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The effect of a 36-session Cardiac Rehabilitation Program on the Framingham Risk Score

Veronica Gonzales

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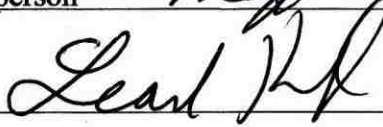
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**The effect of a 36-session Cardiac Rehabilitation
Program on the Framingham Risk Score**

BY

Veronica K. Gonzales

B.S. in Exercise Science

THESIS

Submitted in Partial Fulfillment of the
Requirements for the Degree of

**Masters of Science
Physical Education**

The University of New Mexico
Albuquerque, New Mexico

May, 2010

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ABSTRACT OF THESIS

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ABSTRACT

Cardiac Rehabilitation is widely used in the United States to help individuals who have had a cardiac event that is approved by insurance companies. Factors that lead to the development of cardiovascular disease are lack of exercise, tobacco use, obesity, high blood pressure, high cholesterol levels, and diet comprised of high consumption of saturated fats, salt, refined carbohydrates, and low levels of fruits and vegetables (World Health Organization, 2008). The purpose of this study is to examine the effectiveness of a cardiac rehabilitation program in modifying the patient's Framingham risk score after 36 sessions of cardiac rehabilitation. This is a retrospective study; data were taken from subject files that were acquired during the period of April 2005 to March 2008. The study included 152 male and 46 female subjects.

Baseline and 12-week values for blood pressure (systolic, diastolic), total cholesterol, HDL-C, and LDL-C, smoking status and diabetes status was collected. To evaluate each of the subject's data and form a Framingham risk score for each subject a computer program was developed using the Labview (National Instruments, Austin Texas). Statistical significance was found in all variables (Framingham risk score $p < .001$, systolic blood pressure $p = .049$, diastolic blood pressure $p = .002$, total cholesterol $p < .001$, LDL-C $p < .001$, HDL-C $p = .004$) between baseline and post cardiac rehabilitation program.

There was mild statistical significance between delta fat and the delta Framingham scores $p = .004$. There was no statistical significance found in between delta Framingham and delta MET $p = .817$.

Cardiac rehabilitation programs are an important part in improving an individual's health after a coronary event. Cardiac rehabilitation programs include education to improve an individual's diet skills for handling stress and improve their outlook on life after their recent cardiac event as well as reducing their risk for another coronary event. The Framingham risk score is an assessment tool that is widely accepted to evaluate risk factors that lead to the development of coronary heart disease. However there was research that showed that it underestimated an individual's risk for developing coronary heart disease.

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Chapter I

Introduction

Cardiovascular disease affected 80,000,000 individuals in the United States in 2006 (American Heart Association, 2008). Cardiovascular disease is an umbrella term that includes coronary heart disease, rheumatic heart disease, cerebrovascular disease and renal failure (American Heart Association, 2008). Factors that lead to the development of cardiovascular disease are lack of exercise, tobacco use, obesity, high blood pressure, high cholesterol levels, and diet comprised of high consumption of saturated fats, salt, refined carbohydrates, and low levels of fruits and vegetables (World Health Organization, 2008). Coronary heart disease is the leading cause of death in both men and women in the United States. Coronary heart disease is a form of cardiovascular disease, which includes all illnesses and diseases that affect the vessels going to and from the heart. In cardiovascular disease the vessels become occluded with plaque, making it difficult or impossible for blood to pass through. This occlusion may cause a myocardial infarction, which is damage or death to the areas of the heart not getting enough blood flow.

To qualify for a cardiac rehabilitation program an individual will have had a myocardial infarction, stent, coronary artery bypass graft, valve replacement or repair, heart transplant or new-onset angina. The American Heart Association (AHA), American College of Sports Medicine (ACSM), and American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR) have shown that cardiac rehabilitation programs are beneficial in reducing the reoccurrence of another coronary event or death. However, the exact reasons the programs are beneficial are unclear. In the past, cardiac

rehabilitation programs focused primarily on cardiovascular exercise to reduce the incidence of another coronary event. Cardiac rehabilitation programs have expanded on that theme and now include education and dietary recommendations to provide patients with a multifaceted program. During the cardiac rehabilitation program data are collected from blood tests, exercise tests, and questionnaires.

Cardiac rehabilitation programs are primarily housed in health care institutions such as hospitals. AACVPR has developed guidelines and an accreditation for cardiac rehabilitation programs, providing patients with information and lifestyle changes that will prevent the reoccurrence of a cardiac event. Cardiac rehabilitation programs maintain data per AACVPR policy to assess the efficacy of the program's effectiveness in reducing the risk of another coronary event. AACVPR has identified the major domains that a cardiac rehabilitation program should have in order to acquire and maintain a certification. The domains of AACVPR are behavioral (education, tobacco cessation, stress management, adherence to diet, and exercise), clinical (functional capacity, lipids, MET levels, weight, blood pressure, and heart rate), health (quality of life and loss of work days), and service (patient satisfaction, access and utilization of service, and financial and economic concerns). Even though cardiac rehabilitation programs have been shown to reduce coronary events, more data is needed over a longer period of time to understand and explain their effectiveness.

Framingham Risk Score & Framingham Heart Study

The Framingham risk score is a well-accepted tool in identifying the risk for the development of coronary heart disease. The Framingham risk score is used to evaluate an individual's risk before a coronary event, and assess risk after the cardiac rehabilitation

intervention. Aspects of the Framingham risk score are age, gender, blood pressure, total cholesterol, LDL-C, HDL-C, smoking, and diabetes. These factors are important in assessing an individual's risk for coronary heart disease as they give a physician an idea of what is occurring in the body. Abnormal or high cholesterol values increase the risk for developing atherosclerosis in the vessels of the heart. High blood pressure values increase stress on the heart and can be caused by atherosclerosis. Smoking has been shown to cause irritation in the vessels increasing the presence of plaque that leads to atherosclerosis.

The Framingham Heart Study was a longitudinal study that gave great insight into a variety of risk factors that lead to the development of coronary heart disease. The risk factors that have been identified by the researchers in the Framingham heart study are blood pressure (systolic and diastolic), cholesterol (HDL-C, LDL-C, and total cholesterol); in addition, age and gender are important in identifying cardiovascular disease risk over a 10-year period. The Framingham risk score evaluates each of the values with equal importance and each sub-value is assigned a score based on the distance away from "healthy" values. Since cardiac rehabilitation programs aim at reducing the incidence of coronary heart disease, the Framingham risk score is considered to be a reliable tool in evaluating cardiac rehabilitation programs.

