were observed for body weight ($p=0.001$), body composition (percent body fat) ($p<0.001$), and cardiorespiratory fitness (predicted $VO_2\text{max}$) ($p=0.024$). Although post-hoc analysis failed to reveal significant differences among groups, there were slight trends (which merit further investigation) toward decreasing obesity-related risk within the 2 activity groups, when compared to Control.

THE COMPARISON OF THE EFFECTS OF LIFESTYLE ACTIVITY AND
STRUCTURED CARDIOVASCULAR EXERCISE ON
OBESITY-RELATED RISK FACTORS OF
AFRICAN-AMERICAN WOMEN
AGES 22-55.

By

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CHAPTER I

INTRODUCTION

Overweight, obesity and obesity-related disorders are increasing at alarming rates and recent studies have indicated a particular prevalence among African-American women. Approximately 39.6% of African American women have cardiovascular disease (CVD), 77.3% are overweight, 49.7% are obese, 9.5% have diabetes mellitus, and 44.7% have hypertension (Centers for Disease Control and Prevention, 2002). Additionally, the prevalence of hypertension in African Americans is the highest in the world, and they are more likely to be physically inactive, overweight or obese, and have diabetes (Collins & Winkleby, 2002). Also, more African American women than African American men have hypertension after 55 years of age (Centers for Disease Control, 2002).

Obesity is associated with increased risk for mortality and morbidity as a result of co-morbid conditions including, but not limited to hypertension, dyslipidemia, coronary heart disease, and diabetes (Heyward, 2006). When compared to White women, African-American women are more likely to die of a heart attack, more likely to die of a stroke, and more likely to die from coronary artery disease and are also more likely to be diagnosed with diabetes, particularly non-insulin dependent diabetes (NIDDM) (American Heart Association, 2003). The development of these chronic diseases, largely due to physical inactivity, adversely impacts the mortality rate of African American

women. Cardiovascular disease is the leading cause of death, accounting for 49% of deaths in all women. For African American women,

CVD accounts for 40.6% deaths (Grundy, 2005). The prevalence rate increases even more when it comes to diabetes and obesity. In 2000, the overall death rate from diabetes mellitus in Caucasian women was 20.5% but 48.1% in African American women (Centers for Disease Control, 2002). Obesity also has an unfavorable effect on an African American woman's length of life. For example, a 20-year old Caucasian woman with a body mass index of greater than 45 is estimated to have 8 years of lost life while an African American woman is estimated to have 25 years of lost life (Fountaine, Redden, Wang, Westfall, & Allison, 2003).

The existence of high-quality data from interventions targeting obesity risk reduction and associated chronic disease in minority populations is minimal. This gap in the literature is a major obstacle in the development of programs and policies to deal with the problem of obesity and obesity-related disorders in minority populations. Prior to 1996, most studies utilized small sample sizes and targeted low-income segments of the ethnic groups that were studied. Previous studies generally had high attrition rates (with retention rates approximately 40%) and were usually of relatively short duration (less than 6 months of follow-up data supplied). In addition, many studies investigating obesity and related disorders involved highly selected populations, relatively affluent Caucasian populations, very costly individually targeted educational or behavioral interventions, or, more heterogeneous populations exposed solely to low-intensity mass media interventions, which has limited generalization to ethnic and lower socioeconomic

populations (Yancey et al., 2004). Additionally, the lack of clearly defined theoretical frameworks and the small populations of minorities investigated in previous clinical studies have limited the applicability of their findings (Booth et al., 2001). However, long-term studies that may be more effective are extremely cost prohibitive. The failures and barriers associated with this genre of research has led to the suggestion that researchers should conduct smaller, more focused studies within high-risk populations such as African-American women (Kumanyika, 2002). Data from community-level approaches to obesity and obesity-related risk reduction are needed in order to directly address the determinants of risk and disease burden within high-risk minority communities.

Several recently published studies used multi-modal approaches to obesity risk modification by addressing various combinations of nutritional, behavioral, and exercise variables as a method of intervention, which confounds the effective contribution of each variable. Additionally, successful intervention procedures for any of these modes have yet to be standardized in high-risk populations. Regular moderate-intensity physical activity has been proven to reduce many of the medical co-morbidities associated with obesity, however, the efficacy of low to moderate intensity exercise for the reduction of these risk factors remains to be clearly determined (Kriska et al., 2003). Lifestyle activity (LA) interventions, where moderate forms of activity are incorporated in short bouts within daily routines, are becoming increasingly popular. LA interventions provide another option for attaining the US Surgeon General's recommendation to accumulate 30 minutes of moderate activity each day for the purpose of obtaining health-related benefits (Dunn

et al., 1999). A limited number of studies on LA intervention and accumulated exercise bouts suggest that obesity-related risk factors can be reduced by using these modes of intervention (Dunn, 1999; Farrell et al., 2000; Flegal, Carroll, Kuczmarski & Johnson, 1998; Gallagher, 1996). Furthermore, the American College of Sports Medicine's Guidelines for Exercise Testing and Prescription (7th Ed.) indicates that sedentary individuals can improve fitness with lower-intensity, longer duration exercise sessions (40-49% Heart Rate Reserve or 55-64% Heart Rate Maximum). Although the Surgeon General's recommendations and previous studies of LA interventions have focused on the use of moderate intensity physical activity, research in overweight or sedentary populations suggests greater adherence to low-intensity intermittent activity, these findings have not been duplicated for overweight but otherwise healthy African-American women (Miyatake et al., 2002). More extensive research is needed to prove the efficacy of low to moderate intensity physical activity accumulated through a LA intervention for obesity-related risk factor reduction.

Statement of the Problem

In order to address the issues discussed above, this study examined the effects of accumulated exercise on obesity and obesity-related risk factors using: 1) traditional cardiovascular exercise (Cardio), which uses commonly prescribed aerobic exercise of moderate intensity through multi-modal (treadmill walking, stationary stair stepping or stationary cycling) group sessions in single daily bouts; and 2) Lifestyle Activity Modification (LA), for which participants incorporate short, low to moderate intensity bouts of exercise into activities of daily living to accumulate a recommended total of 150

minutes of exercise each week (Dunn et al., 1999). Based on a thorough review of literature as presented in the following chapter, the aims and corresponding hypotheses were formulated and are listed below.

Specific Aim 1

Compare the effects of LA, Cardio and a control group on the physical activity patterns of sedentary African-American females between the ages of 18 and 55.

Hypothesis 1

Compared to the control group, LA and Cardio will increase physical activity in sedentary African-American females between the ages of 18 and 55.

Specific Aim 2

Compare the effectiveness of LA, Cardio compared to a control group on obesity-related health indicators in sedentary African-American females between the ages of 18 and 55.

Hypothesis 2a

Compared to the control group, both LA and Cardio will have similar positive effects on blood lipid profiles in sedentary African-American females between the ages of 18 and 55.

Hypothesis 2b

Compared to the control group, both LA and Cardio will improve insulin resistance in sedentary African-American females between the ages of 18 and 55.

Hypothesis 2c

Compared to the control group, both LA and Cardio will reduce blood pressure in sedentary African-American females between the ages of 18 and 55.

Hypothesis 2d

Compared to the control group, both LA and Cardio will increase cardiovascular fitness in sedentary African-American females between the ages of 18 and 55.

Hypothesis 2e

Compared to the control group, both LA and Cardio will effect body composition in sedentary African-American females between the ages of 18 and 55.

The anticipated overall outcome of this research is to provide reasonable exercise options for sedentary African-American women that will ultimately promote positive changes in health.

Benefits of Proposed Research

This research will provide much needed data about the impact of lifestyle activity modification on obesity-related risk factors. If lifestyle activity produces comparable results to traditional cardiovascular exercise programs, it could have a significant impact on public health and disease prevention. The accumulation of evidence for the improvement of obesity-related risk factors using lifestyle activity modification is particularly important in this minority population, since increased risk for obesity and obesity-related disorders has already been identified in this group (Whitt, Kumaniyika & Bellamy, 2003). Overweight and obesity is associated with disability, decreased health-

related quality of life and increased health care use, all of which translate into increased health care costs to the American public (Jackson, 2002). There is preliminary evidence indicating greater exercise adherence with intermittent bouts, such as those used in LA interventions, versus continuous bouts of exercise (Church et al., 2007; Darling et al., 2005). The legitimacy of a lifestyle activity program for the reduction of risk factors would establish a realistic option for the initiation of activity in previously sedentary populations, broadening the available physical activity choices and encouraging participation in activities known to have health benefits.

CHAPTER II

REVIEW OF LITERATURE

In this chapter the literature relevant to the problem of obesity, obesity related disorders, physical activity, and minority populations, specifically African-American women, will be reviewed. The identification of limitations associated with the currently published literature was used to inform the development of the specific aims and hypotheses formulated for the current study.

The Scope of the Problem

Globally, obesity is a major health and economic problem. The prevalence is increasing not only in Western and affluent countries, but in third-world countries as well. In 1995, adult mortality attributable to obesity was estimated to be approximately 1 million, more than doubling the mortality rate attributable to under-nutrition. In 1997 the worldwide adult population that was estimated to be overweight was 1.6 billion and this will rise to 2.3 billion by 2015 (WHO, 2008). In addition the population that is obese will rise from approximately 400 million to 700 million (WHO, 2008). In Europe, the range of obesity estimates is highly variable based on the region of interest. Eastern Europe has some of the highest adult obesity rates; averaging 11 percent for men and 28 percent for women in Russia, while Germany has rates of 20 and 23 percent for men and women respectively, and the Czech Republic averages 23 percent for men and 27 percent for

women. At the same time, Central European and Mediterranean countries have some of the lowest rates of obesity (WHO, 2008).

In the United States, the National Center of Health Statistics (NCHS) produced four surveys, [the National Health Examination Survey (NHES, 1960-62) and the National Health and Nutrition Examination Surveys, (NHANES I, 1971-74; II 1976-80; and, III, 1988-94)], that outline the domestic problem of overweight and obesity and its progression over the last several decades. These four surveys have provided data on height and weight and the trends of obesity and overweight for the last 40 years for a representative sample of United States residents. These surveys indicate that the age-adjusted prevalence of overweight, as defined by a BMI equal to or greater than 25, was relatively unchanged from 1960 to 1980, with 48 percent and 39 percent of adult men and women being overweight respectively. Data collected from the NHES (from 1960-1962), indicated that during this time, the prevalence of obesity was 10 percent for men and 15 percent for women. The NHANES II study (1976-1980), demonstrated increases in the prevalence of obesity as proportions for this classification rose to 20 percent and 25 percent of U.S. men and women respectively. Additional increases were noted in NHANES III (1988-1994), with the prevalence of overweight men increasing to 59 percent, and the prevalence of overweight women, increasing to 48 percent.

The Centers for Disease Control (CDC) conducted additional assessments of the obesity trends in the United States. To examine state-specific obesity rates among US adults from 1991-98, Mokdad and colleagues (1999) analyzed data from the Behavioral Risk Factor Surveillance System (BRFSS). Data from all states participating in the

BRFSS was examined for prevalence and trends and the information from each state was pooled to produce nationally representative estimates. BMI was calculated from self-reported height and weight. Participants in the BRFSS were classified as obese if their calculated Body Mass Index (BMI) was equal to or exceeded 30 kg/m². An increase in obesity prevalence was detected in all states regardless of sex, age, race, educational levels and smoking habit. The largest increases, however, were noted in the following groups: 18-29 year olds (7.1 percent to 12.1 percent); participants reporting some college education (10.6 percent to 17.8 percent); and, those of Hispanic ethnicity (11.6 percent to 20.8 percent) (Mokdad et al. 1999).

When Mokdad and colleagues (2003) examined the BRFSS of 2001, the prevalence of obesity in US adults (BMI \geq 30) had increased from 19.8 percent to 20.9 percent during the one-year period from 2000-2001 (an increase of 5.6 percent). Over the previous ten years, from 1991 to 2001, there had been a 74 percent increase in obesity among US adults. Alarmingly, the percentage of adults with a BMI \geq 40 (morbid obesity) increased from 0.9 percent in 1991 to 2.3 percent in 2001. In 2001, although men made up a higher proportion of the overweight category, women made up a higher proportion of the obese category. Of all ethnic groups investigated, Blacks had the highest rate of obesity at 31.1 percent; and among the different states, North Carolina ranked 11th with an obesity rate of 22.4 percent (Mokdad et al. 2003).

By 2007, the highest prevalence of obesity was still among non-Hispanic Black women (39 percent). The prevalence of obesity remained highest in the South (27.3

percent) and the Midwest (26.5 percent), although it was lower in the Northeast (24.4 percent) and West (23.1 percent) (CDC/BRFSS, 2007).

Obesity in America has reached epidemic proportions. Minority populations, especially African-American women, are affected disproportionately; nearly 50 percent of African-American women are overweight. Black women have a higher prevalence of overweight, obesity and physical inactivity than White women. An inverse relationship between the prevalence of overweight and obesity to education and income has been shown in women of all ethnic groups (CDC/NCHS, 2006). Overweight and obese adults are at increased risk for morbidity and mortality associated with many acute and chronic health conditions including diabetes, hypertension, coronary artery disease, elevated serum lipid levels, certain cancers, respiratory disease and arthritis, to name a few (American Heart Association, 2003).

The Cost of Obesity

An estimated 300,000 adults die annually in the US due to obesity related causes (Flegal et al., 2005; Mark, 2005). Nearly all individuals who are overweight display some adverse health risks associated with their increased body weight. While the most evident effect of weight gain on mortality is related to hypertension and elevated serum lipid levels (specifically increases in low-density lipoproteins (LDL)), non-insulin dependent diabetes mellitus (NIDDM) is a rising and costly health concern that is also directly related to the prevalence of obesity. It has been estimated by the National Institutes of Health (NIDDK) that in the US the annual cost of overweight and obesity is \$122.9 billion (\$64.1 billion in direct costs and \$58.8 billion in indirect costs related to the

obesity epidemic), which can be compared to the cost of cigarette smoking (NIDDK, 2004). Direct obesity-related health care costs refer to preventive, diagnostic, and treatment for obesity-related diseases and conditions (i.e., type II diabetes, hypertension and heart disease); while the indirect costs are attributable to the lost wages due to obesity-related disability or illness, and lost productivity and earnings due to premature death as a result of obesity-related illness and disease (WHO 2003). Obesity and obesity-related health costs result in at least \$62.7 million in medical visits and \$39.3 million in lost productivity (due to absenteeism) each year (WHO 2004).

Defining Overweight and Obesity

Much controversy exists over the definition of the terms overweight and obese. While obesity refers to an excess of body fat, most reference data for obesity in health is written with regard to the measurement of body weight (Loos & Bouchard, 2003). In 1953, the Metropolitan Life Insurance Company developed the first height/weight tables to calculate an individual's weight status, classifying them as normal, under or over weight. In 1983 the tables were adjusted to account for individual differences in frame size. Although frame size was subjectively measured at that time, it was an important improvement in the tables, since determining ideal weight was now based on small, medium and large frame size classifications. Further improvements were implemented in 1986 when elbow breadth or wrist circumference measurements were used to classify frame size. Even with the modifications for frame size, the use of the Metropolitan Height/Weight Table was still not discriminatory for an individual's degree of obesity or

for the distribution of body fat. Additionally height/weight tables can provide grossly inaccurate conclusions about an individual's health risk (Loos & Bouchard, 2003).

Today the most commonly used quick reference tool in epidemiological studies is body mass index (BMI). BMI is calculated by dividing total body weight (kilograms), by the square of the individual's height in meters [weight in kilograms/(height in meters)²]. Although it is not a direct measure of body fat, BMI is believed to be a better correlate to overall body fatness than were the height-weight tables (Brown, Miller & Eason, 2006). One of the major limitations of BMI, however, is the failure to account for body fat distribution, which can be a better predictor of health risks (Fox et al., 2008; Votruba & Jensen, 2007). Additionally, an individual with a relatively large lean body mass may frequently be misclassified as obese. Still, BMI uses only an individual's height and weight and no indication of actual leanness or fatness can be determined.

The use of BMI, however, is still a standard widely used to assess health status on a population-wide basis. The third edition of Dietary Guidelines for Americans (1990), suggested an age-adjusted cutoff for overweight based on the indication that increasing age corresponded to an increase in BMI associated with minimum mortality (Andres, 1985). In the fourth edition of Dietary Guidelines for Americans (1995) the age adjustment was deleted based on indication that increased BMI was associated with higher morbidity and mortality rates. (US Department of Agriculture/Department of Health and Human Services, 1995). Even with these modifications advisory organizations could not reach a clear consensus related to BMI. WHO, NHLB, and the NIDDK all published their own individual and differing guidelines over the next three to five years.

These classifications were specifically related to the risks associated with hyperinsulinemia, hyperlipidemia and hypertension, which the current research had associated with increasing BMI. Beginning in 2000, there was finally a convergence of cutoff recommendations, starting with the fifth edition of Dietary Guidelines for Americans following the recommendations outlined by the 1998 NHLB/NIH clinical guidelines. The changing classifications related to weight, by date, have been outlined in Table 1.

The evolution in the cutoffs used to define overweight versus obese, reflect population changes and the realization that discrete comparative standards over time were necessary. While the risks associated with overweight and obesity are on a continuum, rather than tied to discrete cutoff points, these changes in recommendations must be considered when assessing statistical reports concerning prevalence data of overweight and obesity reported over time.

Identification and Quantification of Adiposity

Accurate calculation of the quantity and distribution of adipose tissue is important due to the risks associated with increasing adipose tissue. For this task, both whole body and regional methodology exist. Of the available methods, anthropometry is the most cost-effective and is commonly used for large epidemiological studies. For use in smaller studies, the “gold standard” of body fat estimation is hydrodensitometry. Also convenient for small studies, and less cost-effective, are potassium counting, dual-energy x-ray absorptiometry (DXA), computerized tomography scan (CT), and magnetic resonance imaging (MRI). Each method has advantages and disadvantages, which dictate its use in

Table 1: Changing classification of overweight and obesity (Adapted from Knczmarusk & Flegal, 2000)

Date	Source	Sex	Criteria	Label
1942	Metropolitan Life	M,F	Weight-for-Height tables	Ideal
1980	Dietary Guidelines ^{2,3} (1 st ed)		Weight-for-height tables	Ideal, normal, suggested, acceptable
1985	NIH Consensus Development Panel ⁵	M	(<25–26 kg/m ²) ¹	Overweight, Obesity
		F	(<24–25 kg/m ²) ¹	
1990	Dietary Guidelines ^{2,3} (3 rd ed)	M	≥27.8 kg/m ²	Unhealthy
		F	≥27.3 kg/m ²	
1990	Healthy People 2000 ³	M,F	Weight-for-height tables (≥25.0 kg/m ²) ¹	Overweight
		M,F>34 y	(≥27.0 kg/m ²) ¹	
1995	Dietary Guidelines ^{2,3} (4 th ed)	M	≥27.8 kg/m ²	Healthy weight Moderate overweight Severe overweight
		F	≥27.3 kg/m ²	
1998	NHLBI Expert Panel ⁶	M,F	Weight-for-height chart (<25.0 kg/m ²) ¹	Underweight Normal weight Overweight Obesity 1 Obesity 2 Extreme obesity
			(25.0–28.9 kg/m ²) ¹	
			(≥29.0 kg/m ²) ¹	
			<18.5 kg/m ²	
			18.5–24.9 kg/m ²	
			25.0–29.9 kg/m ²	
2000	Healthy People 2010 ³	M, F	30.0–34.9 kg/m ²	Healthy weight Obese
			35.0–39.9 kg/m ²	
2000	Dietary Guidelines ^{2,3} (5 th ed)	M, F	≥40 kg/m ²	Healthy weight Overweight Obese
			18.5–24.9 kg/m ²	
			≥30.0 kg/m ²	
			18.5–25.0 kg/m ²	
			25.0–29.9 kg/m ²	
			≥30.0 kg/m ²	

¹ Approximate BMI equivalents that correspond to weight-for-height table or chart data.

²⁻⁷ Federal agency using the criteria: ²US Department of Agriculture, ³Department of Health and Human Services, ⁴National Center for Health Statistics, ⁵National Institutes of Health (NIH), ⁶National Heart, Lung, and Blood Institute (NHLBI)/NIH.

the research setting. A full review of each of the available methods is beyond the scope of this paper; therefore, discussion of body composition assessment will be limited to the techniques used in the proposed investigation.

Body Fat Assessment & Quantification

The accumulation of excess body fat can occur due to either increased size or excess number of adipocytes, or both. Past studies in rodents, which were later followed by studies in humans, concluded that there are certain developmental periods throughout the lifespan, including adulthood, during which adipocytes are irreversibly formed (Ailhaud, 2008). Research subsequently suggested that persons who are overweight usually have increases in the size of adipocytes (hypertrophy), while individuals meeting the classification of morbid obesity tend to have above average numbers of adipocytes (hyperplasia) (Garaulet et al., 2006). Those with morbid obesity seem to have a more serious problem, since the same research also suggests that weight loss is effective only for reducing the adipocyte cell size and not the adipocyte cell number.

Along with the need to quantify an individual's total adipose tissue for risk classification, it is also important to describe the location of that tissue within the body. Adipocytes can accumulate in various anatomical sites, and are distinguished according to their location. Most body fat is distributed subcutaneously (approximately 80 percent), while the remaining collections of adipose tissue are located in visceral, retroperitoneal, perirenal, intra- or inter muscular, or orbital regions of the body (Williams & Considine, 2001). Subcutaneous fat distribution is gender-specific, and women have the tendency to accumulate excess fat in the gluteofemoral region and the abdominal wall. As abdominal

fat increases, disruptions in metabolism, morbidity and mortality also increase. There is a wealth of information to support the relationship between this centralized visceral abdominal fat distribution and the distinct features of insulin resistance and hypertension. Although the mechanisms of this relationship have yet to be fully elucidated, certain relational theories (Despres, Lemiex & Prud'homme, 2001; Montague & O'Rahilly, 2000; and, Frayn, 2000) are discussed in other sections of this review.

Anthropometrics

Anthropometric measures comprise some of the most common field measures used in the assessment of body composition. These measures for body fat prediction have been correlated with underwater weighing body fat measurements, and include: 1) skinfold, 2) BMI, and 3) body circumferences. The most commonly used epidemiological method of assessment is BMI, as it is used conventionally for classification of risk, and, epidemiologic comparison data is widely available. The correlation between percent body fat and BMI is both age and sex dependent, but independent of ethnicity (Huxley et al., 2008).

Skinfold thickness, using various equations to calculate predicted body fat, has also been widely used. The multiple site method must be utilized, as no single site measurement can accurately predict percent body fat (Deurenberg & Deurenberg, 2003). If BMI is greater than 40 kg/m^2 , it is likely that the upper limit for most skinfold calipers (50 mm), will be exceeded at several sites. In the case of obese populations, methods other than skinfold usually must be employed (Hans & Lean, 2001).

Body circumferences and ratios of circumferences can be used to describe the distribution of body fat, or in some cases can be combined with skinfold thickness to estimate percent body fat in subjects with increased intra-abdominal thickness (Friedle et al., 2001). As with skinfold measurement, great care must be taken in the identification of site location and measurement to ensure the most accurate, reproducible, and reliable measurements. Circumferences can be used since these measurements are assumed to reflect fat and FFM, and because skeletal size is directly related to lean body mass (Heyward, 2006). A landmark study by Wilmore and Behnke (1969) cross-validated several anthropometric equations with correlations ranging from 0.71 to 0.96. Additionally, other researchers have used anthropometric equations to predict body density (D_b) with correlations ranging from 0.83 to 0.87 and standard estimates of error from 0.0065 to 0.0072 g/cc; with this established data, Behnke (1969) concluded that the use of circumferences was an alternative measure of obesity and concurrently correlated with D_b . In the proposed study, a greater emphasis has been placed on the use of these parameters with regard to their association to health risk, not to negate the valuable relationship to body composition or the use of the measure to assess change over time. Anthropometric measures such as BMI and waist-to-hip ratio (WHR) have been more widely used in recent literature as crude indices of obesity-related health risks. According to the American College of Sports Medicine (2006), a WHR of 0.86 and greater for women indicated an increased risk for disease (American College of Sports Medicine, 2006). Although BMI and percent body fat estimates correlate equally well to blood pressure, waist circumference seems to be a stronger predictor for NIDDM than BMI,

WHR or skinfold measurements (Lofgren et al., 2004). A high waist circumference is associated with an increased risk for type 2 diabetes, dyslipidemia, hypertension, and CVD in patients with a BMI in a range between 25 and 34.9 kg/m² (Lofgren et al., 2004). Waist circumference measures of 88 cm or greater in women, that have been reported to be indicative of the need for weight management and to identify those at risk for cardiovascular disease (Heyward, 2006). In addition to measuring BMI, measurement of changes in waist circumference over time may be helpful, since it can provide an estimate of increased abdominal fat even in the absence of a change in BMI. Furthermore, in obese patients with metabolic complications, changes in waist circumference are useful predictors of change in CVD risk factors (Grundy, 2004a). The table below incorporates both BMI and waist circumference in the classification of overweight and obesity, and provides an indication of disease risk (Table 2).

Table 2. Risk classification by BMI, obesity and waist circumference

Classification	BMI (kg/m²)	Obesity Class	Men ≤102 cm (≤ 40 in.) Women ≤88 cm (≤ 35 in.)	Men >102 cm (>40 in.) Women >88 cm (>35 in.)
Underweight	18.5		----	----
Normal+	18.5 - 24.9		----	----
Overweight	25.0 - 29.9		Increased	High
Obesity	30.0 - 34.9	I	High	Very High
	35.0 - 39.9	II	Very High	Very High
Extreme Obesity	≥40	III	Extremely High	Extremely High

Bioelectric Impedance Analysis

Bioelectric impedance analysis (BIA) measures the opposition of body tissue to the flow of a mild alternating electric current. Electrodes are typically placed on a wrist and an ankle (in the tetra polar method, on the dorsal surfaces of the right hand and foot as well as the wrist and ankle), and change in voltage between electrodes is measured. The method assumes that the body (or the segment of it) is approximately cylindrical and of uniform cross section, with tissues evenly distributed throughout. It is this assumption that allows the calculation of its volume, as proportional to its length squared divided by its impedance (a function of the resistance of tissue composition). Impedance measures vary with frequency of the current used. At low frequencies, current flows primarily through extra-cellular fluids, while at high frequencies it penetrates all body tissues. Application of BIA frequently utilizes a frequency spectrum, to show differences in body composition due to clinical and nutritional status, as well as age, ethnicity, sex, and other variables (Dehghan & Merchant, 2008). Many equations have been developed to estimate total body water (TBW) and fat-free mass (FFM) as a function of impedance, weight, height, gender and age (Cleary et al., 2008). BIA calculations of an individual's body fat reported in the literature may vary by as much as 10 percent of body weight due to the instrumentation and methodologies employed. The standard error estimate for percent body fat from manufacturer formulated equations has been shown to be approximately six percent, with reductions of that error to approximately three percent based on the use of age, gender and fatness to enhance the accuracy of the measurement (Segal, Gutin, Presta, Wange, VanItallie, 1985; Rising, Swinburn, Larson & Ravussin, 1991; Lukaski, Johnson,

Bolonchuk & Lykken, 1985; Segal, Van Loan, Fitzgerald, Hadgdon & Van Itallie, 1988). Despite these accommodations, many studies still indicate limitations in reliability and validity for ethnic-specific BIA predictions (Heyward, 2006; Takasaki, Loy & Juergens, 2003; Sun et al., 2003). There are still fewer studies that examine the influence of bone density differences between Caucasians and African-Americans on the estimates of percent fat from BIA. Several studies indicate that BIA underestimates body fat and overestimate lean body mass and total body water in African –American women (Newton et al., 2006; Schoeller & Luke, 2000).

Obesity Related Health Implications

Several interrelated genetic, environmental and metabolic factors are ultimately responsible for insulin resistance, hypertension, cardiovascular disease and other obesity-related disorders. Some of the earliest symptoms of type II diabetes generally manifest long before fasting hyperglycemia indicates the development of the disease. Several abnormalities that have been associated with insulin resistance are detectable prior to the development of the disease. Particularly problematic are abnormalities in fat metabolism, which have been associated with the progression of insulin resistance to diagnosed diabetes. The consequences of these metabolic abnormalities include, but are not limited to dyslipidemia and impaired glucose tolerance or insulin resistance.

Cardiovascular Disease

The leading cause of adult mortality in the United States is cardiovascular disease, attributed to approximately one million deaths each year (American Heart Association, 2003). The number of women dying from cardiovascular disease has exceeded the

number of men dying from the disease since 1984, surpassing the number of deaths from the next fourteen leading causes of death for women (American Heart Association, 2003). One in ten women are diagnosed with heart disease between the ages of 45-64 and one in four have heart disease over the age of 65. Women's risk of cardiovascular disease is often misleading when compared to that of men. Men generally suffer heart attacks approximately 10 years earlier than women; however, after menopause the incidence of cardiovascular events rises sharply for women, equaling the incidence for men. Approximately one-half of all cardiovascular disease deaths (of the 500,000 annually) are a result of coronary heart disease, one type of cardiovascular disease (American Heart Association, 2003).

The American Heart Association identifies several primary and modifiable risk factors for CVD, including; physical inactivity, overweight and obesity, diabetes mellitus, cigarette smoking, dyslipidemia, and hypertension (American Heart Association, 2003). Other risk factors that may not be modifiable are age, family history, and, for women, menopausal status. While cigarette smoking has been identified as a more powerful risk factor than dyslipidemia, it is important to note that in women, diabetes is a more powerful risk factor than cigarette smoking. As well, being sedentary or maintaining a low physical fitness level are more powerful risk factors for both men and women than was previously thought (American Heart Association, 2003).

Both men and women share the commonly identified risk factors, and until recently, researchers have not discriminated influences of gender as a contributing risk factor. However, a diabetic woman is twice as likely to develop CVD as a diabetic man

(Bittner, 2000). In women who experience a heart attack, 38 percent die within one year compared to only 25 percent of men (AHA, 2003).

Minority women also display greater risk for CVD than their Caucasian counterparts. Winkleby and colleagues (1998) surveyed 1,762 Black, 1,481 Hispanic and 2023 White women ages 25-64, finding that most CVD risk factors occurred with significantly greater frequency among minority women. The study adjusted for socioeconomic status (SES) and educational level, and demonstrated significant differences in BMI, blood pressure, diabetes and physical activity levels for minority women compared to White women ($P < .001$). Similarly, Gerhard et al. (1998) examined risk factors for heart disease in higher SES Black and White premenopausal women (ages 18-45) and observed that young Black women also had significantly more CVD risk factors than young White women.

It is well established that physical activity has two major effects for prevention of cardiovascular disease: 1) it can affect cardiovascular disease through reductions in morbidity and mortality; and 2) it can also positively affect the risk factors associated with CVD (Grundy et al., 2004).

Hypertension

There is a high prevalence of hypertension among obese subjects. The link between blood pressure and insulin sensitivity might be directly related to concomitant obesity. Obesity has been associated with increased sympathetic nervous system activity, an elevation of the plasma levels of the vasoconstrictor ET-1, and decreased insulin-induced endothelium-dependent vasodilation (Scheen & Luyckx, 1999). A complex interaction

between endothelial dysfunction, subsequent abnormal skeletal muscle blood flow, and reduced insulin-mediated glucose uptake may be important to the link between insulin resistance, blood pressure, impaired glucose tolerance and the risk of cardiovascular disease (Cleland et al., 1998). Insulin causes an exaggerated ET-1 peak, which may contribute to the increased vasoconstriction that has been observed during hyperinsulinemic states (Hopfner et al., 1998). The development of insulin resistance may contribute to the onset of hypertension by decreasing the insulin-stimulated release of nitric oxide (NO), a vasodilator (Villa et al., 1999; Walker et al., 1999; van Zwieten, 1999).

Hypertension is a major health concern among African-Americans. Based on the JN-7 guidelines for classifying blood pressure, a recent study found that only 29 percent of Black participants (N=211) met the classification for normal blood pressure. For those classified as “abnormal”, 38 percent were pre-hypertensive. None of the participants had a history of hypertension, nor were any of the participants currently taking anti-hypertensive medications (Brown & Metiko, 2005).

Changes in blood pressure from increased physical activity are theorized to occur via adaptations in the sympathetic nervous system resulting in decreases in catecholamines, total peripheral resistance, as well as alterations in the release of vasodilators and vasoconstrictors (Pescatello et al., 2004).

Lipid Profiles

Abnormalities in triglyceride storage and lipolysis are early manifestations of insulin resistance and are detectable prior to the presentation of diagnosed diabetes.

Increases in free fatty acids (FFA) from adipose tissue contributes to the metabolic complications inherent in individuals with both identifiable insulin resistance, impaired glucose tolerance and diagnosed type II diabetes (Baldeweg et al., 2000). Recently, it has been suggested that abnormal lipid metabolism precedes the conversion from normal glucose tolerance to the development of type II diabetes, as demonstrated by the correlation of elevated plasma FFA and the prediction of type II diabetes (Belfort et al., 2005).

