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The Relationship of a long-term exercise program and selected coronary risk factors

Maryse Wells

University of Nevada Las Vegas

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THE RELATIONSHIP OF A LONG-TERM EXERCISE PROGRAM
AND SELECTED CORONARY RISK FACTORS

by

Maryse Wells

Bachelor of Science
Colorado State University
2002

A thesis submitted in partial fulfillment
of the requirements for the

**Master of Science in Exercise Physiology
Department of Kinesiology and Nutrition Sciences
School of Allied Health Sciences
Division of Health Sciences**

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THE GRADUATE COLLEGE

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Lawrence Golding, Committee Chair

John C. Young, Committee Member

Richard Tandy, Committee Member

J. Wesley McWhorter, Graduate Faculty Representative

Ronald Smith, Ph. D., Vice President for Research and Graduate Studies
and Dean of the Graduate College

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ABSTRACT

The Relationship of a Long-Term Exercise Program and Selected Coronary Risk Factors

By

Maryse Wells

Dr. Lawrence A. Golding, Examination Committee Chair
Distinguished Professor of Exercise Physiology
University of Nevada, Las Vegas

The purpose of this study was to investigate body composition and blood pressure as potential risk factors for coronary artery disease after 3 years of participation in a structured exercise program. The sample group was comprised of 21 male participants (66%) and 11 (34%) female participants with a mean age of 55 years who attended the exercise program and completed health and fitness testing annually. Participants did cardiovascular, strength, and flexibility training 5 days a week with an average attendance of 70%. After 3 years, BMI, waist circumference, and percent body fat increased significantly ($p < .01$); blood pressure levels did not change significantly ($p > .05$). Low control of diet may have contributed to worsening body composition measurements.

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CHAPTER 1

INTRODUCTION

In 2006, American Heart Association (AHA) data indicated that 34% of U.S. deaths were due to cardiovascular disease (CVD) (Lloyd-Jones et al., 2009). Of those, 52% were caused by coronary artery disease (CAD) (Lloyd-Jones et al., 2009). In 2001, among developed nations, the leading cause of death was CAD; in the United States this represented 460,000 deaths (Venet, 2005). In 2005, this figure totaled 445,687 deaths, accounting for one out of every five deaths in the United States (Lloyd-Jones et al., 2009). This represents an undesirable coronary event occurring every 25 seconds and a death from that event occurring every minute (Lloyd-Jones et al., 2009). These coronary events or CAD deaths are usually precipitated by the presence of risk factors in those afflicted by CVD.

The AHA position statement on risk factors and CAD identifies nine major risk factors for the development of CAD (American Heart Association [AHA], 2009). These risk factors include: increasing age, gender, heredity, cigarette smoking, elevated cholesterol levels, high blood pressure, physical inactivity, obesity and overweight, and diabetes mellitus (AHA, 2009).

Not only is physical inactivity one of the risk factors, but it can also negatively affect several other CAD risk factors including high cholesterol levels, high blood pressure, and obesity and overweightness (McArdle, Katch, & Katch, 2007). Several studies have examined the effect of a structured exercise program on CAD risk factors over a period of several weeks to a few years and documented that physical activity can positively

affect overall risk for CAD. The study presented in this paper investigated specific risk factors for CAD after participation in an exercise program for 3 years.

It was hypothesized that levels of selected potential risk factors for CAD would significantly improve after 3 years of participation in a structured exercise program. It was assumed that physical fitness levels would increase, and overall risk for CAD based on selected factors would improve.

The study addressed this hypothesis by assessing changes in blood pressure and body composition that occurred over the 3 years. Changes in physical fitness including flexibility, strength and muscular endurance, and aerobic capacity were also assessed. In addition, attendance was taken to assess actual participation in the daily program.

CHAPTER 2

REVIEW OF LITERATURE

Coronary Artery Disease

The AHA found that in the United States 32% of CVD deaths in 2005 occurred before the age of 75 years (Lloyd-Jones et al., 2009). This statistic indicates that nearly 1 out of 3 people who die from CVD in the United States dies before reaching life expectancy. Although CVD death rates have decreased by 26% between 1995 and 2005, it is a major health concern that too many are still dying from CVD before reaching life expectancy (Lloyd-Jones et al., 2009). Of the CVDs, the incidence of CAD is of particular interest.

CAD is a specific type of CVD characterized by “narrowing of the coronary arteries, usually as a result of atherosclerosis” (Venes, 2005, p. 486). The narrowing of the lumina in the coronary arteries occurs due to the build-up of plaques in the arteries, referred to as the process of atherosclerosis; these lesions occur as a consequence of the repair process of endothelial damage to the arteries (Brubaker, Kaminsky, & Whaley, 2002). As endothelial damage occurs, the resulting lesions build, causing the arteries to narrow, and ending in impairment of blood flow to the heart (Brubaker et al., 2002). If blood flow, and therefore the oxygen supply, to the heart is reduced, permanent damage to the heart muscle occurs (Venes, 2005). Myocardial ischemia describes this condition of reduced blood flow to the heart; if this ischemia is not quickly resolved, myocardial cells die and are replaced with scar tissue (myocardial infarction) (Brubaker et al., 2002). In the United States myocardial infarctions or heart attacks occur in thousands of individuals each year, often ending in sudden death (Venes, 2005).

The Framingham Heart Study

Given the common occurrence of CAD and the corresponding role that it plays in deaths, finding appropriate prevention strategies and treatments is of great importance. Initially many epidemiological studies on CVDs involved infectious and nutritional diseases, while CVD related to hypertension remained uncharted territory (Dawber, Meadors, & Moore, 1951). In 1947 the U.S. Public Health Service planned several epidemiological research programs to study CVDs (Dawber et al., 1951). In 1949 the Framingham Heart Study (FHS) was started, which catalogued the coronary risk factors in the city of Framingham, Massachusetts (Kannel, Brand, Skinner, Dawber, & McNamara, 1967).

Aided by local and state health organizations, the U.S. Public Health Service created a program with a large population of average individuals who were available for regular testing and observation (Dawber et al., 1951). Framingham, Massachusetts was chosen as the research city (Dawber et al., 1951). At the program's inception, there were 5,209 male and female participants between the ages of 30–62 years (Kannel, Dawber, & McGee, 1980).

Participants were examined and tested biennially. A team of physicians obtained information on participants through an extensive medical history and physical examination, specifically designed to diagnose CVD (Dawber et al., 1951). Data were collected to create a complete clinical profile of the participant; data were collected on everything from cholesterol level and eye color to degree of skin freckling and electrocardiograph readings to personal habits and X-ray examinations (Dawber et al., 1951).

Researchers also recorded the development of disease in participants as well as the cause of death in the event of participant mortality (Dawber et al., 1951). A panel of scientists determined specific criteria which resulted in CAD including: (a) angina pectoris, (b) myocardial infarction, (c) ischemic chest pain, or (d) death (Stokes III., Garrison, & Kannel, 1985). When a participant suffered any of the listed criteria, CAD was recorded as developing, allowing researchers to track the development of CAD within the population.

Risk Factors for Coronary Artery Disease

An important contribution of the FHS was the concept of *risk factors*. The use of the term risk factor originated with the FHS in the middle of the twentieth century (Brubaker et al., 2002). The term describes the concept that when disease free individuals are observed over an extended period of time, those who develop a disease can be assessed for degenerative factors that are associated with the development of the disease (Dawber et al., 1951). The AHA lists nine major risk factors for the development of CAD (AHA, 2009). These nine risk factors are: increasing age, gender, heredity, cigarette smoking, hypercholesteremia, hypertension, physical inactivity, obesity and overweightness, and diabetes mellitus (AHA, 2009).

There are also additional factors that may play a part in the development of CAD. These surfacing risk factors may contribute to CAD, however less evidentiary support exists for their actual role in CAD risk at this time (Brubaker et al., 2002). The AHA lists psychosocial factors, such as stress or socioeconomic status, and alcohol consumption as possible contributors to heart disease (AHA, 2009). Brubaker and associates (2002)

additionally list elevated lipoprotein(a) levels, hemostatic factors, left ventricular hypertrophy, high homocysteine levels, increased blood levels of c-reactive protein, decreased blood levels of antioxidants, and certain viral and bacterial infections as potential risk factors.

The nine major CAD risk factors are often categorized based on non-modifiable and modifiable risk factors (Brubaker et al., 2002). Increasing age, male gender, and heredity are non-modifiable risk factors (AHA, 2009). The defining characteristic of non-modifiable risk factors is that drug treatment or lifestyle adjustments cannot change the increased risk of CAD for these factors; knowing these risk factors is simply useful for assessing overall risk and determining course of treatment for patients (Brubaker et al., 2002). The following section discusses these risk factors.

Non-Modifiable CAD Risk Factors

Increasing Age

Since all people age, advancing age is classified as a non-modifiable risk factor. Brubaker and colleagues (2002) explain that there is no specific mechanism by which increasing age contributes to CAD; it is a matter of exposure over time. With aging, arteries endure atherosclerosis as well as other types of arteriosclerosis (Izzo, Levy, & Black, 2000). The older that one becomes, the more extended time that the arteries have been subjected to the atherosclerotic plaques that cause CAD (Brubaker et al., 2002). In addition, the artery walls may harden and lose elasticity as people age (Venes, 2005). Advancing age simply leaves the heart with components that may function at a lower level than in youth.

Gender

Another non-modifiable risk factor is being born a male. It has been shown that men have an increased risk for heart disease when compared to women, and, additionally, men suffer heart attacks at younger ages than women (AHA, 2009). This is supported by the fact that CAD occurs 4 times as often in men than women at ages of 45–49 years (Brubaker et al., 2002). Other evidence supporting gender differences is that the median age of first heart attack was found to be 9 years earlier in men than women (Yusuf et al., 2004). Using FHS data, researchers evaluated the lifetime risk of developing CAD when considered from age 40 and found that while men had a 48% lifetime risk of CAD, the lifetime risk for women came in considerably lower at 32% (Lloyd-Jones, Larson, Beiser, and Levy, 1999). As people age, lifetime risk falls, indicating less time spent at risk in older people due to shorter remaining life expectancy, however the gap between lifetime risk for men and women remains sizeable (Lloyd-Jones et al., 1999).

Heredity

Family history is also a non-modifiable risk factor. To have parents who suffer from early CAD increases the risk of developing CAD (AHA, 2009). Several studies have adjusted for other risk factors and found heredity to be a “strong independent risk factor for CAD” (Brubaker et al., 2002, p. 6). In 1977, 10,269 Harvard College alumni were questioned regarding lifestyle habits and characteristics; their answers were then considered in relation to mortality (Paffenbarger, Jr. et al., 1993). One area of evaluation was early parental death (Paffenbarger, Jr. et al., 1993). Through questioning participants regarding the death of none, one, or both parents before age 65, researchers saw a 64%

increase in risk of CAD was associated with early parental death (Paffenbarger, Jr. et al., 1993).

Race is also a form of genetic risk. The AHA identifies African Americans, Mexican Americans, American Indians, native Hawaiians, and some Asian Americans at higher risk for CAD than Caucasians (AHA, 2009). In addition to race being independently associated with risk levels, often when race is considered, it is also found that certain ethnic groups are more predisposed to other CAD risk factors, such as a prevalence of high blood pressure in African Americans (Lloyd-Jones et al., 2009). However, when CAD risk was studied in Evans County, GA, it was found that although blacks and whites responded to CVD risk factors similarly, there was a lower occurrence of heart disease in the black population (Kleinbaum, Kupper, Cassel, & Tyroler, 1971). These data illuminate the fact that not only does heredity and race play a part in risk of CAD alone, but they also affect response to other risk factors.

Although the non-modifiable nature of increasing age, male gender, and genetics makes their consideration appear of little use, when paired with modifiable risk factors an accurate assessment of overall risk can be found. This assessment can shape lifestyle choices and treatment plans to better prevent CAD.

Modifiable CAD Risk Factors

Modifiable risk factors can be brought into safe levels through lifestyle change or physician managed medication. The modifiable risk factors as described by the AHA are: tobacco smoking, high blood cholesterol, high blood pressure, physical inactivity, obesity and overweight, and diabetes mellitus (AHA, 2009). The INTERHEART study focused on nine different modifiable heart disease risk factors in 52 different countries,

adding alcohol consumption, diet, and psychosocial factors to the six listed by the AHA, and found that 90% of population attributable risk in men and 94% of population attributable risk in women could be explained by those nine modifiable factors (Yusuf et al., 2004). This creates hope in the area of CAD, as although there are risk factors that cannot be improved, several risk factors exist for which people can make changes and increase their odds of CAD prevention. And, even better, these modifiable risk factors seem to account for a large proportion of CAD risk.