Statement of the Problem

Cardiac rehabilitation programs are intended to help an individual after a cardiac event (i.e., myocardial infarction, heart transplant, valve replacement or repair, coronary artery bypass graft, percutaneous transluminal coronary angioplasty or percutaneous transluminal coronary intervention). Much of the research done validating cardiac

rehabilitation programs focuses on the graduation rate and occurrence of another cardiac event. The Framingham risk score appears to be a good instrument, but there may be another instrument or a tool may need to be developed to better evaluate cardiac rehabilitation programs.

Purpose

The purpose of this study is to examine the effectiveness of a cardiac rehabilitation program to modify the patient's Framingham risk score after 36 sessions of cardiac rehabilitation. This research looked at data that has been acquired from a cardiac rehabilitation program and evaluated the program by using the Framingham risk score. This study contributed by identifying whether the current time frame of 12 weeks or 36 cardiac rehabilitation sessions of exercise is adequate.

Primary Research Questions

1. Is there a difference between the baseline and post data for the Framingham risk score, blood pressure (systolic and diastolic), cholesterol (HDL, LDL, and total cholesterol)?

Rationale

In current research exercise, proper nutrition, and smoking cessation have been shown to reduce an individual's risk for developing cardiovascular disease. AACVPR guidelines require cardiac rehabilitation programs to include exercise, nutrition and smoking

cessation. The Framingham risk score is a valid tool developed by National Institutes of Health to identify those who are at risk for developing cardiovascular disease.

2. Do the changes in the Framingham risk score's differ by age or gender?

Rationale

Research has shown that gender plays a role in the development of coronary heart disease (Silander, K. et al., 2008). Females have protective hormones, such as estrogen, that assist in the prevention of coronary heart disease.

Secondary Research Questions

3. Are there gender differences in the mean scores of the variables that make up the Framingham risk score?

Rationale

Research has shown that gender plays a role in the development of coronary heart disease (Silander, K. et al., 2008).

4. Are there age differences in the mean scores of the variables that make up the Framingham risk score?

Rationale

The relationship of age to the mean scores or the variable may shed some light on whether there is a difference in age and the variables.

5. Does gender interact with age for the Framingham risk score?

Rationale

The interaction of age and gender are important in identifying whether the belief that females have a reduced risk for the development of coronary heart disease at younger ages is true.

Assumptions

The following assumptions were made in this study:

1. The subjects were compliant with the cardiac rehabilitation program
2. The data collection was accurate.

Limitations

1. The Framingham risk score is intended to identify individuals who are at risk for developing cardiovascular disease, not those who are diagnosed with cardiovascular disease.
2. The Framingham risk score gives two different percentages for each individual; one percentage uses the total cholesterol values and the other uses the LDL-C value. Current research has shown that LDL values may show a better picture of an individual's future cardiac risk.
3. There are unequal numbers of men and women participants in the data acquired for this study.
4. There was a variety of staff that assisted the subjects from the original walk test through the 36 exercise sessions to the final walk test. As well as over the course of three years the staff had changed.
5. The lab values that were used came from different laboratories.
6. There was a lack of control for the principal investigator and the progression of exercise through out the program.
7. Subjects could attend cardiac rehabilitation anytime between 8am to 12pm and 1pm to 5pm, Monday, Wednesday and Friday. There was no time limit to complete their exercise except for lunch and closing.

List of Terms

Atherosclerosis: A process of progressive thickening and hardening of the walls of medium-sized and large arteries as a result of fat deposits on their inner lining.

Cardiovascular disease: the class of diseases that involve the heart or blood vessels

Carotid artery stenosis: a narrowing of the carotid arteries due to atherosclerosis, also known as carotid artery disease

Catecholamines: hormones that are released during times of physical and emotional stress (dopamine, norepinephrine, and epinephrine)

Coronary heart disease: is a narrowing of the small blood vessels that supply blood and oxygen to the heart

Coronary artery disease: the disease state of the arteries of the heart due to the accumulation of plaque

Coronary artery bypass graft: a surgical procedure performed to relieve angina and reduce the risk of death; arteries or veins from elsewhere in the patient's body are grafted to the coronary arteries to improve the blood supply to the coronary circulation

Cholesterol: a lipid, waxy steroid found in the cell membranes and transported in the blood plasma of all animals

Cardiac rehabilitation program: is a multi-disciplinary program for individuals who have cardiac disease to reduce reoccurrence of a cardiac event

Framingham Heart Study: a 1948 cardiovascular study to identify factors that increase an individual's risk for developing cardiovascular disease

Framingham risk score: an assessment tool that comprises of factors that the Framingham heart study found to be predictors for developing cardiovascular disease

Myocardial infarction: i.e. heart attack that occurs when the blood supply to the heart is disrupted

Occlusion: a blockage in the arteries or veins of the heart Stent: a man-made device used to reopen vessels of the heart

List of Abbreviations

AACVPR: American Association of Cardiac and Pulmonary Rehabilitation

ACC: American College of Cardiology

ACSM: American College of Sports Medicine

AHA: American Heart Association

CABG: coronary artery bypass graft

CDC: Centers for Disease Control

CHD: coronary heart disease

CVD: cardiovascular disease

HDL-C: high-density lipoproteins/ cholesterol

LDL-C: low-density lipoproteins/ cholesterol

MI: myocardial infarction

NO: nitric oxide

PTCA: percutaneous transluminal coronary angioplasty

PTCI: percutaneous transluminal coronary intervention

WHO: World Health Organization

Chapter II

Literature Review

Coronary heart disease is the leading killer in both men and women in the United States (CDC, 2007). In coronary heart disease (CHD) there is a buildup of atherosclerotic plaque in the vessels and arteries in the cardiovascular system. Coronary heart disease is a progressive disease that is caused by a combination of high blood pressure, a diet rich in fat that causes plaque to build up in the veins and arteries of the heart and periphery, and insufficient quality physical activity (exercise) (American Heart Association (AHA), 2008). The diagnosed forms of coronary heart disease are high blood pressure, myocardial infarction and angina, stroke and heart failure (AHA, 2008). Coronary heart disease is preventable through necessary lifestyle changes including diet, exercise, smoking cessation, and diabetes management (Framingham Heart Study, 2008).

Etiology of CHD

The mechanism behind coronary heart disease is called atherosclerosis, the buildup of plaque in the walls of the coronary arteries that are formed from fat, cholesterol, calcium and other substances found in the blood (AHA, 2008). A secondary response to the atherogenic factors leads to the development of a chronic inflammatory response in the walls of the arteries. There is an accumulation of macrophages in the wall of the arteries, caused by the build-up of low-density lipoproteins in the circulatory system. The hardening of the artery is due to atherosclerosis that causes an increased shear stress on the arteries. This shear stress on the smooth muscle cells of the arteries activates the release of nitric oxide (NO) from the endothelial cells causing vasodilation. However, the response to vasodilatation is restricted due to the atherosclerotic plaques

that have formed. In atherosclerosis there is an inability for the arteries to properly relax due to the formation of plaque, which can cause high blood pressure. Atherosclerosis can lead to the development of angina pectoris.