Hormone sensitive lipase (HSL) is the principle enzymatic regulator of FFA release from adipose tissue. Insulin appears to have a suppressive effect on HSL, and suppression of HSL seems to be more prevalent in those who are genetically predisposed to type II diabetes (Shen et al., 2007). Along with the suppression of HSL, insulin may also act to depress the rate of FFA esterification in adipose tissue (Riemens, Sluiter & Dullaart, 2000). The rationale for this effect lies in the dependence of fat cells on glycerol-3-phosphate, derived from insulin-mediated glucose uptake in the adipocyte, for FFA esterification; therefore, with insulin resistance and the decreased availability of glucose, FFA esterification is negatively affected (Kahn & Flier, 2000). Lipoprotein lipase (LPL), as well, is altered in conditions of obesity and obesity-related metabolic disorders. LPL initiates the clearance of triglycerides and is responsible for FFA delivery to the fat cell. Insulin and glucose cause FFA to be shifted away from muscle and towards adipose tissue. In the adipose tissue of obese or type II diabetics, insulin activation of LPL is delayed so that this shift further confounds the uptake of FFA's (Kim et al., 2001). The increase in FFA lipolysis and decreased FFA esterification in those with insulin

resistance, type II diabetes and other obesity-related disorder tends to cause accumulation of FFA in liver, muscle, heart and pancreatic β -cells, ultimately leading to further health complications. It is clear, from numerous research studies, that FFA's impair glucose metabolism in insulin-sensitive tissues such as the muscle and the liver (Shulman, 2000).

Theories to explain the decrease in plasma triglyceride concentrations with exercise training may be through alterations in lipid metabolism. Triglycerides are broken down into free fatty acids that can be used for energy through fatty acid oxidation. Exercise training enhances free fatty acid oxidation and gluconeogenesis during rest and exercise, which can lower the presence of triglycerides in the blood (Bergman et al., 2000). HDL cholesterol levels increase with overall energy expenditure possibly through an intricate relationship with lipid metabolism. Increases in lipoprotein lipase activity also increase HDL formation while simultaneously increasing the use of triglycerides as fuel, increasing their clearance even at rest (Thompson et al., 2001).

In general, Black Americans have a more favorable plasma lipid and lipoprotein profile than White Americans (Johnson et al., 2004), from childhood throughout adulthood despite similar body composition values (Herd et al., 2001). Blacks have a 14% higher HDL-C (Despres, J-P et al., 2000, Srinivasan et al., 2001), 16% lower LDL-C (Srinivasan et al., 2001, Racette et al., 2000), and 21% lower TG (Despres, J-P et al., 2000, Racette et al., 2000) than Whites. Several studies have examined baseline plasma lipid and lipoprotein level differences between overweight and obese Black and White women (Racette et al., 2000; Gerhard et al., 2000; Despres et al., 2000). Racette et al. (2000) and Despres et al. (2000) reported significantly lower TC in Black women when

compared to White women. Considering the reported sedentary lifestyle and the body composition of participants reported at baseline, these results seem to support a lower risk with respect to the lipid profile than was indicated by the anthropometric profile.

McLaughlin and colleagues (McLaughlin et al., 2003; and McLaughlin et al., 2005), in two key studies, have linked triglycerides and HDL-cholesterol concentrations with insulin resistance. In these studies, the investigators identified a risk for hyperinsulinemia at a value of 2.0 for the ratio of TG/HDL in Blacks, indicating a significant ethnicity specific association for triglycerides and HDL-cholesterol. The authors suggested this value could be used in place of plasma insulin concentrations as a predictor of insulin resistance (McLaughlin et al., 2003; and McLaughlin et al., 2005). This relationship was further born out in work related to the HERITAGE study, where investigators found a similar association and also identified ethnic differences with regard to prediction of hyperinsulinemia (Li et al., 2008).

Type II Diabetes

Although there are numerous factors that can account for the rapid rise in type II diabetes worldwide, obesity is a link to which many researchers have dedicated their efforts. More than 80 percent of new cases of type II diabetes worldwide are associated with obesity (Lieberman, 2003). The risk of developing type II diabetes is 60 times greater in women with a BMI $> 33 \text{ kg/m}^2$ when compared to lean women. Type II diabetes risk correlates progressively with the degree, duration and distribution of adiposity. Accompanying these associations with adiposity, are differences in metabolic

activity, measured as changes in lipid metabolism and the processes of glucose homeostasis.

Both overall excess body fat and body fat distributed in the abdominal region independently increase the risk of insulin resistance and dyslipidemia. As previously mentioned, regional abdominal fat increases the risk of cardiovascular disease (National Institutes of Health, National Heart Lung, and Blood Institute, 2004). It has been shown that programs designed for the overall reduction of body fat also decrease abdominal body fat, which concomitantly decreases risk of cardiovascular disease. Studies that focus on energy expenditure through physical activity where reduction of weight is not an outcome, risk for cardiovascular disease is still positively affected (Ross & Janssen, 1999). These and other studies suggest that the reduction of abdominal fat can be an outcome of physical activity interventions with or without weight loss (Ross & Janssen 1999). Ross et al. (2000) and Sopko et al. (1985) demonstrated improvements in insulin sensitivity and increases in HDL cholesterol with physical activity interventions despite the fact that participants did not lose weight. These findings suggest that focusing on weight loss as the singular important outcome overshadows the importance of increasing physical activity as a means for reducing cardiovascular risk.

Normal Glucose Control

During fasting states, independent of insulin, glucose is supplied by the liver for use by the brain. Post-prandial glucose levels stimulate the release of insulin in order to accommodate glucose transport, metabolism and storage by myocytes and adipocytes.

Glucose does not permeate the plasma membrane of either an adipocyte or a myocyte independently; therefore, the entry of glucose must be facilitated by membrane associated carrier proteins such as GLUT- facilitative transporters or SGLT-facilitative transporters. SGLT-transporters, or sodium dependent transporters, are located in the small intestine and generally have a higher affinity for fructose than for glucose. However, in the presence of high sodium concentrations the affinity of SGLT-transporters shifts to favor glucose so that glucose can then be transported against its concentration gradient (Sato et al., 1994). GLUT-transporters, such as GLUT-4, are the major mechanism responsible for glucose uptake in skeletal muscle. The most important stimuli for GLUT-4 mobilization to the plasma membrane are insulin and muscle contraction. Other stimuli that can alter GLUT mobilization are hypoxia, catecholamines and glucocorticoids. Contraction and insulin, of primary concern in this study, increase mobilization through three different mechanisms (Pereira & Lancha, 2004):

1. By causing conformational changes in the cell surface of the glucose transporter, which increase transport activity.
2. By increasing the number of GLUT-4 in the plasma membrane.
3. By causing rapid synthesis of new transporters.

In the muscle, the primary site of glucose uptake, glucose transport can occur via insulin-dependent and insulin-independent mechanisms (Huppertz et al., 2001). Post-prandial increases in blood glucose stimulate the release of insulin. Insulin binds to its receptor in the plasma membrane, resulting in the phosphorylation of the receptor and the

insulin receptor substrate molecules, or IRS molecules. The phosphorylated substrates form complexes with proteins such as phosphoinositide-3 kinase (PI-3 kinase), activating PI-3 kinase, which is a major step with regard to insulin stimulated glucose transport and metabolism. The activation of PI-3 dependent kinases facilitates the activation of protein kinase B and protein kinase C (PKC), which then stimulate the translocation of insulin responsive GLUT-4 molecules to the cell membrane for increased glucose uptake. In particular, this pathway represents events independent of those that facilitate glucose transport mediated by muscle contraction, a secondary and insulin-independent mechanism for glucose transport and metabolism, thought to involve the activation of 5'-AMP-activated kinase without the activation of PI-3 kinase (Zierath, Krook & Wallberg-Henriksson, 2000).

Exercise, or more specifically muscle contraction, can facilitate the transport of glucose from the blood for metabolism independent of insulin action. In response to exercise or contraction, GLUT-4 can also move to the plasma membrane, and “dock”, to form complexes with protein receptors. The vesicles that contain these proteins fuse with the plasma membrane and increase the number of GLUT-4 molecules in the membrane, subsequently increasing the rate of glucose transport into the cell (Zierath, Krook & Wallberg-Henriksson, 2000).

Maintaining circulating blood glucose within normal ranges is a process that requires the balance of both its entry and removal. Circulating glucose is increased after the ingestion of a meal, which stimulates the secretion of insulin from the pancreas, and ultimately reduces blood glucose levels by decreasing output of hepatic glucose and

increasing glucose uptake by the periphery. Any disruption of glucose homeostasis can result in serious health complications.

Consequence of Abnormal Glucose Control

Compared with nondiabetic men and women, mortality from CHD has been reported to be twice as great in diabetic men but four to five times greater in diabetic women (Morrish, Wang & Stevens et al. 2003). Seven and a half million women in the United States have been diagnosed with diabetes, with another 2.3 million estimated to have the disease but are as yet, undiagnosed. Ninety to ninety-five percent of all people with diabetes are type 2, meaning that an estimated 8.8 million women have the disease. Another 24.9 million are considered to be "prediabetic" with either impaired fasting glucose and/or impaired glucose tolerance (Rosamond et al. 2007).

The prevalence of CHD has been estimated to be as high as 55% in diabetic adults, and CVD is responsible for at least two thirds of deaths in persons with type 2 diabetes mellitus (Van de Werf et al. 2003). Most studies have demonstrated an inverse relationship between physical activity and/or fitness and the risk for developing type II diabetes in women. For example, the Nurses' Health Study, an 8-year follow up of 70 102 female nurses without diabetes at baseline, demonstrated that the relative risks of developing type II diabetes decreased across quintiles of physical activity (from 1.0, 0.84, 0.87, 0.77, and 0.74 respectively) despite adjusting for age, BMI, smoking, alcohol use, hypertension, cholesterol level, parental history of diabetes, and menopause status (Hu, Manson, Stampfer et al. 2001).

African American women have a high prevalence of insulin resistance, non-insulin dependent diabetes mellitus and obesity (National Diabetes Statistics, 2007). Prolonged elevation of blood glucose concentrations can result in several health-related impairments. Resistance to the stimulatory effect of insulin on glucose utilization is a key feature of obesity-related disorders and can ultimately lead to type II diabetes (Reaven, 2005). In obese subjects, the [endocrine] pancreas can respond to insulin resistance by an adequate increase in insulin secretion, but when the pancreas response becomes inadequate, glucose intolerance can ensue (Reaven, 2005). Considering this, a sequence of events would be primary insulin resistance, resulting in hyperinsulinemia with corresponding metabolic consequences and ultimate decompensation of the endocrine system with resultant diagnosis of type II diabetes.

Resistance to the stimulatory effects of insulin, with regard to glucose clearance from the blood, is an important feature related to obesity. Glucose transport has been identified as the point of defect for both type I and type II diabetic subjects with regard to increases or lack of homeostatic glucose concentrations in the blood. The availability of GLUT-4 is of primary importance in glucose homeostasis. It has been suggested that GLUT-4 concentrations may be decreased due to an impairment of the insulin-stimulated movement of GLUT-4. This impairment is likely associated with defect(s) in the signaling pathways that regulate its translocation. Additionally, insulin-stimulated glucose uptake in adipocytes has been shown to be defective as a result of down-regulation of GLUT-4 expression. The importance of GLUT-4 with respect to glucose homeostasis has been demonstrated in several rodent studies. In a study by Rossetti and colleagues (1997),

the allele of the glut-4 gene was disrupted so that mice in the study had an approximate 50 percent reduction in Glut-4 concentrations in skeletal muscle, heart and adipocytes. These mice expressed severe insulin resistance and many developed diabetes with age. Several studies have indicated that increases in skeletal muscle's capacity for insulin-stimulated glucose transport are related to the increased expression of Glut-4 (Khan & Pressin, 2002; Bryant et al., 2002; White, 2003).

As well as having diverse mechanisms to increase GLUT transporter mobilization, insulin and muscle contraction accomplish the stimulation of glucose transport through two distinct pathways. Three points of evidence in the research literature provide a clear demonstration of these differing pathways. First, the effects of insulin and muscle contraction with respect to glucose uptake are additive (Wojtaszewski, Nielsen & Richter, 2002). Second, even in situations where insulin has a reduced effect on glucose clearance (such as insulin resistance), rodent models indicate glucose transport is stimulated by muscle contraction (Wojtaszewski, Nielsen & Richter, 2002). And third, when insulin stimulation is inhibited completely, muscle contraction still effectively increases glucose uptake (Wojtaszewski, Nielsen & Richter, 2002).

Improvements in glucose function from chronic exercise stem from increased GLUT4 transporters, capillary density, and muscle fiber conversion to type IIa and enzymes for fatty acid oxidation and gluconeogenesis (Lavrentyev, He & Cook, 2004). These alterations increase glucose uptake into the muscle tissue which improves insulin resistance and utilization of glucose for energy. There is also evidence to suggest that increased physical activity decreases abdominal obesity even in the absence of weight

loss (Kay & Fiatarone Singh, 2006). Reduced visceral fat is associated with improved insulin and glucose homeostasis (Ross, Freeman, Hudson, & Janssen, 2002).

The Metabolic Syndrome

More than two thirds of individuals with type II diabetes have the metabolic syndrome (Smith, 2007; Cameron, Shaw, Zimmet, 2004). The metabolic syndrome, as defined in 2004 by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), is a clustering of interrelated risk factors, including insulin resistance, central obesity, hypertension, and atherosclerosis. It is associated with a 2-fold increase in the risk for cardiovascular events and a 4-fold increase in the development of type II diabetes (Smith 2007; Alexander et al. 2003). Given the significantly increased risk of developing type II diabetes and CVD associated with the metabolic syndrome and its high prevalence in the diabetic population, primary prevention of this clustering of risk factors is of obvious importance.

Many studies indicate that physical activity and/or fitness demonstrates an inverse relationship with the prevalence of the metabolic syndrome (LaMonte et al., 2004; Orakzai, Orakzai & Nasir, 2006; Finey et al., 2006). The association between physical activity/inactivity and the risk for developing the metabolic syndrome in women is not entirely clear. NHANES III (1994) examined the association between leisure time physical activity and the metabolic syndrome, as defined by NCEP ATP III, in US adults. For women inactivity was not associated with the metabolic syndrome, though it was in men. However, in the first 2 years of the most recent NHANES (1999-2000) the association between sedentary behavior, physical activity, and the metabolic syndrome

was re-examined. A gender bias was noted, as women were more than twice as likely to have the metabolic syndrome when reporting no physical activity compared with women reporting ≥ 150 min/wk (odds ratio [OR] = 2.35; 95% CI: 1.19-4.63). Physical inactivity was not associated with the metabolic syndrome in men following stratification.

Physical Activity

Physical activity and cardiorespiratory fitness are interrelated but should be separated with respect to their relationship to health outcomes. Physical activity, defined as, “any bodily movement produced by skeletal muscles that requires energy expenditure”, is a behavior that is highly recommended for cardiovascular fitness (Caspersen, Powell & Christenson, 1985). Cardiovascular fitness, then, is a condition that can be improved through habitual physical activity, but can also be affected by other factors such as age, sex, heredity, and disease state (US Dept. of Health and Human Services, 1996). Tests of cardiorespiratory fitness can be used as an indicator of physical activity, however other factors are acknowledged to influence the results of such tests. Generally changes in cardiorespiratory fitness are used to validate changes in physical activity levels, even though other factors can influence the results of such a test, as Haskell and colleagues (1997) have previously reported. Physical activity has a greater impact on fitness than any of these other factors, so that by engaging in physical activity, the magnitude of their influence on cardiorespiratory fitness can be significantly reduced (Haskell et al., 1997).

Physical Activity and Health

With respect to mortality, primarily from CVD but also all-cause mortality, increasing evidence has indicated an inverse relationship to physical activity (Farrell, Braun, Barlow, & Blair, 2000; Ferrell et al., 2002; Janssen & Jolliffe, 2006). In the past few decades most of these studies have been cohort analyses examining the association between physical activity, all-cause mortality and CVD. Generally, subjects were grouped in physical activity categories based on self report or estimates of job classification and then the mortality rates for each category were determined over a specified time period. However, in the past several years epidemiological studies have begun to examine mortality rates of subject with regard to changes in their physical activity levels over time.

As mentioned previously, a sedentary lifestyle, a significant risk factor for overweight and obesity, is also a risk factor for CVD. Physical activity has been determined to have an inverse relationship with mortality from CVD (Lee & Paffenbarger, 2001). Unfortunately, many of these assessments were biased, as some limited their measures to only occupational activity, while others assessed only leisure activity. Those that did account for both occupational and leisure activity, often assessed only activities with traditionally male participants, failing to recognize leisure or occupational activities commonly associate with females. Additionally, a glaring shortfall of early physical activity assessment was the failure to account for activity pattern variation over time; however, more recent approaches to assessment of physical activity patterns have begun to account for whether patterns are static or dynamic. The use of questionnaires designed to capture recall of activity patterns over longer periods of time

(segmenting time into 1 to 10 year periods) rather than activity patterns at just one time point (i.e. past year, or past week recall of activities) has helped to account for these variations.

Taylor et al (1962) investigated the effects of occupational activity on CHD death rates in men (N=3043; ages 40-64) who were employed by the U.S. Railroad Industry between 1951 and 1956. In their studies they placed male subjects who had been employed for at least 10 years into two activity categories, clerks, whose occupational tasks were considered sedentary, and switchmen or sectionmen, who were considered physically active. The authors investigated mortality rates between 1955 and 1956, and found that there was a statistically significant difference in the age-adjusted death rates (per 1000 workers) for the two job categories (7.62 for section men, 10.29 for switchmen and 11.83 for clerks). A subsequent similar study of the railroad industry included data on male subjects (n=2562; ages 20-79) in seven countries between 1957 and 1960 with follow-up through 1977 (Taylor et al., 1978). In this study, leisure-time physical activity (LTPA), rather than occupational activity was assessed and related to CHD mortality. For assessment of activity level, the subjects completed the Minnesota Leisure-Time Physical Activity Questionnaire (MLTPA) which assessed the frequency and duration of participation in more than 50 different types of LTPA (such as backpacking, jogging, swimming, shoveling, etc.), and converted this data into estimated energy expenditure in kilocalories per week (kcal/week). In this questionnaire, activity assessment was designed to capture not just frequency and duration, but also categorically assessed intensity, dividing activities according to light-to-moderate and intense activities. This

study of LTPA also indicated an inverse relationship between increasing levels of activity and CHD mortality and revealed that the least active men were 1.21 times more likely to die from CHD than the most active men (95% CI 1.03-1.42). Even when CHD relative risk was adjusted to account for other risk factors including, smoking, blood pressure and cholesterol levels, the significant inverse relationship of LTPA and CHD mortality was maintained. Beginning in the early 1980's large investigations of the association of physical activity and CVD and CHD mortality took place. One of the most renowned of these studies was the Multiple-Risk Factor Intervention Trial (MRFIT), a prospective study of 12,138 middle-aged men at high risk for CHD (Leon et al., 1987). Men were classified according to average minutes per day of moderate intensity leisure-time physical activity reported at baseline and data were compared with cumulative CHD and all cause mortality endpoints at the 16 year follow-up. The results of this study indicated an inverse association between physical activity and CHD death rates. Although many of the limitations mentioned in previous paragraphs apply to this methodology (i.e. only accounting for LTPA; only looking at activity at one specific time point, but generalizing it to a pattern of activity for a longer duration; and, failing to account for dynamic nature of activity over time), an important shift in research focus occurred in this study. Although this was not the first research to indicate an inverse association between physical activity and CHD mortality, it was among the first to indicate that moderate intensity activity was related to a greater reduction in mortality rates than was vigorous activity (non-significant relationship to mortality reduction). However, the study did not account for occupational activity and did not include women.

To this point, studies had included primarily White men. In order to address this issue, Blair and colleagues (1989) included women as well as men in their Aerobics Center Longitudinal Study. Subjects were 4,034 men and 967 women who underwent at least three medical examinations [examinations included assessment of cardiorespiratory fitness by maximal exercise tests and measurement of body weight] between 1970 and 1984, and completed a mail-in health questionnaire during an average of ten years of follow-up. The focus of this study was the assessment of cardiorespiratory fitness, rather than physical activity levels, among mostly White middle to upper class men and women. The results indicated that there was a strong inverse association between fitness level and all-cause mortality, with the least fit women four times more likely to die than the most-fit women. In addition to the inclusion of women, the adjustment for smoking, systolic blood pressure, serum cholesterol, serum glucose, BMI and family history of CHD strengthened the applicability of the results and highlighted the far-reaching health implications of being sedentary.

A statement by the US Preventive Services Task Force report on health and exercise in 1989 indicated that the benefits of exercise for the prevention of CHD were well-documented for men, however the efficacy for women was only a presumption made on the basis of “extrapolation”. Even the 1996 US Surgeon General’s report on physical activity highlights this research deficit, as only three citations are included that utilize women as their primary population of study.

A subsequent increase in studies that included or focused on women is reflected in the more recent literature. The Iowa Women’s Health Study indicated an inverse

relationship between self-reported physical activity and mortality among post-menopausal women (Kushi et al., 1997). As well, the findings associated with the Nurses' Health Study (n=72,888), beginning in 1986 also indicated that among women ages 40-65 years old there was a significant inverse relationship between moderate or vigorous intensity exercise and coronary events (Manson et al., 1999). The risk of CHD for women in the Nurses' Health Study who walked briskly for a minimum of three hours per week or exercised vigorously for 90 minutes per week was reduced by 30-40 percent, regardless of obesity status.

The most recent ACSM recommendations for physical activity were published in 2006 in conjunction with the Centers for Disease Control and Prevention (CDC). At this time, several modifications were made to existing components of the ACSM guidelines. The aerobic component was increased to a minimum of 30 minutes of moderate intensity physical activity on most days of the week. Additionally, ACSM recognized that physical activity has an additive effect, and suggested that three 10-minute bouts of physical activity could provide health benefits similar to that obtained with one 30-minute session.

Currently, the 2006 ACSM guidelines remain in use. However, these physical activity guidelines are not uniformly agreed upon due to the introduction of recommendations from other well-regarded scientific groups. For example, the Institute of Medicine (IOM) recommends 60 minutes of moderate intensity physical activity each day. In addition, the President's Council on Physical Fitness and Sports (PCPFS) recommends 20 minutes of vigorous activity at least three times per week, whereas the American Heart Association recommends 30-60 minutes of physical activity five to seven

days per week. These competing recommendations have created a debate as to which guidelines should be adopted for public health initiatives.

Many American adults do not get the recommended amount of daily physical activity, despite its proven benefits. In fact, more than 60% of American adults do not attain adequate physical activity to provide health benefits (CDC/BRFSS, 2003). Moreover, 40% of adults are not physically active at all in their leisure time (ACSM, 2006). Physical inactivity is more prevalent among women than men, among Blacks and Hispanics than Whites, among older than younger adults, and among the less affluent than the more affluent. The statistical prevalence of inactivity also indicates that major public health efforts are needed to promote physical activity in the American population. This has led to the *Healthy People 2010* national health objectives to combat two of the leading United States health indicators: 1) physical inactivity and 2) overweight and obesity. These health indicators, along with eight others, are being used to measure the health of the nation from the year 2000 to 2010. As a group, the leading health indicators reflect the major health concerns in the United States at the beginning of the 21st century (*Healthy People 2010*, May, 2006).

Among the *Healthy People 2010* national health objectives, physical activity is listed as the number one leading health indicator. Thus, public health goals have been developed to improve levels of physical activity among adults, adolescents, and children to reduce sedentary behavior. According to the 1997 National Health Interview Survey (NHIS), only 15% of American adults aged 18 years and older engaged in moderate physical activity for at least 30 minutes 5 or more days per week. A goal of *Healthy*

People 2010 is to increase the proportion of adults who engage in regular, preferably daily, moderate physical activity for 30 minutes per day from 15% to 30% of the United States population. Another goal of *Healthy People 2010* is to reduce the proportion of adults who do not engage in leisure-time physical activity from 40% to 20% of the United States population and to increase the proportion of adults who engage in vigorous physical activity (>20 min, three days per week) from 23% to 30% of the United States population.

In addition to the *Healthy People 2010* initiatives, a recommendation originating in Tokyo, Japan, and adopted by United States public health agencies, involves accumulating 10,000 steps per day. The daily step count is typically recorded by a pedometer. This recommendation of 10,000 steps per day has become a popular means to both monitor and promote physical activity. Instead of engaging in physical activity for a specific duration (i.e. 30 min/day), this recommendation encourages individuals to walk 10,000 steps per day. For an adult, approximately 1,800-2,200 steps equals one mile. It has been found that on average, American adults walk approximately 4,000 – 6,000 steps a day (2-3 miles/day). Thus Americans must, on average, walk an additional 4,000 steps (approximately 2 miles) each day to reach the 10,000 step goal. At a brisk pace, 4,000 steps could be achieved in approximately 30 minutes. This duration is equivalent to the physical activity level recommended by the Centers for Disease Control and Prevention and the American College of Sports Medicine. The use of pedometers to record the number of steps accumulated in a specified time period serves as a principal focus of the present investigation.

Engaging in physical activity on a regular basis reduces the risk of developing the co-morbidities of obesity including coronary heart disease, hypertension, and diabetes. Adults who engage in moderate physical activity, such as walking or gardening, four or more times per week are 33 percent less likely to die than those who are inactive during the same period of time. The recognition of lifestyle physical activity as a preventative mechanism for obesity and other chronic disease by the President's Council was unprecedented. It was unprecedented as an alternative to programmed exercise or activity centered in a gym or health club, and was mainly a recommendation as an alternative to those who did not engage in any form of physical activity.

The beneficial impact of physical activity on various diseases varies widely. Past research links regular physical activity to the reduction of all-cause mortality primarily due to reduced risk of cardiovascular disease (Farrell, Braun, Barlow, & Blair, 2000; Ferrell et al., 2002; Janssen & Jolliffe, 2006). There is an abundance of evidence that links regular physical activity with other positive health outcomes on a variety of diseases and conditions (Bouchard, Shepard, & Stevens, 1994). The evidence for the positive association between regular physical activity and reduction of cardiovascular disease is just the beginning; it has also been implicated in the reduction of risk associated with hypertension, insulin resistance, hyperlipidemia, and obesity (Laaksonen et al., 2002; Franks et al., 2004). Physically active individuals have been associated with a much lower risk of developing adverse CV events compared to inactive people, independent of measures of obesity (Wessel et al., 2004). Physical inactivity has been recognized by the American Heart Association (AHS) as a major independent risk factor for the

development of CAD (Thompson et al., 2003). Due to the overwhelming consensus of health benefits related to physical activity in the literature, several national health agencies have issued statements to that regard. As mentioned previously, the first report of the Surgeon General of the US Public Health Service presented on physical activity and health was released in 1996. This report indicated that physical activity had positive health and economic implications for the country and the report encouraged individuals, families, health professionals, businesses, communities, schools and others to make a commitment toward the inclusion of physical activity in their daily lives.

The National Institutes of Health (NIH) also published a scientific-based consensus that concluded that regular, moderate, daily physical activity had significant health benefits for both children and adults alike. Previous statements from both the Centers for Disease Control and Prevention (CDCP) and the American College of Sports Medicine (ACSM) suggested that every individual accumulate at least 30 minutes or more of moderate-intensity physical activity on most days of the week to improve health (Pate et al., 1995; ACSM, 1990). An important shift should be noted here with respect to these recommendations. The previous ACSM guidelines were designed to improve aerobic capacity; however, the new statements all focused on the health benefits associated with physical activity. These recommendations would dispel previous misunderstandings about the intensity of exercise, as well as the mode of exercise that would cause a beneficial health response as opposed to increases in aerobic capacity for a beneficial fitness response.

Physical Activity and African American Women

African-American women, as previously discussed, have higher levels of overweight and obesity, and have experienced more extreme increases in overweight during the past decade when compared to White populations (Flegal et al., 2002). African American women have more barriers to overcome resulting in higher levels of physical inactivity than Caucasian women (Brady & Nies, 1999). African American women perceive less support for physical activity from their husbands and physician, and have fewer friends who are physically active (O'Brien Cousins, 1995). It is also culturally inappropriate to take time from family and work obligations for physical activity (Kriska & Rexroad, 1998; Marcus & Forsyth, 1998), moreover, many African American women consider rest more important for one's health than physical activity (Airhihenbuwa, Kumanyika, Agurs, & Lowe, 1995). Cultural and daily experiences play an instrumental role in shaping African American women's values and beliefs of the meaning attributed to physical activity. For African American women, physical activity is perceived as movement and being busy. It is regular, intentional, and beyond the typical movement of daily living for the purpose of improving oneself to the demands of daily work and home life (Tudor-Locke et al., 2003).

DiPietro & Capersen (1991) reported the comparison of physical activity patterns among African Americans and other ethnic minorities with those of White Americans. In this study, African American women were least active compared to White men and White women and African-American men. The intervention studies focusing on African-American women have many limitation, including: a) wide variations in study design; b)

inadequate replication of intervention components; c) lack of large sample sizes; and, d) vague descriptions of methodology and associated theoretical framework.

In 1985, Sullivan and Carter studied the effects of an eight-week intervention that included two-weeks of supervised exercise and a one-week educational/motivational component in 10 obese, low-income African-American mothers of young children. In their work the authors found that this minimal intervention strategy provided decreased resting heart rates, but that blood pressure and weight remained unchanged. Although the researchers noted decreases in resting heart rates, their results were not significant, likely due to the small sample size. The exercise and educational session methodology was not clearly identified, obviously prohibiting its replication by other authors in the future.

Many of the studies simply use either educational sessions, for which the description of information disseminated is often vague, or only print media blitzes are used to encourage changes in physical activity patterns. Smith and colleagues (1997) studied 22 obese, diabetic, older women (mean age 62 years, 41% African American) in a longer (16-week) educational program designed to target physical activity and diet. In this study the authors measured exercise frequency, body weight, BMI and glycosylated hemoglobin. Although the treatment group had significant decreases in glycosylated hemoglobin, indicative of improved metabolic control, there were no significant differences in other measured variables.

Additionally, as with many of the studies available for minority populations, the intervention framework is unclear. Kaul and Nidiry (1999) investigated the relationship of physical activity, dietary patterns and weight loss using a seemingly similar

motivational/educational strategy among 16 sedentary, obese, low-income adults (87% African-American women) in a seven-week clinical trial. In this case, rather than group sessions, the sessions were individualized, and although significant weight loss was reported between pre- and post-test data, the theoretical framework still remains unclear.

Investigations integrating exercise sessions as a component of study also emerged more frequently for minority populations beginning in the mid 1980's. A study by Blumenthal et al (1989) reported data on 101 African-Americans, a majority of which were women (n=98; mean age = 67 years), using random assignment to one of four treatment groups including 1 week of yoga, 3 weeks of yoga, 1 week of moderate-intensity aerobics or 3 weeks of moderate-intensity aerobics. The exercise sessions in this study, though short, were supervised and individuals were required to complete homework assignments as an educational component. Though exercise integration was added as a tangible component, regulation of intensity of exercise, and again, the theoretical framework for the educational component were not well defined. The authors did report that each of the 3-week activity groups had significant decreases for systolic blood pressure compared to the groups meeting one-week and controls (those who did not attend sessions).

Cardinal and Sachs (1996) compared the effectiveness of three forms of mail-mediated exercise behavior change strategies on weekly leisure time exercise behavior. The sample consisted of 113 healthy females (Mean Age= 36.9; 62.8 percent African American) employed full-time in a clerical occupation at a major research university. Of those accepted in the study, seventy percent completed all data collections and were

included in the analysis. The authors classified subjects according to a stages of change scale based on the Transtheoretical model of behavior change, and randomized each of the participants to one of three interventions: 1) a lifestyle exercise packet, which encouraged subjects to incorporate more physical activity into their daily lives; 2) a structured exercise packet that encouraged a standard exercise prescription with recommendations for intensity, duration and frequency; 3) a control packet with feedback forms on personal fitness from baseline data. Information was tailored to each stage by using the processes of change associated with the categorized stage. Data was collected after the first and sixth month; otherwise no contact occurred between subjects and investigators. The authors reported a significant weekly leisure-time exercise behavior main effect for both structured and lifestyle groups ($P < 0.05$). Weekly leisure time METS in the lifestyle group (pre= 18.6 ± 23.6 , post 30.7 ± 18.5) were significantly larger than the control group ($P < 0.01$). The Tukey's post-hoc analysis indicated no significant difference in leisure time activity in the structured or control groups ($P > 0.05$). The researchers identified that the leisure packet was more effective than the structure or control packets. Although all subjects, except the control, demonstrated increases in physical activity, only the lifestyle group increases were statistically significant. Subjects that were initially classified in the pre-contemplation/contemplation stages showed the most improvements, with those in the preparation group showing the least improvement. Study adherence rates were 81.1 percent for the lifestyle group, 71.1 percent for the control group and 63.2 percent for the structured group. Although the number of studies designed to promote a

more active lifestyle in minority women is growing, additional research is needed to clarify what interventions are the most effective in this population.

Intensity of Activity Necessary for Health Benefit

With respect to the 1996 U. S. Surgeon General's Recommendations and the focus on health related benefits of physical activity following this report, it is important to include a review of these recommendations for quantity and intensity of physical activity. As mentioned previously, the primary focus of most research prior to 1996 had been vigorous activity or activity that was designed to improve cardiorespiratory fitness. Duncan et al (1991) conducted a 24-week randomized clinical trial of sedentary women involved in a walking program or control group (remaining sedentary) in which the walking groups' distance and frequency remained constant across three treatment groups, while intensity varied between the groups [strollers (4.8 km/hour), brisk walkers (6.4 km/hour), and aerobic walkers (8.0 km/hour)]. Cardiorespiratory fitness changes were assessed via changes in maximal oxygen uptake, which increased across treatment groups relative to the intensity of activity in which participants were engaged. Health indicators, such as cholesterol and percent body fat, decreased similarly in both aerobic and brisk walkers, indicating that moderate activity could result in positive health benefits.