Tobacco Smoking

Several studies confirmed that cigarette smoking is a significant risk factor for CAD. In 1976 the Nurses' Health Study was launched to gain information on lifestyle and disease in a group of 121,700 female nurses between the ages of 30–55 years (Stampfer, Hu, Manson, Rimm, & Willett, 2000). The study identified cigarette smoking as the single most important risk factor for CAD; smoking more than 14 cigarettes daily was associated with an over 5 times higher risk of CAD than not smoking (Stampfer et al., 2000). Other studies also reported dangerous risk levels for smokers. Data from the Harvard College Alumni study revealed that the risk of death for non-smokers was almost 50% lower than heavy smokers (Paffenbarger & Lee, 1998). Overall, the AHA position statement listed the risk of CAD as 2–4 times higher for those who smoke when compared to nonsmokers (AHA, 2009).

Dyslipidemia

For several years epidemiological studies have indicated that an elevated serum cholesterol level is a significant risk factor for CAD; other forms of dyslipidemia such as

depressed HDL levels or elevated triglyceride levels have also shown connections with increased risk (Cleeman, 2001; Lloyd-Jones et al., 2003).

CAD risk based on total cholesterol levels was determined by The National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III); NCEP ATP III determined that elevated total cholesterol was a coronary risk factor (Cleeman, 2001). They stated that total cholesterol greater than $240 \text{ mg}\cdot\text{dL}^{-1}$ is considered high and total cholesterol less than $200 \text{ mg}\cdot\text{dL}^{-1}$ is optimal (Cleeman, 2001). Using FHS data, researchers examined 3,269 men for lifetime risk of CAD to age 80 by total cholesterol level and found a 1.5–2 times higher risk level in men at the highest level of total cholesterol versus at the lowest level (Lloyd-Jones et al., 2003). Correspondingly, when 4,019 women were evaluated, twice the risk was also seen in the highest cholesterol brackets when compared to the lowest (Lloyd-Jones et al., 2003). In addition, several other studies also determined that total cholesterol is an effective tool for evaluating CAD risk; however, it was suggested that using high density lipoprotein (HDL) levels in conjunction with total cholesterol (total cholesterol divided by HDL) could be even more revealing (Kannel, Castelli, & Gordon, 1979).

Kannel, Castelli, and Gordon (1979) suggested that HDL carries low density lipoprotein (LDL) away from atherosclerotic lesions, helping to prevent the build-up of plaques that narrow the arteries and increase CAD. Given the possible preventative effect of HDL on atherosclerosis, it appears that higher levels of HDL would be advantageous. The NCEP ATP III stated that HDL levels below $40 \text{ mg}\cdot\text{dL}^{-1}$ are low and undesirable whereas levels above $60 \text{ mg}\cdot\text{dL}^{-1}$ are high and desirable (Cleeman, 2001). Gordon and associates (1989) analyzed several prospective studies on HDL and CVD and

found lower rates of CAD among those subjects with the highest HDL levels. The protective effect of high HDL levels was seen in several other analyses performed from FHS data (Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977; Wilson & Meigs, 2008).

While high levels of HDL were accepted as desirable, high levels of LDL were accepted as undesirable. As a result the popular literature refers to LDL as “bad” cholesterol and HDL as “good” cholesterol. The NCEP ATP III categorized levels of LDL of less than $100 \text{ mg}\cdot\text{dL}^{-1}$ as optimal, with levels of $160 \text{ mg}\cdot\text{dL}^{-1}$ or above as high; levels of $190 \text{ mg}\cdot\text{dL}^{-1}$ or higher were seen as very high (Cleeman, 2001). These classifications were based upon the NCEP report of a positive relationship between CAD and LDL levels over a wide range of LDL levels (Cleeman, 2001). Based on their study of FHS data Kannel, Castelli, & Gordon (1979) stated that LDL was “superior to the total cholesterol in the measure of atherogenic cholesterol.” Researchers did not suggest eliminating total cholesterol as a coronary risk factor, but advocated that fractional measures of cholesterol may provide more clarity to the relative risk (Kannel, Castelli, & Gordon, 1979).

Triglyceride levels were also often included in the etiology of CAD risk and lipid levels. The NCEP ATP III recommended levels for triglycerides based on available evidentiary support; triglyceride levels of less than $150 \text{ mg}\cdot\text{dL}^{-1}$ were listed as normal and desirable while triglyceride levels of $200 \text{ mg}\cdot\text{dL}^{-1}$ or higher were classified as high and very undesirable (Cleeman, 2001). Nevertheless, research from epidemiological studies on triglyceride levels and disease risk has been varied. However, the NCEP advised that elevated triglyceride levels are an independent risk factor for CAD, and some research

from the FHS supported the concept that elevated triglycerides are a factor in the development of CAD (Cleeman, 2001; Wilson & Meigs, 2008).

Two studies indicated that triglycerides were of minor importance in the development of CAD (Gordon et al., 1977; Kannel, Castelli, & Gordon, 1979). However, another study performed on FHS data determined that those with elevated triglyceride levels did have a higher risk of CAD, but that other lipids were better risk factors (Kannel, Gordon, & Castelli, 1979). Other researchers added to that evidence finding that when adjusted for HDL levels and diabetes, triglyceride levels were no longer a significant risk factor for CAD (Gordon et al., 1977).

Hypertension

There is considerable research supporting high blood pressure as a significant risk factor for CAD. Simply stated, high blood pressure makes the heart work harder, which in turn causes the heart muscle to thicken and develop stiffness (AHA, 2009). Currently most blood pressures are either evaluated from both systolic and diastolic pressure or from systolic blood pressure (SBP) alone.

The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure reported that approximately 50,000,000 people in the United States and 1,000,000,000 people globally suffer from hypertension (Chobanian et al., 2003). Normal blood pressure was classified as less than 120/80 mmHg, while the hypertensive categories began at a level of 140/90 mmHg (Chobanian et al., 2003).

Research supports the fact that individuals with hypertension are at an increased risk for CAD. Researchers have found hypertension to be a strong predictor of CAD (Kaprio, Kujala, Koskenvuo, and Sarna, 2000). Evidence from Paffenbarger and associates (1993)

also found that hypertension was associated with twice the risk of death from CAD. The association of high blood pressure with risk for CAD was compounded by the fact that atherosclerotic lesions are rarely found in the parts of the circulatory system with lower pressure (Kannel, 1995).

Hypertension as a risk factor is independent, continuous, and steady; as the severity or level of hypertension increases, so does the risk of CAD (Chobanian et al., 2003; Kannel, 1995). Data from the FHS showed that in persons aged 40–69 years, for each blood pressure increase of 20/10 mmHg, heart disease mortality doubled. (Kannel & Wolf, 2008). While overall hypertension is a crucial factor in prediction of heart disease risk, SBP readings have become increasingly important.

Isolated systolic hypertension is thought to be related to arteriosclerosis, or the hardening of the arteries, rather than atherosclerosis (Kannel et al., 1980). Izzo and associates (2000) affirmed that the pathogenesis of arteriosclerosis is hastened by systolic hypertension. Between 1960 and 1970 a group of 878 men from 40–59 years were examined for an association between blood pressure and incidence of heart attack (De Vries, Feskens, de Lezenne Coulander, & Kromhout, 1993). It was found that levels of SBP were related to incidence of myocardial infarction (De Vries et al., 2003). When participant SBP data were divided into quartiles, it was found that 25% of the participants in the highest quartile died before the second assessment, while there was only a 10% mortality rate in the lowest quartile (De Vries et al., 2003). Kannel and associates (1980) also reported a great increase in risk level for CVD with isolated systolic hypertension. Similar to the research for overall blood pressure, it was found that there is no need for a

particular SBP because there is a steady linear relationship between SBP and CAD (Izzo et al., 2000).

However, several authors recommended the use of SBP rather than diastolic blood pressure (DBP) or overall blood pressure in risk evaluation. Authors of a meta-analysis using data from 61 prospective studies recommended the use of SBP instead of DBP as a risk factor when just one measurement is used (Lewington et al., 2002). Izzo and colleagues (2000) supported this idea, reporting that in older adults, the use of DBP could even confound results, as there is an inverse relationship between DBP and cardiovascular risk in older adults with isolated systolic hypertension. Given this concept, the authors refuted the recommendation that age-adjusted blood pressure goals are appropriate; they reported that high SBP is dangerous at all ages (Izzo et al., 2000).

Physical Inactivity

Physical inactivity has also been cited as an independent risk factor for CAD. An analysis of 43 studies revealed that an inverse relationship exists between physical activity level and risk of CAD (Powell, Thompson, Caspersen, & Kendrick, 1987).

Two classic studies explored the incidence of CAD as related to physically active jobs versus sedentary jobs. Morris, Heady, Raffle, Roberts, and Parks (1953) found that bus conductors in the London transport system had a lower incidence of CAD than bus drivers. It was hypothesized that the more physically active nature of the bus conductors' jobs, especially since buses were double deckers, contributed to this lower incidence of CAD (Morris et al., 1953). Paffenbarger and colleagues found similar results when comparing physically active longshoremen to physically inactive longshoremen in San Francisco (Paffenbarger, Gima, Laughlin, & Black, 1971). A discernibly lower rate of

death from CAD was seen in the physically active longshoremen than in the physically inactive longshoremen (Paffenbarger et al., 1971).

More contemporary studies also confirmed the connection of physical inactivity and heart disease. Data from the Harvard College Alumni study expressed a 41% lower risk of death from CAD in men who engaged in moderately vigorous activity when compared to sedentary men (Paffenbarger et al., 1993). Further analysis of the data demonstrated that vigorous activity, in excess of $4200 \text{ kJ}\cdot\text{week}^{-1}$, was related to a reduction in risk of CAD, while low to moderate levels of physical activity did not have as clear of an association (Sesso, Paffenbarger, & Lee, 2000).

Obesity and Overweightness

Obesity and overweightness are contributors to development of CAD. It has taken over 20 years for obesity to become apparent in the FHS as an independent risk factor for CAD (Stokes III et al., 1985). The data showed that obesity and overweightness are both associated with cardiovascular diseases. As the degree of overweightness increased, incidence of CAD disease increased as well (Gordon & Kannel, 1976). According to interpretation of the data, there would be 25% less CAD if all people were at their optimal weight (Gordon & Kannel, 1976). Several different measures of obesity have been shown to have association with risk of CAD; studies have used mean reference weight, skinfolds, overweight for height, waist circumference, body mass index (BMI), waist-to-hip girth measurement, and others to gauge the relationship between obesity and disease. These are briefly discussed in the next section.

BMI.

One method of quantifying obesity that has been used consistently, especially in epidemiological studies, is BMI. The AHA Prevention Conference VII states that BMI, although a crude method of estimating body fat, is also convenient and useful for assessing global health trends (Eckel et al., 2004). BMI is a measure of weight in kilograms divided by height in meters squared; classifications of BMI are: normal (19.5–24.9), overweight (25–29.9), and obese (30 and over) (Eckel et al., 2004). Since almost all studies record the height and weight of subjects, the BMI can be calculated long after a study is completed. Using FHS data researchers found an association between BMI and the development of CVD (Kannel et al., 1991). Other notable studies also used BMI to assess the relationship of obesity to CAD risk including the Nurses' Health Study, the Harvard College Alumni Study, and a study of twin-pairs (Kaprio, et al., 2000; Sesso et al., 2000; Stampfer et al., 2000).

Skinfolds.

In several studies skinfold measurements have been used as a more exact method of quantifying overall body composition. As skinfold measurements are often taken at several different sites on the body, they can give a more valid indication of body fat distribution. Skinfolds have been used as an indication of body composition in several community-based studies including both the Tecumseh community study and FHS (Gudbrandsson et al., 1994; Higgins, Kannel, Garrison, Pinsky, & Stokes III, 1988). Skinfold measurements can be used to predict body density and then body fat through prediction equations; Lohman found a theoretical accuracy of prediction equations of 3.3% body fat (as cited by Heyward & Stolarczyk, 1996). Therefore skinfold

measurements can be used as an overall measure of body fatness or as an indication of body fat distribution.