Angina pectoris is caused when the metabolic demand of the tissue exceeds the ability of the compromised blood supply to meet those needs (Boron et al., 2009). There are many things that can mimic angina pectoris within the body, such as acid reflux. One way to diagnose angina is through an exercise stress test. There are a few patients who experience no angina pectoris but can still have a silent ischemia. Common presentations of angina include chest pain and discomfort, pressure, squeezing in the chest or feelings of a heavy weight placed on the chest. Pressure can extend down the arm (left), neck, jaw, shoulder or back (Mayo Clinic, 2009). There are different forms of angina: stable, unstable and variant. Stable angina is exhibited when the heart works harder, for example exertion, (duration is 5 minutes or less) and pain disappears at rest or with the use of angina medication (Mayo Clinic, 2009). Unstable angina occurs even at rest, is unexpected, lasts longer than stable angina and may not disappear with rest or use of angina medication (Mayo Clinic, 2009). Variant angina usually happens when the heart is resting, often is severe and may be relieved by angina medication (Mayo Clinic, 2009). Angina in women can be different from common presentations. For instance, chest pain may feel more stabbing, pulsating or sharp pain in the chest; nausea and abdominal pain may be present (Mayo Clinic, 2009).

Treatment for individuals with stable angina is medication. Nitrates cause the endothelial tissue of the veins and arteries to vasodilate through the release of nitric oxide (Boron et al., 2009). Beta-blockers prevent the sympathetic nervous system from

stimulating β_1 receptors in the myocardium, which in return reduce heart rate, contractility and the metabolic demand (Boron et al., 2009). Calcium channel blockers reduce contractility in the heart muscles and vascular smooth muscle (Boron et al., 2009). If medication does not control angina and if the frequency and the severity increases, medical interventions are needed such as a percutaneous transluminal coronary intervention (PTCI) or percutaneous transluminal coronary angioplasty (PTCA). If the vessel is fully occluded and a PTCI or PTCA cannot be placed in a coronary artery, then a coronary artery bypass graft (CABG) may need to be performed. Even after medical or surgical interventions, it is recommended that all individuals carry nitroglycerin with them to prevent further complications related to the build-up of plaque.

Development of the Framingham Risk Score

The original Framingham risk score study was developed after World War II to identify the causes behind individuals developing cardiovascular disease (Framingham Heart Study, 2008). Cardiovascular disease was the foremost illness of the time since infectious disease had been brought under control (Framingham Heart Study, 2008). By 1950, one out of every three men in the United States developed CVD before reaching the age-sixty (Framingham Heart Study, 2008).

The subjects were recruited from Framingham, Massachusetts from a cohort of the inhabitants. The original cohort was 5,209 men and women between the ages of 30 and 62; all subjects completed questionnaires and extensive physical exams every two years since 1948. In 1971, the Framingham Study recruited 5,124 second-generation children and spouses, of the original cohort. Due to the careful monitoring of the Framingham subjects, important information was uncovered about cardiovascular disease

and the risk factors for developing cardiovascular disease. Risk factors for cardiovascular disease include high blood pressure, high blood cholesterol, smoking, obesity, diabetes and physical inactivity.

The data from the research showed a great deal of valuable information on the effects of related factors such as blood triglyceride and HDL cholesterol levels, age, gender, and psychosocial issues. Although the Framingham cohort was primarily Caucasian, the importance of the major cardiovascular disease (CVD) risk factors identified in this group have been shown in other studies to apply almost universally among racial and ethnic groups, even though the patterns of distribution may vary from group to group (Framingham Heart Study, 2008).

Aims of the Framingham Study are as follows (Dawber and Moore, 1952):

- * First, to secure epidemiological data on atherosclerotic and hypertensive CVD.
- * Second, to secure data on the prevalence of all forms of CVD in a representative population sample.
- * Third, to test the efficiency of various diagnostic procedures.

The first aim was major. The other two were viewed as secondary interests.

Hypotheses for Framingham Heart Study

It was assumed from the start that the CVD did not have a single cause. Rather, it was the result of multiple causes, which worked slowly on the individual to produce the disease.

Factors that were potentially related to CVD were listed in terms of the hypotheses.

These were generated by the Framingham investigators in consultation with an advisory committee composed of specialists representing several branches of the medical sciences.

The medical history and physical examination that would be obtained on the study

subjects would generate data to test these hypotheses. The major hypotheses generated were (Dawber, 1980):

1. CVD increases with age. It occurs earlier and more frequently in males.
2. Persons with hypertension developed CVD at a greater rate than those who are not hypertensive.
3. Elevated blood cholesterol level is associated with an increased risk of CVD.
4. Tobacco smoking is associated with an increased occurrence of CVD.
5. Habitual use of alcohol is associated with increased incidence of CVD.
6. Increased physical activity is associated with a decrease in the development of CVD.
7. An increase in thyroid function is associated with a decrease in the development of CVD.
8. A high blood hemoglobin or hematocrit level is associated with an increased rate of the development of CVD.
9. An increase in body weight predisposes a person to CVD.
10. There is an increased rate of the development of CVD in people with diabetes mellitus.
11. There is higher incidence of CVD in people with gout.

These hypotheses were used directly to determine the medical history obtained and the physical examination taken during the repeated exams of the study. Additional items were tested as the research progressed, including fibrinogen, apolipoproteins (HDL, LDL) echocardiograms and exercise echocardiograms.

The Framingham risk score is an inexpensive tool used in the medical community to assist in the diagnosis of individuals who are at risk for developing CHD. It allows

physicians to identify those who need to be examined further. The risk score is determined by a variety of factors – systolic blood pressure, diastolic blood pressure, total cholesterol, LDL-C, HDL-C, smoking status, diabetic status, age and gender. Each of the factors are assigned different numerical values that are added together to identify the risk as a percentage for the development of coronary heart disease in the next ten years.