Similarly, the Nurses' Health Study also assessed walking patterns of women, and indicated that moderate-intensity activity provided health benefits (Manson et al., 1999). In this study of 70,102 women in the U. S., it was noted that the relative risk of developing diabetes was comparable for both moderate and vigorous intensity walking. When activity levels were divided into quintiles, relative risk for (all-cause or CHD)

associated with the lowest activity to the highest activity levels were 1.0, 0.91, 0.73, 0.69 and 0.58 ($p < .01$) respectively.

More recently, data from the Harvard Alumni study was analyzed to determine the relationship between light, moderate, and vigorous intensity physical activity and mortality (Lee & Paffenbarger, 2000). The data was originally collected from 16,936 men (ages 35-74 between 1962-1966) as a prospective cohort study of CHD predictors among undergraduates free from cardiovascular complications. The original assessments included detailed questionnaires about their daily physical activity, such as number of stairs climbed, city blocks walked, and their participation in sport and recreational activities. Data from the questionnaires were then classified into light (5 kcal/min energy expenditure), vigorous (10 kcal/min energy expenditure) or mixed (7.5 kcal/min energy expenditure) activities. The follow-up study, tracked men until 1992 ($n=13,485$), and a multivariate analysis indicated that distance walked and stairs climbed were independent predictors of longevity.

While vigorous intensity activities demonstrated a clear association with lower mortality rates, moderate intensity activity showed a similar trend for lower mortality rates. In fact, moderate levels of physical activity display significant health benefits in the prevention and treatment of obesity-related diseases (Lee & Paffenbarger, 2000; Manson et al., 1999). Researchers estimate physically active individuals to have half the risk for coronary heart disease as their sedentary counterparts (AHA, 2003; Shephard, 2001). Evidence also suggests other protective benefits of moderate intensity exercise including

the prevention of: hypertension, stroke, type II diabetes, osteoporosis, and some forms of cancer (AHA, 2003; NIH, 1996; Shephard, 2001). Moderate levels of physical activity may also enhance immune function and mental health, in addition to improving flexibility, endurance and muscle strength, which aid in the prevention of injury and disability (AHA, 2003; NIH, 1996; Shephard, 2001; and, DHHS, 2000).

Quantity of Physical Activity Necessary for Health Benefit

According to the U. S. Surgeon General's Report on physical activity and health (1996) most large prospective studies indicate that the benefits of physical activity are dependent on the initial activity level of the participants. Thus, the greatest health benefits associated with activity level are observed in previously sedentary individuals. According to the recommendations of the ACSM and the CDC, the health benefits of physical activity are directly proportional to the quantity of activity performed, measured either by duration of activity in minutes or total kilocalories expended (ACSM, 1990; DHHS, 2000).

The Harvard Alumni Health study indicated that men who expended greater than 2000 calories each week were at significantly lower risk for CHD mortality than men who expended less than 500 calories each. Leon et al (1987) and Slattery et al (1989) reported that average increases in energy expenditure of only 250 calories per week through moderate activity were associated with reduction of CHD mortality by 36 percent and 27 percent, for each study respectively.

While a great deal of evidence exists associating physical activity with decreased risk of cardiovascular disease and all-cause mortality, less is available regarding the

association of exercise intensity and insulin sensitivity. The Insulin Resistance Atherosclerosis Study (N=1467) of Non-Hispanic White, Hispanic and African-Americans (38, 28, and 34 percent participation respectively by race), is one of the few studies that compared the caloric expenditures through physical activity sorted by insulin sensitivity levels in men and women (Rewers et al., 2004). The research involved self-reported vigorous activity, as well as 1-year recall interviews. The reported activity was then classified according to intensity of activity using METs, and energy expenditure was determined. The results, after adjusting for potential confounders, indicated that frequency of participation in vigorous physical activity was associated with greater insulin sensitivity between the lowest activity and highest activity groups (0.90, [95% CI 0.83-0.97]; 1.59 [95% CI 1.39-1.79]); however, neither total energy expended nor non-vigorous activity were associated with increases in insulin sensitivity.

Although there does appear to be a dose-response relationship between energy expended in physical activity and reduced CHD mortality, the absolute difference between the number of calories expended in activity and different intensities of physical activity has not been determined.

Increasing energy expenditure through increased physical activity can also result in weight loss which can indirectly but favorably change the individual cardiovascular risk factors (Dattilo & Kris-Etherton, 1992; Leenen et al., 1993; Chobanian et al., 2003; Yu-Poth et al., 1999). In 1986 a cornerstone study comparing the effects of diet only versus exercise only was carried out by Hagan, Wong and Whittam (1986). In this study the investigators assessed the comparison of the amount of weight lost through diet only

to the amount of weight lost through physical activity in obese men and women. The average weight loss by men and women, 5.5 and 8.4 kg respectively, was achieved by a smaller caloric reduction in women as compared to men, 945 kcal and 1705 kcal respectively. Additionally a group of men and women who exercised through a walk/jog regimen performed 30 minutes each day on 5 days of each week expended 190 kcal and 255 kcal per session [for women and men respectively] to achieve a their weight loss averages of 0.6 kg for women and 0.3 kg for men. Conversely, Sopko et al., (1985) had demonstrated the contrary in a 12 week diet versus exercise program where the male exercise participants expended 500 kcal/day in treadmill walking and reduced their weight by 6.2 kg compared to those who reduced caloric intake by 500 kcal/day to experience 6.1 kg weight reduction. This study demonstrated equivalent response for weight loss through both diet and exercise; however, the number of subject in the study was low, reducing the ability to generalize these finding. Despite these questions, the Surgeon General's Report (1996) suggested, based on the evidence in existence, that participation in activity which leads to an increased daily expenditure of 150 kcal in non-vigorous activity is associated with health benefits. During the same time, Ross et al. (2000) produce a different recommendation based on a review of the current literature. As a conclusion to this review considering the use of exercise without weight loss for health benefit, these authors suggested that moderate-intensity activity (such as brisk walking) for 30-60 minutes per day on most days of the week was a more effective regimen to improve health. Also, the authors suggested 45-60 minutes of moderate intensity exercise each day for "substantial" reductions in obesity and related co-morbidity, with caution

that most of the research can only be generalized to males, which had been the predominant gender of study to that time (Ross et al., 2000).

Continuous Vs. Intermittent Bouts

Prior to the recommendations of the Surgeon General in 1996, only a handful of major research studies directly compared intermittent and continuous bouts of activity (Ebisu, 1985, Debusk et al., 1990; and Jakicic et al., 1995). Of these three studies, the primary outcome measurement was cardiorespiratory fitness. Debusk et al. (1990) compared the effects of three separate 10-minute bouts of moderate to vigorous activity (65 to 75% of peak treadmill heart rate) with one continuous 30-minute bout of activity of equal intensities in two groups of 18 men (N=36; age 45-57 years). During the 8-week study period VO₂ max increased significantly in both groups from 33.3 ± 3.2 to 37.9 ± 3.5 ml/kg/min in men performing long exercise bouts and from 32.1 ± 4.6 to 34.5 ± 4.5 ml/kg/min in men performing short exercise bouts (p < 0.05 within and between groups). Ebisu (1985) randomized men into one of four treatment groups, three exercise groups and one control group. In each of the exercise groups, running distance was the same but the number of sessions used to accumulate that distance varied by group, using a one-, two- or three-session treatment design. This study identified significant increases in cardiorespiratory fitness without significant difference between groups. The relative positive fitness effect on all exercise groups would suggest that short bouts, which could be more conveniently incorporated into activities of daily life, were also beneficial although increases in fitness were greater with longer bouts. In addition to the assessment of cardiorespiratory fitness, this study also noted improvements in LDL-cholesterol levels

for women [but not men] in the one- and two bout group compared to an increase in the control and non-significant reduction in the three-bout groups. Ebisu and colleagues (1985) suggest that walking in 15 minute bouts, at a brisk pace, may be comparative in effect to traditional 20-40 minute bouts of exercise.

Similar results for accumulated exercise bouts were found for women using moderate intensity walking as the exercise mode. Jakicic and colleagues (1995), in a design similar to DeBusk (1990), compared the effect of three 10-minute bouts to one 30 minute bout of moderate intensity walking on cardiorespiratory fitness, exercise adherence and weight loss in a group of overweight women (N=46) between the ages of 35 and 50. After 20 weeks, cardiorespiratory fitness improved significantly in both groups ($p < 0.05$) and weight loss was similar, but not significant in either group. Interesting to note, exercise adherence was greater in the intermittent group than in the continuous group.

After the 1996 Surgeon General's Report more research interest was taken with respect to the accumulation of physical activity, particularly in women, although these studies have produced mixed results. Snyder et al (1997) published their work investigating the effects of long-term moderate intensity intermittent exercise on health (VO_2 max, cholesterol, insulin and glucose) in overweight women. In this work the authors found that moderate intensity, intermittent brisk walking (three 10-minute sessions each day, 5 days per week) for 32 weeks, was not sufficient to significantly increase aerobic capacity, produce weight loss, change body composition or improve blood lipids, insulin or glucose. However, individuals who were older and less fit

individuals did show increases in aerobic capacity and decreases in body fat. Moderately obese women (N=22) who participated in an 18-week study to investigate the effects of intermittent and continuous bouts of moderate walking experienced some significant results (Donnelly, Jacobsen, Heelan et al., 2000). In this study, subjects performed continuous exercise at 60-75% of maximum aerobic capacity, 3 days per week, 30 min per session, or exercised intermittently using brisk walking for two, 15 min sessions, 5 days per week. Significant improvements for aerobic capacity of 8% and 6% were shown for the continuous and intermittent exercise groups, respectively. Weight loss for the continuous exercise group was significant at 2.1% from baseline weight and the intermittent group was essentially unchanged. The continuous group showed a significant decrease in percentage of body fat and fat weight while the intermittent group did not. HDL cholesterol and insulin were significantly improved for both groups. Previously in a study of similar design with respect to duration (18 weeks) and mode of activity (“brisk walking”), Woolf-May et al. (1999) used long (20-40 min/bout), intermediate (10-15 minutes/bout) and short (5-10 min/bout) walking bouts to demonstrate that fitness changes occurred similarly across groups with 149-157 minutes of brisk walking each week. Similar to Debusk et al. (1990), this study revealed a gender bias in that men experienced more positive changes in cholesterol values than women.

Measurement of Physical Activity

Just as the independent terminologies of physical activity and exercise are erroneously used interchangeably, there is similar confusion with regard to the measurement of physical activity and the measurement of energy expenditure. The

measurement of physical activity and energy expenditure are not synonymous, however, many researchers treat them as such. Physical activity, as previously described, is movement. Energy expenditure is related not only to physical activity (movement), but also gender, age, body mass and efficiency of movement (Bursztain, Elwyn, Askanazi et al., 1989). Energy expenditure and/or physical activity may be assessed by various measures including; calorimetry, motion sensors, doubly labeled water, diaries, logs or records, fitness measurements, metabolic measurements, heart rate, or self-report by questionnaires and surveys.

Doubly Labeled Water

Doubly labeled water is the gold standard for assessment of energy expenditure; however the use of doubly labeled water is both cost and time prohibitive for most researchers. The energy expenditure of a single bout of exercise can be estimated by measuring O₂ consumed using an indirect calorimetry system. This method can use either a closed circuit (isolating the individual from outside air) or open circuit (using a collection mechanism to analyze inspired and expired air) technique. In either method, the volume of oxygen consumed is measured to reflect the metabolic rate. The closed circuit is best utilized to establish RMR, but the open circuit method is best suited for prolonged or strenuous conditions of physical activity (Ainslie et al., 2003). Neither method is practical for collection of data during activities of daily living due to the complexity and size of the equipment. Additionally, though portable units are available, the cost and complicated nature of the device makes it unreasonable for use with multiple study participants.

Heart Rate Monitors

Although heart rate does not measure energy expenditure directly, the linear relationship between oxygen uptake and heart rate during exercise allows for the estimation of energy expenditure from recorded heart rate. Since heart rate measurement, specifically through fairly inexpensive monitoring devices, is relatively simply accomplished, this method of data collection has garnered widespread use among researchers. Certain barriers to the estimation of energy expenditure do exist using this method, some of which include, fitness levels, muscle mass, continuous versus intermittent activity, mood, dehydration, illness, ambient temperature, as well as postural changes (sitting, standing, or lying down) to name a few (Davidson et al., 1997; Melanson & Freedson, 1996).

Pedometers

Due to the public health input in developing physical activity recommendations, physical activity monitoring has become increasingly popular. It is recognized that the most common form of physical activity assessment uses self-reported recall questionnaires. However, there are limitations regarding the individuals' ability to accurately recall all physical activity performed over a specified period of time. Thus, physical activity monitors, such as pedometers, are now being used to more objectively measure physical activity.

The use of uniaxial or triaxial accelerometers to measure physical activity has become more common in research literature. The accelerometer relies on the distortion of piezoelectric ceramics to register electrical charges that, depending on the orientation of

the piezoelectric ceramics, can measure movement in one or three planes (Wixted et al., 2005). These small devices may be worn on the waist, hip, low back or ankle in order to record activity with respect to both frequency and intensity (inferred from velocity of movement). The data from some of these devices may be subsequently downloaded for computer analysis. Estimations of energy expenditure may be made based on pre-programmed physical characteristics of each subject. The difficulty of equating energy expenditure and physical activity is highlighted by the failure to validate raw activity data with direct measures of energy expenditure, rather correlations have been shown between estimated total energy expenditure from accelerometry and direct calorimetry or doubly labeled water methods (Swartz et al., 2000; Trost, McIver & Pate, 2005).

Pedometers are inexpensive, light-weight and more feasible accelerometers that measure physical activity by responding to vertical accelerations of the hip during gait cycles (Schneider, 2004). Pedometers are a more basic version of multi-axial accelerometers that include a horizontal, spring suspended lever arm that is displaced in the vertical plane capturing the movement of the hip within the repetitive gait pattern. An electrical circuit closes with each movement detected and an accumulated step count is displayed digitally on a feedback screen. Pedometers do not however, record velocity or intensity of movement, restricting their use to measuring only total accumulated steps/day (President's Challenge Report, 2006).

The accurate measurement of physical activity is essential for research studies in which physical activity is an outcome variable. According to Freedson (2005), observation or surveillance of physical activity must be conducted with a robust activity

measure in order to establish a relationship of physical activity to any specific outcome. In addition, physical activity interventions require an accurate measure of activity dosage in order to establish dose-response relations that explain intervention effectiveness. Such accurate assessments of physical activity are necessary if the physiologic mechanisms linking physical activity and health are to be completely understood (Freedson, 2005).

According to manufacturers' recommendations, pedometers should be worn on a waistband or placed horizontally in a pocket midway between the umbilicus and the hip. Most pedometer models provide data on the quantity of steps taken, distance traveled, and estimated energy expended. Typically, the pedometer user must manually enter a number of variables including gender, stride length, weight, and/or age in order to derive estimated kcal expenditure from accumulated step counts.

Pedometers have been found to be a valid method for assessing ambulatory modes of physical activity such as walking and climbing stairs (Tudor-Locke et al., 2002). When comparing pedometers to others methods of physical activity monitoring, Tudor-Locke and colleagues (2002) found a high correlation between pedometer step counts and accelerometer counts ($r = 0.86$), physical activity observation ($r = 0.82$), energy expenditure ($r=0.68$), but lower correlation with self report physical activity questionnaires ($r = 0.33$). The National Institutes of Health recommends the use of pedometers as a means to ensure 10,000 steps a day for optimal health (NIH, 2005). However, while pedometers accurately measure motion of the hip flexor, they do not accurately measure exercise intensity. So although pedometers can be useful tools, they do not account for

differences in intensity of walking exercise, and thus do not accurately measure kcal expenditure.

Pedometers have been found to capture changes in lifestyle ambulatory behaviors that are not typically considered exercise, but in fact help to increase energy expenditure. Significant improvements have been noted in weight management, insulin sensitivity, blood pressure, and lipid profiles, as a result of participation in physical activity interventions using pedometers as motivational tools (Tudor-Locke et al., 2002; Talbot et al., 2003; Moreau et al., 2001). Although most research to date has involved physical activity assessment, pedometers may also serve to motivate increased activity levels by increasing cognitive awareness (U.S. Dept. of Health and Human Services, 1996) and self efficacy (Tudor-Locke et al., 2002). For example, a pedometer can be used as a tracking device, a feedback tool, and as an environmental cue for physical activity participation. Used in combination with written physical activity tracking logs, pedometers have been found to be an effective way to increase daily physical activity (Tudor-Locke, Myers, & Rodger, 2001).

For example, Croteau (2003) examined the effects of an 8-week, pedometer-based intervention on lifestyle activity. Participants were 37 college employees who volunteered to participate in the study. The intervention consisted of goal setting, pedometer use, self-monitoring, and weekly e-mail reminders. Physical activity measures (pedometer, survey) were taken at baseline and immediately following the intervention. Results indicated a significant increase in average daily steps from 8,565 (SD \pm 3121) steps at baseline to 10,538 (SD \pm 3,681) steps at the end of the program. This study indicates that a

pedometer-based lifestyle intervention was effective in increasing the daily physical activity of adults.

Speck and Looney (2001) investigated the effect of a self report intervention on activity levels in a community sample of working women. Using a longitudinal, pretest-posttest design, 49 working women were randomly assigned to the control (n = 25) or intervention group (n = 24). At pretest and posttest, subjects completed self-report questionnaires that measured psychological, social-environmental, physical activity, and demographic variables. Subjects in the intervention group kept daily records of their activities during the 12-week study, while those in the control group kept no records. In order to compare activity in the two groups, all subjects wore pedometers that recorded number of steps taken daily. The control subjects were blinded to the digital display of their daily step count. In contrast, the intervention group was informed of their daily step counts. Results indicated that the step count was significantly higher in the intervention than control group ($2,147 \pm 636$ steps). It was concluded that pedometer step count monitoring is a cost-effective and acceptable intervention that may increase activity levels in women.

Studies have also indicated that walking speed may have an effect on the accuracy of pedometer counts, distance covered, and kcal expenditure. Crouter et al. (2003) found that pedometers were less accurate at slower speeds (54 m/min) than at faster speeds (80 m/min) due to the less pronounced vertical accelerations of the waist at slow walking speeds (Crouter, 2003). It was also found that pedometers tend to overestimate distance traveled at slower speeds and underestimate distance traveled at faster speeds, with 80

m/min being the most accurate speed for most pedometers (Crouter et al., 2003).

Furthermore, data suggests that pedometers underestimate kcal expenditure during lifestyle activity (i.e. gardening, housework) (Crouter et al., 2003).

Although limited, recent studies indicate that the use of a combination of both accelerometers and heart rate monitors may limit the variability of heart rate data alone. Rennie and colleagues (2000) used this combination resulting in surprisingly close calculations of energy expenditure when compared to results from direct calorimetry. Similar research has shown that these combined methods, using two devices rather than one integrated unit, can also be valuable in decreasing variability of energy expenditure calculations (Haskell, Yee, Evans et al., 1993; Speakman, 2001; Strath, Bassett, Thompson et al., 2002). One advantage of the combined devices is the ability of the accelerometer to act as a backup measure that can be used to verify that elevations in heart rate were in response to physical activity (Ainslie, Reilly, & Weterterp, 2003).

METS

The average adult has a resting metabolic rate (RMR) of approximately 3.5 ml/kg/min of oxygen, or 1 kcal/kg (body weight)/hr (Ainsworth, Leon. Richardson et al., 1993). The expression of a MET, described with regard to quantity of physical activity needed for health benefits, is simply the energy cost of activity in multiples of RMR (Hill and Saris, 1998). As mentioned previously, the advantage of using METs is that it also allows the characterization of the intensity of an activity, rather than frequency and duration alone. Using a log of activities over time, MET values can be used to estimate energy expenditure by reference to extensive resources that provide MET values for all

types of activity. These data are one of the most efficient and economical methods of comparing activity levels between individuals. This method of estimating energy expenditure is frequently employed through the use of self-report methods.

Self Report

The use of self-report can be one of the quickest and most economical methods of physical activity data collection. Self report measures may take the form of surveys, questionnaires or logs used to quantify physical activity. Some of the advantages of surveys are their low cost, the participant time requirement is minimal, and they can be used to assess a wide variety of activities over different life periods. Unfortunately, the existence of large recall bias, and the inability to recall activity accurately can negatively influence the value of these instruments (Ainsworth, Sternfeld, Slattery et al., 1998). Kriska and Casperson (1997) describe more than 30 self report instruments and indicate that no single instrument is appropriate for all populations or purposes. Physical activity surveys and questionnaires often fail to reflect the types of activities done by women, or may perhaps fail to account for variation of cultural interest. Failure to include activities that are specific to the population of interest may lead to inaccuracies in research (Ainsworth et al., 1998). With specific respect to the measurement of physical activity in sedentary populations, few of the questionnaires or surveys include more spontaneous or routine light to moderated activities (Tudor-Locke & Myers, 2001). Additionally, most fail to account for intermittent or incidental activities, which encompass many of those activities that have been recommended for health related benefits (U. S. Department of Health et al., 1996). The Stanford 7-day physical activity recall (7-day PAR) is often used

for the self-reported measurement of physical activity (Blair, Haskell, Ho et al., 1985). The 7-day PAR, however, does not account for activity that is accumulated in bouts less than 10 minutes, nor does it include activity of an intensity which is less than that of brisk walking. Although walking has been reported to be one of the least reliably recalled physical activities, most self-report methods assess walking frequency (Kriska, Knowler, LaPorte et al., 1990; and, Ainsworth, Leon, Richardson et al., 1993). In addition to the lack of reliability of recall, variability has also been reported with respect to difficulty in assessing speed or intensity of walking, which is important since most reported energy expenditures are calculated from MET values which correspond to various intensities of walking activity (Tudor-Locke & Myers, 2001).

While physical activity logs (recorded at the time of activity) can address the concerns of recall, definitions of intensity must be reasonably addressed in order to more accurately reflect energy expenditures from this instrument. Taylor et al. (1984) indicated that a self-report log used in conjunction with an interview-based seven-day recall might maximize accuracy of recall, and also account for discrepancies in defining intensity of activity. The time period over which a log is completed may also account for variation in reported activity patterns. The results of a study by Eason and colleagues (2002) indicated the need for more than seven days of diary self-report to achieve reliable estimates of total activity in African-American and Hispanic women.

Lifestyle Activity Modification

The concept of lifestyle physical activity has emerged as a reaction to the U.S. Surgeon General's Report, directives on health and wellness by the CDC, ACSM and the

NIH, and as a result of a shifting research emphasis from cardiovascular fitness improvements to promoting the health benefits of exercise. The basis of this was in part due to large prospective studies that indicated the positive effect of lower intensity physical activity on cardiovascular disease and all-cause mortality. This methodology has gained increasing momentum due to the association of obesity and obesity related disorders with sedentary behavior. Additionally, research indicating that accumulated bouts of activity promoted greater adherence among sedentary populations also lent credence to lifestyle modification modalities. Dunn and colleagues (1999) defined lifestyle physical activity in light of the research literature as follows:

“Lifestyle physical activity is the daily accumulation of at least 30 minutes of self-selected activities, which includes all leisure, occupational, or household activities that are at least moderate to vigorous in their intensity and could be planned or unplanned activities that are a part of everyday life.”

The amount and accumulation of activities set forth in this definition are a reflection of the concepts of physical activity for the promotion of health as described by the U.S. Surgeon General’s Report on Physical Activity. Individuality is important to the concept of lifestyle physical activity as the self-selection of activities by the participant and integration in daily life make up two key components in the definition. The flexibility of the lifestyle activities methodology allows for inclusion of not only individual differences, but also differences in culture and environment of participants.

The Finnish Diabetes Prevention Study was a controlled trial in which 522 persons with impaired glucose tolerance (172 men and 350 women; mean age, 55 years; mean body-mass index 31) were randomly assigned to either a two-year control or

intervention group (Tuomilehto, Lindstrom, Eriksson et al., 2001). The goals of the intervention group were: 1) a five percent or greater reduction in weight; 2) decrease total fat intake to less than 30 percent of energy consumed, while reducing the intake of saturated fat of less than 10 percent of energy consumed; 3) an increase in fiber intake to at least 15 g per 1000 kcal; and, 4) participation in moderate exercise for at least 30 minutes per day. Exercise could be either aerobic exercise (such as walking, jogging, swimming, aerobic ball games, or skiing) or resistance training (circuit training). In the five years following baseline assessment the absolute incidence of diabetes was 32 cases per 1000 person-years in the intervention group and 78 per 1000 person-years in the control group. In this trial, lifestyle modification addressed a combination of variables including, cardiovascular endurance, muscular strength and diet and therefore, the effect of any single variable cannot be analyzed.

Another trial, also incorporating individuals with impaired glucose tolerance was conducted in China (Pan, Li, Hu et al., 1997). In this study 577 persons were randomly assigned to one of four groups: 1) control, 2) diet, 3) exercise, or 4) diet and exercise. Instructions to the diet group were to maintain a diet composed of 25-30 kcal/kg body weight, 55-65 percent carbohydrate, 10-15 percent protein and 25-30 percent fat. Additionally, participants who had a BMI of 25 or more were encouraged to lose 0.5- 1.0 kg per month until they attained a BMI of 23 kg/m². For the exercise group, participants were encouraged to increase their level of physical activity one unit per day, and at least 2 units per day for individuals under 50 years of age (A unit of physical activity is defined in Table 3). The incidence of diabetes at six years was 67.7 percent in the control group,

43.8 percent in the diet only group, 41.1 percent in the exercise only group, and 46.0 percent in the diet and exercise group. This study allows a greater comparison among intervention variables than the previously mentioned study, however the trials, which were conducted in over 33 centers across China, were not well controlled. Clinic staff was involved in a two-day training seminar; however, it does not appear that standardized educational materials were disseminated and clinicians were free to adapt nutritional and exercise prescriptions. The studies were randomized by clinic rather than within individuals at each clinic, which could have created some bias in the study results. The origin of the definition of one “unit” of exercise is unclear, and therefore, results are difficult to compare with other studies.

Table 3. Activities required for one unit of exercise (From Pan et al., 1997)

Intensity	Time (min)	Exercise
Mild	30	Slow walking, traveling by bus, shopping, housecleaning
Moderate	20	Faster walking or walking down stairs, cycling, doing heavy laundry, ballroom dancing (slow)
Strenuous	10	Slow running, climbing stairs, disco dancing for the elderly, volleyball or tennis
Very Strenuous	5	Jumping rope, basketball, swimming

Project Active was a longitudinal study that evaluated whether a structured exercise group (S) versus a lifestyle group (L) would meet the recommended 30 minutes

of accumulated physical activity (Dunn, Garcia et al., 1997). A sample of 235 healthy, community dwelling women and men were randomized into either the structured or lifestyle group. The structured group (n=114) exercised vigorously 3-5 times per week at local health club. The lifestyle group (n=121) attended classes to learn how to incorporate more daily physical activity one hour per week for 16 weeks, then one hour every other week for the next 8 weeks. There is no mention of the ethnicity or gender make up of either group. However, the study included 14% African Americans in the overall sample. At 6 months, both groups demonstrated similar and significant changes for decreased sitting time (L=9.3%; S=12.2%); increased stair climbing (L=64%; S=30.2%); increased minutes walked (L=25%; S= 20.9%); and increased maximal oxygen uptake (L=18%; S=31%). Both groups had significant reductions in serum cholesterol and lipid levels, blood pressure, and body fat. By 24 months, both groups significantly maintained their increased level of physical activity (L=25%; S=30%) and significantly increased stair climbing (L: p=.003; S: p=.009) (Dunn, Blair et al., 1997). The principle finding was that the lifestyle group had similar changes in all measures compared to the structured group, and accumulating 30 minutes of physical activity would be a means to encourage sedentary individuals to adopt and maintain physical activity.

The authors concluded that the lifestyle activity is as, if not more, effective than structured exercise and should be widely disseminated. This conclusion may be inappropriate given the study design and results. First, the changes were not clinically impressive given the length of the study and the modes of activity for the lifestyle group were unclear. Determining the activities of the lifestyle group is imperative, as they may

have chosen to engage in structured activities independently. Also unreported in these findings was information specific to accumulated or continuous bout activities for the lifestyle group, which is a distinguishing guideline of the current lifestyle recommendations. The intervention procedures, related to the behavioral component of the lifestyle activity group specifically also remains unclear; it is therefore difficult to conclude whether the lifestyle activity itself promoted adherence or if better continued adherence to the lifestyle activity mode was a result of cognitive/educational intervention among the study participants.

Another study examined the short and long-term changes in body composition and maximal oxygen uptake by comparing a diet with structured aerobic group (A) to a diet with lifestyle physical activity group (L) (Andersen et al., 1999). Forty sedentary, obese women, including 10 African Americans, age range 21-60 years, were randomized to either group. The lifestyle physical activity group (n=20) was taught to incorporate short sessions of physical activity into their daily lives, such as taking stairs more frequently and walking short distances instead of driving. The diet and structured aerobic program group (n=20) attended a one-hour step aerobic class, 3 times per week for 16 weeks. By the end of the 16-week intervention, the lifestyle group increased their daily activity by 28%. At 16 weeks, both groups demonstrated significant changes in weight loss (L=7.9 kg; A=8.3 kg); reduced body fat (L=6.2%; A=4.3%); and increased maximal oxygen uptake (L=16.2%; A=18.8%). At 68 weeks, there were still significant changes in maximal oxygen uptake (L=24.2%; A=16.3%) and neither group significantly regained any weight (L=0.08kg; A=1.6kg) from baseline. The authors concluded that diet plus

lifestyle physical activity offered similar health benefits as diet and vigorous activity.

Increasing lifestyle physical activity may be a better alternative than vigorous activity for sedentary, obese, African-American women.

One of the most significant trials, bolstering the effects of exercise over pharmacological treatments in the prevention of type II diabetes, was the three-year multi-center, randomized clinical trial to determine whether the effects of lifestyle intervention would prevent or delay the onset of diabetes in high risk individuals (DPP Research Group, 2002). The Diabetes Prevention Program (DPP) demonstrated a decrease in the incidence of type II diabetes by 58 percent in the lifestyle group, compared to a decrease of only 31 percent in the metformin group (DPP Research Group, 2002). The two major goals of the study were focused on weight loss and physical activity. The goal for weight loss was a reduction in baseline body weight by seven percent; and, the goal for physical activity was the increase in energy expenditure from baseline of at least 700 kcal per week from physical activity, or the participation in at least 150 minutes of moderate-intensity physical activity each week. On average, the lifestyle intervention group maintained their physical activity at 30 minutes per day, usually with walking or other moderate intensity exercise, and lost 5-7 percent of their body weight. This landmark study, on the advice of the DPP's external data monitoring board, ended a year early because the data had clearly answered the main research questions. Although the results were similar to the smaller Chinese and Finnish studies, this was the first major trial to show that diet and exercise could effectively delay diabetes in a diverse American population of overweight people with impaired glucose tolerance (IGT). Of the 3,234

participants enrolled in the DPP, 45 percent were from minority groups that suffer disproportionately from type 2 diabetes: African Americans, Hispanic Americans, Asian Americans and Pacific Islanders, and American Indians.

The Activity Counseling Trial (ACT) was a multi-site trial that examined the effects of three physical activity interventions, *advice*, *assistance*, or *counseling*, on fitness and physical activity (The Writing Group for the Activity Counseling Trial Group, 2001). The study consisted of an intervention group solely given advice for recommended care from a physician (Advice, n=290), assistance, which was advice from a physician plus interactive mail and behavioral counseling during the physician visits (Assistance, n=290), or counseling (Counsel, n=290), which was Assistance and Advice plus telephone counseling and behavioral classes (The Writing Group, 2001). The physical activity recommendations for all groups was 30minutes of moderate intensity 5 days a week or 20 minutes of vigorous intensity activity 3 or more days each week. As a result of the intervention female participants' VO₂max levels increased significantly in the Assistance and Counsel groups when compared to the Advice group. There were no fitness differences between the Assistance and the Counsel groups and no differences in reported physical activity. For men there were no significant differences among any of the three arms in fitness or physical activity.

Walking Studies

Most lifestyle intervention studies use “moderate” intensity or “brisk walking” as their primary mode of physical activity, but many also include other intervention components such as restricted diet and counseling interventions. Physical activity alone is

not often the variable of research, so studies that concentrate on the relationships of this variable specifically, or separate this variable within the intervention studies are of the utmost importance. Researcher in one of these studies found that walkers can significantly improve their walking times on a submaximal walking test after a 12 week program, indicating an increase in fitness, which could be a clear indication of increased efficiency in “brisk walking” or could also be indicative of changes in fitness levels (Tucker & Mortell, 1993). A similar study compared the fitness benefits of walking regimens [3 miles, 5 days per week] utilizing various intensities: at either a stroll (20 minutes/mile), a brisk walk (15 minutes/mile) or an aerobic walk (12 minutes/mile) (Duncan, Gordon, & Scott, 1991). The results indicated that all walkers had significant improvements in their VO_{2max} as compared to a control group and that the aerobic walkers had a significant improvement over the strollers but not the brisk walkers. Duncan et al. (1991) reported modest improvements in the HDL cholesterol in all three groups, though no other significant differences were revealed among other cardiovascular risk factors. From these findings the authors concluded that vigorous exercise is not necessary to obtain meaningful lipoprotein differences, although this conclusion seems excessive given the marginal changes in lipoproteins. In a review article Winett (1998) notes the differences of the effect size for the three groups [0.25 for the strollers, 0.54 for the brisk walkers, and 0.91 for the aerobic walkers] as a limitation of the study. In fact only the brisk walkers, and aerobic walkers met the recommended increases in VO_{2max} for health benefits suggested by the authors (with the limited effect size). There is obvious importance in examining the health impacts gained from walking and the purported

inverse relationship of decreased risk that is suggested to accompany increased fitness levels. Winett (1998) suggests that the authors errantly interpret their results and suggests changes in VO₂ max for the brisk and aerobic walkers as the most important variable.