Waist circumference.

Waist circumference has been a common and popular method of assessing obesity in research protocols. One review of research on CAD with regard to obesity and body fat distribution found that “obesity, particularly when associated with a preferential deposition of fat in the abdominal region, predisposes to the development of CAD,” suggesting that waist circumference would be an excellent measure of the relationship of obesity to CAD (Brochu, Poehlman, & Ades, 2000). Lakka, Lakka, Tuomilehto, and Salonen (2002) supported this evidence with their study, indicating that abdominal obesity specifically is an independent risk factor for CAD. Their data revealed that twice the risk for coronary events was seen in males with waist circumferences of 90 cm (35 in.) or greater when compared to men whose waists measured less than 83.5 cm (33 in.) (Lakka et al., 2002).

The FHS data were evaluated for the relationship of regional obesity to CVD, and it was found that the obesity measures were too highly related to single out a specific obesity measurement as the best measure of risk (Kannel et al., 1991). Bigaard and colleagues (2005) also recommended that adiposity measurements be used in conjunction with one another to determine overall risk. The data showed that obesity was associated with risk of CAD, but examining data on more than one obesity measure could be useful due to the interrelatedness of obesity measures (Bigaard et al., 2005).

Diabetes Mellitus

The last major risk factor as listed by the AHA is diabetes mellitus (AHA, 2009). The AHA classifies the risk of developing CVD when diabetes is present as serious; substantiated by the fact that a disease of the blood vessels or heart is responsible for the death of around 75% of diabetics (AHA, 2009). When FHS data were examined for incidence of type II diabetes mellitus over a 20-year period, a nearly 2.5% increase was seen from the 1970s to the 1990s; which is severe in light of the risk associated with the disease (Fox et al., 2006). Gordon and associates (1977) looked at women in the FHS and found supporting evidence to the AHA risk assessment, showing diabetes to be a risk factor for CAD in women 30 years and older. Analogous to the women's risk, when male twin-pairs from the Finnish twin cohort discordant for CAD were evaluated for an association between diabetes and risk for CAD, a clear association between the two was found (Kaprio et al., 2000).

CAD Risk Factor Interaction

The noticeable feature about coronary risk factors is that not only do they increase the risk for CAD, but often having one risk factor increases the risk for another risk factor. Kannel and Wolf (2008) recently reaffirmed that no solitary crucial risk factor is responsible for CVD; it is the product of many risk factors. Wilson and Meigs (2008) investigated the concept of cardiometabolic risk using FHS data and found interesting results on risk factor clustering. They examined HDL, BMI, SBP, triglycerides, glucose, and total cholesterol and found that "clusters of three or more risk factors occurred at twice the rate predicted by chance" (Wilson & Meigs, 2008). This would suggest that there is some relationship between the risk factors themselves.

Risk factor interaction was often seen when considering obesity with other risk factors. FHS researchers examined data from 5,127 male and female participants aged 30–62 years for a relationship between blood pressure and adiposity (Kannel et al., 1967). A marked relationship was found between adiposity, as defined by mean reference weight, weight change over time, and skinfold measurements, and blood pressure (Kannel et al., 1967). As the degree of obesity, or one risk factor, increased, the risk for hypertension, an independent risk factor, increased as well, showing that the two risk factors for CAD were interrelated (Kannel et al., 1967). The evidence for the association between hypertension and obesity was duplicated by researchers of the Framingham Offspring Study who found that those with higher body fat levels have an increased risk for hypertension (Garrison, Kannel, Stokes III, & Castelli, 1987).

Other researchers have found connections between obesity and unhealthy lipid levels as well as obesity and diabetes. Using the FHS data, a link between obesity and lipid levels was found; people with higher body fat had higher levels of all lipid values, except for HDL (Kannel, Gordon, & Castelli, 1979). HDL had an inverse relationship with obesity (Kannel, Gordon, & Castelli, 1979). When trends in diabetes were examined with the FHS data, it was discovered that the majority of the increase in diabetes incidence between the 1970s and 1990s was seen in the obese group, rather than the overweight or normal group (Fox et al., 2006). These types of results were seen in studies of many risk factors versus obesity as well.

Stokes III, Garrison, and Kannel (1985) found correlations in the FHS data between several measures of weight, size, and body fat, including BMI, mean reference weight, skinfold measurements, and two body circumference measurements, and SBP, serum

total cholesterol, and blood sugar. Upon further investigation of FHS data, these results were seen again; weight gains and losses were significantly associated with corresponding increases and decreases in blood sugar, cholesterol, and blood pressure over two year intervals in 32 years of follow-up data (Higgins et al., 1988).

Physical Activity and Coronary Artery Disease

Physical activity is another independent CAD risk factor that often has an impact on other CAD risk factors. There have been several long-term studies as well as shorter term training studies investigating the effect of physical activity on risk of CAD and on risk factors for CAD.

Classic Research in Physical Activity and Coronary Artery Disease

It is suggested that current research on exercise and CAD began with the research on the London bus system by Jeremy N. Morris in 1949, close to the time that the FHS was launched (Paffenbarger, Blair, & Lee, 2001). Morris and colleagues (1953) began to question the relationship of physical activity and heart disease by collecting information on different occupations that could be classified as physically active or inactive and the incidence of heart disease within those occupations. Since Morris first began his investigative work on this topic, his research has made generous contributions to the field of exercise as a protective factor against CAD (Paffenbarger et al., 2001). Morris and associates (1953) collected data on 31,000 employees of the London Transport study, including job title, recorded illnesses, deaths, and retirement, and as mentioned earlier, discovered that bus conductors (a physically active job) had a lower rate of CAD than bus drivers (a physically inactive job). During the same timeframe, Morris and associates

(1953) reinforced their London Transport findings with a comparison of the incidence of CAD in mailmen, who walked delivering the mail, to post office workers in sedentary jobs; they found that the physically active postmen suffered a much lower rate of heart disease than their sedentary counterparts.

In 1971 Paffenbarger and associates (1971) added evidence to the findings of Morris and colleagues with their study on the association between CAD deaths and the physical activity of San Francisco longshoremen (also mentioned earlier). They studied 3,263 longshoremen for risk of CAD and stroke (Paffenbarger et al., 1971). After determining that cargohandlers expended about 925 calories more during a work day than their more sedentary coworkers, the death rates for the two occupations were evaluated against each other. It was found that the CAD death rate of physically active cargohandlers was 20% lower than that of the more sedentary longshoremen (Paffenbarger et al., 1971).

Paffenbarger and colleagues' (1993) research continued with the Harvard College Alumni study. 52,000 men who attended Harvard College or the University of Pennsylvania in 1916–1950 were used for evaluation of morbidity and mortality (Paffenbarger & Lee, 1998). Participants from this pool had completed survey questionnaires regarding lifestyle habits and health information from 1962 to as recently as 1993 (Paffenbarger & Lee, 1998). Again, this research showed a distinct connection between physical activity and lowered CAD risk. The research of both Morris and Paffenbarger has proved the fact that exercise improves health and reduces the risk of CVD (Andrade & Ignazewski, 2007).

Other Research in Physical Activity and Coronary Artery Disease

Epidemiological and Questionnaire-based Research

Since, and concurrent to, the studies of Morris and Paffenbarger many other epidemiological studies have realized an inverse relationship between physical activity and CAD. Physical inactivity as an independent risk factor for CAD has been examined in the FHS, the INTERHEART Study, the Women's Health Study, and the Finnish Twin Cohort Study (Kannel, Wilson, & Blair, 1985; Kaprio et al., 2000; Weinstein et al., 2008; Yusuf et al., 2004). Physical fitness was not objectively measured in the FHS, so researchers used indications of fitness as a measurement tool (Kannel et al., 1985). Vital capacity, heart rate, and obesity were used as gauges of physical fitness in the FHS (Kannel et al., 1985). The other studies mentioned used questionnaires to assess physical activity or fitness (Kaprio et al., 2000; Weinstein et al., 2008; Yusuf et al., 2004).

In the FHS, selected participant physical fitness measures were evaluated against the percent of participants who developed CAD or experienced cardiovascular mortality (Kannel et al., 1985). Researchers found a 5 times higher risk of CAD in those people who were short of breath, had a high resting heart rate, and had high body fat levels (Kannel et al., 1985). As a low resting heart rate is often associated with higher fitness levels, the researchers reasoned that physical fitness may yield a protective effect from CAD (Kannel et al., 1985).

The INTERHEART study yielded similar results with the evaluation of 15,152 cases and 14,820 controls; when cases of myocardial infarction were considered against age and gender matched controls, exercise was found to be protective against CAD (Yusuf et al., 2004). The population attributable risk associated with physical activity, when

adjusted for age, sex, and smoking, was calculated to be 25.5%, meaning that if all people in the population were physically active, there would be 25.5% less incidence of CAD (Yusuf et al., 2004).

These findings were confirmed by the Women's Health Study conclusion that increases in total physical activity significantly reduced risk of CAD and the results of the Finnish Twin Cohort study which revealed that conditioning exercisers had a 48% lower relative risk of CAD than sedentary individuals, $p = .0001$, and occasional exercisers had a 23% lower relative risk of CAD than sedentary individuals, $p = .0001$ (Kaprio et al., 2000; Weinstein et al., 2008). Contrary to the early studies by Morris and associates (1953) and Paffenbarger and associates (1971) on occupational physical activity and CAD, Kaprio and associates (2000) did not find an association between increased caloric expenditure at work and lowered risk of CAD. The researchers hypothesized that the differing nature of occupational physical activity and recreational physical activity may have caused the difference in CAD manifestation (Kaprio et al., 2000). Job-related physical activity was seen as a requirement that was often monotonous and long-term, while recreational activity was considered more dynamic and transitory (Kaprio et al., 2000).

Structured Exercise Program Research

In addition to research protocols designed using self-reported levels of physical activity or other indirect methods of physical activity measurement, there have also been several studies on the effect of structured exercise programs on risk of CAD. One such lifestyle modification program looked at the effect of exercise training, with nutritional counseling, stress management, and education on cardiac event rate and CAD risk factors

over 10 years (Kappagoda et al., 2006). A total of 137 patients who had been diagnosed with CAD or suffered a heart attack were initially enrolled in the 2-year lifestyle modification program, however, only 77 patients completed the program, the others dropped out during the 2 years (Kappagodda et al., 2006). After 10 years, an analysis of cardiovascular event rate during that time period revealed that the patients who completed the program had an event rate of 1.5%, while the event rate in drop-outs was 18%, $p < .02$ (Kappagodda et al., 2006). This suggested that participation in a program for a time span as short as 2 years could have lasting effects on cardiac event rate well into the future, even after program participation has ended.

Physical Activity and Other Risk Factors for CAD

In addition to studies on the effect of physical activity on risk for CAD, research has also focused on the relationship between physical activity and specific CAD risk factors. Studies on the effect of physical activity on obesity and blood pressure are addressed below.

Physical Activity and Obesity

Research on the relationship between physical activity and obesity has been varied in both structure and results (Table 1). Researchers have often looked for a negative relationship between physical activity and obesity; it was generally hypothesized that as physical activity increased, obesity rates or levels would decrease. However, further examination did not always prove this hypothesis correct. The paragraphs below address both epidemiological-based research as well as training program-based research on the relationship of physical activity and obesity.

Epidemiological and Questionnaire-based Research

The relationship of obesity and physical activity was examined in the Fels Longitudinal Study, the Tecumseh Community Study, and the Finnish Twin Cohort Study (Gudbrandsson et al., 1994; Guo, Zeller, Chumlea, & Siervogel; 1999; Waller, Kaprio, & Kujala, 2008). From 1976–1996, 210 white men and women participated in the Fels Longitudinal Study, during which data were collected biennially; included in these data were measurements of BMI, body fat percentage, and results from a physical activity questionnaire (Guo et al., 1999). Body fat measurement was obtained through underwater weighing and physical activity levels were separated into three categories (low, medium, and high) based on participant answers to questions regarding physical activity levels (Guo et al., 1999). Researchers found that while lower BMI was associated with lower body fat percentage in males, BMI was not significantly associated with body fat percentage in females (Guo et al., 1999). In both men and women, physical inactivity was associated with high body fat percentages (Guo et al., 1999).