Use of the Framingham Risk Score

Taylor, et al. (2000) investigated 79 subjects that experienced sudden death associated with coronary heart disease. The researchers looked at the plaque development in the subjects' arteries through coronary segments and noticed that there were stable plaque, plaque ruptures and erosions present. The researchers compared the evidence of coronary heart disease (coronary segments) to the Framingham risk score to see if the Framingham risk score accurately captured the subjects' risk for coronary heart disease. The study showed that there was significant disagreement between the coronary segments and the Framingham risk score. The Framingham risk score seemed to underestimate risk associated with atherosclerosis. In the Taylor study there were some limitations, including a small sample size, and the mean age of subjects were 49 years, a generalization to a larger population is difficult. The researchers investigated the relationship between the Framingham risk score and coronary calcification and sudden death. They measured age, gender, height, weight, total cholesterol, HDL-C, tobacco use, and hypertension. The subject's Framingham risk score was evaluated after death. There was a modest but significant relationship between the calcification and Framingham risk score. This study found that the highest Framingham risk scores in cases of sudden death were due to plaque rupture, which is consistent with data linking hypercholesterolemia

and smoking with a coronary event. This study did have some limitations. Since the all 79 patients died due to a coronary thrombosis, it cannot be applied to those that survived. The research confirmed a relationship between coronary calcification and Framingham risk scores. The researchers also stated that this study did underestimate the Framingham risk score in the subjects.

Richardson et al. (2008) assessed whether a community-based intervention for men and women aged between 45-64 years without pre-existing coronary heart disease condition prevented the development of coronary heart disease. The subjects were chosen from an area that has a high level of social deprivation and standardized mortality rate of CHD. This data looked at weight, body mass index (BMI), and waist circumference, as well as behavior (i.e., smoking, alcohol, fruit and vegetable consumption and exercise). Richardson and colleagues investigated weight, BMI, and waist circumference pre and post intervention (exercise guidelines, smoking cessation services and a dietician) and noticed that at the one-year follow-up weight, BMI, and waist circumference had worsened. However pulse, systolic blood pressure, total cholesterol, HDL-C, and glucose had improved. There were also documented behavioral changes including a decrease in smoking, alcohol consumption and an increase in exercise, and fruit and vegetable intake. This research showed that there could be either an overestimation or underestimation of the Framingham risk score in identifying the development of coronary heart disease.

Vasan et al. (2005) researched adults between the ages of 35-74 with no coronary heart disease to estimate the contributions of borderline and elevated risk factors to the development of coronary heart disease. The researchers used both the Framingham risk score and the Third National Health and Nutrition Examination Survey (NHANES-III).

The five risk factors examined were blood pressure, LDL-C, HDL-C, glucose intolerance, smoking status. Blood glucose is used to identify whether an individual is at risk for developing diabetes, two ways of testing is through obtaining blood when they have eaten recently and thorough fasting, no food or fluids except for water in the last 8-12 hours. These were categorized at optimal, borderline and elevated levels. Additional grouping was done with each of the risk factors into three groups—optimal risk factors, borderline or elevated.

- Characteristics that identify a subject in the optimal risk factors group include: blood pressure value between <120/80mmHg, LDL-C value between <100mg/dL, HDL-C value between >59, blood glucose value between <110mg/dL, fasting blood glucose value between <140mg/dL, and a nonsmoker.
- Characteristics that identify a subject in the borderline group include: blood pressure value between 120/80-139/89mmHg, LDL-C value between 100-159mg/dL, HDL-C value between 40-59mg/dL, impaired glucose intolerance if blood glucose value was between 110-125mg/dL, fasting blood glucose value was between 140-199mg/dL, and a former smoker (unidentified when subject quit).
- Characteristics that identify a subject in the elevated levels group include: blood pressure value between >140/90mmHg, LDL-C value between >159mg/dL, HDL-C value between <40mg/dL, blood glucose value between >125mg/dL, fasting blood glucose value between >199mg/dL and a current smoker.

The results showed that two-thirds of men and a one-third of women between the ages of 35-44 years of age had elevated modifiable risk factors, with a low prevalence of optimal risk factors in the United States. This research showed that currently in the

United States those who fall in the elevated risk factors group can be modify their lipid values, blood pressure and blood glucose values through diet and exercise.

Domains of cardiac rehabilitation programs

Smoking and cardiovascular disease

Since the 1960s and 1970s, there have been important studies that have contributed to the understanding of what smoking can do to the body and it's relation to variety of diseases including cardiovascular disease. There is an increased risk for smokers versus nonsmokers for the development of coronary heart disease. The Multiple Risk Factor Intervention Trial (MRFIT) researched the effects of smoking and the development of CHD (Kuller et al., 1991, Shaten et al., 1991, Ockene et al., 1990). Results showed a clear dose response to cigarette smoke and CHD and mortality, an increased risk among diabetics who smoke versus nonsmoking diabetics, an additive effect of smoking, high cholesterol levels in total cholesterol and LDL-C and elevated blood pressure on CHD mortality (Kuller et al., 1991, Shaten et al., 1991, Ockene et al., 1990). Shaten et al. (1991) evaluated the effect of HDL-C, LDL-C, blood pressure and cigarette smoke as predictors for CHD. The nonsmoking subjects included had similar blood pressure or serum total cholesterol compared to the smoking subjects. Smoking status was self-reported. Former smokers who quit more than 12 months before the study and nonsmokers had similar rates of CHD deaths (Shaten et al.). Smokers had an increased rate of death due to CHD when compared to nonsmokers. LDL-C levels were elevated among nonsmokers and when the two groups were compared they had the same LDL levels, however smokers had a higher rate of death due to CHD (Shaten et al.).

Benfante et al. (1991) stated that cigarette smoke is a major risk factor for CHD in middle-aged populations; older populations had a diminished effect of cigarette smoke. To investigate this claim, subjects between the ages of 65 to 74 years with no diagnosed cardiovascular disease were divided into three smoking status groups (current, former and never). Current elderly smokers smoked an average of 20 cigarettes per day compared to 24 cigarettes per day by middle-aged smokers (age 51-59 years). The elderly smoked on average 10 years longer (Benfante et al.). The relative risk for cigarette smoke and CHD were similar between the elderly and middle-aged groups. There was an increase in overall risk in the elderly for the development of CHD since the risk for CHD complications increase with age (Benfante et al.).

Powell (1998) investigated the vascular damage from smoking. The products that were combusted and absorbed into the systemic circulation caused damage to the arterial walls through a variety of ways. The absorbed nicotine activated the release of catecholamines (hormones that are released during times of physical and emotional stress) while other products from the smoking injured the endothelium and promoted atherogenesis. Smoking also increased the release of LDL-C, which adhered to the artery walls causing the development of atherosclerosis (Powell). Smoking appeared to affect the body on a very direct level by impairing the body's response to the inflammation.

Nutrition and cardiovascular disease

Nutrition is an important part of preventing CHD, improving overall health and is an important part of cardiac rehabilitation programs. Currently, the average American diet included large amounts of salt, fat and very little fruit and vegetables (CDC, 2008).

Researchers have investigated a variety of diets that have shown a relationship with the development of atherosclerosis and CVD.