Walking also appears to be a popular choice for exercisers as reflected by the Nurses Health Study, where 60% of the women walked while only 26% reported participation in vigorous exercise, suggesting walking may be a more acceptable and more prevalent mode of exercise (Manson et al., 1999). Another study found that walking was the most prevalent type of exercise and that most individuals who report moderate-intensity activity participation, like walking, met moderate to high levels of fitness classifications (Stofan, DiPeitro, Daves, Kohl, & Blair, 1998). In both studies more emphasis is placed on the positive health impact of brisk rather than very brisk walking. A “very brisk” pace is a pace similar to a 20 minute/mile pace, which is also attainable for many individuals. Lastly from the cohort of women in the Nurses Health Study increased levels of physical activity were associated with decreased risk of developing Type 2 Diabetes and a pace faster than a normal walking pace was also inversely associated with risk (Hu et al., 1999). Similarly, increased physical activity was associated with decreased risk of ischemic stroke and a brisk walking pace was associated with a lower risk of ischemic stroke when compared with a normal walking pace (Hu et al., 2000).

In pedometer-based studies employing multiple methodologies by several authors, a wide range of risk variables were studied. Of these some common study characteristics emerge. In largely healthy populations, free of diagnosed disease, populations of

overweight or obese women were placed in intervention groups for four, eight and 36 weeks (Hultquist et al., 2005; Swartz et al., 2003; Scheider et al., 2006) respectively. Each of these groups exhibited weight change (-0.5 kg significant difference; +0.3 kg NS; and -2.4 kg significant respectively. The difference between effectiveness of the three programs begins with the family history of the population used in the study by Swartz and colleagues. The Swartz cohort examined a population with a family history of type II diabetes, while the other two studies did not target this group. Perhaps individuals with Type II diabetes have other underlying genetic predispositions that alter their response to this type of intervention.

Insulin concentrations have been shown to be positively associated with age and negatively associated with physical activity (Hu et al., 2003 and Gustat et al, 2002). In a recent pedometer based walking intervention to discriminate between difference in response of sedentary and active young, middle age and older women, insulin concentrations were significantly negatively correlated with activity (steps per day). However the midlife group had significantly lower insulin concentrations than the young and older women. The active women [participating in 357 ± 243 minutes/week of programmed physical activity or approximately 50 minutes per day] had significantly lower insulin concentrations than the sedentary group. Overall this study revealed significantly different insulin concentrations between the lowest and highest tertile groups. No significant differences in glucose concentrations were observed between the tertiles. Plasma glucose level was positively associated with age; however, no relationship

was seen between activity and plasma glucose, serum triglycerides, or serum HDL cholesterol levels.

In a meta-analysis of walking studies to address cardiovascular risk factors, Albright et al. (2006) noted that the average participant in a step-based walking study can expect to lose a modest amount of weight, amounting to about 0.05 kg per week or 1 pound every 10 weeks. Since most people enter with the goal of weight loss, it is likely that relatively small decreases in body weight with these types of interventions attributes to the attrition in these studies.

Physical Activity and Chronic Disease Risk

In 1999 the ACSM constructed a panel of experts to review the literature on physical activity for the treatment of obesity and obesity-related disorders. Several large-scale longitudinal studies cited by the ACSM panel indicated that physical activity and fitness attenuates age-related weight gain, but may not promote weight loss. An attenuation of weight gain through regular physical activity, however, seems to reduce or mitigate the co-morbidities of obesity such as glucose intolerance, hypertension, and dyslipidemia (DiPietro, Kohl, Barlow & Blair, 1998; Grundy et al., 1999).

The relationship between obesity and dyslipidemia, insulin resistance, and hypertension is well established. Current research described by the 1996 U. S. Surgeon General's Report indicates that physical activity levels exhibit proportional benefits with regard to cardiovascular mortality. In addition, individuals who participate in physical activity have a lower prevalence of cardiovascular risk factors. Research supports the reduction of blood pressure or prevention of hypertension for individuals who participate

in regular physical activity (Blair, 1994; and, Blair, Kampert & Kohl, 1996). Physical activity has similar beneficial effects on diabetic risk factors through the reduction of glucose production, increased insulin sensitivity, and the reduction of obesity (Dunn, 1999; Tuomilehto, Lindstrom, Eriksson et al., 2001). The blood lipid profile demonstrates the most varied response to physical activity and is likely due in large part, to the heterogeneity of methodology, populations of study, intervention protocol, and the use of combination therapies including both diet and pharmacological strategies (Shepard & Balady, 1999). Changes in lipoprotein levels as a result of exercise are often similar to those achieved by weight loss (Hardman, 1999). As suggested by longitudinal epidemiological studies, changes in body composition may contribute to changes in lipid metabolism. Two separate epidemiological studies lasting over ten years assessed the role of physical activity associated weight loss and the lipoprotein changes related to changes in body fat (Sternfeld, 2003). Conversely, previous intervention studies have produced physical activity mediated lipid profile changes with no associated changes in body weight or composition (Haapanen et al., 1997; Williamson et al., 1993; Schwartz et al., 1992; and, Hardman et al., 1989). The relationship between physical activity and obesity-related risk factors of interest in this study will be discussed in the following sections.

Overweight and Obesity

The Obesity Education Initiative (OEI) sponsored by the NHLBI, reviewed 12 well-designed clinical trials to address the issue of the ability of physical activity to produce weight loss. In ten of the 12 articles, the reported mean weight loss was 2.4 kg or a mean reduction of BMI of 0.7 kg/m² in the exercise group compared to a control group.

In two of twelve studies, weight loss was less than 2 kg. It seems that when evaluated by itself, physical activity produces only modest weight loss.

The efficacy of weight regulation is supported by a variety of work. A study by DiPietro and colleagues (1998) supports the use of physical activity for the prevention of age-related weight gain. In cooperation with the Cooper Institute, the investigators followed 4,599 men and 724 women from 1970 to 1994. Body weight and cardiorespiratory fitness (assessed by maximal graded treadmill test) was determined at three different time points during the study. This study concluded that for each 1-minute improvement in treadmill time from the first to the second testing session there was a 9 percent and 14 percent decrease in the odds of gaining 5 kg in women and men, respectively. The odds reduction for gaining 10 kg was 21% for both women and men with each 1-minute increase in treadmill time.

Other evidence supporting the role of physical activity in body weight regulation is a product of cross-sectional and population studies which have consistently shown a negative relationship between levels of habitual physical activity and BMI (French et al., 1994; Slattery, McDonald & Bild, 1992; Williamson et al., 1993). Further information indicating an important role of physical activity for weight regulation has been compiled from the National Weight Control Registry, a data collection resource that includes individuals who have lost at least 30 lbs and maintained that loss for at least one year. The Registry includes data from 1047 men and women, and it reveals that one of the most consistent traits of the registrants was the participation in daily physical activity.

Registered individuals report an average of at least one hour of moderate to vigorous intensity activity on a daily basis.

Body Fat Distribution

Another important aspect of body weight and body fat is the distribution of that fat. The effect of physical activity on body fat distribution, especially abdominal fat, is of particular interest. Higher levels of abdominal fat, or visceral adipose tissue (VAT), are associated with several metabolic and cardiovascular disorders. The ACSM panel examined five studies that assessed the independent effect of exercise on abdominal girth. Of the five, each reported reductions in both visceral and subcutaneous abdominal fat with exercise alone, and only one of the five studies indicated accompanying weight loss. Additionally the Obesity Education Initiative (OEI) organized by the National Institutes of Health (NIH) indicated small to modest reductions in abdominal girth with exercise. One study indicated a reduction of 0.9 cm in men with no reduction for women (Stefanick et al., 1998). The OEI also showed that studies with only modest weight loss (<2%) resulted in a 12 to 16 percent increase in cardiorespiratory fitness.

Research by Mayo and colleagues (2003) examined the effect of a 16-week exercise program on body fat distribution in a group of young obese men (N=30; mean age 19.8±0.6 years). This study employed gradual increases in exercise intensity over the 16-week period, and anthropometric data (FFM, WHR, hip circumference (HC)) was collected both at baseline and after the sixteenth week. Data indicated a preferential reduction in abdominal fat and maintenance of FFM with exercise. Similarly, Miyatake et al (2002) found that daily walking was effective for reducing visceral adipose tissue area

and improving insulin resistance in Japanese males (Ages 32 to 59; N=31). Subjects in this study were equipped with pedometers and instructed to increase their daily step count by 1000 steps from baseline. Dietary patterns were monitored for consistency throughout the 1-year study utilizing food diaries. Data indicated a correlation in changes in steps taken and abdominal visceral adipose tissue area. The information provided by each of these studies lends an important research prospective to exercise as it relates to the cardiovascular risk associated with abdominal obesity, since a strong correlation of visceral abdominal fat and waist circumference is related to such CVD risk factors as dyslipidemia and insulin resistance (Wei et al., 1997; Pouliot, Despres, Nadeau et al., 1992; Hong et al., 1998).

Influence of Physical Activity on Blood Lipids

Controversy also exists concerning the amount of physical activity necessary for measurable changes in blood lipid profiles associated with obesity. Disagreement remains with respect to type, amount, pattern, and intensity of physical activity to produce healthy lipid profiles. Morris et al. (2004) concluded that energy expenditure is the key determinant in the relationship between physical activity and health related biochemical risk. Generally, increases in physical activity will result in decreased TG levels, increased levels of HDL cholesterol (HDL-C) and decreased levels of LDL cholesterol (LDL-C), while total cholesterol is not consistently changed (Hardman, 1999).

Plasma lipids and lipoproteins are reported to be quite heterogeneous among obese individuals as well. Some obese individuals exhibit normal plasma lipid and lipoprotein profiles while many others exhibit impaired profiles (Sims, 2001, Vierhapper

et al., 2000). The type of obesity, defined by the location with the most accumulation of fat, determines who may exhibit impaired lipid and lipoprotein profiles. The evidence to suggest changes in LDL-C and total cholesterol (TC) in response to increase physical activity is limited. Although a meta-analysis by Tran & Weltman (1985) indicated that intensity of exercise to elicit changes in blood lipid profiles were not as great as those required to demonstrate changes in cardiovascular fitness. LDL-C and TC levels tend to be the least affected by interventions that employ increases in physical activity. In large part, differences noted in LDL-C and TC lack significance when confounding lifestyle factors are controlled (i.e. smoking and drinking habits) (Durstine et al., 2001). It is for this reason that more interest has been focused on HDL-C and TG levels. Conflicting statements still abound with regard to HDL-C changes as a result of exercise or physical activity programs. In a 24 week walking study, Duncan and colleagues (1991) reported increases in HDL-C levels for pre-menopausal women, regardless of the intensity of physical activity; whereas, Santiago et al. (1995) indicated no change in HDL-C levels for women engaging in brisk walking at distances similar to that of Duncan's study. Kokkinos and Fernhall (1999) indicated that the impetus for this difference could be attributed to the variation in baseline measures of HDL-C for each population, so that women with higher baseline levels (55 versus 65 ml/dl) may not exhibit the same increases, despite the duration of the study.

When activity patterns are assessed, HDL-C levels are higher in the most active individuals and lower in the least active individuals. Moore et al. (1982) established this relationship through the comparison of recreational joggers (J), long distance runners

(LD) and inactive (I) women. Controlling for adiposity and nutrient intake, HDL-C levels were significantly higher in LD (78 mg/dl) as compared to J (62 mg/dl) or I (62 mg/dl) ($p > 0.001$). There were no significant differences in TC between groups. In addition, TG levels were significantly lower in LD compared to I ($p < 0.002$), but when adjustments for adiposity were made, differences in TG were no longer significant. Duncan et al. (1991) reported similar increases in HDL-C levels in women (29-40 years) following 24 weeks of walking (4.8 km/session) regardless of intensity, suggesting that moderate exercise will raise HDL-C levels as much as intense exercise. For a walking program involving African-American women (45-70 years), Keller and colleagues (2004) demonstrated clinically significant changes in HDL-C levels from baseline, for each of two walking frequency groups (3 and 5 days per week). However, these changes were not statistically significant. Statistically significant changes from this intervention included decreases in body fat (estimated by BIA) and regional fat distribution (estimated by WHR), with the greatest improvements among subjects moving from sedentary to active categories. The changes that were statistically significant were noted only at 36 weeks from baseline with no changes noted to be statistically significant 12 weeks from baseline. In women, the volume of exercise seems to be more important than the intensity of exercise for influencing HDL-C levels. Most studies suggest a large volume of exercise is necessary for significant HDL-C changes in women; however, the exercise volume threshold has not yet been defined. Generally, physically active women exhibit higher levels of HDL-C when compared to their sedentary counterparts (Kikkinos & Fernhall 1999). The HERATIGE studies revealed that Blacks generally had higher plasma HDL cholesterol

levels than Whites, specifically when comparing abdominally obese Black versus White individuals (Depres et al., 2000).

Most studies suggest that endurance exercise is positively associated with increases in HDL-C levels in men. However, in women the relationship between endurance exercise and HDL-C levels is less clear. The response of HDL-C levels will differ for each individual depending on the intensity, duration and frequency of exercise, the initial HDL-C level, and the length of the training period.

Physical Activity and Hypertension

Independent of obesity and cardiorespiratory fitness, research indicates that physical activity is associated with decreases in systolic blood pressure of women when comparing the most active to the least active (10.7 to 5.9 mmHg respectively) (Wareham et al., 2000). In a nine-week randomized controlled trial 44 hypertensive, overweight adults on anti-hypertensive medication were randomized to a lifestyle or control group. The lifestyle group also participated in a moderate-intensity exercise program 3 times per week. The lifestyle group also received a dietary intervention which consisted of a reduced-sodium diet. The control group received no intervention. At the end of the intervention, mean weight loss in the lifestyle group was 4.9 kilograms. In the lifestyle group mean net reductions in systolic and diastolic blood pressures were 12.1 mm Hg ($P<0.001$) and 6.6 mm Hg ($P<0.001$), respectively. A recent study involving African-American women, reported clinically significant reductions in systolic blood pressure from baseline (143 mmHg) at 12- (137 mmHg) and 36-weeks (131 mmHg) in the least active group. While these findings were not statistically significant, they suggest that low

intensity exercise can result in clinically significant blood pressure reductions (Wareham et al., 2000).

Physical Activity and Insulin Resistance

The Insulin Resistance Atherosclerosis Study, attempted to determine whether habitual, non-vigorous physical activity, as well as vigorous and overall activity, is associated with better insulin sensitivity (S_I) (Mayer-Davis et al, 1998). A total of 1467 men and women of African American, Hispanic, and non-Hispanic White ethnicity, aged 40 to 69 years, with glucose tolerance ranging from normal to mild non-insulin-dependent diabetes mellitus participated in this study. After adjusting for potential confounders, the mean S_I for individuals who participated in vigorous activity 5 or more times per week was 1.59 compared with 0.90 for those who rarely or never participated in vigorous activity ($P < 0.001$). When habitual physical activity was assessed by 1-year recall of activities, the correlation coefficient between S_I and total exercise energy expenditure was 0.14 ($P < 0.001$). After adjustment for confounders, vigorous and non-vigorous levels of exercise energy expenditure (metabolic equivalent levels ≥ 6.0 and < 6.0 , respectively) were each positively associated with S_I ($P \leq 0.01$ for each).

In summary, there is significant evidence to support the use of physical activity for the reduction of obesity-related disorders; however, much of the research that indicates successful outcomes actually demonstrate the effects of various combinations of intervention strategies, and cannot be used to support the effects of increased physical activity alone. In addition, many of the interventions include activity levels above and beyond the levels that have been recommended for health benefits. Thus, the current

proposal will try to isolate the ability of PA to reduce obesity-related risk factors using both traditional exercise and lifestyle activity modification.

CHAPTER III

METHODS

Introduction and Design

The overall goal of this research study was to compare changes in obesity-related risk factors after participation in either a twelve-week traditionally prescribed cardiovascular exercise program or a lifestyle activity modification program for previously sedentary African-American women. A primary goal of the activity groups was to determine the effectiveness of the lifestyle activity modification intervention to change physical activity levels among sedentary African-American women through the use of both traditional exercise and lifestyle activities. A secondary goal of the activity groups was to compare the effects of the Lifestyle and Traditional exercise program on obesity-related risk factors (physical activity, blood pressure, blood lipids and lipoproteins, insulin resistance, and body composition).

Study Procedures

The target of participant recruitment for the study was obese, sedentary, but otherwise healthy women between the ages of 18 to 55. Sedentary individuals were defined as individuals who have not participated in regular physical activity for at least the past 3 months (< 2 days/week; >20 minutes/day). The minimum BMI cutoff for participation in the study was 23 kg/m². Specific exclusion criteria included:

1. History of cardiovascular disease, stroke, or diagnosed diabetes (Type 1 or 2).
2. Currently engaging in physical activity at least 3 days/week for 20 minutes or more each time.
3. Currently participating in as organized weight management program such as Weight Watchers.
4. Having a plan to move from the local area within the time span of the study.
5. Pregnant or planning to be pregnant.
6. Taking medication that could alter exercise performance, metabolism or appetite (except birth control pills, blood pressure medication and statins).
7. Smoking or recent history of smoking (within 3 months of study).

Methods of recruitment included newspaper advertisements, electronic e-mail announcements, and posters at a university and its surrounding community. In addition, radio announcements were made on the campus radio station. Respondents were scheduled for an orientation session during which time the details of the study purpose, time commitment, and eligibility criteria was explained. Subjects that expressed interest in the study were asked to complete an eligibility questionnaire that included the aforementioned exclusion criteria, as well as medical history and physical activity readiness (PAR-Q) questionnaires.

During the orientation sessions eligible subjects were asked to review and sign a consent form, after which height and weight for each participant was measured for

calculation of Body Mass Index (BMI). Written consent was obtained for all eligible participants in accordance with the policy statement regarding the use of human subjects for research at the University of North Carolina at Greensboro.

Assessment Procedures

All groups participated in pre-screening/orientation, pre-participation and post-participation testing for the assessment of study variables (anthropometric, physical activity, biochemical and sub-maximal fitness data). The following assessments were conducted at baseline and at 12 weeks.

Anthropometric Measurements

Anthropometric data was gathered for waist circumference. Body Mass Index (BMI) was calculated using each subject's height and weight. Body weight was measured, barefoot, to the nearest 0.1 pound using a standard balance beam scale (Health-o-meter) and converted to mass in kilograms (kg). Height was measured to the nearest 0.1 cm. For the height measurement, participants stood barefoot on a flat surface with heels together and body mass evenly distributed on both feet. The head was positioned so that the line of vision was approximately horizontal and the sagittal plane of the head was vertical. BMI was calculated from these measurements by dividing the weight in kilograms by the square of height in meters.

Waist circumferences were measured using a Gulick measuring tape with tension device to ensure consistent circumference measurement. Participants stood on a flat surface with heels together and body mass evenly distributed on both feet. Three

measurements to the nearest 0.1 cm were taken. The three measurements were then averaged for each subject. Waist circumference was assessed as the narrowest measured circumference of each subject's trunk.

Body Composition

Body composition was assessed via 3-site skin fold measurement (triceps, suprailliac, and thigh) and bioelectric impedance analysis (BIA). The bioelectrical impedance analysis was used to assess percent body fat through a prediction model of the fat free mass and total body water. All skinfold measures were taken on the right side of the body with Lange skinfold calipers by the same investigator. Body density was calculated using Jackson et al's (1980) three-site equation for body density $[1.0994921 - 0.0009929 \times \text{Sum of Skinfolds}^2 + 0.0000023 \times \text{Sum of Skinfolds}^2 - 0.001392 \times \text{Age}]$. Body density was converted to percentage body fat (%bf) using an ethnic specific equation $[\% \text{ fat } (483.2/D - 436.9)]$ (Ortiz et al., 1992). Participants were instructed to remove all metal items from their body prior to testing. Subjects were then instructed to walk for approximately four minutes, following which they were to lie down in a supine position on a level surface for two to five minutes as preparations were made for the assessment. Researchers then applied four electrodes, one each to right-side hand, wrist, foot and ankle respectively and the BIA equipment was then used to estimate intracellular water, extra-cellular water, lean body mass and total body water as the subject remains motionless. The percent body fat was then assessed by calculating fat mass from predicted lean body mass and dividing fat mass by total body weight.

Heart Rate

Heart rate and blood pressure were measured manually according to guidelines set forth in the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, commonly referred to as JNC-7, with the exception of an extended rest period prior to measurement [10 minutes as opposed to the recommended 5 minutes] (Chobanian, Bakris, Black et al., 2003).

Biochemical Markers

Fasting blood samples were obtained from subjects by a trained phlebotomist at both baseline and 12 weeks. Participants sat in a chair, or could lie down or sit up in a bed so that the blood samples can be taken. Standard venipuncture techniques were used to collect the blood from the antecubital vein. The total blood collection for each testing session was 60 cc per subject, thus the total donation for the study was 120 cc or approximately ¼ pint per subject. Serum cholesterol, high-density lipoprotein cholesterol (HDL-C) concentrations, low-density lipoprotein cholesterol (LDL-C) concentrations, triglycerides, fasted blood glucose and insulin were assessed for each subject. Cholesterol was measured enzymatically in serum through a series of coupled reactions that hydrolyze cholesterol esters and oxidize the 3-OH group of cholesterol. The byproduct of the reaction (H_2O_2) was measured to determine, through proportionality, cholesterol concentration. Triglycerides were also measured enzymatically to produce glycerol, which was subsequently oxidized and measured as described for cholesterol. HDL cholesterol was measured in the HDL-containing

supernate, following the precipitation of the other lipoproteins LDL cholesterol was calculated from the measured values of total cholesterol, triglycerides and HDL cholesterol according to the relationship: $[\text{LDL-C}] = [\text{TC}] - [\text{HDL-C}] - [\text{TG}]/5$. Insulin was determined by ELISA from Mercodia and glucose was determined by colorimetric assay from Roche Diagnostics Systems. Insulin resistance was calculated using the HOMA formula. The HOMA estimate of insulin resistance was assessed by the formula $[\text{fasting insulin (mU/l)} \times \text{fasting glucose (mmol/l)}] / 22.5$.

Energy Expenditure

Energy expenditure was measured using Physical Activity Recall (PAR) after participants have been provided written and verbal instruction for instrument completion. All listed activities were converted into metabolic equivalent units (MET), for which one MET is approximately equal to an oxygen consumption of 3.5 ml/kg/minute or the estimation of the metabolic rate while sitting quietly in a chair. MET values were used to describe exercise intensity for occupational activities and exercise prescription, and estimated MET values for household chores, office activity, industrial jobs and various modes and intensities of exercise are commonly available in many physiology texts. MET equivalents from the compendium of physical activities were matched to activities logged by participants in their daily activity logs or their daily cardiovascular logs by activity mode and intensity (Ainsworth et al., 2004). MET equivalents are reported as kcal/kg/hr by activity and energy expenditure

is calculated according to minutes logged for that activity and the body weight of the individual performing the activity (Heyward, 2006).

Cardiorespiratory Fitness

Cardiorespiratory fitness was assessed using the YMCA submaximal cycle ergometer protocol, using the guidelines established by the American College of Sports Medicine (ACSM). During the orientation session, subjects were introduced to the testing equipment and the cycle ergometer was adjusted for each individual (with all settings recorded for future testing during this study). Just prior to being seated on the cycle ergometer for testing subjects were fitted with a heart rate monitor. Heart rates were monitored during testing and the heart rate was not blinded for participants.

The sub-maximal cycle ergometer protocol proceeds as follows:

- 1) A 2-3 minute warm-up was used to acquaint the subject with the cycle ergometer and prepare her for the first stage of the 2-4 (3-minute) stages test. The workload for the warm up session was the same intensity used for stage one.
- 2) The workload for the first stage of the test was 150 kgm/min. (0.5 kg at 50 rpm. The test was continued until two consecutive heart rates (within 6 bpm of each other) were achieved between 110 bpm and 85% of the age-predicted maximal HR of each individual.
- 3) The heart rate (HR) in the third minute of the stage dictates the workload for the second stage as follows: less than 80 bpm, the second stage was set at 750 kgm/min (2.5 kg at 50 rpm); 80-89, the second stage was set at 600

kgm/min (2.0 kg at 50 rpm); 90-100, the second stage was set at 450 kgm/min (1.5 kg at 50 rpm); greater than 100, the second stage was set at 300 kgm/min (1.0 kg at 50 rpm)

- 4) The third and fourth (if required) stages were set according to the same criteria as the second.
- 5) Upon completion of two consecutive stages that meet the criteria for cessation listed above, the subject then drop the workload back to that of the warm up or less for at least a 2-minute cool-down stage.
- 6) The subject's heart rate was monitored following exercise, and subject was asked to remain in the lab until it reaches an individual's pre-exercise level.
- 7) The heart rate measured during the last stage of the exercise protocol was plotted against work rate as outlined by the American College of Sports Medicine's Guidelines for Exercise Testing and Prescription (6th edition, pp74-77). The line generated from the plotted points were then extrapolated to the individual's age-predicted maximum HR (220-age), and a perpendicular line was dropped to the x-axis to estimate the work rate that would have been achieved if the person had worked to maximum. VO₂max can then be estimated from the work rate using the American College of Sports Medicine's Guidelines for Exercise Testing and Prescription (Appendix D).

Intervention Procedures

Forty-five healthy, sedentary, African-American female volunteers, 22-55 years of age were recruited and randomly assigned to one of three 12-week groups described in the following paragraphs, each consisting of 15 participants. Two individuals taking blood pressure medications were randomized using a computer-generated block design. Pre-participation and 12 week data were coded and recorded by researchers throughout the study. Code keys, that identify individuals with coded data, were available only to the principle investigator. Individual participants were advised of their results upon completion of the study. All HIPAA guidelines were followed. From time of recruitment, individuals were involved in the study a total of 16 weeks (two weeks for pre-testing, two weeks for post testing, and 12 weeks for treatment). All groups were asked to record daily physical activity, fill out Stanford 7-day Physical Activity Recall Questionnaires (pre- and post-intervention) for the estimation of energy expenditure; use pedometers to record daily steps per day, and record dietary patterns using a three-day dietary record (two weekdays and one weekend day). All groups were asked to maintain current dietary patterns throughout the study. Medications were tracked during the study, and individuals taking medication were asked to maintain a consistent dosing schedule.

Lifestyle Activity Modification (Lifestyle): The participants were advised and encouraged to increase levels of low to moderate intensity activity in order to accumulate approximately 150 minutes of activity weekly, which was approximately 30 minutes of accumulated activity on most days of the week. This group participated

in a lifestyle activity curriculum during weekly one-hour meetings for the first six weeks and alternating weekly meetings during the second six weeks of the study, in which they were educated about the incorporation of exercise into activities of daily living. Information for the Lifestyle group meetings were disseminated according to the curriculum recommended by the CDC's Division of Nutrition and Physical Activity in its Guide for Community Action which can be found via internet access at the following internet address:

(<http://www.cdc.gov/nccdphp/dnpa/physical/index.htm>). Additionally modules from the Diabetes Prevention Program, which are based on the aforementioned CDC information, were used. The DPP modules, participant manuals and scripts for presentation of materials can be found at the following site:

(http://www.bsc.gwu.edu/dpp/lifestyle/dpp_acor.html). Physical activity was assessed indirectly through the use physical activity recall questionnaires (MET minutes/day) and pedometers (steps/day). Program compliance was tracked through the use of daily physical activity logs.

Traditional group exercise (Traditional): This treatment provided traditionally prescribed exercise using common modes of exercise through group exercise sessions. Exercise modes available to and used by the participants included treadmills, stationary cycle ergometers or elliptical machines. This group participated in four low to moderate level (50-65 percent of heart rate maximum) exercise sessions per week (averaging 40 minutes per session) to accommodate the recommendation that individuals accumulate at least 150 minutes of low-moderate intensity exercise

each week. Exercise protocol for traditional group consisted of 5-10 minutes of low intensity warm-up and stretching, 30-40 minutes of moderate intensity exercise, and 5-10 minutes of low intensity cool-down. Group participants were asked to exercise for a minimum of 20 minutes during their first session and progressively increase by 5 minutes each week until they were able to exercise for a 40 minute continuous session. All participants met the 40 minute continuous exercise by the third week of exercise and maintained these bouts throughout the exercise intervention period. Physical activity, beyond the group sessions, was assessed through the use of physical activity recall questionnaires, physical activity records, pedometers (number of steps walked). Heart rate monitors were used to monitor intensity of exercise during group exercise sessions. Participants were assigned individual heart rate ranges for exercise sessions, which they also recorded (as an average heart rate) at the completion of each exercise session.

Control Group: A control group met the first, third, sixth, ninth and twelfth weeks of the study to verify continued participation in the study, to verify records and answer questions about record-keeping during the course of the study. This group was asked to make no changes in pre-study levels of physical activity during the twelve-week period. Physical activity was assessed through the use of physical activity questionnaires and pedometers (which were given to participants during pre-participation assessment). Individuals in this group were offered free participation in a similar exercise program upon completion of this study. This group met every other week to receive risk factor information and to complete forms.

Session outlines for each group can be found in Appendix A and B. Upon completion of the study, subjects were advised of their results. Subjects were also given their results if they were prematurely removed from the study due to exclusion criteria or if they voluntarily drop out of the study.

Intervention Attrition and Adherence

Participation requirements were different for each of the three intervention groups, therefore adherence to intervention requirements were calculated differently. Adherence to the Lifestyle and the Control treatments were based on group meetings, not exercise participation. Attending at least 9 of the group meeting (75%) was considered a criterion for adherence, similar to methods for assessing adherence and attrition used in Project Active (Dunn et al., 1997). For the Traditional exercise group, attendance at 3 of the weekly meetings (75%) and 36 of the exercise sessions (75%) was considered a criterion for adherence.

Attrition rates were determined by the number of participants who dropped out of any of the three treatment groups. Lifestyle or Control participants were considered “dropouts” if they attend less than 75% of the class meetings and Traditional participants were considered as dropouts if they failed to attend 75% of either exercise or group meeting sessions. Additionally, if any participant failed to attend orientation or at least one pre-testing sessions, or failed to complete informed consent paperwork, that individual’s participation in the study was terminated and that individual was not included in data analysis. If a study participant, as well, did not attend post-testing sessions, that individual’s data was included in the study for purposes of data analysis.

Statistical Analysis

Data analysis was conducted using SPSS version 16.0 software. Descriptive statistics were calculated to determine the nature and variability of participants' measures of health and physical fitness indicators. The general plan for statistical analysis was to first provide statistical comparisons between intervention groups for all dependent variables. Repeated measures analysis of variance was used to determine if any significant differences exist between groups at either time point. Specific statistical procedures are outlined below for each aim and accompanying hypothesis. Variables with statistically significant measurements between groups at baseline were assessed using an analysis of covariance model, with the baseline as the covariate.

Specific Aims

Specific Aim 1

The first aim of this study was to compare the effects of LA, CARDIO and a control group on the physical activity patterns of sedentary African-American females between the ages of 18 and 55.

Hypothesis 1. Compared to the control group, LA and Cardio would increase physical activity in sedentary African-American females between the ages of 18 and 55.

A repeated measures analysis of variance (ANOVA) for three groups over two time points (baseline and 12 weeks) was used to determine if any significant

differences exist between groups at each time point, as well as within groups over time for physical activity levels in MET minutes per day.

Specific Aim 2

The second aim of the study was to compare the effectiveness of LA, CARDIO and a control group on obesity-related health indicators in sedentary African-American females between the ages of 22 and 55.

Hypothesis 2a. Compared to the control group, both LA and Cardio would have similar positive effects on blood lipid profiles in sedentary African-American females between the ages of 22 and 55.

Hypothesis 2b. Compared to the control group, both LA and Cardio would have similar positive effects on insulin resistance in sedentary African-American females between the ages of 22 and 55.

Hypothesis 2c. Compared to the control group, both LA and Cardio would have similar positive effects on blood pressure in sedentary African-American females between the ages of 22 and 55.

Hypothesis 2d. Compared to the control group, both LA and Cardio would have similar positive effects on cardiovascular fitness in sedentary African-American females between the ages of 22 and 55.

Hypothesis 2e. Compared to the control group, both LA and Cardio would have similar positive effects on body composition in sedentary African-American females between the ages of 22 and 55.