Obesity was measured in the Tecumseh Community Study through percentage overweight and sum of skinfolds; the results were parallel to those in the Fels Longitudinal study (Gudbrandsson et al., 1994). In Tecumseh, Michigan 857 men and women with an average age of 30 years were evaluated for antecedents of high blood pressure and CVD (Gudbrandsson et al., 1994). Based on a questionnaire, it was determined that 70% of the population was sedentary (Gudbrandsson et al., 1994). When obesity was considered in relation to physical activity, there was a significant difference

Table 1

Research on the Relationship of Physical Activity and Obesity/Overweight

Study	<i>n</i>	Sex	Age (<i>M</i>)	DV	Result	<i>p</i> value
Andersen et al., 1999	40	F	43	BF%	16 weeks aerobics training, 4.3% decrease	< .001
Guo et al., 1999	102	M	44	BMI	<i>M</i> = 0.83 less in high versus low activity	< .05
				BF%	<i>M</i> = 2.5% less in high versus low activity	< .05
	108	F	44	BMI	No difference between high versus low activity	-
				BF%	<i>M</i> = 8.9% less in high versus low activity	< .05
Gudbrandsson et al., 1994	857	M/F	30	OW%	<i>M</i> = 6.1% less in exercisers versus non-exercisers	.0003
				SSF	<i>M</i> = 12.2 mm less in exercisers versus non-exercisers	.00001
King et al., 1995	269	M/F	- ^a	BMI	2 years exercise training, no significant change	-
				WH	2 years exercise training, no significant change	-
				BF%	2 years exercise training, no significant change	-
Santa-Clara et al., 2003	36	M	56	BF%	1 years aerobics and resistance training or aerobics training, <i>M</i> significantly lower in exercise groups than control group	< .05
Takeda et al., 1996	11	F	55	BF%	36 mos exercise training, no significant change	-
				SSF	36 mos exercise training, no significant change	-
Waller et al., 2008	84	M/F	29	WC	<i>M</i> = 8.4 cm (3.3 in.) less in active versus inactive twins	< .001

Note. DV = dependent variable; BF% = body fat percentage; BMI = body mass index; OW% = overweight percentage; SSF = sum of skinfolds; WH = waist-to-height ratio; WC = waist circumference.

^a*M* was not reported; participants were between the ages of 50 – 65 years.

between physically active participants and their sedentary counterparts based on percentage overweight, $p = .0003$, and sum of skinfolds in mm, $p = .00001$; indicating that there was an inverse relationship between obesity and physical activity (Gudbrandsson et al., 1994).

This relationship between obesity and physical activity was again observed in 146 twin-pairs from the Finnish Twin Cohort who were different for both intensity of physical activity and time spent exercising (Waller et al., 2008). Obesity was measured by waist circumference (Waller et al., 2008). In the twin-pairs who were consistently different on measures of physical activity, an 8.4 cm (3.3 in.) smaller waist circumference was seen in the more active co-twin, $p < .001$ (Waller et al., 2008).

Structured Exercise Program Research

Various studies also focused on the evaluation of physical activity and obesity based on structured training programs rather than self-reported questionnaires or epidemiological data. Two training studies were conducted on participants suffering from heart disease (Santa-Clara, Fernhall, Baptista, Mendes, & Sardinha, 2003; Takeda, Tanaka, Unno, Hiyama, & Asano; 1996). Santa-Clara and associates studied 36 male patients between the ages of 45–68 years who had completed a phase II cardiac rehabilitation program after diagnosis of CAD. Patients were divided between three different treatment groups: (a) a weight training and aerobic training group, (b) an aerobic training group, and (c) a control group with no exercise (Santa-Clara et al., 2003). The exercise program ran for 1 year with both exercise groups participating in group exercise programs three times per week (Santa-Clara et al., 2003). Participant body composition was assessed using dual-energy radiographic absorptiometry (Santa-Clara et al., 2003).

Takeda and associates (1996) designed a similar protocol using 11 female CAD patients with a mean age of 55 years, however there was no control group and all 11 women completed the 36-month exercise training program. The exercise program was created with both a supervised exercise element and a home-based exercise element (Takeda et al., 1996). Participants spent 1–2 days per week in 1-hour supervised exercise sessions and zero to five times per week practicing home exercise sessions lasting 15–60 min; the exercise program was geared toward aerobics and movements of large muscle groups (Takeda et al., 1996). Body composition was assessed in this study with bioelectrical impedance and four-site skinfold measurements (Takeda et al., 1996).

Differing results were found between the two exercise program studies with CAD patients (Santa-Clara et al., 2003; Takeda et al., 1996). Santa-Clara and associates (2003) found that after 1 year of study, the aerobic and strength training group had a significant decrease in total body fat, $p < .0001$ and the aerobic training group remained at starting body fat levels. The control group who remained sedentary had a significant increase in total body fat, $p < .05$ (Santa-Clara et al., 2003). Alternately, Takeda and associates (1996) saw changes in body composition over 8–12 months, however the differences were not significant, and after 36 months, body fat percentages returned to their initial values. These results do not clearly explain the relationship between exercise and obesity.

Two other studies, one on 40 obese women with a mean age of 43 years, and another on 269 previously sedentary adults aged 50–65 years, provided further results on obesity versus physical activity (Andersen et al., 1999; King, Haskell, Young, Oka, & Stefanick, 1995). In the study from Andersen and colleagues, the women participated in either a

program focused on diet and structured group aerobic activity or diet and lifestyle activity changes. Structured group activity included three weekly aerobic stepping classes, while the lifestyle modification group was encouraged to increase physical activity levels to 30 minutes per day for most days of the week (Andersen et al., 1999). Participant body composition was assessed with dual x-ray absorptiometry at baseline and at the end of the 16-week program (Andersen et al., 1999).

The 2-year study by King and colleagues (1995) evaluated participant body composition using body fat calculated with underwater weighing, BMI, and waist-to-hip ratio. Participants in this study were separated into three treatment groups: (a) higher-intensity, group-based exercise training, (b) higher-intensity home-based training, and (c) lower-intensity home-based training (King et al., 1995). After 2 years, no significant differences were found in body fat percentage, waist-to-hip ratio, or BMI between the treatment groups or within the treatment groups from the inception of the program (King et al., 1995). Andersen and colleagues (1999) found no difference between the lifestyle group and the exercise group, however between baseline and 16-weeks a significant difference was found within both groups; body fat percentage was decreased by 6.2% in the lifestyle group and by 4.3% in the aerobic group, $p < .001$.

Based on these four studies, results concerning the effect of structured exercise programs on the CAD risk factor of obesity are conflicting, although often positive. These studies demonstrate that several different methods of evaluating obesity have shown a negative association between exercise and obesity.

Physical Activity and Blood Pressure

Much like research on physical activity and obesity, research examining physical activity and blood pressure has been varied also. As shown in Table 2, researchers have examined blood pressure using differing methods which have yielded varying results.

Epidemiological and Questionnaire-based Research

Epidemiological data on the effect of exercise on blood pressure or hypertension is varied. Researchers of the Harvard College Alumni study reported less occurrence of hypertension in physically active men versus sedentary men, $p < .001$ (Sesso et al., 2000). However, contrary to these results, the Tecumseh Community Study did not find a significant difference between systolic blood pressure in exercising participants versus sedentary participants, $p = .20$, or between diastolic blood pressure in exercising participants versus sedentary participants, $p = .15$ (Gudbrandsson et al., 1994).

Structured Exercise Program Research

Several studies in which one group of participants underwent a physical activity program of varying length found decreases in systolic and diastolic blood pressures at the end of the programs. Takeda and colleagues' (1996) female CAD patients began the program with a mean blood pressure in the pre-hypertensive (mean DBP: 88 mmHg) or hypertensive (mean SBP: 141 mmHg) range, but both blood pressures lowered significantly to the normotensive range by the fourth month of the program, $p < .05$, and remained there to the end of the program at 36 months.

Table 2

Research on the Relationship of Physical Activity and Blood Pressure

Study	<i>n</i>	Sex	Age (<i>M</i>)	DV	Result	<i>p</i> value
Cox et al., 2001	126	F	48	SBP	18 mos moderate intensity exercise, 2.8 mm Hg decrease	.049
					18 mos vigorous intensity exercise, no change	-
				DBP	18 mos moderate intensity exercise, 2.7 mm Hg decrease	.004
					18 mos vigorous intensity exercise, no change	-
Daubenmeir et al., 2007	869	M/ F	59	SBP	3 mos lifestyle changes, significant decrease	.001
				DBP	3 mos lifestyle program, significant decrease	.001
Gudbrandsson et al., 1994	857	M/ F	30	SBP	No difference between active and inactive	-
				DBP	No difference between active and inactive	-
Jennings et al., 1986	12	M/ F	22	SBP	Significantly lower in active regimen	.01
				DBP	Significantly lower in active regimen	.01
Kasch et al., 1995	24	M	46	SBP	28 years, significant increase in sedentary group with no change in exercise group	< .001
				DBP	28 years, significant increase in sedentary group with no change in exercise group	< .001
Sesso et al., 2000	12,516	M	58	HTN	Active males less likely than inactive males to suffer from hypertension	< .001
Tabara et al., 2007	40	M/ F	67	SBP	6 mos exercise training, decrease of 7 mm Hg	.0056
				DBP	6 mos exercise training, decrease of 5 mm Hg	.0008
Takeda et al., 1996	11	F	55	SBP	36 mos exercise training, significant decrease	< .05
				DBP	36 mos exercise training, significant decrease	< .05

Note. DV = dependent variable; SBP = systolic blood pressure; DBP = diastolic blood pressure.

The Multisite Cardiac Lifestyle Intervention Program was an investigation of risk factor management in 869 male and female participants (Daubenmier et al., 2007). Patients participated in a program based on diet, exercise, and stress management for 3 months; the exercise component involved 1 hour of weekly supervised exercise program participation (Daubenmier et al., 2007).

Similar to the study on 40 obese women by Andersen and colleagues (1999), the Sedentary Women Exercise Adherence Trial examined a participant pool of 126 sedentary women (Cox et al., 2001). The women were recruited to exercise three times per week for 30 minutes over 18 months and risk factors were periodically evaluated over that time period (Cox et al., 2001). Participants were randomly assigned to either a supervised exercise group or a home-based exercise group for the first 6 months of the study, and then both groups continued home-based exercise for the remaining 12 months of the study; the exercise consisted of walking, aerobics training, and circuit training sessions (Cox et al., 2001).

Both participants in the lifestyle intervention program and in the sedentary women's program saw similar significant reductions; researchers in the lifestyle intervention program found a reduction in mean SBP of about 12 mmHg, $p = .001$ and a reduction in mean DBP of about 7 mmHg, $p = .001$ (Cox et al., 2001; Daubenmier et al., 2007). Participants in the sedentary women's program who exercised at moderate intensity significantly decreased their SBP by 2.81 mmHg, $p = .049$ and their DBP by 2.70 mmHg, $p = .004$ (Cox et al., 2001). Contrastingly, vigorous intensity exercisers from the same program did not see the same significant blood pressure decreases (Cox et al., 2001). Another study examined the effect of 30 minutes of mild-to-moderate intensity exercise

twice weekly for 6 months in 40 adults with a mean age 67 years on blood pressure and arterial stiffness (Tabara et al., 2007). This study also confirmed earlier findings; a significant decrease was found in both SBP and DBP after 6 months of training, with p values of .0056 and .0008, respectively (Tabara et al., 2007).

Two other studies were designed with controls and also found positive effects on blood pressure levels with exercise. In the study 12 men at four different activity levels over 4 months by Jennings, Nelson, & Nestel (1986), both systolic and diastolic blood pressures fell significantly from the sedentary and below-sedentary activity levels to the three times per week regimen and the seven times per week regimen, $p < .01$. Additionally, blood pressure was significantly less at the activity level of daily exercise versus three time weekly exercise, $p < .05$ (Jennings et al., 1986).