Millen et al. (2005) investigated a variety of factors, which included nutritional intake, carotid ultrasound, lipid analysis, BMI, diabetes status, physical activity, and hypertension status to identify the cause of the development of atherosclerosis. The research subjects were from the Framingham heart study first subject pool, the offspring cohort and the offspring-spouse cohort. The subjects were followed and evaluated every four years for the development of carotid artery stenosis. The daily intake that was recommended $\leq 30\%$ of total energy as fat, $<10\%$ of energy as saturated fat and $<300\text{mg}$ of cholesterol to lower risk for developing coronary artery disease. The results showed that smoking and dietary noncompliance doubled the odds for having carotid stenosis compared to those with dietary compliance. Research also showed that individuals who had carotid stenosis had a diet that was higher in total fat, saturated fat, increased sodium intake, with lower consumption of fiber and a lower consumption of vitamins and minerals. The subject groups, those with stenosis and those did not develop stenosis, had low levels of physical activity. This study looked at compliance and noncompliance for healthy nutrition and an additional grouping of smoking and nonsmoking subjects. The results showed that a combination of a poor diet and smoking is associated with the largest increase in atherosclerosis.

A diet that includes a high consumption of saturated and trans fats, salt, refined carbohydrates and low levels of fruit and vegetables are associated with an increased risk of CHD (Woodside et al., 2008). Diets high in saturated fatty acids and trans fatty acids have been associated with CHD (Woodside et al.). Saturated fats increased LDL-C and

total cholesterol whereas polyunsaturated fats decreased LDL-C and total cholesterol. Within the grouping of saturated fats, there are a few that have little effect on plasma cholesterol levels (i.e. stearic acid since it gets converted to oleic acid) (Woodside et al.). Simply lowering fatty acid intake may not reduce CHD incidence or lower lipid profile. However optimizing the fatty acid composition of the diet could have major benefits (Woodside et al.). Replacing saturated fatty acids and trans fatty acids with unsaturated fats and an increased consumption of omega-3 fatty acids, a diet high in fruits vegetables and whole grains improved CHD risk (Woodside et al.).

Obesity and Coronary Heart Disease

Increased adipose tissue especially around the waist causes an increase of adipokines and hormones increases an individual's risk for CHD. Research had shown that increased amounts of adipose tissue (especially visceral) affect the body at a cellular level (Choi et. al., 2004). Adipose tissue is an active organ that secretes various hormones and metabolites that are believed to regulate energy metabolism and insulin sensitivity (Choi, et. al.). Adiponectin, a protein that is released by adipose tissue, had shown to be important in metabolic and vascular diseases. Adiponectin increased morbidity, C-reactive proteins (CrP), IL-6 (Interleukin 6) and levels of TNF- α (Tissue Necrosis Factor-alpha) (Moller, 2000). Adipose tissue released TNF- α and IL-6 into the circulation system and both molecules modified insulin sensitivity by interfering with the intracellular insulin-signaling pathway (Moller, 2000). IL-6 initiated the release of CrP from the liver to the circulation system and within the circulation system IL-6 that caused the release of IL-1 (Interleukin-1). CrP is produced in the liver and is a reliable marker

for systemic inflammation (Lu et al., 2004). IGFBP-1 has been related to poor glucose control and increased mortality (Anselmino et al., 2008).

Diabetes and CHD

Diabetes affected 20.8 million individuals in America and approximately 90-95 percent of those diagnosed have type 2 diabetes (ADA, 2008). Insulin resistance, similar to type 2 diabetes, was found in 25% of the general population (Johnstone, 2005). Eighty percent of individuals with type 2 diabetes are overweight. Diabetes is more prevalent among African-Americans and Hispanics populations in the United States (CDC, 2008).

Diabetes is a disease that affects metabolism. Once food enters the body, it begins to break down. Carbohydrates are digested to form molecules of glucose, which the body can store or use for energy. High levels of glucose cause the release of the hormone insulin from the β islet cells of the pancreas. In individuals with type 2 diabetes, either there is a deficiency of insulin released or the cells ignore insulin (ADA, 2009). Since there is an inadequate response to the β islet cells to glucose followed by a decreased response of the peripheral tissues to insulin, this could eventually lead to a reduction of β cells (ADA, 2009). Type 2 diabetes has led to an increased amount of glucose in the bloodstream and impaired response to removing the glucose. Increased amounts of glucose can cause systemic inflammation and damage to tissue and organs, causing neuropathy, loss of eyesight, inability to repair damage to the endothelial tissue, and eventually death (ADA, 2009).

Diabetes is considered a risk factor for the development of atherosclerosis and eventual diagnosis of CVD. A hypothesis for the development of atherosclerosis in diabetic individuals was that there are chronic low levels of inflammation (Wilund,

2007). Research had shown that exercise modified CVD risk and type 2 diabetes by reducing systemic inflammation (Wilund, 2007). In patients that are at risk for developing diabetes are those that are classified as obese, there was an increase risk for cardiovascular disease. Obesity and lack of exercise had a causal link to the development of diabetes. In diabetic individuals, exercise improved insulin sensitivity and reduced the amount of glucose in the blood stream since the muscle requires the energy. The mechanisms for developing diabetes are unclear and not fully understood (Wilund, 2007).

There was a direct correlation of the risk of cardiovascular mortality and increased levels of plasma glucose concentration and HbA1c levels (Bianchi et al., 2008). In individuals with diabetes, there was an increase in basal heart rate and cardiac output, as well as an abnormal heart function due to autonomic neuropathy (Johnstone, 2005). Insulin resistant individuals release higher levels of insulin for the same glucose uptake exhibited in healthy individuals. Insulin levels approximately 100 μ U/mL showed increased heart rate in obese individuals, compared to healthy individuals insulin levels (approximately 70 μ U/mL) (Johnstone, 2005).

The San Antonio Heart Study is the most comprehensive study to date that has looked at multiple factors that can lead to cardiovascular disease (Bianchi et al., 2008). One component of the study examined the role diet plays in various forms of cardiovascular disease. San Antonio Heart Study subjects who did not have diabetes at baseline, but developed type 2 diabetes by the 8-year follow-up had higher total cholesterol and LDL values than subjects who did not develop type 2 diabetes (Bianchi et al.). This study showed there was an increase in atherogenic factors in diabetic individuals. Atherogenic factors are lipid oxidation products, such as cholesterol

oxidation products, trans-fatty acids and some saturated fatty acids (Addis, 1995).

Diabetes increases the risk of having cardiovascular disease factors, including uncontrolled cholesterol, LDL, and saturated fats.