A repeated measures analysis of variance (ANOVA) for three groups over two time points (baseline and 12 weeks) was used to determine if any significant differences exist between groups at each time point, as well as within groups over time for all of the health and physical fitness data. Since glucose and insulin data was not analyzed for all participants completing the intervention, these results were reported as raw data, rather than analyzed inferentially. Any significant differences at baseline were analyzed using an analysis of covariance model, with the baseline measure as the covariate. A series of Tukey's post hoc tests was run to determine the difference among the means of each group.

CHAPTER IV

RESULTS

In response to announcements about the program, one hundred and twenty-two women expressed interest either by telephone or e-mail. Ninety-seven African-American women attended one of 10 interest meetings, which outlined the details and requirements for eligibility for the research study. As a result of the interest meetings, 74 women made appointments for pre-intervention assessments. Sixty-two women participated in non-biochemical variable assessments (everything except the blood draw); while only fifty-three attended a biochemical assessment. Forty-eight African-American women voluntarily began participation in the study as a part of one of the three intervention groups: 18 in the Lifestyle Group; 15 in the Cardio Group; and 15 in the Control group. The data of one subject was eliminated from the final analysis for failure to meet the attrition criteria for the LA group meetings. The data from two more LA group members was dropped from the study for failure to report diagnosis of type II diabetes prior to initiation of the intervention protocol. At the completion of the project, 15 individuals in each of the three groups had completed the program, including pre and post testing for all the non-biochemical assessments. Two subjects (1 Control and 1 LA) were currently prescribed anti-hypertensive medications. Ten subjects [3 Control (n=12), 4 Cardio (n=11) and 3 LA (n=12)] were excluded from the blood analysis data due to incomplete

pre/post blood collection and some samples for the glucose and insulin variables (and thus the HOMA measure) could not be included due to missing data. Therefore, six Control (n=6), eight Cardio (n=8) subjects and nine LA (n=9) subjects completed pre and post testing for all variables in the study. Due to the decreased power associated with the low subject number analyzed for the glucose, insulin and HOMA variables, these data were excluded from inferential statistical analysis. Baseline descriptive statistics for all participants are given in Table 4. Group means (SD) for baseline measurements can be found in Table 5. Summary means for the ANOVA analysis are reported in Table 6. Significant differences at baseline were indicated only for LDL cholesterol. No significant differences in post assessment LDL were revealed using an analysis of covariance model, with the baseline LDL as the covariate. Changes from baseline to post intervention assessment in physical activity, cardiorespiratory fitness, anthropometrics, and blood lipids are also presented below (Tables 7 and 8). The variables were analyzed in a 3 X 2 analysis of variance (ANOVA) with pre-post as the within-subject factor and treatment groups (Cardio, LA, and Control) as the between subject factor.

Table 4: Baseline values for all participants.

Baseline Measure	Minimum	Maximum	Mean	Std. Deviation
Participant's Age in years	22	55	39.3	10.1
Weight (kg)	55.0	127.3	82.7	17.6
Body Mass Index (kg/m ²)	23.2	46.4	30.8	5.9
% Body Fat (skinfold)	17.6	48.4	34.0	7.6
% Body Fat (BIA)	24.3	48.4	39.3	6.7
Waist (cm)	70.7	127.5	85.2	18.7
Systolic Blood Pressure (mmHg)	98	180	118.6	15.4
Diastolic Blood Pressure (mmHg)	57	98	73.4	10.3
Average Daily Steps	1381	16182	5976.8	3396.8
Predicted VO ₂ Max (ml/kg/min)	14.7	39.3	23.5	6.6
Dietary Intake (Kcal)	1235.9	2242.7	1728.7	248.9
Total Cholesterol (mg/dl)	170.9	308.2	239.47	38.9
HDL-Cholesterol (mg/dl)	42.6	92.3	63.4	11.67
LDL-Cholesterol (mg/dl)	91.9	216.7	160.8	35.7
Triglycerides (mg/dl)	54.2	200.4	93.8	30.0

Table 5: Baseline measures for each intervention group. Values listed as mean (SD).

	Control	Lifestyle	Cardio	p-value
Participant's Age (yrs)	39.8 (8.5)	36.5 (10.8)	41.7 (10.6)	0.361
Weight (kg)	87.2 (16.0)	82.5 (21.5)	78.5(14.7)	0.407
Body Mass Index (kg/m ²)	32.6 (5.1)	30.4 (7.2)	29.3 (5.3)	0.307
% Body Fat (skinfold)	34.3 (6.8)	33.7 (8.9)	34.0 (7.6)	0.980
% Body Fat (BIA)	39.9 (5.3)	37.4 (7.9)	40.7 (6.7)	0.375
Waist (cm)	86.3 (16.5)	88.8 (16.2)	80.7 (22.9)	0.489
Systolic BP (mmHg)	124.3 (19.0)	115.4 (10.2)	116.1 (15.2)	0.213
Diastolic BP (mmHg)	75.7 (9.7)	71.3 (10.2)	73.1 (11.2)	0.502
Steps	5851 (2966)	6566 (4468)	5343 (2711)	0.663
Predicted VO ₂ Max (ml/kg/min)	24.1(7.8)	23.2 (6.6)	23.2 (5.7)	0.926
Dietary Intake (Kcal)	1777.6(219.2)	1708.9 (319.8)	1699.4 (201.2)	0.654
Total Cholesterol (mg/dl)	234.1 (37.3)	228.5 (42.6)	257.4 (32.8)	0.173
HDL-Cholesterol (mg/dl)	61.0 (10.8)	69.0 (11.0)	59.8 (12.0)	0.115
LDL-Cholesterol (mg/dl)	161.8 (33.6)	143.1 (32.0)	179.0 (34.1)	0.050
Triglycerides (mg/dl)	95.7 (26.5)	86.8 (27.1)	100.5 (35.7)	0.206

ANOVA demonstrated significant differences between groups from baseline to post assessment for body weight ($p=0.001$), body composition (percent body fat) ($p<0.001$), and cardiorespiratory fitness (predicted VO₂max) ($p=0.024$). Significant effects for pre-post by treatment condition interaction were observed for body weight

[$F(1, 45) = 8.133, p = 0.001$]. Neither the Cardio group [Baseline, $\bar{X}=78.5\pm 4.6$ kg vs. Post, $\bar{X}=77.0\pm 4.5$; ($p=0.269$)] nor the LA group [Baseline, $\bar{X}=82.5\pm 4.5$ kg vs. Post, $\bar{X}=82.0\pm 4.5$; ($p=0.664$)] displayed a significant decrease in weight from baseline to post assessment when compared to the control group [Baseline, $\bar{X}=87.2\pm 4.6$ kg vs. Post, $\bar{X}=88.5\pm 4.5$]. However, the Control group trended very slightly upward, while the exercise groups trended very slightly downward or stayed the same (Table 7). Tukey's post hoc analysis indicated homogeneity among groups ($p=0.269$).

Table 6: Repeated measures ANOVA p-values for group, time and group by time.

	Group	Time	Group x Time
Weight (kg)	0.255	0.243	0.001*
SBP (mmHg)	0.428	0.928	0.903
DBP (mmHg)	0.281	0.886	0.277
Waist (cm)	0.164	0.543	0.630
BMI (kg/m^2)	0.299	0.866	0.060
Predicted MaxVO ₂ (ml/kg/min)	0.121	0.024*	0.002*
% Body Fat (skinfold)	0.105	0.013*	0.000*
Steps	0.276	0.000*	0.079
Dietary Intake	0.136	0.413	0.961
Total Cholesterol	0.508	0.006*	0.202
HDL-Cholesterol	0.344	0.017*	0.240
LDL-Cholesterol	0.295	0.181	0.253
Triglycerides	0.307	0.781	0.576

*indicates significance at $p<0.05$

Table 7: Non-biochemical baseline and post assessment measures. Values are mean (SD).

Measure	Control		LA		Cardio	
	Pre	Post	Pre	Post	Pre	Post
Weight (kg)	87.2 (4.6)	88.5 (4.5)	82.5(4.5)	82.0(4.5)	78.5 (4.6)	77.0 (4.5)
Body Mass Index (kg/m ²)	32.6 (5.1)	33.1 (5.0)	30.4 (7.2)	30.3 (7.1)	29.3 (5.3)	29.1 (5.4)
% Body Fat (skinfold)	34.3(2.0)	35.6(2.0)	33.7 (2.0)	32.4(33.5)	34.0 (2.1)	31.4(2.1)
% Body Fat (BIA)	39.9(5.3)	40.2(5.9)	37.4(7.9)	37.3(8.6)	40.7(6.7)	38.4(6.8)
Waist (cm)	87.3 (16.7)	88.9(18.0)	88.8(16.2)	87.7(16.9)	80.7(22.9)	82.8(11.8)
Systolic BP (mmHg)	124.3 (19.0)	123.8 (12.6)	115.4 (10.2)	116.2 (10.3)	116.1 (15.2)	115.4 (10.7)
Diastolic BP (mmHg)	75.7 (9.7)	77.6 (10.0)	71.3 (10.2)	70.7 (7.6)	73.1 (11.2)	72.1 (10.5)
Steps	6217.3(3132.9)	7067.3(2728)	6566.3(4567.7)	9989.7(4181.4)	5343.5(2711.4)	9421.0(6320.7)
Predicted VO ₂	24.0(7.8)	22.3(6.3)	23.2(6.6)	25.4(5.7)	23.2(5.7)	27.6(8.0)
Dietary Intake (kcal)	1777.6(219.2)	1794(158.9)	1708.9(319.8)	1739.4(198.1)	1699.4(201.2)	1714.2(259.0)

Table 8: Biochemical baseline and post assessment measures. Values are mean (SD).

Measure	Control		LA		Cardio	
	Pre	Post	Pre	Post	Pre	Post
Total Cholesterol (mg/dl)	235.0(36.0)	242.5(45.6)	232.1(42.9)	219.6(39.6)	258.2(33.4)	258.7(37.6)
HDL-Cholesterol (mg/dl)	61.9(11.7)	62.5(12.4)	67.2(12.4)	69.7(12.7)	61.2(11.0)	65.4(12.9)
LDL-Cholesterol (mg/dl)	161.9(33.4)	161.4(38.3)	147.6(34.7)	134.4(33.5)	180.3(36.3)	171.8(39.7)
Triglycerides (mg/dl)	95.7(26.5)	96.7(28.4)	86.8(27.1)	89.3(28.1)	100.5(35.7)	97.1(28.2)
Glucose (Mmol/L)	5.268(0.85)	5.174(1.10)	4.021(1.10)	4.626(1.12)	5.629(0.67)	4.287(1.12)
Insulin (uU/L)	5.86(4.55)	6.78(2.88)	9.16(10.82)	9.88(9.13)	11.33(4.18)	7.50(2.86)
HOMA	1.732(1.02)	1.57(0.49)	1.649(1.92)	1.794(1.32)	2.074(1.57)	1.681(0.92)

Body Composition

Body composition as measured by skinfold calipers (percent body fat) also had a significant effect by group over time [$F(2,44)=6.779$, $p=0.013$], as well as significant differences from baseline to post assessments overall [$F(1, 44) = 12.761$, $p=0.001$]. Though not significantly different when compared to the control group, the mean body fat percentages (skinfold) decreased from baseline to 12 weeks for both exercise groups [Cardio: $X= 34.0 \pm 2.1$ (baseline) and $X=31.4 \pm 2.1$ (post), $p=0.714$; and, LA: $X=33.7 \pm 2.0$ (baseline) and $X=32.4 \pm 2.0$ (post), $p=0.774$], while the control group tended toward increased body fatness from baseline to post testing ($X=34.3 \pm 2.0$ and $X=35.6 \pm 2.0$).

Body composition as measured by BIA and skinfold assessment were not highly correlated ($r=0.245$), and BIA measures were generally higher than skinfold measures. Body composition as measured by BIA (percent body fat) had a significant effect by group over time [$F(2,45)=6.224$, $p=0.017$] as well as significant differences from baseline to post assessments overall [$F(1, 45) = 7.450$, $p=0.002$]. Trends for BIA assessment of body composition across the intervention period were similar to those assessed by skinfold calipers.

BMI changes were not significantly different across groups. In this case, neither the Cardio group nor the LA groups showed marked directional changes. The two physical activity groups showed only slight changes in BMI from baseline to 12 weeks (Cardio: $X=29.3 \pm 5.3$ kg/m² and $X=29.1 \pm 5.4$ kg/m²; LA: $X=30.4 \pm 7.2$ kg/m² and $X=30.3 \pm 7.1$ kg/m²). Likewise, the Control group demonstrated a similar pattern of BMI consistency for the intervention (Control: $X=32.6 \pm 5.1$ kg/m² and $X=33.1 \pm 5.0$ kg/m²).

Fitness

Cardiorespiratory fitness (VO_2max) had a significant difference from baseline to post assessments overall [$F(2,45) = 5.48, p=0.024$] and a significant time by treatment effect was also observed [$F(2,45) = 6.956, p=0.002$], though there were no significant differences between any specific groups. Again, both physical activity groups increased cardiorespiratory fitness as measured by predicted VO_2max (ml/kg/min), compared to decreases in the control group (Figure 1). The Cardio group demonstrated the largest increase, with a mean increase from 23.2 ± 5.7 ml/kg/min at baseline to 27.6 ± 8.0 ml/kg/min, the LA group had a smaller increase from 23.2 ± 6.6 ml/kg/min to 25.4 ± 5.7 ml/kg/min, while the control group trended downward from 24.0 ± 7.8 ml/kg/min to 22.3 ± 6.3 ml/kg/min, respectively for pre & post (Figure 1).

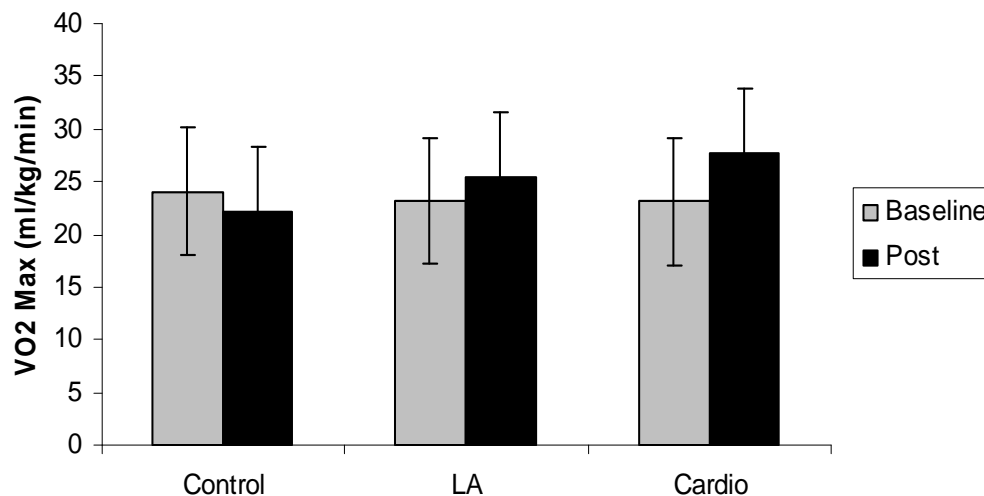


Figure 1. Changes in cardiorespiratory fitness (predicted VO_2max) by group at baseline

Physical Activity

Daily number of steps measured by pedometers showed an increased number of steps from baseline to 12 weeks for all groups. The Cardio group averaged 5343.5 ± 2711.4 daily steps upon enrollment and at the completion of the study had increased steps per day to 9421.0 ± 6320.7 ($p=0.008$). The LA group began the study at 6566.3 ± 4567.7 and completed the study logging 9989.7 ± 4181.4 ($p=0.002$). The control group averaged 6217.3 ± 3132.9 steps per day at baseline, also increasing to 7067.3 ± 2728.1 at week twelve. Significant differences for time [$F(2,45) = 16.80, p=0.000$] but not time by intervention group [$F(2,45) = 2.016, p=0.148$], were noted. Average daily steps were divided into quartiles, with no statistical differences in inter-quartile means when examined by treatment group. Steps recorded were also divided by month, and no significant differences existed within months. Daily steps by week have been graphically reported in Figure 2.

Physical activity logs from the LA group and structured exercise logs from the Cardio group were translated in to METS and further converted into average weekly kilocalories (kcal) expended in physical activity/exercise (Figure 3). The two exercise groups were significantly different when compared by independent t-test analysis [$t(28)=4.214; p=0.001$]. The Cardio group had an average expenditure of 1557.1 ± 338.4 kcal per week from participation in the structured exercise sessions, while the activity expenditure for the LA group was 1029.4 ± 347.5 kcal per week from incorporation of physical activity in to their daily schedules.

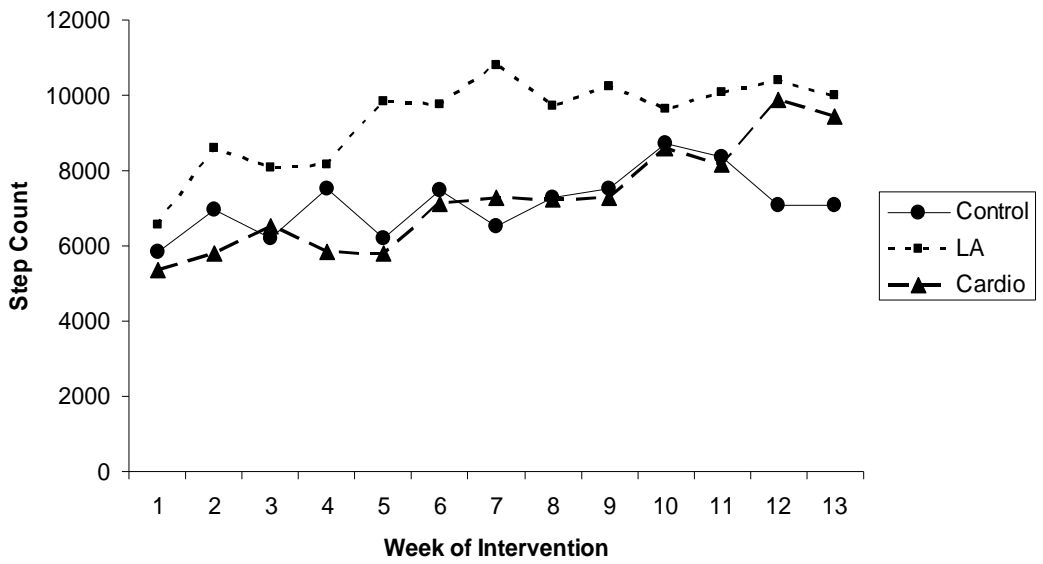


Figure 2: Average daily step counts for activity groups by week.

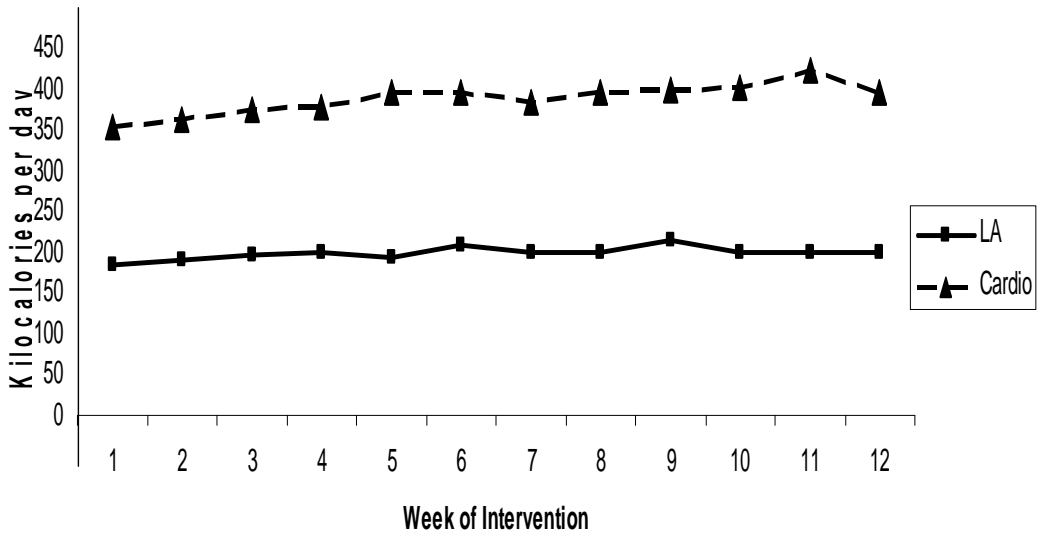


Figure 3. Average daily step counts for activity groups by week.

Blood Pressure

Neither diastolic (DBP) nor systolic blood pressure (SBP) measures were significantly altered by either exercise intervention. Though close to statistically significant, neither SBP or DBP were different baseline to post assessments overall [SBP: $F(2,45) = 0.008, p=0.051$; DBP: $F(2,45) = 0.021, p=0.052$]. Significant effects of pre-post measurements by treatment condition were more notably not indicated for either SBP [$F(2,45) = 0.103, p=0.065$], or DBP [$F(2,45) = 1.322, p=0.270$], though there were trends that emerged with slight increases in DBP for the Control group compared to decreases in mean DBP for both the LA and Cardio groups.

Biochemical Markers

For the lipid markers, only one variable demonstrated any significant effects: HDL cholesterol from baseline to post assessment [$F(2,32) = 8.84, p=0.006$]. HDL showed little change in either the control or the LA groups; however, the largest difference was demonstrated in the baseline to post-testing assessment of the Cardio group (with the Cardio group demonstrating an upward trend). Other biochemical measures have been reported below with baseline and post assessment measurements by group (Table 7). Individual pre-post values for glucose and insulin have been reported graphically by group (Figures 4 and 5 respectively). Since the number of subjects analyzed for these variables was small, statistical analysis was not completed for these variables, but the mean (SD) for each group is presented in Table 8.

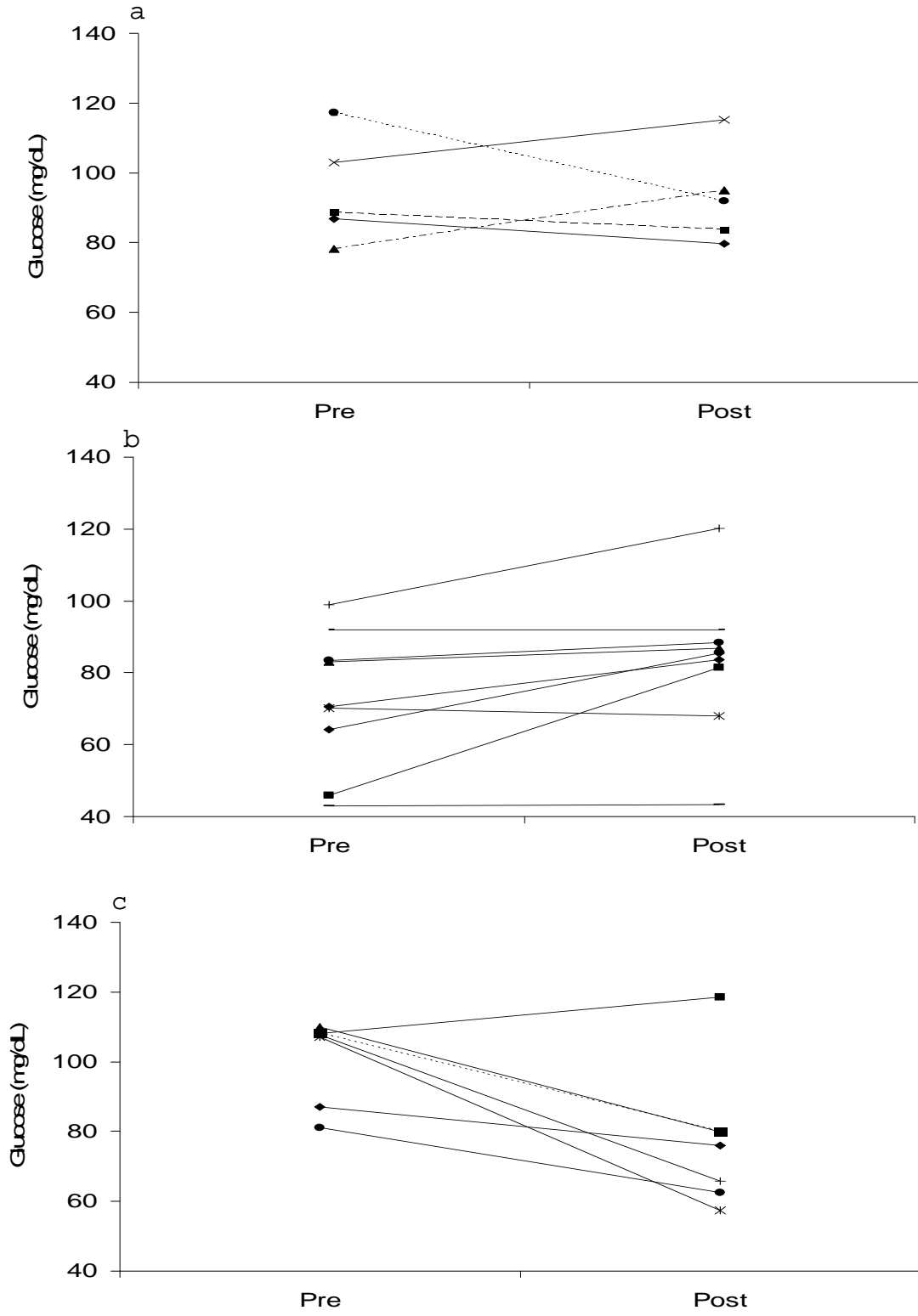


Figure 4. Baseline and post assessment glucose values (mg/dl) (a) Control, (b) LA and (c) Cardio

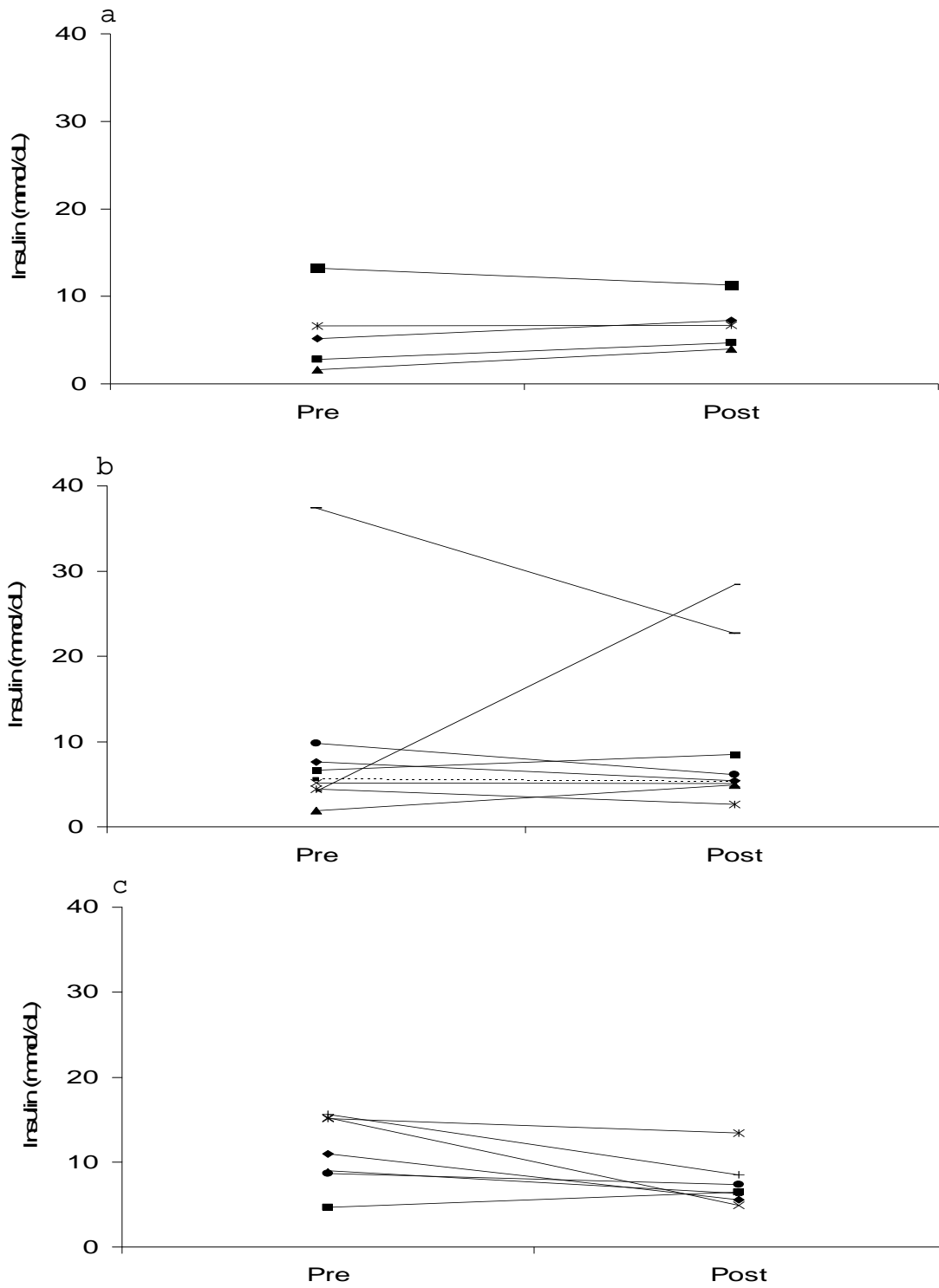


Figure 5. Baseline and post assessment insulin values (uU/ml) (a) Control, (b) LA and (c) Cardio

CHAPTER V

DISCUSSION

Results of the current investigation revealed slight trends toward improvements in body weight, body fat percentage, step count, and cardiorespiratory fitness. Specifically, with regard to body weight and percent body fat (skinfold and BIA), the physical activity groups tended to decrease, while the control group displayed a slight increase. Interestingly, increased step counts were noted among all groups, but the increases were greater in the LA and Cardio groups compared to the Control group. In addition, while cardiorespiratory fitness tended to improve in the Cardio and LA groups, the Control group tended to decrease. These trends clearly suggest small, positive changes or a halting of more deleterious obesity related outcomes resulting from traditional physical activity or lifestyle activities compared to no intervention. Pertinent to these trends are the descriptive changes in activity of each of the activity-based intervention groups. Both the LA and the Cardio groups demonstrated activity increases through pedometer step counts. Congruent with the clearly defined goals of the lifestyle modification program, the LA group had the largest increase in overall step counts, but conversion of activities from the physical activity recall (PAR) and exercise logs to MET values indicated that the Cardio group expended more calories from intentional physical activity than did the LA group.

Although many of the group differences were not statistically significant at 12 weeks, the trends in the data suggest that a similar program performed over a longer duration would participants.

Despite the initial enthusiasm and interest in an activity program among the African-American female community, many of the potential subjects were lost prior to initiation of participation in the intervention groups. While there was commonality of characteristics between women who made initial contact and those that attended interest meetings, it is the researcher's estimation that only half of the initial contacts actually attended interest meetings. Many of the participants attending interest meetings accompanied friends or co-workers. Attrition was lowered dramatically once participants actually started an intervention group. In addition, participants who were randomly assigned to the same intervention group as a co-worker were more likely to complete the intervention. This is no surprise, since studies have shown that individuals who exercise using a buddy system and/or who have accountability to another person or group were more likely to stick with an exercise program (Sharma et al., 2005). Most women expressed interest in being randomized to the traditional exercise group, but the randomized control trial design of the study did not allow randomization based on preference of the participant. The most commonly stated reason for not continuing upon randomization was displeasure with the group assignment. Control subjects were the least likely to continue, followed by LA and then Cardio.

All participants met the BMI inclusion criteria of 23 kg/m^2 , and with an average BMI of $30.8 (\pm 5.9)$ most of the participants were appropriately categorized into the target

group of overweight or obese (National Heart, Lung, Blood Institute & World Health Organization Clinical Guidelines for Weight, 1998). As well, according to the NHLBI's Obesity Education Initiative the body composition values (% body fat), BMI classifications and waist circumference measures placed these subjects in risk categories ranging from increased to very high risk for CVD, hypertension, type II diabetes and other obesity-related disorders (Heyward, 2006). In general, short-term studies that reported changes in body weight and body composition averaged 16 weeks in duration and increased energy expenditure by approximately 2000 kcal/week (Ross et al., 2001; Slentz et al., 2004). Using either of these standards, the current study fell short of these generalized goals for duration of the intervention and caloric expenditure. Despite this, three Control participants increased risk categories (1 from normal- to elevated-risk and 2 from elevated- to high-risk), while seven activity-based participants decreased risk categories (2 LA and 5 Cardio: 3 from high- to elevated-risk and 3 from elevated- to normal-risk) as measured by percent body fat via skinfold and BIA (Heyward, 2006). Though changes in risk classification were reported in this study, it should be noted that cut-offs for risk classification are arbitrarily set based on previous research and that many of the changes within a given individual were relatively small and may not be associated with physiologically significant reductions in overall risk.

While statistical analysis revealed a significant interaction for pedometer step counts among the 3 groups over the 12 week intervention, there were no significant differences among groups when post-hoc analysis was completed. However, inspection of Figure 3 shows a clear pattern of increasing pedometer steps for the LA group, with the

mean approaching 10,000 steps per day as recommended by the US Surgeon General's 1996 report. One of the key goals of the LA curriculum utilized in this intervention was the attainment of the 10,000 steps per day goal (NIH, 2005).