These findings were supported by a 28-year study on cardiovascular changes with aging and exercise (Kasch et al., 1995). Using a cohort selected from a university-based fitness program, 12 men were placed into a group that exercised consistently for 28 years and 12 men who had dropped out from the exercise program were placed in a control group (Kasch et al., 1995). Over the 28-year time period, the blood pressures of exercise group participants remained the same, while those of the drop-outs increased significantly, $p < .001$ (Kasch et al., 1995). These results demonstrate the moderating effect that exercise can have on blood pressure. Overall, exercise training studies have shown a very positive effect of regular exercise on blood pressure levels, either decreasing or maintaining blood pressure levels.

Exercise Program Attendance Versus Participant Results

Several structured exercise program studies examined the relationship between program attendance rates and participants' results. In the study on lifestyle activity compared to a structured exercise program, Andersen and colleagues (1999) found that the participants in the structured exercise program condition had an overall mean attendance rate of 87.7% over the 16 week program. It was then confirmed that attendance was significantly related to participant weight loss, $r = .53$, $p = .02$ (Andersen et al., 1999).

King and colleagues (1995) found similar results with more obvious increases in HDL levels over 2 years in the lower-intensity, home-based training treatment group than in other treatment groups. The lower-intensity, home-based training group averaged 3.0 training sessions per week over the 2 years of the program, while the higher-intensity, home-based group averaged 2.2 exercise sessions per week and the higher-intensity, group-based condition averaged only 1.3 sessions per week (King et al., 1995). This demonstrated that the treatment group with higher program adherence achieved better results in HDL level improvement (King et al., 1995).

It was found that attendance or adherence rates were often not compared to the changes in levels of potential risk factors for CAD in structured exercise program studies. Often attendance rates were reported for the group as a whole, but not necessarily analyzed with the results. Attendance rates were also used as inclusion criteria, rather than as a basis for analysis. For example, in the lifestyle modification program study by Kappagodda and colleagues (2006), participants who attended 60% of the sessions were

considered to have completed the program, however further breakdown of attendance versus results was not performed.

Research-based Generalizations on Risk Factor Management

Although evidence for physical activity as a management tool for the CAD risk factors of obesity and high blood pressure appears positive; the evidence from the studies discussed is not solely positive. The overall risk profile for Tecumseh Community Study participants was reported as better for the physically active than the sedentary; the sedentary group was heavier, was more overweight, had higher cholesterol, triglycerides, blood pressure, heart rates, and insulin, and had lower HDL (Gudbrandsson et al., 1994). However, even considering general improvements to overall risk for CAD, within the studies examined, individual risk factor levels were not always significantly different between exercising and non-exercising groups.

Additional Research and Limitations

With some mixed results as to the effect of physical activity on risk factors for CAD such as obesity and hypertension, additional research could help provide clarity to the topic. Possibly some of the variations in findings could be accounted for by limitations caused by changes in physical activity over time or changes to other factors such as nutrition or stress levels (Sesso et al., 2000). Differences may have also been seen due to varying methods of physical activity classification and differences in survey questionnaire design between studies. Several studies were also performed without control groups, which can lead to difficulty in making true inferences from data. It seems

evident that the area of physical activity and risk factors for CAD could only benefit from further research.

CHAPTER 3

METHODS

Data for this study were collected during the University of Nevada, Las Vegas (UNLV) Adult Exercise Program (Golding, 2007). The daily exercise program was initiated in 1976 and continued until 2007 (Golding, 2007). The program was initially approved by the UNLV Institutional Review Board in 1976, and approval was maintained for the entire course of the program (Golding, 2007). The program consisted of organized exercise classes and periodic health and fitness testing for participants (Golding, 2007).

Exercise Class Structure

The exercise classes were held Monday through Friday during the academic year and Monday through Thursday during the academic summer session, with the exception of public holidays or university closures (Golding, 2007). The exercise classes were directed by university instructors or graduate assistants and consisted of 45-minute daily sessions focusing on stretching and flexibility, muscular fitness, and aerobics (Golding, 2007). Each session included a warm-up segment, a muscular strength and endurance segment, an aerobic exercise segment, and a cool down segment (Golding, 2007).

Participants

The program participants ranged in age from 30–80 years (Golding, 2007). There were not specific age restrictions for participation, however participation was restricted by the health of the applicant and the ability of the applicant to take part in vigorous

exercise (Golding, 2007). All admitted participants were in general good health; they were required to pass an initial health screening before participation (Golding, 2007). A written approval from a physician was required if deemed necessary after the completion of the initial screening (Golding, 2007). The program group contained both male and female participants (Golding, 2007). There were two separate exercise groups, classified by sex; the males exercised from 12:00 p.m. to 1:00 p.m., and the females exercised from 11:00 a.m to 12:00 p.m. (Golding, 2007).

Over the 31 years of the program, approximately 10 participants were enrolled for the entire time period (Golding, 2007). The group of participants varied from year to year, meaning that the same group of participants did not complete or participate in the entire 31 years of the program (Golding, 2007). Each year participants were gained and lost, so the overall participant composition was variable (Golding, 2007). Each year there was between 70 to 80 participants in the class (Golding, 2007). There were about 300 participants who spent at least 1 year in the program (Golding, 2007). Many participants attended the program intermittently over the course of the program; it was not uncommon for participants to attend sessions for a semester or a year and then drop out to return at a later date (Golding, 2007). Attendance was recorded daily (Golding, 2007). There was an effort for class members to attend 70% of the time, or 3 out of the 5 days (Golding, 2007). The actual average attendance over the year was usually 3.5 times per week (Golding, 2007).

Data Collection

In addition to organized exercise sessions, the program also included health and physical fitness testing with corresponding data collection of the results (Golding, 2007). In the fall semester of the university (September) after being accepted into the exercise program, participants were tested; after the academic year (May/June), participants were retested (Golding, 2007). Participants with continued enrollment were then tested annually thereafter, in the spring semester (Golding, 2007). The data were released to participants for personal use, as well as collected by the Exercise Physiology lab staff for analysis (Golding, 2007). Participant measurements were taken on physical fitness parameters, body composition, electrocardiogram, and blood profiles (Golding, 2007).

The physical fitness testing included a group of tests closely following those of the YMCA Physical Fitness Testing Battery (YMCA of the USA [YMCA], 2000). Participants' ages on the day of testing and birth dates were recorded and anthropometric measurements of weight and height were taken (Golding, 2007). Resting heart rate and resting blood pressure were measured by trained technicians (Golding, 2007). Body composition was evaluated by skinfold measurements in conjunction with the Jackson-Pollock sum-of-four prediction equation (YMCA, 2000). However, seven skinfold sites (chest, triceps, abdomen, back, hip, thigh, and midaxillary measurements) were measured and waist circumference was measured (Golding, 2007). In some participants dual x-ray absorptiometry was also used for body composition (Golding, 2007). All participants had a somatotype photograph taken (Golding, 2007). Cardiovascular fitness was assessed using the 3-minute step test and Physical Working Capacity Test as described in the *YMCA Fitness Testing and Assessment Manual* (YMCA, 2000). Flexibility was

measured using the YMCA sit-and-reach test (Golding, 2007). During some years of the study, hip flexion was also measured in participants (Golding, 2007). Strength was evaluated with number of repetitions of bench presses, bicep curls, and abdominal crunches (Golding, 2007). These tests were chosen to evaluate main components of physical fitness including strength, flexibility, aerobic fitness, and body composition (Golding, 2007).

Resting electrocardiogram (EKG) readings were obtained for each participant during semi-annual testing (Golding, 2007). EKG readings were performed using a three channel EKG machine from Fukuda Denshi (Golding, 2007). The machine produced a print-out including EKG tracings, heart rate, various interval measurements, axis measurements, and wave height measurements (Golding, 2007). Additionally, the print-out included a recommendation for or against exercise based on the reading (Golding, 2007). The readings were assessed by laboratory staff and forwarded to the personal physicians of the participants as necessary (Golding, 2007).

Blood profiles were performed at a local diagnostic laboratory, and results were released to the exercise physiology laboratory staff (Golding, 2007). A vast range of tests were completed including levels of: glucose, creatinine, minerals, proteins, lipids, blood cells, and other blood testing parameters (Golding, 2007). Some participants were also evaluated for homocysteine and prostate specific antigen (Golding, 2007).

Participant testing was conducted twice each year of the program's existence and data were stored both electronically and in physical hard-copy format (Golding, 2007). For this research study, coded data that were stored electronically were used for analysis.

Data Analysis

Relevant data were selected from the existing electronic data set. Participants' attendance information, demographic information, and measurements affecting CAD risk were extracted for analysis. Physical fitness testing data were also selected from the existing data set. The demographic information included participants' age and sex. In addition the occupation of each participant was noted. Body composition measurements and blood pressure measurements were extracted as potential CAD risk factors. Resting heart rate, bench press repetitions, bicep curl repetitions, sit-and-reach score, 3-minute step test recovery heart rate, and abdominal crunches were selected as measures of physical fitness.

The included participants were then limited by the length of participation in the program. In order for research study inclusion, the participants had 3 consecutive years of study participation, with physical fitness testing in the first year and third year of participation. This 3-year time period was chosen for several reasons. Although over the course of the study, several participants did not remain consecutively within the program for more than a year, there was a sample of 32 participants who participated in 3 consecutive years of the structured exercise program. In addition, literature has shown that exercise program participation for a time period as short as 2 years, can have protective effects from heart disease as far out as 10 years beyond the time of program participation (Kappagodda et al., 2006). An initial evaluation of program data also revealed that in participants of longer than 3 years, changes to measured variables leveled out, so examining a time period beyond 3 years was not expected to yield different results than those at 3 years. Participants were not excluded based on age or gender.

The main possible CAD risk factors examined were blood pressure level and obesity. SBP and DBP were chosen for evaluation of blood pressure level. Measurements of BMI, waist circumference, and skinfolds were representative of obesity. Skinfolds were evaluated both as individual measurements and as a composite body fat percentage calculated by the Jackson-Pollock sum-of-four prediction equation (YMCA, 2000).

The levels of the variables at the first year selected for each program participant were compared to the levels of the variables 3 years later. The 3-year time span was not necessarily the same span of years for each participant. This comparison was performed using the statistical method of one-way repeated measures analysis of variance (Warner, 2008).

The levels of the variables that may contribute to CAD risk level were then considered in conjunction with physical fitness variables and attendance data. Pearson's correlation was used to assess the association between physical fitness and attendance, and levels of potential CAD risk factors (Warner, 2008).

CHAPTER 4

RESULTS AND DISCUSSION

Introduction

Between the years 2000 and 2006, 45 people participated in the UNLV Adult Exercise Program for 3 consecutive years or more. Of those 45 participants, 32 participated in the health and fitness testing procedures for the 3 years of program participation. These 32 participants were used in this paper's evaluation. Their average age at the beginning of the evaluation was 55 years. Twenty-one participants (66%) were male and 11 (34%) were female. All participants were business and professional workers with minimal occupational physical activity; many were employed on the UNLV campus. During the 3 years studied, their average attendance in the exercise program was 70%.

Repeated measures ANOVA was used to assess whether there was a significant change in body composition and blood pressure during the program. Data were screened for normality using the Shapiro-Wilk W test as well as by visual examination of distribution. Outliers were also investigated by visually examining the distribution.

Analysis of Overall Fitness and Attendance Data

Data for age, resting heart rate, weight, repetitions of abdominal crunches, sit-and-reach score, recovery heart rate after the 3-minute step test, and attendance were found to be normally distributed. No significant skewness or kurtosis was found in the data sets. After data evaluation for normality, resting heart rate, weight, abdominal crunches, sit-and-reach score, recovery heart rate, and attendance were analyzed using one-way repeated measures ANOVA. The Huynh-Feldt correction to the degrees of freedom was

used in instances where the sphericity assumption was violated. All post hoc tests were conducted using the Sidak correction for multiple comparisons.

During the study, the participants' mean resting heart rate increased significantly from 68 to 71 beats per minute, $F(1.64) = 3.75, p = .038$. However, post hoc tests revealed that the differences between mean resting heart rates for individual years (i.e. comparing year 1 to year 2), were not significantly different, $p > .05$.

Mean weight also increased significantly from 77.71 to 80.38 kg, $F(1.41) = 7.09, p = .005$. Post hoc results indicated a significant difference between years 1 and 3, $p = .004$, and years 2 and 3, $p = .011$, while there was no significant difference between years 1 and 2, $p > .05$.