Another physiological factor that affects the arteries and vessels in individuals with diabetes is vasoconstriction due to lack of insulin response. In type 2 diabetes and hypertensive patients, there is a decrease in NO production (Johnstone, 2005). The mechanisms of insulin-mediated vasodilation in type 2 diabetes or obesity are unknown. Nitric oxide is released from the endothelial cells and activates cyclic GMP (cGMP), causing smooth muscle relaxation. Nitric oxide has various properties besides a vasodilator response, including antiatherogenic properties.

Diabetes therapy included a diabetic diet, medications, weight loss and cardiovascular exercise. When an individual was first diagnosed with diabetes, the physician encouraged lifestyle changes to prevent uncontrolled type 2 diabetes (ADA, 2009). These changes included talking to a diabetic consultant or a registered dietitian that developed a proper meal plan and began an exercise program, if not currently participating in one (ADA, 2009). Prescribing insulin or secretagogue (insulin-secreting) medications are postponed due the effects they have on the body and the difficulty they cause with losing weight (ADA, 2009).

Individuals with type 2 diabetes are a special concern in cardiac rehabilitation programs since exercise increases glucose uptake (American Dietetics Association, 2008). Several factors in individuals with diabetes complicated nutrition recommendations since blood glucose levels must be regulated. According to the ADA, there had not been much research on individuals with type 2 diabetes that examined the

amount needed for carbohydrate, fluids and calories to perform during an exercise session. Nutrition guidelines that were developed are based on studies in individuals who are non-diabetic. Carbohydrate intake before, during and after exercise is important to ensure that blood glucose does not get low. In type 2 diabetics, weight loss through diet and exercise is important in treatment and possible prevention in borderline diabetics.

Exercise benefited patients with diabetes management by helping with glycemic control and weight reduction. It has also been shown to reduce HbA1c, which decreased the risk of diabetic complications (Boule et al., 2001). Exercise also improved muscle and liver insulin sensitivity and muscle glucose uptake and utilization. Improvements in insulin sensitivity are beneficial in type 2 diabetics. Consistent exercise caused hypoglycemia in individuals taking insulin or insulin secretagogues. To prevent hypoglycemia and other health complications associated with hypoglycemia, patient medication may need to be adjusted entirely (Hayes et al., 2007). Even though exercise has shown to be beneficial, it is still an underutilized therapeutic intervention in diabetes management (AADE, 2007). In individuals with cardiovascular disease and diabetes, exercise has been shown to reduce mortality and the progression of these diseases. The exercise program needed to be monitored by the individual's physician or exercise professional to ensure that the individual is not harmed. Lifestyle changes are an important factor in controlling and possibly reversing both diseases. Individuals diagnosed with borderline type 2 diabetes and begin an exercise routine and make other necessary lifestyle changes can slow the progression of the disease (Lavie, 1993).

Exercise and cardiovascular disease

Exercise has been shown to reduce the deleterious effects of cardiovascular disease. It is an important lifestyle change that should occur along with proper nutrition. Cardiovascular disease can lead to an increase in inflammatory responses associated with atherosclerosis and chronic inflammation (Wilund et al., 2007). Researchers have investigated whether exercise can improve the effects of chronic inflammation. Acute exercise has been shown to increase circulating levels of anti-inflammatory cytokines (Wilund et al.). IL-6, TNF- α and CRP are strong independent risk factors of CVD (Wilund et al.). IL-6 is secreted by a variety of cell types in response to IL-1 and TNF- α . IL-6 is believed to be a traditional pro-inflammatory cytokine that increases CrP production and is associated with unstable angina and increases the risk of future MI (Wilund et al.). Exercise can increase the amounts of reactive oxygen species (ROS), which are free radicals that are released by reactions to produce energy for exercise. However it is unclear if this oxidative stress is associated with CVD risk (Wilund et al.). Increased levels of IL-6 released during acute exercise have been shown to stimulate the reaction of anti-inflammatory cytokines – IL-1ra, sTNFR and IL-10 (Wilund et al.).

Kohl (2001) examined five observational studies (eight publications) to evaluate the dose response relationship between physical activity and the risk for CVD. Two of the eight publications in the review showed no evidence for a dose response relationship between CVD and physical activity. One study reported a mixed response between dose response physical activity and CVD. The remaining studies observed a causal relationship with credible evidence for a dose-response physical activity. Two out of the five studies that observed a causal relationship were also the most recent studies, and the subjects included elderly and women. A limitation of these studies is that CVD

encompasses a variety of diseases and illnesses. The sample sizes of the research examined varied in size from 36 subjects to over 4000 subjects. The exercise that the subjects participated in was undisclosed. However, they were categorized by active or inactive in their jobs, leisure activity and if active how much time was spent.

Besides investigating CVD and physical activity, the relationship between CHD and physical activity has been researched. Kohl's (2001) literature review encompasses approximately 50 years' worth of research. In thirty-one articles that were reviewed, twenty articles provided support the argument that dose related response of physical activity and its relationship to CHD, three studies showed mixed results and eight showed no relationship between dose response physical activity and CHD (Kohl, 2001). Some of the results showed a "U"-shaped relationship with higher relative risk of CHD at the highest physical activity levels (Kohl, 2001). There was an inverse relationship between exercise and blood pressure, clotting factors, glucose tolerance, and smoking habits (all of these factors are associated with the an increased risk of CVD).

Physical activity reduces the likelihood of developing cardiovascular disease (CVD), controls blood pressure and cholesterol (if a healthy diet is also maintained) (Haskell, 2007). Guidelines for healthy individuals over the age of 65 and adults 50-64 years with chronic conditions should participate in moderate aerobic physical exercise for 30 minutes, five days a week or vigorous aerobic exercise for 20 minutes, three days a week, according the position paper from AHA and ACSM (Thomas, et al., 2007). The position paper of AACVPR, American College of Cardiology (ACC), and AHA indicates that adherence to physical activity has been associated with a 20%-30% reduction in

mortality in patients with cardiovascular disease (Thomas, et al). Exercise has been shown to reduce the development and progression of atherosclerosis (Wilund, 2007).

Treatment and Care provided through Cardiac Rehabilitation Programs

Cardiac Rehabilitation programs are designed to assist patients to make necessary lifestyle changes to prevent another cardiac event and return their regular daily activities. In-hospital and post-hospital cardiac rehabilitation programs have been shown to be beneficial in reducing the likelihood of having another event, these programs also increase quality of life. Cardiac rehabilitation programs are shown to be beneficial in reducing the reoccurrence of another coronary event or death (AACVPR, AHA, ACC). However, the exact reasons the programs are beneficial are unknown. In the past, cardiac rehabilitation programs focused primarily on cardiovascular exercise to reduce the incidence of another coronary event. Cardiac rehabilitation programs have expanded on that aspect of treatment and now include education to provide patients with a multifaceted program. Cardiac rehabilitation programs reduce the risk of another coronary event through education and cardiovascular exercise (Thomas, et al., 2007).