Several other studies have also noted mean post-intervention step counts below the daily 10,000 step recommendation, but the mean age of the participants in these studies was consistently older than the population in the current study and included cancer survivors (Wilson et al., 2005), overweight diabetic women (Tudor-Locke et al., 2004) and hypertensive postmenopausal women (Moreau et al., 2004). However, investigations that included middle-aged populations with greater similarity to participants in the current study also have reported failure to attain the daily recommended step count (Schneider et al., 2006). Of the studies whose mean step totals met the criteria of 10,000 steps by post assessment, both included men and women in their intervention groups (Chan et al., 2004; Hulquist et al., 2005). Hulquist and colleagues (2005), had significant changes in weight and VO_2 max within their four week intervention, but they monitored step count in a laboratory environment, and also controlled exercise intensity. Chan et al., (2004) included a counseling module for modification of diet, exercise and behaviors based on self-efficacy and social support, which may have improved adherence to the intervention. Results from these studies suggest that significant reduction in risk factors and attainment of the 10,000 steps per day goal may require more intensive intervention than the current investigation employed. One confounding variable in the current study may have been the relatively low baseline step values in the LA group. While the baseline step counts of the participants fell at the

upper-end of the average daily steps for American adults (4,000-6,000), they were much lower than baseline levels reported in other LA intervention trials (Croteau 2003; Speck and Looney 2001).

Similar to the LA group, the Cardio group also increased step counts from baseline to post intervention. Thus, alterations in the dependent variables of interest in this study may be confounded by the compounding effect of structured exercise and increased step counts in the Cardio group. Simply allowing participants to be aware of quantifiable measures of their activity habits has been shown to impact activity patterns (Lindberg, 2000; Swartz et al., 2003; Tudor-Locke & Bassett, 2004; Tudor-Locke et al., 2002; and, Tudor-Locke et al, 2001) and likely resulted in the Cardio participants increasing their activity level outside of the structured exercise sessions. Though not statistically significant, the Cardio group had seemingly larger differences from baseline to post assessment than the LA group for most of the variables of interest. It is possible that if the intervention period had been extended, the summative volume of activity of the Cardio group could have resulted in more significant changes compared to the LA group (Snyder et al., 1997; Donnelly et al., 2000; and, Hagan et al., 2005). Conversely, as Tudor-Locke and colleagues (2002) described, pedometer steps do not account for differences in intensity of walking during exercise, and thus do not accurately measure kcal expenditure. Since the focus of the Cardio group was not the accumulation of moderate intensity activity, the increase in accumulated steps likely does not reflect increased “brisk walking” and thus, likely provided minimal contribution to the positive health trends that emerged in the Cardio group at the end of this 12-week study. For

comparative purposes, many of the interventions that achieved significant changes in risk factors (i.e., decreased weight, increased VO₂max, decreases in blood pressure) with pedometer walking alone were either of longer duration (range; 16 weeks to 18 months) (Duncan et al., 1991; Jakicic et al., 1995; Snyder et al., 1997; Woolf-May et al., 1999; and, Donnelly et al., 2000; Tudor-Locke et al., 2004; DPP, 1998), dealt with different ethnic populations (Chan et al., 2004) or different disease categorization (Moreau et al., 2001; Tudor-Locke et al., 2004; DPP, 1998). This study was fairly unique in the utilization of a younger, healthy, African-American population and a short-duration intervention.

The Control group also logged slight increases in the total steps accumulated during the 12 week intervention. As suggested above for the Cardio group, allowing participants to be aware of their activity level may have influenced their behavior and increased their step counts (Lindberg, 2000; Swartz et al., 2003; Tudor-Locke & Bassett, 2004; Tudor-Locke et al., 2002; and, Tudor-Locke et al., 2001). Thus, it may have been prudent to blind the Cardio and Control groups to their accumulated step counts. This simple procedure would likely have reduced the positive behavioral effect of immediate feedback on step counts in these 2 groups.

With regard to energy expenditure it is important to note that the Cardio group did not wear pedometers during their exercise sessions, since pedometers may not accurately assess activity that does not mimic normal gait (i.e., stationary bicycle, elliptical machines or step machines). While step counts were greater among the LA participants (though not significantly different among the two activity groups), the Cardio group had greater

overall caloric expenditure. When MET equivalent equations were used to transform exercise sessions and activity logs in to intentional physical activity caloric expenditure (Figure 2), the Cardio group consistently recorded exercise with greater MET values than the LA group.

The Cardio group expended an average of 389 kcals each exercise session, whereas the LA group had an average daily expenditure of 198 kcals. Factoring in frequency of exercise for both groups, total weekly expenditure for the Cardio group was considerably higher than that of the LA group (1556 vs 1107 kcals). Though these reported expenditures exceeded the target of 700 kcals per week set by the landmark DPP study, the DPP intervention was significantly longer than the current study. The subjects in this study fell within the ACSM (1998) recommendation of 700-2000 kcals of physical activity expenditure each week for positive effects on aerobic fitness and body composition, yet no significant benefits were noted in these variables for either exercise group, perhaps due to the relatively short length of the intervention.

Although some studies of comparable or even shorter length have noted changes in variables such as cardiorespiratory fitness, the populations studied were largely Caucasian male (Debusk et al. 1990) or produced significantly greater deficits in caloric expenditure via exercise (Sopko et al. 1985; Hagan et al., 1986). Conversely, several other walking interventions lasting 12 weeks reported no changes in cardiorespiratory fitness (Shinkai et al., 1994; Murtagh et al., 2005).

Few randomized controlled trials have systematically examined the effects of exercise alone on obesity-related risk. Several studies have demonstrated greater success

in the reduction of risk using diet and exercise over exercise alone; however, these interventions also included concomitant weight loss (Anderssen et al., 2007; Okura et al., 2007). Weight loss alone improved many of the individual metabolic and lipid variables of risk, making it difficult to identify the successful component(s) of the intervention (Yu-Poth et al., 1999). In addition, many of the published studies in this area did not have a control group for comparison, which limited the interpretation of some results (Roberts et al., 2006; Muzio et al., 2005; Esposito et al., 2004; Okura et al., 2007). The current study clearly showed repetitive, small, positive trends for the dependent variables in both the LA and Cardio groups, with trends toward no change or increased risk in the Control participants.

In concordance with studies of similar length and exercise energy expenditure, participants in the current study did not achieve significant weight loss (Hagan et al., 1986, Woolfe-May et al., 1999; Murtagh et al., 2004). When average energy expenditures from physical activity ranged from 190 to 500 kcal per day and the population evaluated was similar to the current study, weight loss was also similar to what was observed in this study (Hagan et al., 1986, Woolfe-May et al., 1999; Murtagh et al., 2004). According to a meta-regression conducted for 9 pedometer-based studies similar to the current study, the expected weight loss for a sedentary adult would average approximately 1 lb. every 10 weeks. Based on this, the expectation of weight loss in a 12 week study that utilized moderate physical activity would be a decrease of approximately 0.6 kg in body weight (Richardson et al., 2008), which is in agreement with the results of this investigation.

The initial mean cardiorespiratory fitness value of all participants (23.5 ml/kg/min) met the “poor” classification for all age ranges of women who participated in the study (Heyward, 1998). Compared to women in high cardiorespiratory fitness categories, a low cardiorespiratory fitness level was an important predictor of all-cause mortality risk ($p=0.002$) (Farrell et al., 2002). Previous research has indicated that an intervention period of twelve weeks was not generally sufficient to change cardiovascular fitness and that a minimum of 15 weeks was required to elicit changes in this variable (ACSM, 2000).

Obesity has been shown to negatively impact plasma lipids and lipoproteins and is one of the pathways through which obesity influenced CVD risk (Howard, 1999). Obesity has been consistently associated with higher TG and lower HDL-C (Richelsen et al., 1993, Nicklas et al., 1997, Denke, Sempos, & Grundy, 1993) and HDL-C of less than 40 mg·dL⁻¹ was an independent CAD risk factor (ACSM, 2000). However some obese individuals exhibited normal plasma lipid and lipoprotein profiles, while others exhibited impaired profiles (Sims, 2001, Vierhapper et al., 2000). This variability in lipid profiles appeared to be associated with the type of obesity, defined by the location of fat accumulation (McLaughlin et al., 2003; McLaughlin et al., 2005; and Li et al., 2008). For example, individuals with accumulated visceral abdominal fat of less than 100 cm² (Despres et al., 1990; Seidell et al., 1991; Haffner et al., 1987; Dowling & Pi Sunyer, 1993; Sims, 2001), who had early onset obesity and marked hyperplasia of adipocytes as they became obese (Sims, 2001), had seemingly normal lipid and lipoprotein profiles. On the other hand, obese individuals with adult onset obesity that displayed marked

adipocyte hypertrophy as they became obese (Sims, 2001) and accumulated visceral abdominal fat greater than 130 cm² (Despres et al., 1990), tended to exhibit impaired plasma lipid and lipoprotein profiles. While these types of longitudinal assessments are telling with regard to obesity and lipid profiles, they are far beyond the scope of the current investigation. The mean TC (239.5 mg·dL⁻¹) and LDL-C (160.8 mg·dL⁻¹) for participants in the current study straddled the classification between borderline high and high, while TG means (93.8 mg·dL⁻¹) were at desirable levels. Mean participant HDL-C levels (63.4 mg·dL⁻¹) fell above the recommended level of 60 mg·dL⁻¹ set by the National Cholesterol Education Program Committee (Heyward, 2006).

While the overweight women in the present study exhibited abnormally high baseline plasma HDL-C, exhibiting levels considered to be cardioprotective (~ 50 mg·dL⁻¹), they also exhibited high TC and LDL-C levels (Heyward, 2006). In addition, while obesity has been reported to be much more prevalent in African American than White or even Hispanic American women, these women also tended to have lower blood TG, TC and LDL-C and higher blood HDL-C levels (Albu et al., 1999; Racette et al., 2000; Lovejoy et al, 1996). In the current study population, only three women had HDL-C below 50 mg·dL⁻¹ (1 Control and 2 Cardio participants) and no women had an HDL-C below the critical level of 40 mg·dL⁻¹, a value which constitutes increased CVD risk (ACSM, 2000). Since all of the women in the current study were sedentary at baseline, the elevated HDL-C in this group was not due to regular participation in physical activity. As mentioned previously, an association between increased age and increased visceral and intramuscular fat has been reported (Ohkawa et al., 2005). Participants in the current

study were both relatively young and had mean waist circumferences below the threshold for increased risk for CVD (88 cm) (Heyward, 2006). Lastly, with regard to the assessment of cholesterol, it should be noted that measurement of cholesterol subfractions was beyond the scope of this study. Particle size of HDL-C has been shown to be a better indicator of CVD risk than traditional lipid panels (Kraus et al., 2002).

While the energy expenditure of the Cardio group was on the lower end of the recommended range for alterations in lipid profiles (Durstine et al. 2001), the physical activity participants in the current study did have significant changes in HDL-C from baseline to post intervention testing, with larger increases associated with larger energy expenditure from physical activity. While literature has shown that a consistent exercise expenditure of 1200-2200 kcal/week was needed for chronic changes in TG and HDL (Durstine et al., 2001), some studies have shown acute changes in HDL-C in healthy populations with as little as 230-800 kcal. Further, one study suggested that exercise interventions were more successful for reducing obesity related risk in adults with abdominal obesity and hypertension compared with individuals with lipid abnormalities (Orchard et al., 2005). This group of women had both higher HDL-C values at baseline and mean waist circumference values that did not exceed the risk criteria for cardiovascular disease and were generally regarded as normotensive.

While the women in the present study did not exhibit severely impaired plasma lipid and lipoprotein profiles, the average TC and LDL-C concentration for each group was above the desirable range set by NCEP (NCEPATP III, 2004). 36 women had a TC level greater than 200 mg·dL⁻¹ and all but one woman had LDL-C levels higher than 100

mg·dL⁻¹. A total of 7 women met the high classification and another 11 had very high LDL levels (NCEPATP III, 2004). These high LDL-C levels have been shown to be risky by themselves, as excess LDL-C can disrupt the normal state of the endothelium and lead to plaque build-up, ultimately resulting in a rupture of the arteries or thrombosis (NIH, 2002). Also, it is important to note the increased association with myocardial infarction related to high LDL-C levels in combination with other factors of comorbidity (i.e., hypertension, obesity) (Wilhelmsen, 1997). Several studies have indicated that moderate exercise without weight loss or changes in dietary patterns was not sufficient to produce changes in either TC or LDL levels, similar to the results of the current investigation (Hinkleman & Neiman, 1993; Andersen et al., 1995; and, Neiman et al., 2002). As with HDL-C subfractions, better prediction of risk for CVD has been related to the sub-fractional particles of LDL-C (Kraus et al., 2002). Even low amounts of exercise similar in volume to those logged by the LA group, have clearly been shown to be beneficial with regard to concentrations of LDL particles, small LDL particles, large HDL particles and large VLDL particles, all of which have been reported to be better indicators of risk (Zilversmit, 1995; Lemarche et al., 1997; Kamigaki et al., 2001; Vakkilainen et al., 2000; Pascot et al., 2001). There is a preponderance of evidence that suggests exercise can reduce small, dense LDL and excess TG, which have been associated with insulin resistance (Lemarche et al., 1997; Howard, 1999; Sanchez, 2001; and, Kraus, 2002). It has been suggested that individuals with TG between 70 and 140 (which would include all but 5 subjects in the current study) may require a direct measure of particle size to

establish the presence of small, dense LDL, as TG levels in this range were not predictive of LDL-subfractions (Lemarche et al., 1997; and, Lemanski, 2004).

The impact of the two physical activity interventions employed in this investigation on plasma blood glucose and insulin concentration were mixed and generalization of the data was difficult due to small sample size. Even so, case wise observations were equally as valuable here. Six study participants (1 Control, 2 LA and 3 Cardio), were classified as having impaired glucose homeostasis according to blood glucose measures (greater than 5.5 mmol/L or 100mg/dL) (ADA, 2004). Of these six participants, three participants reduced glucose levels to normal values by the post-intervention testing. With regard to insulin levels, five participants (1 Control, 1 LA and 3 Cardio group participants) recorded baseline measures above 10 uU/L or 60 pmol/L [Insulin Resistance (IR) (ADA, 2004)]. Regardless of exercise intervention, all of the participants in an exercise group who met the IR criteria reduced insulin values by the completion of the study. In addition, 3 of these participants (1 LA and 2 Cardio) no longer met the criteria for IR post-intervention. Utilizing the ratio of TG:HDL set forth by Liu et al. (2008) as a predictor of IR, most participants in the current study who logged high IR values ($>10\text{uU/L}$), exceeded the 2.0 TG:HDL (TG:HDL range: 1.5 to 2.2). One participant in the LA group dramatically increased plasma insulin values from baseline to post assessment. This individual met the TG:HDL cutoff for African-Americans set by Li et al. (2008), of 2.0 at both testing points (pre=2.16 and post=2.20). This suggested that this particular subject was likely close to being IR at baseline despite the fact that the initial insulin value was below the IR cut-off set by ADA (ADA 2004). While this

particular subject did not respond in the hypothesized fashion to the exercise intervention, it is difficult to know exactly how severe the rise in insulin may have been during the 12 week intervention if the subject had been assigned to the control group instead of to the LA group. With consideration of all the groups, the highest baseline insulin levels were in the Cardio group and the lowest in the Control. The greatest changes in insulin values were noted among Cardio participants, but perhaps this goes back to the fact that they had the highest values at baseline, with more room for change.

In opposition to most studies with similar activity interventions, this study did not reveal significant changes in blood pressure over the 12 week intervention. Though a trend for decreased DBP was noted in both activity groups, that change was neither clinically nor statistically significant. Previous aerobic training studies have clearly established significant reductions in blood pressure in as little as a 4 week-intervention (Murphy et al., 2002; Seals et al., 1997; Swartz et al., 2003; Hultquist et al., 2005). The intensities of each of these studies vary, but most fall within the intensity range recommended to the LA group or recorded by the Cardio groups in this study. For those studies that found changes in SBP/DBP, the prescribed intensities varied from 50 -70 % of heart rate reserve to 60 - 80 % of predicted heart rate maximum (Seals et al., 1997 & Murphy et al., 2002). Smaller changes were noted in groups where goals of walking 30 minutes each day or walking 10,000 steps were given (Swartz et al., 2003 & Hultquist et al., 2005). Many of these studies had small N values for their groups (10-20 subjects) (Murphy et al., 2002; Seals et al., 1997), used subjects at greater risk [Seals et al.1997; Swartz et al., 2003] or did not use minority populations of women. The subjects in this

study were largely characterized as normotensive, and perhaps without the concomitant accumulation of risk factors, the duration of the intervention or intensity of activity in the study was insufficient to produce measurable changes in blood pressure in most subjects.

The two activity interventions did not produce significant improvements in cardiorespiratory fitness, but participants in both exercise groups demonstrated trends toward improvement or at the very least, maintained the same level of cardiorespiratory fitness from baseline to post-intervention. Previous research has shown that low levels of cardiorespiratory fitness were accompanied by greater risk of all-cause mortality, were more predictive of that risk than BMI (Ferrell, 2002), and that the greatest health benefits were gained when sedentary individuals became active (Pate et al., 1995). Exercise regimens that have improved cardiorespiratory fitness (range: 2.5 to 7 ml/kg/min), have an average exercise session duration ranging from 15-60 minutes, with prescribed intensities of 48-84% VO_2max , and activity frequency of 2 to 5 days per week, with minimal study length of 10 weeks up to 2 years (Shinkai et al., 1994; King et al., 1995; Leon et al., 1996; Woolf-May et al., 1997; Schmidt et al., 2001; and, Murtagh et al., 2005). Similar to the current study, 12-week walking studies produced no changes in cardiorespiratory fitness (Shinkai et al., 1994; Murtagh et al., 2005), while several studies of 18-weeks or longer produced changes in cardiorespiratory fitness (King et al., 1995; Leon et al., 1996; Woolf-May et al., 1997; and, Schmidt et al., 2001). Thus, as suggested by ACSM (ACSM 2000), a minimal intervention period of 15 weeks appears to be necessary to improve cardiorespiratory fitness.

Weight loss can also favorably change the risk factors related to obesity, whereby the greater the weight loss, the larger the magnitude of change in cardiovascular risk factors (Dansinger et al., 2005; Yu-Poth et al., 1999; Muzio et al., 2005). The current intervention did not promote significant weight loss. Thus, the results from this randomized controlled trial suggest that changing lifestyle activity in overweight individuals, without an emphasis on weight loss may be insufficient to alter obesity-related risk status in a twelve week intervention. Conversely, both activity groups showed positive trends toward improvements among several risk factors; it is therefore possible to consider that the duration of the study was simply insufficient to produce weight loss and its associated significant reductions in risk.

Regarding intervention physical activity levels, it was interesting to note that both activity groups reported increased pedometer step activity during the intervention. Although the reason for the increased physical activity via walking during the later weeks of the intervention in the Cardio group was unclear, it is possible that the engagement in cardiorespiratory fitness increased energy levels and resulted in increased pedometer step counts. There are several other potential explanations for the elevated activity level of the participants in this study related to the sedentary lifestyle index proposed below (Tudor-Locke & Bassett, 2004).

Tudor-Locke and Bassett (2004) defined a sedentary individual as one who takes fewer than 5,000 steps per day (sedentary lifestyle index). Participants in all groups began the study logging approximately 6000 steps each day. First, it is possible that “sedentary”, as measured by the assessment techniques employed in this study, did not

accurately describe the baseline physical activity patterns of the individuals enrolled.

Self-report of physical activity may have been complicated by several specific characteristics of this sample. Many of the participants in this investigation worked on a college campus, which was transitioning to a “walking campus” at the time of this intervention, so activity levels may have been higher than the general African American female population. Secondly, a significant number of the participants were mental health workers. In conversation with these individuals, many of them were actively engaged in home visits, which may have had them walking to and from parking areas and housing units on a regular basis which is a form of elevated activity compared to the general population. In addition, many of the study participants were enrolled between October and January, and participation in activity may have been influenced by holidays.

The physical activity self-report assessment used in this study has been used measure in physical activity in previous research, but the reliability of self-report within a population similar to this study has not yet been published (Bock et al., 2001; Blair et al., 1985). PAR has been considered the gold standard of self-report measures and has been validated against objective measures of physical activity, including accelerometer data (Hayden-Wade et al., 2003), VO₂max (Dishman & Steinhart, 1988), and heart rate monitors. It has also been validated in at least one ethnic minority sample (i.e., Latinos), but the applicability of those findings to the current sample is uncertain (Rauh et al., 1992). In the current study the physical activity by self-report (in MET equivalency) did not always relate to increased pedometer step counts through accelerometry. A possible reason for this was that the LA group might have simply altered the intensity of activities

that were already a part of their daily routines in order to meet the objectives of accumulated moderate intensity activity. It is possible that participants found it difficult to estimate the intensity and duration of activities that did not represent typical, structured exercise (e.g., jogging, cycling). This difficulty may have resulted in inflated estimates of weekly activity. In fact, research with African American women has suggested that a large proportion of this group's physical activity was accounted for by non-traditional exercise, such as household chores and occupational activities (Banks-Wallace & Conn, 2002; Whitt, DuBose, Ainsworth, & Tudor Locke, 2004), while men were more likely to engage in structured, recreational physical activities (Singh et al., 2001) that were more easily quantified. An alternative explanation with regard to physical activity reporting could be related to social desirability (the desire to meet perceived expectations of the social situation), which is an inherent limitation of most self-report measures (Herbert et al., 2002; Adams et al., 2005). Every effort was made by the researcher to maintain an unbiased study, but over-reporting of PA is still a possibility that must be considered, given the observed lack of a relationship between pedometer steps and self-reported PA in the LA group. Participants were not given monetary compensation for their participation in the study; instead, participants had the option of choosing an alternate activity intervention at the completion of their assigned intervention group, which may have encouraged over-reporting if subjects perceived that some threshold of activity was necessary to allow them to join the structured exercise group. Another factor related to social desirability may have existed as participants developed a friendship with the investigator and felt the need to please that individual.

Though most physical activity was to be logged upon completion, the study did, in particular reference to the LA group, rely on recall as a mode of assessment for convenience of the participant. Participants logged type of activity, intensity and duration (minutes). When relying on recall to log activities for an entire day, discrepancies between the actual activity and the recorded activity could occur. Logs for Cardio participants were completed at the end of each exercise session, limiting errors related to logging of activity. In addition to logging type of activity, intensity and duration, the Cardio group also recorded average heart rate, which could be used to verify the accuracy of the logged activities.. There is also a lack of research examining the validity of the PAR with African-Americans. Interestingly, one study used the PAR as the “reference” measure of activity in order to validate a newly created physical activity questionnaire among African Americans (Singh, Fraser, Knutsen, Lindsted, & Bennett, 2001), despite the fact that the PAR itself has not specifically been validated as an accurate assessment of PA in this population. In general, there has been very limited study of the psychometric properties of any physical activity measure with this population, precluding the use of a better alternative for the current study. In fact, a review of physical activity interventions for African American women reported that only 7 of 18 reviewed studies included a previously developed instrument such as the PAR, while most prior studies have used non-validated measures created for a specific project (Banks-Wallace & Conn, 2002).

Though group cohesion was not measured in the current study, a clear indication of some social support linkage was noted by the researcher. A genuine enthusiasm for interaction was clearly demonstrated within the exercise sessions of the Cardio group,

which was muted in the LA group and not possible for the Control group. Individual participants in the Cardio group often fostered friendships and social interactions beyond simply conversing during intervention sessions. Research has indicated that the social support system available within the recreation and fitness environment is one of the factors that increased adherence and attendance, particularly to a specific program or facility (Iwasaki & Havitz, 2004). Further, research has found that social factors may be particularly important to exercise adherence within the African American community (Izquierdo-Porrera, Powell, Reiner, & Fontaine, 2002). When group cohesion and social factors were investigated as a component of exercise, the participants indicated that social interaction was one of the three most important factors motivating them to attend fitness classes (along with functional fitness and general health), and group integration was significantly related to class attendance (Estabrooks, 2000). Likewise, Gillett (1988) demonstrated in an exercise intervention with moderately overweight women that the social environment affected adherence; of eight factors identified as affecting adherence, three had social components (group homogeneity, carpooling, and social networks within the class). These studies indicate that social factors play a critical role in motivation and adherence to exercise. In addition, it is unclear if beneficial health effects can occur from social interaction alone, beyond the beneficial effects of exercise itself.

Another limitation of the study could be the seasonal variations of the participants eating habits during the course of the intervention period. Unfortunately, the most likely time for completion of the LA or Cardio interventions was between the months of October and January. The holiday period that coincides with this time of year could have

led to tremendous variations in dietary intake. Although caloric intake and macronutrient percentages remained unchanged from baseline to post-assessment, these time points may have missed changes within the intervention. It would be advisable to assess dietary intake at more frequent time points.

Overall, the study promoted positive, albeit small, changes in obesity-related risk among both Cardio and LA participants as compared to Controls. The changes that did occur within the study seemed to largely be associated with energy expenditures during exercise bouts, which were mode dependent (LA versus Cardio). One of the distinct limitations of the study was intervention duration, with 12 weeks seemingly insufficient to cause statistically significant changes. Despite relatively small absolute changes in some variables, clinical risk categorizations did improve among a few of the activity participants, while there were rarely changes in classifications among Control subjects. As noted by Kraus and colleagues (2002) changes can occur in small, dense LDL-C, HDL-C and HDL-C even in the absence of weight loss; therefore, future research should exam cholesterol subfractions in order to better understand changes that may occur with this type of intervention.

REFERENCES

- 1997 Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care*, 20, 1183-1197.
- Ailhaud, G. (2008). Omega-6 fatty acids and excessive adipose tissue development. *Nutrition and Fitness: Cultural, Genetic and Metabolic Aspects*, 98, 51-61.
- Ainslie, P., Reilly, T. & Weterkerp K. (2003). Estimating human energy expenditure: A review of techniques with particular reference to doubly labeled water. *Sports Medicine*, 33(9), 683-698.
- Ainsworth, B. E., Haskell, W. L., Whitt, M. C. (2004). Compendium of physical activities: an Update of activity codes and MET intensities. *Medicine and Science in Sports and Exercise*, 32, S498-S504.
- Ainsworth, B. E. (2000). Challenges in measuring physical activity in women. *Exercise and Sports Science Reviews*, 28, 93-96.
- Ainsworth, B. E., Sternfeld, B., Slattery, M. L., Daguise, V., & Zahm, S. H. (1998). Physical Activity and breast cancer: Evaluation of physical activity assessment. *Cancer*, 83(3), S611-S620.
- Ainsworth, B. E., Leon, A. S., Richardson, M. T., Jacobs, D. R., & Paffenbarger, R. S. (1993). Accuracy of the College Alumnus Physical Activity Questionnaire. *Journal of Clinical Epidemiology*, 46(12), 1403-1411.
- Albu, J. B. Curi, M., Shur, M., Murphy, L., Matthews, D. E., & Pi-Sunyer, F. X. (1999). Systemic resistance to anti-lipolytic effect of insulin in black and white women with visceral obesity. *American Journal of Endocrinology and Metabolism*, 277, E551-E560.
- Alexander, C. M., Lansman, P. B., Teutsch, S. M., & Haffner, S. M. (2003). NCEP-defined metabolic syndrome and prevalence of coronary heart disease among NHANES III participants age 50 years and older. *Diabetes*, 52, 1210-1214.
- American College of Sports Medicine (ACSM). (1990). Position stand on the recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness in healthy adults. *Medicine and Science in Sports and Exercise*, 22, 265-274.

- American College of Sports Medicine (ACSM). (2006). **ACSM Guidelines for Exercise Testing and Prescription, 7th Edition**, (M. Whaley editor) Lipincott Williams and Wilkins. Philadelphia: PA.
- American Dietetic Association (ADA) (1997). Weight management: A position of the American Dietetic Association, *Journal of the American Dietetic Association*, 97, 71-74.
- American Heart Association. (1991). 1992 Heart and stroke facts. Dallas: American Heart Association.
- American Heart Association. (2000). *2000 Heart and stroke statistical update*. Dallas: American Heart Association, 2000, pp. 1-29.
- American Heart Association. (2003). *Heart and stroke statistical update*. Dallas: American Heart Association, 2000, pp. 1-34.
- Andersen, R. E., Wadden, T. A., Bartlett, S. J., Zemel, B., Verde, T. J., & Franckowiak, S. C. (1999). Effects of lifestyle activity versus structured aerobic exercise in obese women. *Journal of the American Medical Association*, 281, 335-340.
- Andersen, R. E., Wadden, T. A., Bartlett, S. J., Vogt, R. A., & Weinstock, R. S. (1995). Relation of weight loss to changes in serum lipids and lipoproteins in obese women. *American Journal of Clinical Nutrition*, 62(2), 350-357.
- Anderssen, S. A., Carroll, S., Urdal, P., & Holme, I. (2007). Combined diet and exercise intervention reverses the metabolic syndrome in middle-aged males: Results from the Oslo Diet and Exercise Study, *Scandinavian Journal of Medicine Science and Sports*, 17(6), 687-695.
- Andres, R., (1985). Mortality and obesity: The rationale for age-specific height-weight tables. In: Andres, R. Bierman, E. L., Hazzard, W.R., eds. **Principles of Geriatric Medicine**, New York, NY: McGraw-Hill Co; 311-318.
- Baldeweg, S. E., Pink, A. M., Yudkin, J. S., & Coppack, S. W. (2000). The relationship between obesity, vascular reactivity and endothelial dysfunction in subjects with non-insulin dependent diabetes mellitus. *International Journal of Obesity Related Metabolic Disorders*, 24, S134-S135.
- Banks-Wallace, J., & Conn, V. (2002). Interventions to promote physical activity among African American women. *Public Health Nursing*, 19, 321-335.

- Belfort, R., Mandarino, L., Kashyap, S., Wirfel, K., Pratipanawatr, T., Berria, R., & Defronzo, R. A. (2005). Dose-response effect of elevated plasma free fatty acid on insulin signaling. *Diabetes*, *54*(6), 1640-1648.
- Bittner, V. (2000). Perspectives on dyslipidemia and coronary heart disease in women, *Journal of the American College of Cardiology*, *46*(9), 1628-1635.
- Blair, S.N. (1994). Physical activity, fitness and coronary heart disease. In: **Physical Activity, Fitness and Health**. Shepphard, R. J. & Stephens, T. (Eds.) Campaign: Human Kinetics, Inc. 579-590.
- Blair, S. N., Kampert, J. B., & Kohl, H. W. (1996). Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all cause mortality in men and women. *Journal of the American Medical Association*, *276*, 205-210.
- Blair, S. N., Haskell, W. L., Ho, P., Paffenbarger, R. S., Vranizan, K. M., Farquhar, J. W. et al. (1985). Assessment of habitual physical activity by a seven-day recall in a community survey and controlled experiments. *American Journal of Epidemiology*, *122*, 794-804.
- Blumenthal, J. A., Emery, C. F., Madden, D. J., George, L. K., Coleman, R. E., Riddle, M. W., McKee, D. C., Reasoner, J., & Williams, R. S. (1989). Cardiovascular and behavioral effects of aerobic exercise training in healthy older men and women. *Journal of Gerontology*, *44* (5), M147-M157.
- Bock, B. C., Marcus, B. H., Pinto, B. M., & Forsyth, L. H. (2001). Maintenance of physical activity following an individualized motivationally tailored intervention, *Annals of Behavioral Medicine*, *23*, 79-87.
- Booth, S., Sallis, J., Ritenbaugh, C., Hill, J., Birch, L., & Frank, L. (2001). Environmental and societal factors affect food choice and physical activity: Rationale, influences and leverage points. *Nutrition Review*, *59* (3), S21-39.
- Bouchard, C., Shepard, R. J., & Stephens, T. (Eds.) (1994). **Physical activity, fitness and health**. Champaign, IL: Human Kinetics.
- Bray, G. A., Bouchard, C., & James, W.P.T., (1998). Definitions and proposed current classification of obesity. In: Bray, G.A., Bouchard, C., & James, W.P.T., eds. **Handbook of Obesity**. New York, NY: Marcel Dekker Inc.; 1998: 31-40.
- Bryant, N., Grovers, R., & James, D. (2002). Regulated transport of the glucose transporter GLUT-4. *National Review of Molecular and Cellular Biology*, *3*, 267-277.

- Brown, D. J., & Metiko, E. B. (2005). Prevalence of hypertension in a sample of Black American adults using JNC-7 classifications, *Journal of the National Black Nurses Association*, 16(2), 1-5.
- Brown, P. S., Miller, W. C., & Eason, J. M. (2006). **Exercise physiology: Basis of human movement in health and disease**. Lippincott, Williams & Wilkins. 311.
- Bursztain, S., Elwyn, D., Askanazi, J., & Kinney, J. (1989). **Energy metabolism and indirect calorimetry**. Baltimore, MD: Williams & Wilkins.
- Cameron, A. J., Shaw, J. E., & Zimmet, P. Z. (2004). The metabolic syndrome: Prevalence in worldwide populations. *Endocrinology Metabolism Clinics of North America*, 33(2), 351-375.
- Chan, C. B, Ryan, D. A., & Tudor-Locke, C. (2004). Health benefits of pedometer based physical activity intervention in sedentary workers. *Preventative Medicine*, 39(6), 1215-1222.
- Chobanian, A. V., Bakris, G. L., Black, H. R. et al. (2003). The seventh report of the Joint National Committee on the prevention, detection, evaluation, and treatment of high blood pressure. *Journal of the American Medical Association*, 289 (19), 2560-2572.
- Church, T. S., Earnest, C. P., Skinner, J. S., & Blair, S. N. (2007). Effects of different doses of physical activity on cardiorespiratory fitness amount sedentary, overweight or obese post menopausal women with elevated blood pressure: A randomized controlled trial, *Journal of the American Medical Association*, 297, 2081-2091.
- Cleland, S. J., Petrie, J. R., Ueda, S., Elliot, H. L., & McConnell, J. (1999). Insulin as a vascular hormone: Implications for the pathophysiology of cardiovascular disease. *Clinical Experimental Pharmacology and Physiology*, 25, 175-184.
- Collins, T., & Winkleby, M. A. (2002). African American women and men at high and low risk for hypertension: A single detection analysis of NHANES III, *Preventative Medicine*, 35 (4), 303-312.
- Conway, J. M., Yanovski, S. Z., Avla, N. A., & Hubbard, V. S. (1995). Visceral adipose tissue differences in black and white women. *American Journal of Clinical Nutrition*, 61, 765-771.