During the 3 years the mean number of abdominal crunches decreased significantly from 59 to 52 repetitions, $F(1.92) = 4.67, p = .013$. Post hoc testing showed that the significant difference was between years 1 and 3, $p = .011$. There was no significant change in the sit-and-reach scores, $p > .05$; mean sit-and-reach scores remained at 14 cm (5.5 in.) during the study. The recovery heart rate after the 3-minute step test increased significantly from 88 to 91 beats per minute, $F(2) = 5.62, p = .006$; correspondingly post hoc testing showed that the significant difference was, again, between years 1 and 3, $p = .005$.

Attendance was also evaluated during the study. It was thought that attendance would remain constant during that time period. As hypothesized, there was no significant difference in attendance over 3 years of testing, $F(1.55) = 1.04, p = .345$. Mean attendance was 70% in year 1, 71% in year 2, and 69% in year 3.

Changes in seven skinfold sites were also evaluated: chest, triceps, abdomen, back, hip, thigh, and midaxillary (Table 3). Two mean skinfold measurements (chest and midaxillary) showed no changes over 3 years of assessment.

Table 3

Changes in Skinfold Measurements

Skinfold Site	Year 1 <i>M</i> (mm)	Year 2 <i>M</i> (mm)	Year 3 <i>M</i> (mm)	Change (mm)	<i>p</i> value
Chest	18.28	19.66	18.59	↑ 0.31	.254
Triceps	15.56	16.78	18.75	↑ 3.19	< .001
Abdomen	28.13	31.50	29.00	↑ 0.87	.015
Back	19.38	20.25	21.41	↑ 2.03	.043
Hip	19.03	20.13	23.47	↑ 4.44	< .001
Thigh	20.03	19.87	21.39	↑ 1.36	.027
Midaxillary	18.97	20.28	20.50	↑ 1.53	.103

There was no significant change in the mean chest skinfold measurement; it remained unchanged between 18.25 and 19.75 mm during the 3 years, $F(2) = 1.54$, $p = .254$. There was also no significant change in the mean midaxillary skinfold measurement with the

measurements staying relatively constant between 19.00 and 20.50 mm $F(2) = 2.36, p = .103$.

There were however, several skinfold measurements that reflected increasing body mass. There was a significant increase from 15.50 to 18.75 mm in the mean triceps skinfold measurement, $F(2) = 13.21, p < .001$. Post hoc results showed significant differences between years 1 and 3, $p < .001$, and years 2 and 3, $p = .011$, while the year 1 and 2 results were non-significant, $p > .05$. The mean back skinfold measurement also increased significantly from 19.50 to 21.50 mm, $F(2) = 3.32, p = .043$; post hoc testing revealed that the significant difference was between years 1 and 3, $p = .026$. Similarly, the mean hip skinfold measurement increased significantly from 19.00 to 23.50 mm, $F(2) = 12.77, p < .001$. Post hoc testing showed that there was a significant increase between both years 2 and 3, $p < .001$, and years 1 and 3, $p < .001$.

Two mean skinfold measurements also showed an overall increase over 3 years, however, post hoc testing was less conclusive. There was a significant increase in the mean abdominal skinfold measurement, $F(2) = 4.50, p = .015$. However, post hoc testing showed that there was no significant difference between years 1 and 3 individually, $p > .05$. The mean abdominal skinfold measurement increased significantly from year 1 (28.00 mm) to year 2 (31.50 mm), $p = .025$, however it decreased significantly from year 2 (31.50 mm) to year 3 (29.00 mm), $p = .035$. The mean thigh skinfold measurement significantly increased from 20.00 to 21.50 mm, $F(2) = 3.85, p = .027$. However, post hoc testing revealed that the differences for individual pairs of years were not significantly different, $p > .05$. Overall, the mean skinfold measurements reflected either steady body fat levels or increasing body fat levels over 3 years of program participation.

Exercise Program Participation and Potential CAD Risk Factors

Changes in BMI, waist circumference, percent body fat, SBP, and DBP over 3 years of program participation were evaluated with one-way repeated measures ANOVA. Post hoc testing was performed using Sidak correction for multiple comparisons.

Exercise Program Participation and BMI

In order to assess whether there was a significant decrease in the mean BMI measurements, a repeated measures ANOVA test was performed using BMI as the dependent variable. Visual examination of histograms and box plots for each year of data revealed a relatively normal distribution and no extreme outliers. The BMI data were not significantly skewed or kurtotic. The Shapiro-Wilk W test was non-significant for all 3 years examined; W statistics for years 1 through 3 were $W(32) = .98, p = .872$, $W(32) = .95, p = .414$, and $W(32) = .97, p = .583$, respectively. Mauchly's test indicated that the assumption of sphericity had been violated ($p = .032$), therefore degrees of freedom were corrected using the Huynh-Feldt correction.

The mean BMI measurements for years 1 through 3 were 25.38, 25.84, and 26.31, respectively. The results showed that the mean BMI measurements increased significantly over the 3 years of testing and program participation, $F(1.74) = 5.45, p = .009, \eta^2 = .15$. Post hoc testing revealed that the mean BMI in year 1 was significantly lower than the mean BMI in year 3, $p = .023$, however, the differences between years 1 and 2 and years 2 and 3 were not significant, $p > .05$ (Figure 1).

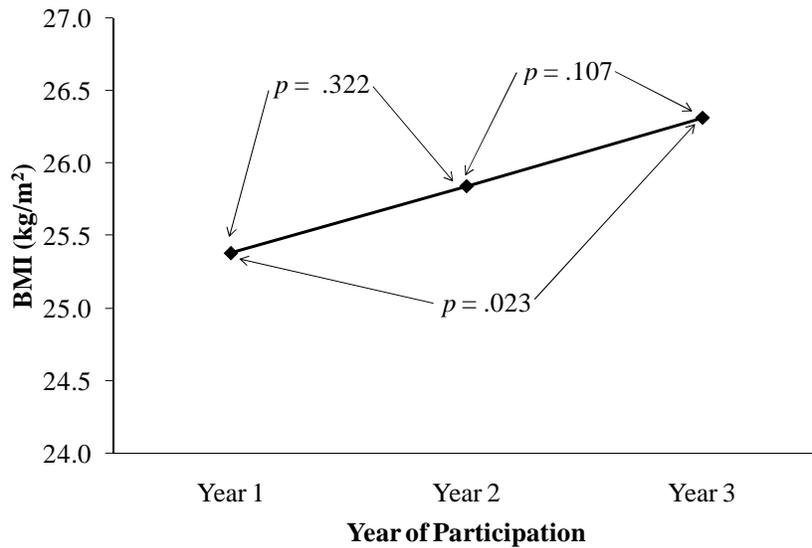


Figure 1. Exercise program participation and BMI.

Although mean BMI measurements increased over the course of participation, the mean BMI measurements remained within the AHA Prevention Conference VII category of overweight (25 – 29.9) for the duration of the 3 years (Eckel et al., 2004). While it was expected that participants would decrease BMI measurements and possibly score mean BMI measurements below 25, in the normal category, it is still important to note that the mean BMI measurements may not have increased to a level of high clinical significance and risk. Brochu and colleagues (2000) found that 75% of CAD patients entering a rehabilitation program between the ages of 30 and 85 years had a BMI of greater than or equal to 27, and exercise program participants did remain below this level. However, ideally participants would have lowered their BMIs to a desirable level below 25, as this is the level at which overweight and obese adults are considered to be at risk (Expert Panel on the Identification, Evaluation, and Treatment of Overweight and

Obesity in Adults, 1998).

Exercise Program Participation and Waist Circumference

In order to assess whether there was a significant decrease in the mean waist circumference measurements, a repeated measures ANOVA test was performed using waist circumference as the dependent variable. Visual examination of histograms and box plots for each year of data revealed a relatively normal distribution and no extreme outliers. The waist circumference data were not significantly skewed or kurtotic. The Shapiro-Wilk W test was insignificant for all 3 years examined; W statistics for years 1 through 3 were $W(32) = .96, p = .351$, $W(32) = .97, p = .396$, and $W(32) = .98, p = .908$, respectively. Mauchly's test indicated that the assumption of sphericity had not been violated, $p = .564$, therefore degrees of freedom were not corrected as sphericity was assumed.

The mean waist circumference measurements for years 1 through 3 were 88.25 cm, 88.63 cm, and 91.19 cm (34.74 in., 34.89 in. and 35.90 in.), respectively. The results show that the mean waist circumference measurements increased significantly over the 3 years of testing, $F(2) = 5.63, p = .006, \eta^2 = .15$. Post hoc testing revealed that the mean waist circumference in year 1 was not significantly different from the mean waist circumference in year 2, $p = .974$, nor was the mean waist circumference in year 2 significantly different from the mean waist circumference in year 3, $p = .051$. However, the mean waist circumference measurement increase between years 1 and 3 were statistically significant, $p = .005$ (Figure 2).

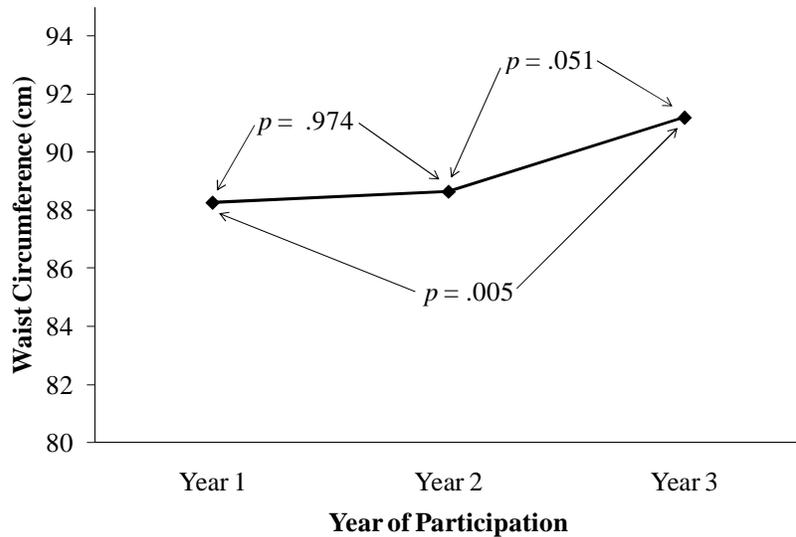


Figure 2. Exercise program participation and waist circumference.

Over the 3 year period of testing, there was an overall mean increase of 2.9 cm (1.1 in.) in waist circumference. CAD risk level based on waist circumference measurement is classified based on gender (Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults [PIETOO], 1998). According to this panel, waist circumference measurements of greater than 102 cm (40 in.) in males and 88 cm (35 in.) in females indicate high-risk (PIETOO, 1998). Over 3 years of program participation, the mean male waist circumferences for years 1, 2, and 3 were 93.95 cm, 95.24 cm, and 96.42 cm (36.99 in., 37.50 in., and 37.96), respectively. Correspondingly, mean female waist circumferences were 77.36 cm, 76.00 cm, and 81.18 cm (30.46 in., 29.92 in., and 31.96 in.), respectively over the 3 years. Although waist circumferences did increase significantly, the mean waist circumferences measurements for both males and females remained below the threshold of high-risk as defined by the above

referenced panel.

Exercise Program Participation and Percent Body Fat

In order to assess whether there was a significant decrease in the body fat percentages, a repeated measures ANOVA test was performed using percent body fat, as calculated by the sum-of-four skinfold sites equation, as the dependent variable (YMCA, 2000). Visual examination of histograms and box plots for each year of data revealed a relatively normal distribution and no extreme outliers. There was no significant skewness or kurtosis for years 1 or 3, however, body fat data for year 2 was slightly positively skewed with a statistic of 2.30. The Shapiro-Wilk W test was non-significant for all 3 years examined; W statistics for years 1 through 3 were $W(32) = .96, p = .323$, $W(32) = .94, p = .084$, and $W(32) = .95, p = .164$, respectively. As the test for normality was non-significant, the data for year 2 was not adjusted for skewness. Mauchly's test indicated that the assumption of sphericity had not been violated, $p = .662$, therefore degrees of freedom were not corrected as sphericity was assumed.