Cardiac rehabilitation programs that maintain data per AACVPR may provide insight regarding whether or not cardiac rehabilitation programs are in fact reducing the risk of another coronary event. AACVPR recommends the major domains of care and education that a cardiac rehabilitation program should have in order to acquire and maintain a certification. Those four domains are behavioral (education, tobacco cessation, stress management, adherence to diet, and exercise), clinical (functional capacity, lipids, METs, weight, blood pressure, and heart rate), health (quality of life and loss of work

days), and service (patient satisfaction, access and utilization of service, and financial and economic concerns).

Summary

Cardiac rehabilitation programs require proper data collection techniques and acquisition that is standard in all programs to be able to compare them. Since the United States government has required all cardiac rehabilitation programs to become a certified program through AACVPR or another government certified organization. The thesis research discussed will look at data acquired from a cardiac rehabilitation program and evaluate the program by using the Framingham risk score. The Framingham risk score is considered a standard form of measurement in diagnosing individuals that are at risk for developing coronary heart disease.

Chapter III

Methodology

This is a retrospective study looking at individuals who have completed a 36-session cardiac rehabilitation program at New Heart Cardiac Rehabilitation Center to see if that program reduced an individual's Framingham risk score. The Framingham risk score identifies risk factors that are known to contribute to the development and reoccurrence of cardiovascular disease. The risk factors in the Framingham risk score are blood pressure (systolic, diastolic), cholesterol (HDL-C, LDL-C, total cholesterol), smoking status and diabetes.

Institutional Review Board

This research has Institutional Review Board approval from the University of New Mexico (Appendix A).

Subjects

Subjects were recruited from a cardiac rehabilitation program in Albuquerque, New Mexico. As this is a retrospective study, data were taken from subject files that were acquired during the period of April 2005 to March 2008. The study included 152 male and 46 female subjects. All subjects signed a waiver that explained the risks of the initial walk test and the cardiac rehabilitation program (Appendix B). The waiver also had a section stating that all of their records may be used for statistical analyses or scientific purposes. All subjects completed a questionnaire that asked about their tobacco use, medication compliance, etc., and all subjects completed a submaximal walk test (Appendix C).

Instrument

The instrument used in this investigation was the Framingham risk score.

Inclusion/Exclusion Criteria

Individuals who had a cardiac event (myocardial infarction, PTCI, PTCA, CABG, valve replacement and repair or a heart transplant), who completed a pre- and post-walk test, (with lab values for both) and completed the 36-session cardiac rehabilitation program at New Heart Cardiac Rehabilitation Center were included in the data analysis.

The exclusion criteria for the study consisted of those individuals that had a secondary disease or illness that inhibited their ability to complete the 36-session cardiac rehabilitation program. Data was not included for persons who could not perform the walk test and/or did not want their data used for this research.

Experimental Protocol

Patient information was retrospectively taken from their files with approval from the Medical Director at New Heart Cardiac Rehabilitation Center. Study approval was granted from the University of New Mexico human subjects review committee (Appendix A). All tests were performed at the New Heart Cardiac Rehabilitation Center by trained staff. After their initial visit with the physician and the walk test, subjects were scheduled for a one-on-one exercise orientation where they were shown the facility and an exercise program was designed for them based on their needs and limitations. All subjects were instructed to wear EKG monitors each time they exercised. Staff was

present to help them with any questions that they may have. The subjects had a 2-week consultation with the behavioral specialist, 4-week appointment with the cardiac rehabilitation program coordinator and 8-week follow up with the physician and a nutrition consultation. The time varied for each subject due to a variety of factors. All were encouraged to exercise up to an hour 3 days per week. If patients were able and staff felt comfortable patients were encouraged to exercise at home. After 36 sessions, all subjects completed a post-program walk test and were evaluated by the physician.

Cardiac Rehabilitation Program

The program started with a new patient orientation where the cardiac rehabilitation program was explained and a tour of the facility was given. All subjects at that time scheduled their walk test and first doctor's appointment. Each subject was given a folder that had the necessary paperwork to be completed: a questionnaire, consent forms for the walk test, their data and the exercise program and other items they needed to bring with them at their next appointment. Following the walk test and the doctor's appointment an exercise orientation was scheduled. The staff reviewed the information in the file and developed an exercise program based on the subject's capabilities and interests. If a patient wanted to participate in a triathlon in six months, the exercise staff assisted them to achieve their goal if possible and in a healthy manner. The patient had three additional appointments with a behavioral specialist, cardiac rehabilitation coordinator, and the physician to assess how they were progressing and recommended changes to their program that assisted with achieving their goals. All subjects meet with a

registered dietician at least once to assess their diet and help them make better food choices.

Retrospective Procedures and Data Analysis

Subjects' data was retrospectively attained from the New Heart Cardiac Rehabilitation Center from April 1, 2005 to December 31, 2008 from patients who entered and completed the cardiac rehabilitation program during that time period. Pre and post values for blood pressure (systolic, diastolic), total cholesterol, HDL-C, and LDL-C, smoking status and diabetes status was collected by the principal investigator and placed on to a spread sheet. The data that was collected did have the subjects name to protect their identity a number was assigned to each subject and the spread sheet that had the subjects named was password protected and stored on external hard drive that only the principal investigator had access to. Using the variables listed above, a Framingham risk score (Appendix D) was calculated and the LDL-C percentage was used. In identifying the blood pressure's numerical value needed for the Framingham, the systolic and diastolic blood pressure has a table that groups the values to get a score however if the systolic or diastolic have a higher value that value was used. The continuous variable age was used to form a categorical variable treated as two factors (≤ 50 and > 50 years old) to be able to evaluate the age range that has a greater risk in comparison to the Framingham risk score.

To evaluate each of the subject's data and form a Framingham risk score for each subject a program was developed using the program Labview (National Instruments, Austin Texas). All the data that was collected during the subjects' cardiac rehabilitation was through a variety of cardiac rehabilitation employees and from questioning staff only

the same treadmill was used for each subject. Delta fat intake and delta MET were obtained by a cardiac rehabilitation employee and put on a spreadsheet. Delta fat intake was the difference between baseline and post values of self recorded fat intake at the current moment. Delta MET was the difference between baseline and post values that were obtained through their walk test, which was MET level they achieved.