- Cooper, R., Cutler, J., Desvigne-Nickens, P., Fortmann, S., Freidman, L., & Havilik, R. (2000). Trends and disparities in coronary disease, stroke and other cardiovascular diseases in the United States: Findings of the national conference on cardiovascular disease and prevention, *Circulation*, *102*(25), 3137-3147.
- Crouter, S. E., Schnieder, P. L., Karahulu, M. (2003). Validity of 10 electronic pedometers for measuring steps and distance. *Medicine and Science in Sports and Exercise*, *35*, 1455-1460.
- Dansinger, M. L., Gleason, J. A., Griffith, J. L., Selker, H. P., & Schaefer E. J. (2005). Comparison of the Atkins, Ornish, Weight Watchers and Zone diets for weight loss and heart disease risk reduction. *Journal of the American Medical Association*, *293*(1), 43-53.
- Darling, J. L., Linderman, J. K., & Laubach, L. L. (2005). Energy expenditure of continuous and intermittent exercise in college students. *Journal of Exercise Online*, *8*(4), 1-8.
- Dattilo, A. M., & Kris-Etherton, P. M. (1992). Effects of weight reduction on blood lipids and lipoproteins: A meta-analysis. *American Journal of Clinical Nutrition*, *56*(2), 320-328.
- Davidson, L., McNeill, G., Haggarty, P., Smith, J. S., & Franklin, M. E. (1997). Free-living energy expenditure of adult men assessed by continuous heart-rate monitoring and doubly-labelled water. *British Journal of Nutrition*, *78*(5), 695-708.
- DeBusk, R. F., Stenestrand, U., Sheehan, M., & Haskell, W. L. (1990). Training effects of long versus short bouts of exercise in healthy subjects. *American Journal of Cardiology*, *65*, 1010-1013.
- Dehghan, M., & Merchant, A. (2008). Is bioelectric impedance accurate for use in large epidemiological studies? *Nutritional Journal*, *7*, 26.
- Denke, M. A., Sempos, C. T., & Grundy, S. M. (1993). Excess body weight: An underrecognized contributor to high blood cholesterol levels in white American men. *Archives of Internal Medicine*, *153*(9), 1093-1103.
- Despres, J. P., Lemieux, I., Prud'homme, D. (2001). Treatment of obesity: Need to focus on high risk abdominally obese patients. *British Medical Journal*, *322*, 716-20.

- Despres, J. P., Couillard, C., Gagnon, J., Bergeron, J., Leon, A. S., Rao, D. C., Skinner, J. S., Wilmore, J. H., & Bouchard, C. (2000). Race, visceral adipose tissue, plasma lipids, and lipoprotein lipase activity in men and women: The health, risk factors, Exercise training, and genetics (HERITAGE) Family Study. *Arteriosclerosis Thrombosis and Vascular Biology*, 20, 1932-1938.
- Despres, J. P., Moorjani, S., Lupien, P. J., Tremblay, A., Nadeau, A., & Bouchard, C. (1990). Regional distribution of body fat, plasma lipoproteins and cardiovascular disease risk. *Arteriosclerosis*, 10(4), 497-511.
- Diabetes Prevention Program Research Group. (2002). Reduction of the incidence of type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine*, 346, 393-403.
- DiPietro, L., Kohl, H. W., Barlow, C. E., & Blair, S. N. (1998). Improvements in cardiorespiratory fitness attenuate age-related weight gain in healthy men and women: the Aerobics Center Longitudinal Study. *International Journal of Obesity and Related Metabolic Disorders*, 22 (1), 55-62.
- Dishman, R. K., & Steinhardt, M. (1988). Reliability and concurrent validity for a 7-day recall of physical activity in college students. *Medicine and Science in Sports and Exercise*, 20, 14-25.
- Donnelly, J. E., Jacobsen, D. J., Heelan, K. S., Snyder-Heelan, K. A., Seip, R., & Smith, S. (2000). The effects of 18 months of intermittent vs. continuous exercise on aerobic capacity, body weight and composition, and metabolic fitness in previously sedentary, moderately obese females. *International Journal of Obesity and Related Metabolic Disorders*, 24 (5), 566-572.
- Dowling, H. J., & Pi-Sunyer, F. X. (1993). Race-dependent health risks of upper body obesity. *Diabetes*, 42(4), 537-543.
- Duncan, J. J., Gordon, N. F., & Scott, C. B. (1991). Women walking for health and fitness. How much is enough? *Journal of the American Medical Association*, 266 (23), 3295-3299.
- Dunn, A. L., Marcus, B. H., Kampert, J. B., Garcia, M. E., Kohl, H. W., & Blair, S. N. (1999). Comparison of lifestyle and structured interventions to increase physical activity and cardiorespiratory fitness: a randomized trial. *Journal of the American Medical Association*, 281(4), 327-334.
- Deurenberg, P., & Deurenberg-Yap, M. (2003). Validity of body composition methods across ethnic population groups. *Acta Diabetologica*, 40(1), S246-S249.

- Durstine, J., Grandjean, P., Davis, P., Ferguson, M., Alderson, N. & DuBose, K. (2001). Blood lipid and lipoprotein adaptations to exercise: A quantitative analysis, *Sports Medicine*, 31, 1033-1062.
- Durstin, J. L., Grandjean, P. W., Davis, P. G., Ferguson, M. A., Alderson, N. L., & DuBose, K. D. (2001). Blood lipid and lipoprotein adaptations to exercise: A quantitative analysis. *Sports Medicine*, 31(15), 1033-1062.
- Eason, K. E., Masse, L. C., Kelder, S. H., & Tortolero, S. R. (2002). Diary days needed to estimate activity among older African-American and Hispanic women. *Medicine and Science in Sports and Exercise*, 34(8), 1308-1315.
- Eriksson, J. W., Smith, U., Waagstein, F., Wysocki, M., & Jansson, P. A. (1999). Glucose turnover and adipose tissue lipolysis are insulin-resistant in healthy relatives of type 2 diabetes patients: is cellular insulin resistance a secondary phenomenon? *Diabetes*, 48 (8), 1572-1578.
- Esposito, K. Marfella, R., Ciotola, M., Di, P. C., Giugliano, F., & Gugliano, G. (2004). Effect of Mediterranean-style diet on endothelial dysfunction and markers of vascular inflammation in the metabolic syndrome. *Journal of the American Medical Association*, 292, 1440-1446.
- Farrell, S., Braun, S., Kampert, J., Barlow, C., & Blair, S. N. (2000). The relation between body mass index, cardiorespiratory fitness, and all-cause mortality in women. In *American College of Sports Medicine Annual Meeting*. Indianapolis, IN.
- Farrell, S., Braun, L. Barlow, E. E., Cheng, Y., J., & Blair, S. N. (2002). The relation of body mass index, cardiorespiratory fitness and all-cause mortality in women. *Obesity Research*, 10(6), 417-423.
- Finley, C. E., LaMonte, M. J., Waslien, C. L. Barlow, C. E., Blair, S. N., & Nichaman, M. Z. (2006). Cardiorespiratory fitness, macronutrient intake, and the metabolic syndrome: The Aerobics Center Longitudinal Study. *Journal of the American Dietetics Association*, 106. 673-679.
- Flegal, K. M., Graubard, B. I., Williamson, D. F., & Gail, M. H. (2005). Excess deaths associated with underweight, overweight, and obesity. *Journal of the American Medical Association*, 293, 1861-1867.
- Flegal, K. M., Carroll, M. D., Kuczmarski, R. J., & Johnson, C. L., (1998). Overweight and obesity in the United States prevalence and trends, 1960-1994. *International Journal of Obesity*, 22, 39-47.

- Flegal, K. M., Carroll, M. D., Ogden, C. L., & Johnson, C. L. (2002). Prevalence and trends in obesity among US adults, 1999-2000. *Journal of the American Medical Association*, 288 (14), 1723-1727.
- Fountain, K. R., Redden, D. T., Wang, C., Westfall, A. O., & Allison, D. B. (2003). Years of lost life due to obesity, *Journal of the American Medical Association*, 289(2), 187-193.
- Fox, C.S., Pencina, M. J., Wilson, P. W., Paynter, N. P., Vasan, R. S. & D'Agostino, R. B. (2008). Lifetime risk of cardiovascular disease among individuals with and without diabetes stratified by obesity status in the Framingham heart study, *Diabetes Care*, 31(8), 1582-1584.
- Franks, P., Ekelund, U., Brage, S., Wong, M., & Wareham, N. (2004). Does the association of habitual activity with the metabolic syndrome differ by level of cardiorespiratory fitness? *Diabetes Care*, 27, 1187-1193.
- Frayn, K. N. (2000). Visceral fat and insulin resistance--causative or correlative? *British Journal of Nutrition*, 83, S71-S77.
- Friedl, K. E., Westphal, K. A., Marchitelli, L. J., Patton, J. F., Chumlea, W. C., & Guo, S. S. (2001). Evaluation of anthropometric equations to assess body composition changes in young women. *American Journal of Clinical Nutrition*, 73(2), 268-275.
- Gallagher, D., Visser, M., Sepulveda, D., Pierson, R. N., Harrison, T., & Heymsfield, S. B., (1996). How useful is body mass index for comparison of body fatness across age, gender and ethnic group. *American Journal of Epidemiology*, 143, 228-239.
- Garaulet, M., Hernandez-Morante, J. J., Lujan, J., Tebar, F. J., & Zamora, S. (2006). Relationship between fat cell sized and number of fatty acid composition in adipose tissue from different fat depots in overweight/obese humans. *International Journal of Obesity*, 30, 899-905.
- Gerhard, G. T., Sexton, G., Malinow, M. R., DeLoughery, T. G., Evans, A. J., Connor, S. L., Wander, T. C., & Connor, W. E. (1998). Premenopausal black women have more risk factors for coronary heart disease than white women, *American Journal of Cardiology*, 82, 1040-1045.
- Gillett, P. A. (1988). Self-reported factors influencing exercise adherence in overweight women. *Nursing Research*, 37(1), 25-29.

- Grundy, S. M., Blackburn, G., Higgins, M., Lauer, R., Perri, M. G., & Ryan, D. (1999). Physical activity in the prevention and treatment of obesity and its comorbidities. *Medicine and Science in Sports Exercise*, 31 (11), S502-S508.
- Grundy, S. M. (2004a). Obesity, metabolic syndrome, and cardiovascular disease. *The Journal of Clinical Endocrinology & Metabolism*, 89(6), 2595-2600.
- Grundy, S. M., Cleeman, J. I., Merz, C. N., Brewer, H. B., Jr., Clark, L. T., Hunninghake, D. B. et al. (2004). Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III Guidelines. *Circulation*, 110, 227-239.
- Grundy, S. M., Blackburn, G., Higgins, M., Lauer, R., & Ryan, D. (2005). Diagnosis and management of the metabolic syndrome: An American Heart Association/National Heart, Lung and Blood Institute Scientific Statement, *Circulation*, 112(17), 2735-2752.
- Gustat, J., Srinivansan, S. R., Elkasabany, A., & Berenson, G. S. (2002). Relation of self-rated measures of physical activity to multiple risk factors of insulin resistance syndrome in young adults: the Bogalusa Heart Study. *Journal of Clinical Epidemiology*, 55(10), 997-1006.
- Haapanen, N., Milunpalo, S., Vuori, I., Oja, P., & Pasanen, M. (1997). Association of leisure time physical activity with risk of coronary heart disease, hypertension and diabetes in middle-aged men and women. *International Journal of Epidemiology*, 26, 739-747.
- Haffner, S. M., Stern, M. P., Hazuda, H. P., Pugh, J., & Patterson, J. K. (1987). Do upper-body and centralized adiposity measure different aspects of regional body-fat distribution? Relationship to non-insulin-dependent diabetes mellitus, lipids and lipoproteins. *Diabetes*, 36(1), 43-51.
- Hagan, R. E., Upton, S. J., Wong, L., & Whittman, J. (1986). The effects of aerobic conditioning and/or caloric restriction in overweight men and women. *Medicine and Science in Sports and Exercise*, 18, 87-94.
- Hagan, R. E., Williams, H. C. & Whittman, J. (2005). Aerobic training overweight men and women diagnosed with type 2 diabetes. *Medicine and Science in Sports and Exercise*, 37(4), 684-690.
- Hammond, T. Tanguay, J. F., & Bourassa, M. G. (2000). Management of coronary artery disease: Therapeutic options in patients with diabetes. *Journal of the American College of Cardiology*, 36, 355-365.

- Hans, T. S. & Lean, M. E. J. (2001) Anthropometric indices of obesity and regional distribution of fat depots. In: **International textbook of obesity**. Bjorntorp, P. (Ed.) New York: John Wiley & Sons Ltd., 51-65.
- Hardman, A. E. (1999). Accumulation of physical activity for health gains: what is the evidence? *British Journal of Sports Medicine*, 33, 87-92.
- Hardman, A. E., Hudson, A., Jones, P. R., & Norgan, N. G. (1989). Brisk walking and plasma high density lipoprotein cholesterol concentration in previously sedentary women. *British Medical Journal*, 299(6709), 1204-1205.
- Haskell, W. L., Leon, A. S., Caspersen, C. J., Froelicher, V. F., Hagberg, J. M., Harlan, W., Holloszy, J. O., Regensteiner, J. G., Thompson, P. D., Washburn, R. A., & Wilson, P. W. (1997). Cardiovascular benefits and assessment of physical activity and physical fitness in adults. *Medicine and Science in Sports and Exercise*, 24, S201-S220.
- Haskell, W. L., Yee, M. C., Evans, A., & Irby, P. J. (1993). Simultaneous measurement of heart rate and body motion to quantitate physical activity. *Medicine and Science in Sports and Exercise*, 25(1), 109-115.
- Hayden-Wade, H. A., Coleman, K. J., Sallis, J. F., & Armstrong, C. (2003). Validation of the telephone and in-person versions of the 7-day PAR. *Medicine and Science in Sports and Exercise*, 35, 801-809.
- Herd, S. L., Gower, B. A., Dashti, N., & Goran, M. I. (2001). Body fat, fat distribution and serum lipids, lipoproteins and apolipoproteins in African American and Caucasian-Americans, *International Journal of Obesity*, 25, 198-204.
- Heyward, V. H. (1996). Evaluation of body composition, *Sports Medicine*, 22(3), 146-156.
- Heyward, V. (2006). The Physical Fitness Specialist Certification Manual, The Cooper Institute for Aerobics Research, Dallas TX, revised 1997 printed in Advance Fitness Assessment & Exercise Prescription, 4th Ed.p.48.
- Hinkleman, L. L., & Neiman, D. C. (1993). The effects of a walking program on body composition and serum lipids and lipoproteins in overweight women. *Journal of Sports Medicine and Physical Fitness*, 33, 49-58.
- Hong, C. Y., Chia, S. E., & Fong, S. K. (1998). Neuropathy in non-insulin-dependent diabetes mellitus: The significance of symptoms. *Scandinavian Journal of Primary Health Care*, 16(4), 233-237.

- Hopfner R. L., Hasnadka, R. V., Wilson, T. W., McNeill, J. R., & Gopalakrishnan, V. (1998). ETA receptor expression in rat aortic smooth muscle cells, *Diabetes*, *47*, 937-944.
- Howard, B. V. (1999). Insulin resistance and lipid metabolism. *American Journal of Cardiology*. *84*, 28J-32J.
- Hu, F. B., Li, T. Y., Colditz, G. A., Willett, W. C., & Manson, J. E. (2003). Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *The Journal of the American Medical Association*, *289*, 1785-1791.
- Hu, F. B., Manson, J. E., Stampfer, M. J., Colditz, G. A., Lui, S., Solomon, C. G., & Willett, W. C. (2001). Diet, lifestyle and the risk of type 2 diabetes mellitus in women, *New England Journal of Medicine*, *345*, 790-797.
- Hu, F. B., Stampfer, M. J., Colditz, G. A., Ascherio, A., Rexrode, K. M., Willett, W. C., & Manson, J. E. (2000). Physical activity and risk of stroke in women. *The Journal of the American Medical Association*, *283*(22), 2961-2967.
- Hu, F. B., Sigai, R. J., & Rich-Edwards, J. W. (1999). Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a Prospective study. *The Journal of the American Medical Association*, *282*, 1433-1439.
- Hulquist, C. N., Albright, C., & Thompson, D. L. (2005). Comparison of walking recommendations in previously inactive women. *Medicine and Science in Sports and Exercise*, *37*(4), 676-683.
- Huppertz, C., Fischer, B., Kim, Y-B., Kotani, K., Vidal-Puig, A., Slieker, L., Sloop, K., Lowell, B., & Kahn, B. (2001). Uncoupling protein 3 stimulates glucose uptake in muscle cells through a phosphoinositide-3-kinase-dependent mechanism. *Journal of Biological Chemistry*, *276*(16), 12520-12529.
- Huxley, R. H., Huxley, R., Barzi, F., Lee, C. M., Janus, E., Lam, T. H., et al. (2008). Is central obesity a better discriminator of the risk of hypertension than body mass index in ethnically diverse populations? *Journal of Hypertension*, 169-177.
- Jackson, F. M. (2002). Considerations for community-based research with African American women, *American Journal of Public Health*, *92*(4), 561-564.
- Jakicic, J. M., Wing, R. R., Butler, B. A. & Robertson, R.J. (1995). Prescribing exercise in multiple short bouts versus one continuous bout: Effects on adherence, cardiorespiratory fitness, and weight loss in overweight women. *International Journal of Obesity and Related Metabolic Disorders*, *19* (12), 893-901.

- Jakicic, J. M., Wing, R. R., & Lang W. (1998). Bioelectrical impedance analysis to assess body composition in obese adult women: The effect of ethnicity, *International Journal of Obesity*, 22, 243-249.
- Janssen, I., & Jolliffe, C. J. (2006). Influence of physical activity on mortality in elderly with coronary artery disease. *Medicine and Science in Sports and Exercise*, 38(3), 418-427.
- Jeffery, R. W., Drewnowski, A., Epstein, L. H., Stunkard, A. J., Wilson, G. T., Wing, R. R., & Hill, D. R. (2000). Long-term maintenance of weight loss: Current status. *Health Psychology*, 19, S5-S16.
- Johnson, J. L., Slentz, C. A., Duscha, B. D., Samsa, G. P., McCartney, J. S., Houmard, J. A., & Kraus, W. E. (2004). Gender and racial differences in lipoprotein subclass distributions: the STRRIDE study, *Atherosclerosis*, 176, 371-377.
- Kamigaki, A., Siscovick, D. Schawarts, S., Psaty, B. M., Edwards, K. L., Raghunathan, T. E., & Austin, M. A. (2001). Low density lipoprotein particle size and risk of early-onset myocardial infarction in women, *American Journal of Epidemiology*, 153, 939-945.
- Kahn, A. H., & Pressin, J. E. (2002). Insulin regulation of glucose uptake: A complex interplay of intracellular signaling pathways. *Diabetologica*, 45, 1475-1483.
- Kahn, B. B., & Flier, J. S. (2000). Obesity and insulin resistance. *Journal of Clinical Investigation*, 106 (4), 473-481.
- Kaul, L., & Nidiry, J. J. (1999). Management of obesity in low-income African Americans. *Journal of the National Medical Association*, 91(3), 139-143.
- Kay, S. J., & Fiatarone, M. A. (2006). The influence of physical activity on abdominal fat: a systematic review of the literature, *Obesity Research*, 7, 183-200.
- Keller, C. S., Robinson, B., & Pickens, L. (2004). Comparison of two walking frequencies in African-American postmenopausal women. *Association of Black Nursing Faculty Journal*, 15(1), 3-9.
- Kim, H. S., Abbasi, F., Lamendola, C., McLaughlin, T., & Reaven, G. M. (2001). Effect of insulin resistance on postprandial elevations of remnant lipoprotein concentrations in postmenopausal women. *American Journal of Clinical Nutrition*, 74 (5), 592-595.

- King, A., Haskell, W., Young, D., Oka, R., & Stephanick, M. (1995). Long-term effects of varying intensities and formats of physical activity rates on participation rates, fitness and lipoproteins in men and women aged 50 to 65 years, *Circulation*, *91*, 2596-2604.
- Kokkinos, P. F., & Fernhall, B. (1999). Physical activity and high density lipoprotein cholesterol levels: What is the relationship? *Sport Medicine*, *28*(5), 307-314.
- Kraus, W. E., Houmard, J. A., Duscha, B. D., Knetzger, K. J., Wharton, M. B., McCartney, J. S., Bales, C. W., Henes, S., Samsa, G. P., Otvos, J. P., Kulkarni, K. R., & Slentz, C. A. (2002). Effects of amount and intensity of exercise on plasma lipoproteins, *New England Journal of Medicine*, *347* (19), 1483-1492.
- Kriska, A., Aramesh, S., Hanson, R., Bennet, P., Kobes, S., Williams, D., & Knowler, W. (2003). Physical activity, obesity and the incidence of type 2 diabetes in a high-risk population. *American Journal of Epidemiology*, *158* (7), 669-676.
- Kriska, A. M., & Rexroad, A. R. (1998) The role of physical activity in minority populations. *Women's Health Issues*, *8*(2), 98-100.
- Kriska, A. M., Knowler, W. C., La Porte, R. E., Drash, A. L., Wing, R. R., Blair, S. N., Bennett, P. H., & Kuller, L. H. (1990). Development of a questionnaire to examine relationship of physical activity and diabetes in Pima Indians. *Diabetes Care*, *13*(4), 401-411.
- Kuczmarsk, R. J., & Flegal, K. M. (2000). Criteria for definition of overweight in transition: Background and recommendations for the United States. *American Journal of Clinical Nutrition*, *72*(5), 1074-1081.
- Kumanyika, S. (2002). Obesity treatment in minorities. In: **Obesity: Theory and therapy**. Wadden, T. & Stunkard, A. (Eds), New York: Guilford Publications, Inc., 377.
- Laaksonen, D., Lakka, H., Salonen, J., Niskanen, L., Rauramaa, R., & Lakka, T. (2002). Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome. *Diabetes Care*, *25*, 1612-1618.
- LaMonte, M. J., Ainsworth, B. E., & Durstine, J. L. (2004). Influence of cardiorespiratory fitness on the association between C-reactive protein and disease in health, obese middle-aged and older men. *Metabolism*, *46*, 1441-1447.

- Lavrentyev, E., He, D., & Cook, G. (2004). Expression of genes participating in regulation of fatty acid and glucose utilization and energy metabolism in developing rat hearts. *American Journal of Physiology: Heart and Circulatory Physiology*, 287, H2035-H2042.
- Lean, M. E., Han, T.S., & Morrison, E. E. (1995). Waist circumference as a measure for indicating need for weight management. *British Medical Journal*, 311, 158-161.
- Leon, A., Casal, D., & Jacobs, D. (1996). Effect of 2,000 kcal per week of walking on physical fitness and risk factors for coronary heart disease, *Journal of Cardiopulmonary Rehabilitation*, 16, 183-192.
- Leon, A. S., Connett, J., Jacobs, D. R., & Rauramaa, R. (1987). Leisure-time physical activity levels and risk of coronary artery disease and death: The multiple risk factor intervention trial. *Journal of the American Medical Association*, 258(17), 2388-2395.
- Lean, M. E., Han, T. S., & Morrison, C. E. (1995). Waist circumference as a measure for indicating need for weight management, *British Medical Journal*, 311, 158-161.
- Lee, I. M., & Paffenbarger, J. R. S. (2000). Associations of light, moderate, and vigorous intensity physical activity with longevity. *American Journal of Epidemiology*, 151, 293-299.
- Leenen, R., van der Kooy, K., Meyboom, S., Seidell, J. C., Deurenberg, P., & Weststrate, J. A. (1993). Relative effects of weight loss and dietary fat modification on serum lipid levels in the dietary treatment of obesity. *Journal of Lipid Research*, 34(12), 2183-2191.
- Leibel, R. I., Berry, E. M., & Hirsch, J. (1983). Biochemistry and development of adipose tissue in man. In: **Health and Obesity**. Conn, H. L., DeFelice, E. A., & Kuo, P. (Eds), New York: Raven Press, 21-48.
- Lemanski, P. E. (2004). Beyond routine cholesterol testing: The role of LDL particle size assessment, CDPHP Medical Messenger: "LDL Partical Size 1370/20.000404".
- Lemarche, B., Tchernof, A., Moorjani, S., Cantin, B., Dagenais, G., Lupien, P., & Despres, J-P. (1997). Small, dense low-density lipoprotein particles as a predictore of the risk of ischemic heart disease in men. *Circulation*, 95, 69-75.
- Leon, A. S., Connett, J., Jacobs, D. R., & Rauramaa, R. (1987). Leisure-time physical activity to reduced risk of coronary heart disease. In: **Physical activity and cardiovascular health: A national consensus**. A. S. Leon (Ed.) Champaigne: Human Kinetics, Inc., 57-66.

- Li, C., Ford, E. S., Meng, Y-X., Mokdad, A. H., & Reaven, G. M. (2008). Does the association of the triglyceride to high-density lipoprotein cholesterol ratio with fasting serum insulin differ by race/ethnicity? *Cardiovascular Diabetologica*, 7, 4.
- Lieberman, L. S. (2003). Dietary, evolutionary, and modernizing influences on the prevalence of type 2 diabetes. *Annual Review of Nutrition*, 23, 345-77.
- Lindberg, R. (2000). Active living: On the road with the 10,000 steps program. *Journal of the American Dietetics Association*, 100(8), 878-879.
- Lofgren, I., Herron, K., Zern, T., West, K., Patalay, M., Shacher, N., et al. (2004). Waist circumference is a better predictor than body mass index of coronary heart disease risk in overweight women. *The American Society for Nutritional Sciences Journal of Nutrition*, 134, 1071-1076.
- Loos, R. J. F., & Bouchard, C. (2003). Obesity: Is it a genetic disorder? *Journal of Internal Medicine*, 254, 401-425.
- Lovejoy, J. C., de la Bretonne, J. A., Klemperer, M., & Tulley, R. (1996). Abdominal fat distribution and metabolic risk factors: Effects of race, *Metabolism*, 45, 1119-1124.
- Lukaski, H. C., Johnson, P.E., Bolonchuk, W. W., & Lykken, G. I. (1985). Assessment of fat-free mass using bioelectrical impedance measurements of the human body. *American Journal of Clinical Nutrition*, 41, 810-817.
- Manson, J. E., Hu, F. B., Rich-Edwards, J. W., Colditz, G. A., Stampfer, M. J., Willett, W. C., Speizer, F. E., & Hennekens, C. H. (1999). A prospective study of walking as compared with vigorous exercise in the prevention of coronary heart disease in women. *New England Journal of Medicine*, 341, 650-658.
- Mark, D. H. (2005). Deaths attributable to obesity. *Journal of the American Medical Association*, 293, 1918-1919.
- Mayer-Davis, E. J. D'Agostino, R., Karter, A. J., Haffner, S. M., Rewers, M., Saad, M., & Bergman, R. N. (1998). Intensity and amount of physical activity in relation to insulin sensitivity: the Insulin Resistance Study. *The Journal of the American Medical Association*, 279, 669-674.
- Mayo, M. J., Grantham, J. R., & Balasekaran, G. (2003). Exercise-induced weight loss preferentially reduces abdominal fat. *Medicine and Science in Sports and Exercise*, 35 (2), 207-213.

- McLaughlin, Abbasi, F., Cheal, K., Chu, J., Lamendola, C., & Reaven, G. (2003). Use of metabolic markers to identify overweight individuals who are insulin resistant. *Annals of Internal Medicine*, *139*, 802-809.
- McLaughlin T, Reaven G, Abbasi F, Lamendola C, Saad M, Waters D, Simon J., & Krauss R .M. (2005). Is there a simple way to identify insulin-resistant individuals at increased risk of cardiovascular disease? *American Journal of Cardiology*, *96* (3), 399-404.
- Melanson, E., & Freedson, P. (1996). Physical activity assessment: a review of methods. *Critical Reviews in Food Science and Nutrition*, *36*(5), 396.
- Miyatake, N., Nishikawa, H., Morishita, A., Kunitomi, M., Wada, J., Suzuki, H., Takahashi, K., Makino, H., Kira, S., & Fujii, M. (2002). Daily walking reduces visceral adipose tissue areas and improves insulin resistance in Japanese obese subjects. *Diabetes Research and Clinical Practice*, *58* (2), 101-107.
- Mokdad, A. H., Serdula, M. K., Dietz, W. H., Bowman, B.A., Marks, J. S., & Koplan, J. P. (1999) The spread of the obesity epidemic in the United States, 1991-1998, *Journal of the American Medical Association*, *282*, 1519-1522.
- Mokdad, A. H., Ford, E. S., Bowman, B. A., Dietz, W. H., Vinicor, F., Bales, V. S., & Marks, J. S. (2003). Prevalnce of obesity, diabetes and obesity-related health risk factors, 2001. *Journal of the American Medical Association*, *289*, (1), 76-79
- Montague, C. T., & O'Rahilly, S. (2000). The perils of portliness: Causes and consequences of visceral adiposity. *Diabetes*, *49*(6), 883-888.
- Morearu, K., Degarmo, R., Langley, J., McMahon, C., Howley, E., Bassett, D., & Thompson, D. (2001). Increasing daily walking lowers blood pressure in postmenopausal women. *Medicine and Science in Sports and Exercise*, *11*, 1825-1831.
- Morris, A. M., Noakes, M., & Clifton, P. M. (2004). In vitro mononuclear cell production of tumor necrosis factor alpha and weight loss. *Diabetes Research and Clinical Practice*, *63*(3), 179-184.
- Morrish., N. J., Wang, S. L., Stevens, L. K., Fuller, J. H., & Keen, H. (2001). Mortality and causes of death in the WHO multinational study of vascular disease and diabetes. *Diabetologia*, *44*, S14-S21.
- Murphy, M., Nevill, A., Nevill, C., Biddle, S., & Hardman, A. (2002). Accumulating brisk walking for fitness, cardiovascular risk and psychological health. *Medicine and Science in Sports and Exercise*, *34*(9), 1468-1474.

- Murphy, M., & Hardman, A. (1998). Training effects of short and long bouts of brisk walking in sedentary women, *Medicine and Science in Sports and Exercise*, 30, 152-157.
- Murtagh, E. M., Boreham, C. A., Nevill, A., Hare, L. G., & Murphy, M. H. (2005). The effects of 60 minutes of brisk walking per week, accumulated in two different patterns, on cardiovascular risk. *Preventative Medicine*, 41(4), 92-97.
- Muzio, F., Mondazzi, L., Sommariva, D. & Branchi, A. (2005). Long-term effects of low calorie diet on the metabolic syndrome in obese non-diabetic patients. *Diabetes Care*, 28(6), 485-486.
- National Institute of Diabetes and Digestive and Kidney Diseases. Nation Diabetes Statistics, 2007 fact sheet. Bethesda, MD: U. S. Department of Health and Human Services, National Institutes of Health, 2008.
- National Institutes of Health. (1996). Bioelectrical impedance analysis in body composition measurement: National Institutes of Health Technology Assessment Conference Statement. *American Journal Clinical Nutrition*, 64, 524S-532S.
- National Institutes of Health: National Institute of Diabetes, Digestive and Kidney Diseases. (2004). Statistics related to overweight and obesity: The Economic costs. Accessed June 2005. URL: <http://www.win.niddk.nih.gov/statistics/index.htm>
- Neiman, D. C., Brock, D. W., Butterworth, D., Utter, A. C., & Nieman, C. C.. (2002). Reducing diet and/or exercise training decreases the lipid and lipoprotein risk factors of moderately obese women. *Journal of the American College of Nutrition*, 21(4), 344-350.
- Newton, R. L., Alfonso, A., York-Crowe, E., Walden, H., White, M. A., Ryan, D., & Williamson, D. A. (2006). Comparison of body composition methods in obese African American women, *Obesity*, 14, 415-422.
- Nicklas, B. J., Katznel, L. I., Busby-Whitehead, J., & Goldberg, A. P. (1997). Increases in high density lipoprotein cholesterol with endurance exercise training are blunted in obese compared with lean men. *Metabolism*, 46(5), 556-561.
- Ohkawa, S., Odamaki, M., Ikegaya, N., Hibi, I., Miyaji, K., & Kumagai, H. (2005). Association of age with muscle mass, fat mass and fat distribution in non-diabetic haemodialysis patients. *Nephrology, Dialysis and Transplantation*, 20(5), 945-951.