The mean body fat measurements for years 1 through 3 were 24.10%, 25.31%, and 25.97%, respectively. The results show that the mean body fat percentages increased significantly over the 3 years of testing, $F(2) = 7.56, p = .001, \eta^2 = .196$. Post hoc testing revealed that the mean percent body fat in year 3 was not significantly different than the mean percent body fat in year 2, $p = .488$. However, both the mean percent body fat increases between years 1 and 2, $p = .032$, and years 1 and 3, $p = .003$, were statistically significant (Figure 3).

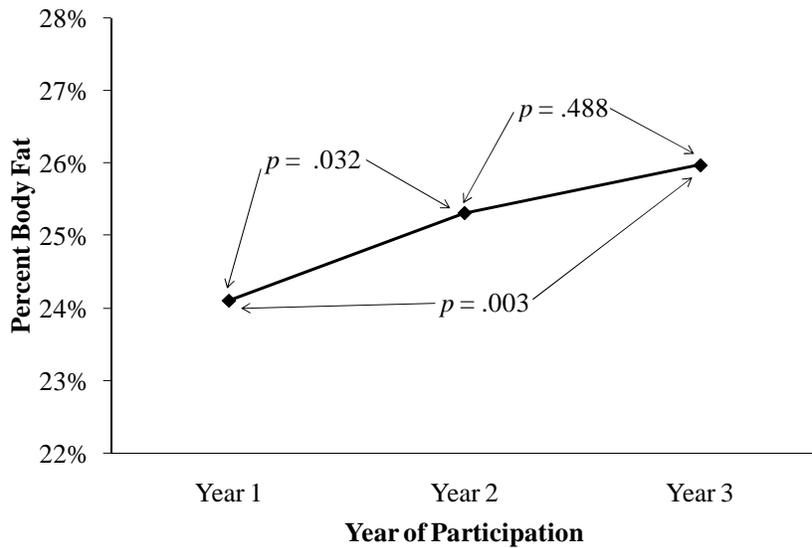


Figure 3. Exercise program participation and percent body fat.

The mean increase in percent body fat was 1.87%. Similar to the waist circumference, when evaluating percent body fat, males and females are considered based on different percent body fat scales as well. Males at a level of 25% body fat or greater and females at a level of 32% body fat or greater are considered at risk for obesity-related diseases, including CAD (Lohman, 1992). Over 3 years of program participation, the mean male percent body fat measurements for years 1, 2, and 3 were 24.96%, 26.08%, and 26.43%, respectively. Correspondingly, mean female percent body fat measurements were 22.46%, 23.84%, and 25.10%, respectively over the 3 years. Although there was an overall increase in mean percent body fat, during the 3 year timeframe, the women remained below the “at risk” percent body fat level. The men began the 3 year program in the “at risk” category, and remained there for the duration of the program.

Exercise Program Participation and SBP

In order to assess whether there was a significant decrease in the mean SBP, a repeated measures ANOVA test was performed using SBP as the dependent variable. Visual examination of histograms and box plots for each year of data revealed a relatively normal distribution and no extreme outliers. There was no significant skewness or kurtosis for years 1 or 2, and the Shapiro-Wilk W test was insignificant for those 2 years as well. W statistics for years 1 and 2 were $W(32) = .98, p = .672$ and $W(32) = .96, p = .247$, respectively. There was no significant kurtosis for year 3, however, the year 3 data were positively skewed and the Shapiro-Wilk W test was significant, $W(32) = .93, p = .032$. Although this indicated that the data were not normally distributed, the data were not adjusted for this to keep continuity in testing methods between data sets. The positive skewness was caused by two high participant blood pressure readings on testing days, and the scores were kept in the data set. Mauchly's test indicated that the assumption of sphericity had been violated, $p = .045$, therefore degrees of freedom were adjusted using the Huynh-Feldt correction.

The mean SBP measurements for years 1 through 3 were 122.19 mmHg, 123.78 mmHg, and 123.56 mmHg, respectively. The results show no significant change to the mean SBP measurements over the 3 years of testing, $F(1.77) = .23, p = .765$.

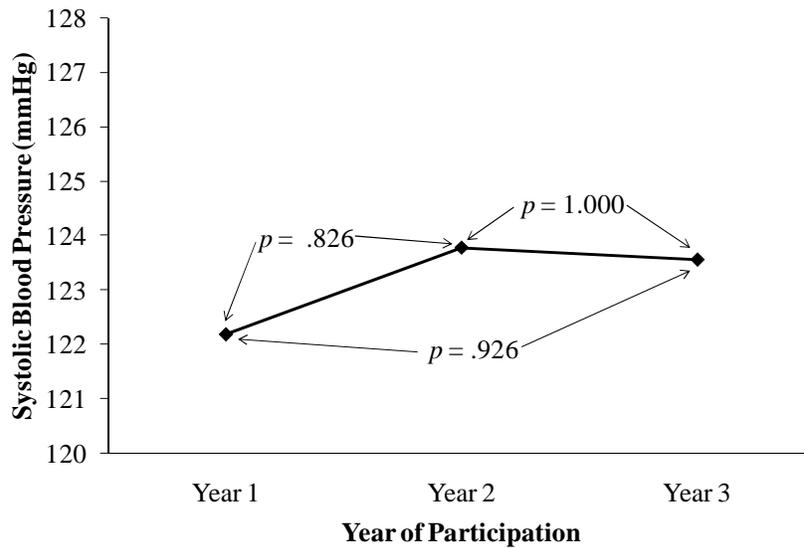


Figure 4. Exercise program participation and systolic blood pressure.

The mean SBP remained relatively constant between 122 and 124 mmHg. These mean SBP measurements are classified as slightly above the normal or optimal systolic blood pressure level of 120 mmHg, in the prehypertensive category (Chobanian et al., 2003).

Exercise Program Participation and DBP

In order to assess whether there was a significant decrease in the mean DBP, a repeated measures ANOVA test was performed using DBP as the dependent variable. Visual examination of histograms and box plots for each year of data revealed a relatively normal distribution and no extreme outliers. Corresponding with the two outlying high SBPs in year 3, there was a group of three outlying high DBPs. The highest blood pressure was 20 mmHg or 2.23 standard deviations ($\sigma = 8.97$) above the 75th percentile. These scores were checked for accuracy in recording and kept in the data set. There was

no significant skewness or kurtosis for years 1 or 2, and the Shapiro-Wilk W test was non-significant for those 2 years as well. W statistics for years 1 and 2 were $W(32) = .97$, $p = .525$ and $W(32) = .95$, $p = .174$, respectively. There was no significant kurtosis for year 3, however, the year 3 data were positively skewed with a statistic of 2.58 and the Shapiro-Wilk W test was significant, $W(32) = .88$, $p = .002$. Although this indicated that the data were not normally distributed, the data were not adjusted for this to keep continuity in testing methods between data sets. Mauchly's test indicated that the assumption of sphericity had not been violated, $p = .307$, therefore sphericity was assumed.

The mean DBP measurements for years 1 through 3 were 79.78 mmHg, 78.13 mmHg, and 77.28 mmHg, respectively. The results show that the mean DBP measurements did not change significantly, $F(2) = 1.54$, $p = .224$, $\eta^2 = .05$ (Figure 5).

Over the 3-year period of testing and program participation, the mean DBP remained relatively constant between 77 and 80 mmHg. These mean DBP measurements are all within the normal category of DBP of less than 80 mmHg (Chobanian et al., 2003).

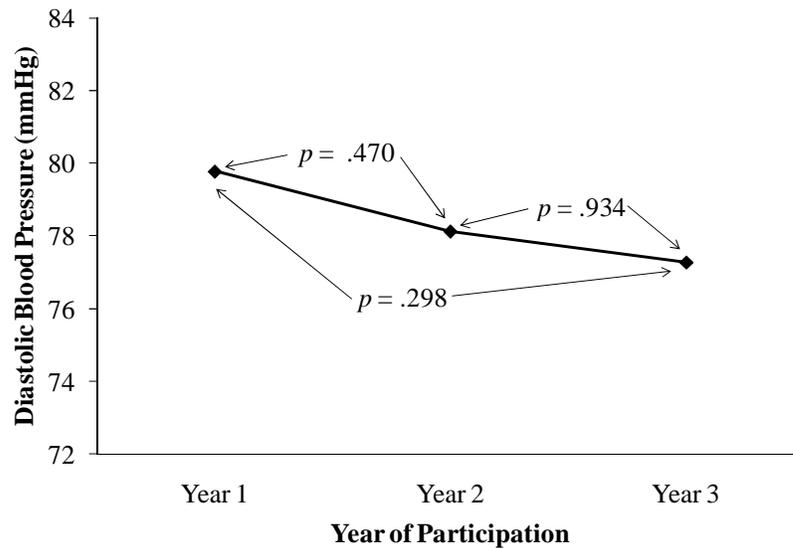


Figure 5. Exercise program participation and diastolic blood pressure.

Exercise Program Participation and Lipid Levels

A subset of data consisting of 17 participants was analyzed for changes in lipid levels using one-way repeated measures ANOVA. The group consisted of 11 males and 6 females. Over the 3 years of the exercise program, lipid levels remained steady (Table 4). Only the changes in HDL levels were significant, $F(2) = 3.78$, $p = .034$, $\eta^2 = .19$; HDL levels increased by $5 \text{ mg}\cdot\text{dL}^{-1}$ over 3 years. The significant difference was found between years 2 and 3, $p = .046$. These results concerning HDL levels were very consistent with those found by both King and associates (1995) as well as Takeda and associates (1996). Both groups of researchers found a significant improvement to HDL levels after 2 years of exercise program participation (King et al., 1995; Takeda et al., 1996).

Table 4

Changes in Lipid Levels

Measurement	Risk Level	Year 1 <i>M</i>	Year 2 <i>M</i>	Year 3 <i>M</i>	<i>p</i> value
Total Cholesterol (mg•dL ⁻¹)	≥ 240	185.35	186.65	192.06	.260
Low Density Lipoprotein (mg•dL ⁻¹)	≥ 160	112.35	113.35	113.53	.948
High Density Lipoprotein (mg•dL ⁻¹)	≤ 40	57.53	56.47	62.53	.034
Triglycerides (mg•dL ⁻¹)	≥ 150	77.24	83.65	79.59	.426

Other research showed some improvement to other lipid levels as well within time periods of 3 months to 2 years (Daubenmeier et al., 2007; Kappagodda et al., 2006). However, the research of King and associates was very similar to the present study, as they found no changes to LDL, total cholesterol, or triglycerides with an exercise regimen. It is possible that improvement in other factors was influenced by other variables including diet or drug therapy.

Overall it was promising that HDL levels improved, and other lipid levels did not get worse over the 3-year program duration. As body mass measurements increased, lipid levels remained constant.

Attendance, Physical Fitness, and CAD Risk

Participant data on BMI, waist circumference, percent body fat, SBP, and DBP were all analyzed for correlation with program attendance and physical fitness factors. The physical fitness factors examined were resting heart rate, repetitions of abdominal crunches, sit-and-reach score, and recovery heart rate after a 3-minute step test. For a majority of the relationships, there was no significant correlation found above .6. There was no significant relationship between program attendance and any of the possible risk factors for CAD.

Only two relationships between possible risk factors for CAD and physical fitness factors were found to be both strong and significant. Both recovery heart rate after a 3-minute step test and waist circumference (Table 5) and recovery heart rate after a 3-minute step test and BMI had strong significant relationships (Table 6). It was found that as recovery heart rate increased in beats per minute, generally waist circumference and BMI also increased.