- Okura, T., Nakata, Y., Ohkawara, K., Numano, S., Katayama, Y., & Matsuo, T. (2007). Effects of aerobic exercise on metabolic syndrome improvements in response to weight reduction. *Obesity, 15*, 2478-2484.
- Orchard, T. J., Temprosa, M., Goldberg, R., Haffner, S., Ratner, R., & Marcovina, S. (2005). The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: The Diabetes Prevention Program randomized trial. *Archives of Internal Medicine, 142*, 611-619.
- Orakzai, R., Orakzai, S. H., & Nasir, K. (2006). Association of increased cardiorespiratory fitness with low risk for clustering of metabolic syndrome components in asymptomatic men. *Archives of Medical Research, 37*, 522-528.
- Paffenbarger, R. S., Hyde, R. T., Wing, A. L., & Hsieh, C. C. (1986). Physical activity, all-cause mortality, and longevity of college alumni. *New England Journal of Medicine, 314* (10), 605-613.
- Pan, X. R., Li, G. W., Hu, Y. H., Wang, J. X., Yang, W. Y., An, Z. X., Hu, Z. X., Lin, J., Xiao, J. Z., Cao, H. B., Liu, P. A., Jiang, X. G., Jiang, Y. Y., Wang, J. P., Zheng, H., Zhang, H., Bennett, P. H., & Howard, B. V. (1997). Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: The Da Qing IGT and Diabetes Study. *Diabetes Care, 20*, 537-544.
- Paolisso, G., Tataranni, P. A., Foley, J. E., Bogardus, C., Howard, B. V., & Ravussin, E. (1995). A high concentration of fasting plasma non-esterified fatty acids is a risk factor for the development of NIDDM. *Diabetologia, 38* (10), 1213-1217.
- Pate, R. R., Pratt, M., Blair, S. N., Haskell, W. L., Macera, C. A., Bouchard, C., Buchner, D., Ettinger, W., Heath, G. W., King, A. C., Kriska, A., Leon, A. S., Marcus, B. H., Morris, J., Paffenbarger, R. S., Patrick, K., Pollock, M. L., Rippe, J. M., Sallis, J., & Wilmore, J. H. (1995). Physical activity and health: A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Journal of the American Medical Association, 273*, 402-407.
- Pereira, L. O., & Lancha, A. H. (2004). Effect of insulin and contraction upon glucose transport in skeletal muscle. *Progress in Biophysics and Molecular Biology, 84*(1), 1-27.
- Pescatello, L. S., Franklin, B. A., Fagard, R., Farquhar, W. B., Kelley, G. A., & Ray, C. A. (2004). American College of Sports Medicine Position Stand: Exercise and hypertension. *Medicine and Science in Sports & Exercise, 36*, 533-553.

- Pouliot, M. C., Despres, J. P., Nadeau, A., Moorjani, S., Prud'Homme, D., Lupien, P. J., Tremblay, A., & Bouchard, C. (1992). Visceral obesity in men: Associations with glucose tolerance, plasma insulin and lipoprotein levels. *Diabetes*, *41*(7), 826-834.
- Racette, S. B., Horowitz, J. F., Mittendorfer, B., & Klein, S. (2000). Racial differences in lipid metabolism in women with abdominal obesity. *American Journal of Physiological Regulatory Integrative Comparative Physiology*, *279*, R944-R950.
- Rauh, M. J., Hovell, M. F., Hofstetter, C. R., Sallis, J. F., & Gleghorn, A. (1992). Reliability and validity of self-reported physical activity in Latinos. *International Journal of Epidemiology*, *21*(5), 966-971.
- Reaven, G. (2005). Insulin resistance, type 2 diabetes mellitus and cardiovascular disease: The end of the beginning. *Circulation*, *112*, 3030-3032.
- Rennie, K., Roswell, T., Jebb, S. A., Holburn, D., & Wreham, N. J. (2000). A combined heart rate and movement sensor: Proof of concept and preliminary testing study. *European Journal of Clinical Nutrition*, *54*(5), 409-414.
- Rewers, M., Zaccaro, D., D'Agostino, R., Haffner, S., Saad, M. F., Selby, J. V., Bergman, R., & Savage, P.; Insulin Resistance Atherosclerosis Study Investigators. (2004). Insulin sensitivity, insulinemia, and coronary artery disease: the Insulin Resistance Atherosclerosis Study. *Diabetes Care*, *27* (3), 781-787.
- Richardson, C. R., Newton, T. L., Abraham, J. J., Sen, A., Jimbo, M., & Swartz, A. M. (2008). A meta-analysis of pedometer-based walking interventions and weight loss. *Annals of Family Medicine*, *6*(1), 69-77.
- Richelsen, B., Prdersen, S. B., Moller-Pedersen, T., Schmitz, O., Moller, N., & Borglum, J. D. (1993). Lipoprotein lipase activity in muscle tissue influenced by fatness, fat distribution and insulin in obese females. *European Journal of Clinical Investigation*, *23*(4), 226-233.
- Riemens, S. C., Sluiter, W. J., & Dullaart, R. P. (2000). Enhanced escape of non-esterified fatty acids from tissue uptake: its role in impaired insulin-induced lowering of total rate of appearance in obesity and type II diabetes mellitus. *Diabetologia*, *43* (4), 416-426.
- Rising, R., Swinburn, B., Larson, K., & Ravussin, E. (1991). Body composition in Pima Indians: Validation of bioelectrical impedance. *American Journal of Clinical Nutrition*, *53*, 594-598.

- Roberts, C. K., Won, D., Pruthi, S., Kurtovic, S., Sindhu, R. K., & Vaziri, N. D. (2006). Effect of a short-term diet and exercise intervention on oxidative stress, inflammation, MMP-9, and monocyte chemotactic activity in men with metabolic syndrome factors. *Journal of Applied Physiology*, *100*, 1657-1665.
- Rosamond, W., Flegal, K., & Furie, K. (2007). Heart disease and stroke statistics 2008 update: a Report fro the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*, December, *17*, 2007.
- Ross, R., Dagnone, D., Jones, J. H., Smith, A., Paddags, Hudson, R., & Janssen, , I. (2000). Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise induce weight loss in men: A randomized controlled trial. *Annals of Internal Medicine*, *133*, 92-103.
- Ross, R., Freeman, J. A., & Janssen. (2000). Exercise alone is an effective strategy for reducing obesity and related comorbidities. *Exercise and Sports Science Reviews*, *28*, 165-170.
- Ross, R., & Janssen, I. (1999). Is abdominal fat preferentially reduced in response to exercise-induced weight loss? *Medicine and Science in Sports and Exercise*, *31*, S568-S572.
- Sanchez, S. S., Aybar, M. J., Velarde, M. S., Prado, M. M., & Carrizo, T. (2001). Relationship between plasma Endothelin-1 and glycemic control in type 2 diabetes mellitus. *Hormone and Metabolic Research*, *33(12)*, 748-751.
- Santiago, M. C., Leon, A. S., & Serfass, R. C. (1995). Failure of 40 weeks of brisk walking to alter blood lipids in normolipemic women. *Canadian Journal of Applied Physiology*, *20(4)*, 417-428.
- Scheen, A. J., & Luyckx, F. H. (1999). Medical aspects of obesity, *Acta Chirurgica Belgica*, *99(3)*, 135-139.
- Schoeller, D. A., & Luke, A. (2000). Bioelectrical impedance analysis prediction equations differ between African Americans and Caucasians, *Annals of New York Academy of Science*, *904*, 225-226.
- Schmidt, W., Biwer, J., & Kalscheuer, L. (2001). Effects of long versus short bout exercise on fitness and weight loss in overweight females, *Journal of the American College of Nutrition*, *20(5)*, 494-501.

- Schwartz, R. S., Cain, K. C., Shuman, W. P., Larson, V., Stratton, J. R., Beard, J. C., Kahn, S. E., Cerqueira, M. D., & Abrass, I. B. (1992). Effect of intensive endurance training on lipoprotein profiles in young and older men. *Metabolism, 41*(6), 649-651.
- Schneider, P. L., Bassett, D. R., Thompson, D. L., Pronk, N. P., & Bielak, K. M. (2006). Effects of a 10,000 steps per day goal in overweight adults. *American Journal of Health Promotion, 21*(2), 85-89.
- Sharma, A. M., & Chetty, V. T. (2005). Obesity, hypertension and insulin resistance. *Acta Diabetologica, 1*, S3-S8.
- Shen, W. J., Liang, Y., Wang, J., Harada, K., Patel, S., Michie, S. A., Osuga, J., Ishibashi, S., & Kraemer, F. B. (2007). Regulation of hormone sensitive lipase in islets. *Diabetes Research and Clinical Practice, 75*(1), 14-26.
- Seals, D. R., Silvrman, H. G., Reiling, M. J., & Davy, K. P. (1997). Effect of regular aerobic exercise on elevated blood pressure in postmenopausal women. *American Journal of Cardiology, 80*(1), 49-55.
- Segal, K. R., Gutin, B., Presta, E., Wang, J., & Van Itallie, T. B. (1985). Estimation of human body composition by electrical impedance methods: A comparative study. *Journal of Applied Physiology, 58*, 1565-1571.
- Segal, K. R., Van Loan, M., Fitzgerald, P. I., Hodgdon, J. A., & Van Itallie, T. B. (1998). Lean body mass estimation by bioelectrical impedance analysis: a four-site cross-validation study. *American Journal of Clinical Nutrition, 57*, 7-14.
- Seidell, J. C. (1998). Effects of obesity. *Medicine, 4*-8.
- Seidell, J. C., Cigonlini, M., Deslypere, J. P., Charzewska, J. A., Ellsinger, B. M., & Cruz, A. (1991). Body fat distribution in relation to serum lipids and blood pressure in European men: the European fat distribution study. *Atherosclerosis, 86* (2-3), 251-260.
- Sharp, P. C., & Konen, J. C. (1997). Women's cardiovascular health. *Primary Care, 24*(1), 1-14.
- Shephard, R. J. (2001). Compliance: the Achilles heel of cardiac rehabilitation programs? *Medicine and Science in Sports and Exercise, 33*(11), 1976-1978.

- Shephard, R. J. (2001). Absolute versus relative intensity of physical activity in a dose-response context. *Medicine and Science in Sports and Exercise*, 33(6), S400-S418.
- Shephard, R. J., & Balady, G. J. (1999). Exercise as cardiovascular therapy. *Circulation*, 99(7), 963-972.
- Shepherd, P. R., & Kahn, B. B. (1999). Glucose transporters and insulin action. Implication for insulin resistance and diabetes mellitus. *New England Journal of Medicine*, 341, 248-257.
- Shinkai, S., Watababe, S., Kurokawa, Y., Torii, J., Asai, H., & Shephard, R. J. (1994). Effects of 12 weeks of aerobic exercise plus dietary restriction on body composition, resting energy expenditure and aerobic fitness in mildly obese middle-aged women. *European Journal of Applied Physiology and Occupational Physiology*, 68(3), 258-265.
- Shulman, G. I. (2000). Cellular mechanisms of insulin resistance. *Journal of Clinical Investigation*, 106 (2), 171-176.
- Shuman, W., Morris, L., Leonetti, D. et al. (1986). Abnormal body fat distribution detected by computed tomography in diabetic men. *Investigative Radiology*, 21, 483-487.
- Sims, E. A. H. (2001). Are there persons who are obese, but metabolically healthy? *Metabolism*, 50(12), 1499-1504.
- Slattery, M. L., Jacobs, J. D., & Nichaman, M. Z. (1989). Leisure-time physical activity and coronary heart disease death: The U. S. Railroad Study. *Circulation*, 79, 304-311.
- Slattery, M. L., McDonald, A., & Bild, D. E. (1992). Associations of body fat and its distribution with dietary intake, physical activity, alcohol, and smoking in blacks and whites. *American Journal of Clinical Nutrition*, 55, 943-949.
- Slentz, C., Duscha, B., & Johnson, J. (2004). Effects of the amount of exercise on body weight, body composition and measures of central obesity. *Archives of Internal Medicine*, 164, 31-39.
- Smith, S. C. (2007). Multiple risk factors for cardiovascular disease and diabetes mellitus. *American Journal of Medicine*, 120(3), S3-S11.

- Snyder, K. A., Donnelly, J. E., Jabobsen, D. J., Hertner, G., & Jakicic, J. M. (1997). The effects of long-term, moderate intensity, intermittent exercise on aerobic capacity, body composition, blood lipids, insulin and glucose in overweight females. *International Journal of Obesity and Related Metabolic Disorders*, 21 (12), 1180-1189.
- Sopko, G., Leon, A. S., Jacobs, D. R., Foster, N., Moy, J., Kuba, K., Anderson, J. T., Casal, D., McNally, C., & Frantz, I. (1985). The effects of exercise and weight loss on plasma lipids in young obese men. *Metabolism*, 34(3), 227-236.
- Speakman, J. R., Perez-Camargo, G., McCappin, T., Frankel, T., Thompson, P., & Legrand-Defretin, V. (2001). Validation of the doubly-labelled water technique in the domestic dog. *British Journal of Nutrition*, 85(1), 75-87.
- Speck, B. J., & Looney, S. W. (2001). Effects of a minimal intervention to increase physical activity in women: Daily activity records. *Nursing Research*, 50(6), 374-378.
- Srinivasan, S. R., Wattigney, M. S., Webber, L. S., Berenson, G. S. (1991). Race and gender differences in serum lipoproteins of children, adolescents, and young adults-emergence of an adverse lipoprotein pattern in white males: The Bogalusa Heart Study. *Preventive Medicine*, 20, 671-684.
- Stefanick, L., Mackey, S., Sheehan, M., Ellsworth, N., Haskell, W. L., & Wood, P. D. (1998). Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *New England Journal of Medicine*, 339 (1), 12-20.
- Sternfeld, B. (2003). Changes in physical fitness, physical activity and lipid profile in the CARDIA study. *Annals of Epidemiology*, 9(1), 25-33.
- Stofan, J. R., DiPietro, L., Davis, D, Kohl, H. W., & Blair, S. N. (1998). Physical activity patterns associated with cardiorespiratory fitness and reduced mortality: the Aerobics Center Longitudinal Study. *American Journal of Public Health*, 88(12), 1807-1813.
- Strath, A., Bassett, D., Swartz, A., & Thompson, D. (2002). Validity of simultaneous heart rate-motion sensor technique to predict energy expenditure. *Medicine and Science in Sports and Exercise*, 34(4), 888-894
- Sun, S. S., Chumlea, W. E., Heymsfield, S. B., Lukaski, H. C., Schoeller, D., Friedl, K., Kuczmarski, R. J., Flegal, K. M., Johnson, C. L., & Hubbard, V. S. (2003). Development of bioelectrical impedance analysis prediction equations for body composition with the use of a multicomponent model for use in epidemiologic surveys, *American Journal of Clinical Nutrition*, 77(2), 331-340.

- Swartz, A. M., Bassett, D. R., Moore, J. B., Redwine, B. A., Groer, M., & Dixie, L. (2003). Effects of body mass index on the accuracy of an electronic pedometer. *International Journal of Sports Medicine, 24*, 588-592.
- Swartz, A. M., Strath, S. J., Bassett, D. R., O'Brien, W. L., King, G. A., & Ainsworth, B. E. (2000). Estimation of energy expenditure using CSA accelerometers at hip and wrist sites. *Medicine and Science in Sports and Exercise, 32*(9), S450-456.
- Takasaki, Y., Loy, S. F., & Juergens, H. W. (2003). *Journal of Physiological Anthropology and Applied Human Science, 22*, 233-235.
- Talbot, L. A., Gaines, J. M, Huynh, T. N., & Metter, E. J. (2003). A home-based pedometer-driven walking program to increase physical activity in older adults: a Preliminary study. *Journal of American Geriatrics Society, 51*(3), 387-392.
- Taylor, H. L., Klepetar, E., Keys, A., Parlin, H., Blackburn, H., & Pchner, T. (1962). Death rates among physically active and sedentary employees of the railroad industry. *American Journal of Public Health, 52*, 1696-1707.
- Taylor, C. B., Coffey, T., Berra, K., Iaffaldano, R., Casey, K., & Haskell, W. L. (1984). Seven-day activity and self-report compared to a direct measure of physical activity. *American Journal of Epidemiology, 120*(6), 818-824.
- The Writing Group for the Activity Counseling Trial Research Group. (2001). Effects of physical activity counseling in primary care: the activity counseling trial, a randomized controlled trial. *The Journal of the American Medical Association, 286*, 677-687.
- Thompson, P. D., Buchner, D., Pina, I. L., Balady, G. J., Williams, M. A., Marcus, B. H., Berra, K., et al. (2003). Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease. *Circulation, 107*, 3109.
- Thompson, P. D., Crouse, S. F., Goodpaster, B., Delley, D, Moyna, M., & Pescatello, L. (2001). The acute versus chronic response to exercise. *Medicine and Science in Sports and Exercise, 33*, S438-S445.
- Tran, Z. V., & Weltman, A. (1985). Differential effects of exercise on serum lipid and lipoprotein levels seen with changes in body weight. *Journal of the American Medical Association, 254*(7), 919-924.
- Trost, S. G., McIver, K. L., & Pate, R. R. (2005). Conducting accelerometer-based activity assessments in field-based research. *Medicine and Science in Sports and Exercise, 37*(11), S531-S543.

- Tucker, L. A., & Mortell, R. (1993). Comparison of the effects of walking and weight training programs on body image in the middle-aged women: an Experimental study. *American Journal of Health Promotion, 8*(1), 43-42.
- Tudor-Locke, C., & Bassett, D. (2004). How many steps are enough?: Preliminary pedometer indices for public health, *Sports Medicine, 34*(1), 1-8.
- Tudor-Locke, C., Henderson, K. A., Wilcox, S., Cooper, R. S., Durstine, L., & Aisworth, B. (2003). In their own voices: Definitions and interpretations of physical activity. *Women's Health Issues, 13*(5), 194-199.
- Tudor-Locke, C., Williams, J. E., Reis, J. P., & Pluto, D. (2002). Utility of pedometers for assessing physical activity: Convergent validity. *Sports Medicine, 32*(12), 794-808.
- Tudor-Locke, C., & Myers, A. M. (2001). Challenges and opportunities for measuring physical activity in sedentary adults. *Sports Medicine, 31*(2), 91-100.
- Tudor-Locke, C., Myers, A. M., & Rodger, N. W. (2001). Development of a theory based daily activity intervention for individuals with type 2 diabetes. *Diabetes Education, 27*(1), 85-93.
- Tumiolehto, J., Lindstrom, J., & Eriksson, J. (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine, 344*, 1343-1350.
- U. S. Department of Agriculture/Department of Health and Human Services, (1990). Dietary Guidelines fro Americans, 3rd Ed. Washington, D.C.: US Department of Agriculture/Department of Health and Human Services Home and Garden Bulletin.
- U. S. Department of Agriculture/Department of Health and Human Services, (1995). Dietary Guidelines fro Americans, 4th Ed. Washington, D.C.: US Department of Agriculture/Department of Health and Human Services Home and Garden Bulletin.
- U. S. Department of Agriculture/Department of Health and Human Services, (2000). Dietary Guidelines fro Americans, 5th Ed. Washington, D.C.: US Department of Agriculture/Department of Health and Human Services Home and Garden Bulletin.

- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, President's Council on Physical Fitness and Sports. (1996). Physical activity and health: A report of the Surgeon General. Washington D. C.: U. S. Government Printing Office.
- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, (2002). Overweight among U.S. adults. Washington D. C.: U. S. Government Printing Office.
- U.S. Department of Public Health and Welfare. National Center for Health Statistics Division of Health Examination Statistics.(1969). Factors related to response in a health examination survey: United States 1960-1962. Washington D. C.: U. S. Government Printing Office.
- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics,(1975). Health, United States 1971-1974 with chartbook on trends in the health of Americans: NHANES II. Hyattsville, MD: U. S. Government Printing Office.
- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics,(1981). Health, United States 1976-1980 with chartbook on trends in the health of Americans: NHANES III. Hyattsville, MD: U. S. Government Printing Office.
- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics,(1995). Health, United States 1988-1994 with chartbook on trends in the health of Americans. Hyattsville, MD: U. S. Government Printing Office.
- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics,(2007). Health, United States 2007 with chartbook on trends in the health of Americans. Hyattsville, MD: U. S. Government Printing Office.
- U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Behavioral Risk Factor Surveillance System Survey Data (BRFSS)1984-2007, accessed June 26, 2008. Available at <http://apps.nccd.cdc.gov/brfss>

- U. S. Department of Health and Human Services. *Healthy People 2010*. 2nd Ed. Washington DC: U. S. Government Printing office, November 2003.
- Vakkilainen, J., Makimattila, S., & Seppala-Lindroos, A. (2000). Endothelial dysfunction in men with small LDL particles, *Circulation*, *102*, 716-721.
- VanZwieten, P. A. (1997). Endothelial dysfunction in hypertension: A critical evaluation. *Blood Pressure (Supplement 2)*, 67-70.
- Vierhapper, B., Nardi, A., & Grosser, P. (2000). Prevalence of paradoxically normal serum cholesterol in morbidly obese women. *Metabolism*, *49*(5). 607-610.
- Villa, E., Glonzalez,-Albarran, O., & Rabano, A. (1999). Effects of hyperinsulinemia on vascular blood flows in experimental obesity, *Journal of Steroid Biochemistry and Molecular Biology*, *69*(1), 273-279.
- Vortuba, S. B., & Jensen, M. D. (2007). Sex differences in abdominal, gluteal, and thigh LPL activity, *American Journal of Physiology, Endocrinology and Metabolism*, *292*, E1823-1828.
- Walker, A. B., Chattington, P. D., Buckingham, R. E., & Williams, G. (1999). Thiazolidinedione rosiglitazone lowers blood pressure and protects against impairment of endothelial function in Zucker fatty rats, *Diabetes*, *48*, 1148-1453.
- Wareham, N. J., Wong, M. Y., Hennings, S., Mitchell, J., Rennie, K., Cruickshank, K., & Day, N. E. (2000). Quantifying the association between habitual energy expenditure and blood pressure. *International Journal of Epidemiology*, *29*(4), 655-660.
- Wei, M., Gaskill, S. P., Haffner, S. M., & Stern, M. P. (1997). Waist circumference as the best predictor of noninsulin dependent diabetes mellitus (NIDDM) compared to body mass index, waist/hip ratio and other anthropometric measurements in Mexican Americans--a 7-year prospective study. *Obesity Research*, *5* (1), 16-23.
- Wessel, T. R., Arant, C. B., Olson, M. S., Johnson, D., Reis, S. E., Sharaf, B. L. et al., (2004). Relationship of physical fitness vs. body mass index with coronary artery disease and cardiovascular events in women. *Journal of the American Medical Association*, *292*(10), 1179-1187.
- White, M. F. (2003). Insulin signaling in health and disease. *Science*, *302*, 1710-1711.

- Whitt, M. C., DuBose, K. D., Ainsworth, B. E., & Tudor-Locke, C. (2004). Walking patterns in a sample of African American, Native American and Caucasian women: the Cross-cultural activity participation study. *Health Education and Behavior, 31*(4), S45-S56.
- Whitt, M., Kumanyika, S., & Bellamy, S. (2003). Amount and bouts of physical activity in a sample of African-American women. *Medicine and Science in Sports and Exercise, 35*(11), 1887-1893.
- Wilhelmsen, L. (1997). ESC Population Studies Lecture 1996: Cardiovascular monitoring of a city over 30 years. *European Heart Journal, 18*(8), 1220-1230.
- Williams, L. B., & Considine, R. V. (2001). Etiology of obesity. In: **Obesity**. Gumbiner, B., Eds, Philadelphia: American College of Physicians. 23-49.
- Williamson, D. F., Madans, J., Anda, R. F., Kleinman, J. C., Kahn, H. S., & Byers, T. (1993). Recreational physical activity and ten-year weight change in a US national cohort. *International Journal of Obesity, 17*, 279-286.
- Wilmore, J. H., & Behnke, A. R. (1969). An anthropometric estimation of body density and lean body weight in young men. *Journal of Applied Physiology, 27* (1), 25-31.
- Wilson, D.B., Porter, J. S. Parker, G., & Kilpatrick, J.(2005). Anthropometric changes using a walking intervention in African American breast cancer survivors: a pilot study. *Prevention of Chronic Disease, 2*(2), A16.
- Winkleby, M. (1994). The future of community-based cardiovascular disease intervention studies. *American Journal of Public Health, 84* (9), 1369-1372.
- Wixted, A., Theil, D., James, D., Hahn, A., Gore, C., & Pyne, D. (2005). Signal processing for estimating energy expenditure of elite athletes using accelerometers. *Sensors, 13*(3), 4-8.
- Wojtaszewski, J. R., Neilsen, J. N., & Richter, E. A. (2002). Exercise effects on muscle insulin signaling and action: Invited review. *Journal of Applied Physiology, 93*, 384-392.
- Wolf, A. M., & Golditz, G. A. (1999). Social and economic effects of body weight in the United States. *American Journal of Clinical Nutrition, 63*, 466S-469S.
- Wolf-May, K., Bird, S., & Owen, A. (1997). Effects of an 18 week walking programme on cardiac function in previously sedentary or relatively inactive adults. *British Journal of Sports Medicine, 37*, 48-53.

- World Health Organization. (1998). Obesity: Preventing and managing the global epidemic. Report of a WHO Consultation on Obesity. Geneva, June 3-5, 1997. Geneva: World Health Organization, 1998. WHO/NUT/NCD/98.1.
- World Health Organization. (2008). The Surf report 2: Surveillance of chronic disease risk factors. Accessed June 16, 2008, URL: http://www.who.int/ncd_surveillance/infobase/web/surf2/start.html
- Yancey, A. K., Kumanyika, S. K., Ponce, N. A., McCarthy, W. J., Fielding, J. E., Leslie, J. P., & Akbar, J. (2004). Population-based interventions engaging communities of color in healthy eating and active living: A review, *Preventing Chronic Disease, 1(1)*, [electronic journal] Accessed June 2005: URL: http://www.cdc.gov/pcd/issues/2004/jan/03_0012.htm
- Yu-Poth, S. Zhao, G., Etherton, T., Naglack, M., Jonnalagadda, S., & Kris-Etherton, P. M., (1999). Effects of the National Cholesterol Education Program's Step I and II dietary intervention programs on cardiovascular disease risk factors: a meta-analysis. *American Journal of Clinical Nutrition, 69*, 632-646.
- Zierath, J. R., Krook, A., & Wallberg-Henriksson, H. (2000). Insulin action and insulin resistance in human skeletal muscle. *Diabetologia, 43*, 821-835.
- Zilversmit, D.(1995). Atherogenic nature of triglycerides, postprandial lipidemia, and triglyceride-rich remnant lipoproteins. *Clinical Chemistry, 41*, 153-158.

APPENDIX A

Outline of Sessions

Lifestyle Activities Group Sessions:

Session 1A:	Welcome	Defining LA Group
Session 1B:	It's All About THE FORM	Forms for the Study-HOW TO's
Session 2:	Keep It Safe	Stretching, Preventing Injury, When to Stop & RICE; Pedometers
Session 3:	Getting Started Being Active	Activity Options
Session 4:	Being Active: A Way of Life ; Just Do It: Getting Going At Work or After Work	Working Activity into YOUR day, Your Night, Your Weekend, YOUR LIFE!
Session 5:	FITT	FITT Principle/Ways to determine exercise intensity (Heart Monitors/Borg Scale)
Session 6:	Jump Start Your Activity Plan	Activity Assessment and Activity Plan
Session 7:	Staying Active on Vacation/Holidays; Make it Fun, Make it Fit; Before and After Work	Non-Traditional Exercise
Session 8:	Be a step detective	Strategies for Extra Steps
Session 9:	TRACKING	Plan for Activity & Track Progress

CARDIO Group Sessions:

Session 1A:	Welcome to the Lifestyle Activities Program	Definitions, Quantity, Quality of Life
Session 1B:	It's All About THE FORM	Forms for the Study-HOW TO's
Session 1C:	Fitness Center Orientation	How TO's: Cardio Equipment and HR Monitors
Session 2:	A Safe Start to Activity	Healthy Activity & Pedometers

Control Group Sessions:

Session 1A:	Welcome	Defining Control Group
Session 1B:	It's All About THE FORM	Forms for the Study-HOW TO's
Session 2:	Physical Activity and Health	Risks of Being Inactive
Session 3:	Physical Activity Recommendations	How Much is Enough
Session 4:	FITT	
Session 5:	Keep It Safe	Stretching, Preventing Injury, When to Stop & RICE; Pedometers
Session 6:	Being Active: A Way of Life ;Staying Active on Vacation/Holidays; Make it Fun, Make it Fit; Before and After Work	Working Activity into YOUR day, Your Night, Your Weekend, YOUR LIFE!

APPENDIX B

Session Activities

Lifestyle Activities Sessions:

Session 1a/b:

Meet the research and programming team.

Review the Standard Healthy Lifestyle Guidelines.

Be given the Lifestyle Activities notebook.

Discuss the participant's initial reaction to being assigned to the Lifestyle Activities group.

Receive an overview of the Lifestyle Activities Program.

Learn the two Lifestyle Activities goals and why they are important.

Have an explanation of how to fill out the forms.

Have someone explain how to use pedometers (if not explained already)

Discuss key aspects of the director-participant relationship and sign a related agreement.

Session 2:

Focus on physical activity goal

Be given an initial physical activity goal of "doing something to be physically active" on 3 to 4 days next week.

Be encouraged to attend the Lifestyle Activity sessions.

Be assigned self-monitoring of physical activity.

Session 3:

Receive the Lifestyle Activities activity goal.

Discuss why the activity goal is important.

Discuss current level of physical activity.

Be encouraged to participate in the Lifestyle Activity sessions.

Identify other activities equivalent to brisk walking that the participant enjoys.

Discuss the importance of injury prevention.

Discuss the importance of preparing for exercise (stretching, warm-up and cool-down)

Discuss when it is appropriate to STOP exercising.

Develop an activity plan for the coming week (for most participants, this will be a total for the week of 60 minutes of activity) that includes the Lifestyle Activities activity sessions and other moderate activities that the participant enjoys.

Session 4:

Begin to graph activity.

Discuss time as a barrier to activity.

Learn two different ways to find the time to be active.

Discuss lifestyle activity.

Discuss ways to prevent injury and receive handouts on how to do some simple stretches and when to stop exercising.

Develop an activity plan for the coming week (for most participants, this will be a weekly total of 90 minutes).

Session 5:

Discuss ways to add interest and variety to the participant's activity plans.

Learn the definition and benefits of "aerobic fitness."

Session 6:

Check Track Logs and discuss the use of variety within the last week.

Group feedback on different activities in which they participated.

Learn the F.I.T.T. Principles (frequency, intensity, time, and type of activity) as related to heart (aerobic) fitness.

Session 7:

Check Tracking Books

Planning for Activity on Vacations and Holidays

Group Brainstorm: Activity appropriate for business travel, pleasure travel, and holidays.

Session 8:

Check tracking progress and activity variety

Where are the extra steps in my life?

Handouts from 10,000 step program

Session 9:

Review the participant's progress since Session 1, and if not at goal, develop a plan to improve progress.

Discuss the importance of motivation and ways to stay motivated.

Discuss & Sign Up for Post-Testing

Cardio Group Sessions

Session 1a/b/c:

Meet the research and programming team.

Review the Standard Healthy Lifestyle Guidelines.

Be given the Cardio notebook.

Discuss the participant's initial reaction to being assigned to the Cardio group.

Receive an overview of the Cardio Program.

Learn the Cardio group goals and why they are important.

 Have an explanation of how to fill out the forms.

 Have someone explain how to use pedometers (if not explained already)

 Have someone explain how to use the heart rate monitors.

Fitness Center Orientation and Machine Orientation

Discuss key aspects of the director-participant relationship and sign a related agreement.

Session 2:

Discuss the importance of injury prevention.

Discuss the importance of preparing for exercise (stretching, warm-up and cool-down)

Discuss when it is appropriate to STOP exercising.

Control Group Sessions:

Session 1:

Meet the research and programming team.

Review the Standard Healthy Lifestyle Guidelines.

Be given the Control Forms.

Discuss the participant's initial reaction to being assigned to the Control group.

Receive an overview of the Control Program.

Have an explanation of how to fill out the forms.

Have someone explain how to use pedometers (if not explained already)

Discuss key aspects of the director-participant relationship and sign a related agreement.

Session 2:

Review the Standard Healthy Lifestyle Guidelines.

Check weekly forms.

Session 3:

Discuss why activity is important.

Discuss current level of physical activity.

Be encouraged to participate in the Control sessions.

Identify other activities equivalent to brisk walking that the participant enjoys.

Check weekly forms.

Session 4:

Learn the definition and benefits of "aerobic fitness."

Learn the F.I.T.T. Principles (frequency, intensity, time, and type of activity) as related to heart (aerobic) fitness.

Check weekly forms.

Session 5:

Discuss the importance of injury prevention.

Discuss the importance of preparing for exercise (stretching, warm-up and cool-down)

Discuss when it is appropriate to STOP exercising.

Session 6:

Discuss time as a barrier to activity.

Learn two different ways to find the time to be active.

Discuss lifestyle activity.

Discuss ways to prevent injury and receive handouts on how to do some simple stretches and when to stop exercising.

Develop an activity plan for the coming week (for most participants, this will be a weekly total of 90 minutes).

Discuss & Sign Up for Post-Testing