Table 5

Recovery Heart Rate and Waist Circumference

Measurement	Statistic	Waist Circumference (cm) <i>Year 1</i>	Waist Circumference (cm) <i>Year 2</i>	Waist Circumference (cm) <i>Year 3</i>
Recovery Heart Rate (bpm) <i>Year 1</i>	Pearson Correlation	.916	.932	.929
	Significance (2-tailed)	< .001	< .001	< .001
	<i>N</i>	32	32	32
Recovery Heart Rate (bpm) <i>Year 2</i>	Pearson Correlation	.874	.901	.893
	Significance (2-tailed)	< .001	< .001	< .001
	<i>N</i>	32	32	32
Recovery Heart Rate (bpm) <i>Year 3</i>	Pearson Correlation	.897	.919	.944
	Significance (2-tailed)	< .001	< .001	< .001
	<i>N</i>	32	32	32

Table 6

Recovery Heart Rate and BMI

Measurement	Statistic	BMI (kg/m ²) <i>Year 1</i>	BMI (kg/m ²) <i>Year 2</i>	BMI (kg/m ²) <i>Year 3</i>
Recovery Heart Rate (bpm) <i>Year 1</i>	Pearson Correlation	.761	.756	.726
	Significance (2-tailed)	< .001	< .001	< .001
	<i>N</i>	32	32	32
Recovery Heart Rate (bpm) <i>Year 2</i>	Pearson Correlation	.742	.775	.732
	Significance (2-tailed)	< .001	< .001	< .001
	<i>N</i>	32	32	32
Recovery Heart Rate (bpm) <i>Year 3</i>	Pearson Correlation	.831	.850	.850
	Significance (2-tailed)	< .001	< .001	< .001
	<i>N</i>	32	32	32

CHAPTER 5

CONCLUSIONS

Summary

The purpose of this study was to investigate the levels of specific potential risk factors for CAD after participation in a structured exercise program for 3 years. Data were collected on 32 participants in the UNLV Adult Exercise Program. The exercise program focused on stretching and flexibility, muscular fitness, and aerobic capacity. Changes in blood pressure and body composition were assessed over the 3-year time period. These changes were considered in comparison to changes in physical fitness measures as well, including flexibility, strength and muscular endurance, and aerobic capacity. Attendance rates were also compared to changes in the potential CAD risk factors of body composition and blood pressure. It was thought that with steady program attendance, improvement in CAD risk profile based on blood pressure and body composition would be seen.

Conclusions

Within the limits of this study, it was found that body composition measures and blood pressure did not significantly improve, thereby improving overall CAD risk profile, with exercise program participation. Table 7 summarizes the overall changes in risk profile based on body composition and blood pressure in relationship to the associated levels of risk. Over the duration of 3 years of program participation, the general trend was for less favorable body compositions and steady blood pressure levels. Improvement

was also not seen in the physical fitness measures examined, and attendance was not associated with the outcomes of possible CAD risk factor measures.

Table 7

Coronary Artery Disease Risk Profile Results

Measurement	Sex	Risk Level	Year 1 <i>M</i>	Year 2 <i>M</i>	Year 3 <i>M</i>	Change
BMI (kg/m ²)	M/ F	≥ 25	25.38	25.84	26.31	↑ 0.93
Waist Circumference (cm)	M	> 102	93.95	95.24	96.42	↑ 2.47
	F	> 88	77.36	76.00	81.18	↑ 3.82
Percent Body Fat (%)	M	≥ 25	24.96	26.08	26.43	↑ 1.47
	F	≥ 32	22.46	23.84	25.10	↑ 2.64
Systolic Blood Pressure (mmHg)	M/ F	140	122.19	123.78	123.56	↑ 1.37
Diastolic Blood Pressure (mmHg)	M/ F	90	79.78	78.13	77.28	↓ 2.5

Body composition, as evaluated by BMI, waist circumference, and percent body fat, did not improve over 3 years of program participation. Mean BMI, waist circumference, and percent body fat all increased significantly during the time period.

There was also no significant improvement in blood pressure levels in 3 years of program participation. Both DBP and SBP measurements remained at the same levels,

very near the cutoff for optimal or normal blood pressure as defined by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (Chobanian et al., 2003).

While it was anticipated that blood pressure levels would decrease with exercise program participation, it is still important to note that blood pressures did not rise and did not become CAD risk factors for participants. Data from the FHS has shown that from age 30 – 84 linear rises are seen in both SBP and DBP; and this effect was not observed in this sample (Franklin et al., 1997). Additionally, obesity has been shown to be an independent risk factor for hypertension development, with BMI specifically being a good predictor of hypertension (Mertens & Van Gaal, 2000; Spiegelman, Israel, Bouchard, & Willett, 1992). Again, in this case, although BMI and other indicators of obesity increased, blood pressure levels remained steady and below defined levels of hypertension. In this case, it seems possible that the exercise program did yield a protective effect from increasing blood pressures and hypertension with increasing age and BMI.

Given the generally unfavorable changes in potential risk factors for CAD, except for HDL cholesterol, with exercise program participation in this study, it is important to examine the possible reasons that these results were obtained. There are many possible explanations for this phenomenon of observing increasing levels of body fat and steady levels of blood pressure.

Diet

Weight and body mass measures may have increased due to insufficient control of participant diet. Participants were not required to follow a specific diet or level of caloric

intake for program participation. As diet was not controlled, it is possible that participants began increasing their caloric intake as they were increasing their energy expenditure in the exercise program. This increase in caloric intake may have diminished improvements in body composition. While participants were counseled on the importance of good nutrition, meal or nutritional measurements were not documented. Upon discussing caloric intake with participants after 1 year of participation in the program, participants often had general feelings of “being hungry all the time” due to exercising regularly (Golding, 2007). These feelings of hunger may have promoted increased calorie intake above and beyond the increased energy expenditure of the program.

This tendency was discussed in detail in a controversial article meant for the general public published in Time magazine in August of 2009, much to the disappointment of exercise physiologists around the world (Cloud, 2009). Exercise suddenly became the “bad guy” when it came to weight loss. However, these results are not an argument against exercise, but rather an argument for nutritional counseling, the importance of diet, and experimental control.

Exercise Frequency and Intensity

Another possible explanation for the increases seen in body mass may be an insufficient level of intensity or exercise frequency. Recommendations on the appropriate intensity level and frequency of exercise can vary substantially (Blair, LaMonte, & Nichaman, 2004; Institute of Medicine, 2005; Lee, Djoussé, Sesso, Wang, & Buring, 2010; U.S. Department of Health and Human Services, 2008). While the U.S. Department of Health and Human Services currently recommends 150 weekly minutes of

moderate-intensity exercise or 75 weekly minutes of vigorous-intensity exercise for adults, the Institute of Medicine recommends over twice that amount with a recommendation of 420 weekly minutes of moderate-intensity exercise to maintain a normal BMI level. A recent study by Lee and associates found that women exercising at a moderate-intensity level for 60 minutes daily were able to maintain a healthy BMI and gain less than 2.3 kg over 13 years; women exercising with less frequency and intensity gained weight and had unhealthy BMIs. After reviewing physical activity recommendations, Blair and colleagues also agreed that the recommendation of 30 minutes of daily exercise may not be enough to prevent unhealthy weight-gain. Considering this information, it is possible that the program participants did not exercise enough or at an appropriate intensity level to prevent an increase in body mass. Participants averaged 158 weekly minutes of exercise in the program, which according to the research discussed above may simply not be enough to prevent weight gain. Additionally, participants may not have always come to the program with the focus to exercise at the necessary intensity level either.

Drug Therapy

Participant use of drug therapy was not recorded or used in the study, nor were participants excluded from participation based on drug therapy. It is highly probable that participants using prescription or over-the-counter drugs related to blood pressure or weight loss had results that were affected by those factors.

Participants may have used over-the-counter or prescription medications related to weight loss and then discontinued use upon joining the exercise program. This may have

affected the ability to maintain or lose weight in the program. Diet drugs used during the program may also have affected results.

It is also known that there were participants on blood pressure medication. This medication could have influenced physical fitness results in particular, as blood pressure medications often influence heart rate and other cardiovascular factors.

Additional Exercise and Activities

Participants were not questioned regarding their prior experience with exercise or their exercise regimen outside of the exercise classes. Prior activity levels as well as exercise and activity performed concurrent to the exercise program would influence total caloric expenditure and ultimately the program results.

It is possible that some participants were exercising more outside of the exercise class. This means that not all participants may necessarily have been doing the same amount or intensity of exercise each week; the results were dependent not only on the exercise program, but on that additional exercise.

Participants also may have exercised prior to joining the exercise program and then abandoned their prior regimen upon beginning the structured program. Prior exercise regimens may not have been the only way that the participants modified their lifestyles after joining the program. They also may have substituted the adult exercise program for other activities that they did before joining the program, i.e. taking the stairs or walking to the mailbox. Any decrease in other activity level done before beginning the program would affect the total balance of calories taken in and expended, and would therefore influence the body mass results of the program.

Time of Data Collection

The overall program results may also have been affected by the time at which the results were collected for each participant. Due to the nature of the program and the tendency of participants to join for a period of time, leave the program, and then return, the 3 consecutive years that were examined for each participant did not always begin with the participant's true first year in the program. Some participant data were collected beginning with the first year of program participation, while other participant data were collected somewhere in the middle of program participation or after an interruption in program participation. This lack of consistency in the timing of participant data collection may have influenced the perceived participant responsiveness to the program.

Low Initial Risk

One final observation regarding the results of the study is that in the area of blood pressure in particular, the participants began in a low-risk classification for both SBP and DBP. In this case, it would be difficult to see any decrease or improvement in blood pressure as a measure of risk profile, when the participants were already in a normal category. At some point, it becomes impossible to get "more normal."

Recommendations

The limitations to this study were broad. Several modifications have the potential to improve future studies on structured exercise programs and CAD risk profile.

1. Diet was uncontrolled in this study. With control of diet, it would be more possible to isolate a cause and effect relationship between exercise and the levels of potential risk

factors. Tracking or controlling for diet would help alleviate similar problems in evaluating the effect of exercise on CAD risk factors.

2. Use of drug therapy was not controlled in this study. Creating exclusion criteria for these types of participants would help control this variable. It may also be possible to track changes to potential CAD risk factors in participants grouped based on use or non-use of pharmacologic substances.

3. Additional exercise outside of the structured exercise program was not considered or controlled. This may have altered their response to the program, depending on the structure of their prior or outside regimen.

Although this study did not provide a strong argument for exercise as a prevention strategy for CAD, it is still believed that exercise is beneficial and worthwhile. Due to the discussed extraneous variables of diet, exercise frequency and intensity, drug therapy, additional exercise and activities, time of data collection, and low initial risk it was determined that exercise was not able to be isolated as having a cause and effect relationship with CAD risk profile in this study. The obstacles in creating a long-term study to effectively demonstrate a cause and effect relationship are many, and this study was affected by several of them. Despite the study results, it is maintained that exercise has a healthy and positive effect on the body, including on CAD risk profile.

APPENDIX



**Biomedical IRB – Expedited Review
Approval Notice**

NOTICE TO ALL RESEARCHERS:

Please be aware that a protocol violation (e.g., failure to submit a modification for any change) of an IRB approved protocol may result in mandatory remedial education, additional audits, re-consenting subjects, researcher probation suspension of any research protocol at issue, suspension of additional existing research protocols, invalidation of all research conducted under the research protocol at issue, and further appropriate consequences as determined by the IRB and the Institutional Officer.

DATE: February 5, 2010
TO: **Dr. Lawrence Golding**, Kinesiology
FROM: Office for the Protection of Research Subjects
RE: Notification of IRB Action by Dr. Charles Rasmussen, Co-Chair
Protocol Title: **The Relationship of Participation in a Long-Term
Exercise Program and Selected Risk Factors for Coronary Artery
Disease**
Protocol #: 1001-3323

This memorandum is notification that the project referenced above has been reviewed by the UNLV Biomedical Institutional Review Board (IRB) as indicated in regulatory statutes 45 CFR 46. The protocol has been reviewed and approved.

The protocol is approved for a period of one year from the date of IRB approval. The expiration date of this protocol is February 1, 2011. Work on the project may begin as soon as you receive written notification from the Office for the Protection of Research Subjects (OPRS).

Should there be *any* change to the protocol, it will be necessary to submit a **Modification Form** through OPRS. No changes may be made to the existing protocol until modifications have been approved by the IRB.

Should the use of human subjects described in this protocol continue beyond February 1, 2011 it would be necessary to submit a **Continuing Review Request Form** *60 days* before the expiration date.

If you have questions or require any assistance, please contact the Office for the Protection of Research Subjects at OPRSHumanSubjects@unlv.edu or call 895-2794.

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VITA

Graduate College
University of Nevada, Las Vegas

Maryse Wells

Degrees:

Bachelor of Science, Business Administration, 2002
Colorado State University

Thesis Title: The Relationship of a Long-Term Exercise Program and Selected Coronary Risk Factors

Thesis Examination Committee:

Chairperson, Lawrence A. Golding, Ph.D.
Committee Member, John C. Young, Ph.D.
Committee Member, Richard D. Tandy, Ph.D.
Graduate Faculty Representative, J. Wesley McWhorter, Ph.D., MPT