Statistical Analysis

This study employed a mixed design repeated measures analysis. Gender and age were the between-subject factors and the pre and post Framingham risk scores were the within-subjects factor. Type 1 family-wise error was controlled with a $p \leq .05$ for each dependent variable. Using SPSS version 16, the data was analyzed using in multi-variant analysis and multiple regression. The dependent variables in this research are delta Framingham risk score, the baseline and 12-week scores of systolic blood pressure, diastolic blood pressure, total cholesterol, LDL-C, HDL-C and Framingham risk score. Independent variables are delta fat, delta MET, age binned, sex and raw age.

Chapter IV

Results

The characteristics of the subject population are listed in Table 1. The total population (N=198) includes women (n=46), and men (n=152).

Table 1

Summary Table

Characteristics (mean, (SD))	Women	Men	Total	
	n=46	n=152	N=198	
Age	68.7 (8)	66.47 (10.3)	67 (9.8)	
Baseline Systolic Blood Pressure	126.6 (17.8)	117.5 (14.4)	119.7 (15.7)	*
Post Systolic Blood Pressure	122.1 (17.6)	116.9 (15.8)	118.2 (16.2)	*
Baseline Diastolic Blood Pressure	76.6 (12)	74.9 (9)	75.4 (9.8)	*
Post Diastolic Blood Pressure	72.7 (7.6)	73.4 (7.9)	73.3 (7.8)	*
Baseline Total Cholesterol	172 (48.7)	149.4 (40.3)	154.5 (43.4)	*
Post Total Cholesterol	150.7 (29)	136.1 (30.1)	139.5 (30.5)	*
Baseline LDL-C	96.5 (43.6)	85.9 (34.9)	88.3 (37.2)	*
Post LDL-C	78.1 (26)	71.4 (24.2)	72.9 (24.8)	*
Baseline HDL-C	46.4 (11.5)	39.5 (12)	41.1 (12.3)	*
Post HDL-C	47.8 (8.8)	42.7 (11)	43.9 (10.7)	*
Baseline Framingham Risk Score	9.1 (4.7)	7.9 (5.1)	8.14 (4.9)	
Post Framingham Risk Score	7 (4.3)	6.1 (3.6)	6.30 (3.8)	
Delta Framingham Risk Score	2.11 (3.27)	2.92 (4.41)	1.84 (3.61)	
Delta MET	-1.08 (1.15)	-2.07 (1.48)	-1.82 (1.47)	
Delta Fat	3.14 (5.68)	2.64 (6.39)	2.45 (6.01)	
Baseline Smoking Status, n	0	11	11	
Post Smoking Status, n	0	3	3	
Diabetes Status, n	6	19	25	

*Identifies a statistically significant *p* value of < 0.05

Framingham Risk Scores

The difference between the baseline Framingham risk score (N=198) and 12-week Framingham risk score (N=198) was statistically significant $F(1,193) = 36.37, p < .001$; partial eta squared = .15. The interaction between age and time was not significant $F(1,193) = .125, p = .725$; partial eta squared = .001. The interaction between time and sex was not statistically significant $F(1,193) = 1.310, p = .254$; partial eta squared = .007.

In the secondary analyses of sex and age differences, the main effect of sex was not statistically significant $F(1, 193) = 1.807, p = .180$; partial eta squared = .009. The main effect of age as a categorical variable was statistically significant $F(1, 193) = 11.472, p = .001$; partial eta squared = .056. The interaction of sex and age was statistically significant $F(1, 193) = 5.579, p = .019$; partial eta squared = .028.

Figure 1. Comparison of baseline and post values for the Framingham risk score

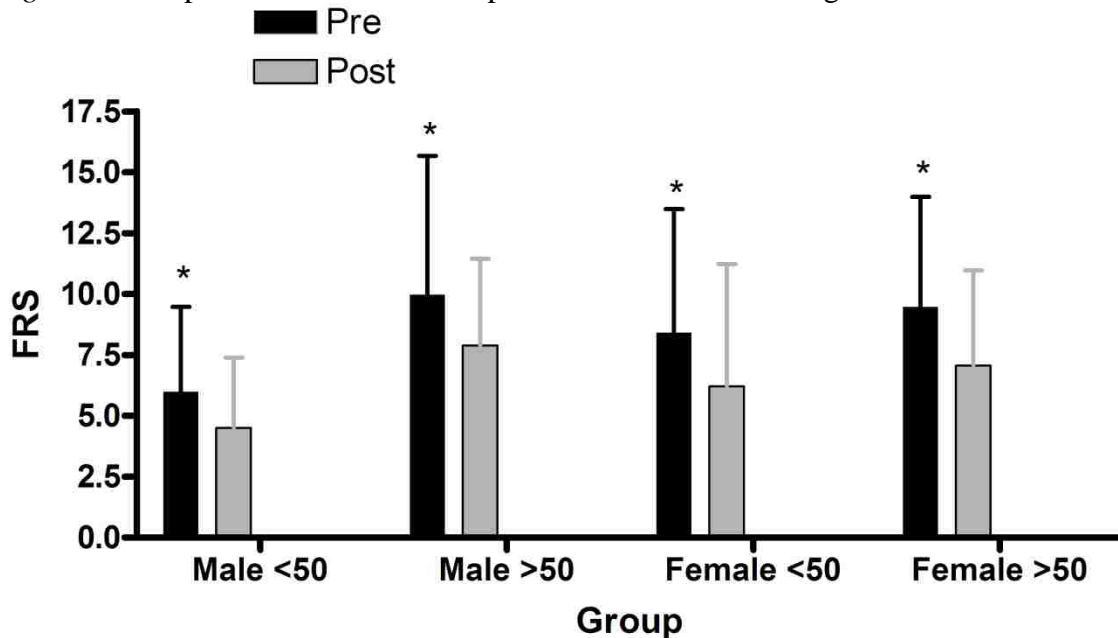


Figure 1. This graph shows that the cardiac rehabilitation program had a statistically significant affect on the Framingham Risk Score and that age and gender had an effect.

Systolic Blood Pressure

The difference between baseline systolic blood pressure (N=198) and 12 week systolic blood pressure (N=198) was statistically significant $F(1,193) = 3.391, p = .049$; and partial eta squared .020. The interaction between age and time was not significant $F(1,193) = .130, p = .719$; partial eta squared = .001. The interaction between time and sex was not statistically significant $F(1,193) = 2.522, p = .114$; partial eta squared = .013.

In the secondary analyses of sex and age differences, the main effect of sex is statistically significant $F(1, 193)= 6.731, p= .010$; partial eta squared = .034. The main

effect of age was not statistically significant $F(1, 193) = 2.831, p = .004$; partial eta squared = .014. The interaction of sex and age was not statistically significant $F(1, 193) = .831, p = .363$; partial eta squared = .004.

Figure 2. Comparison of baseline and post values for systolic blood pressure

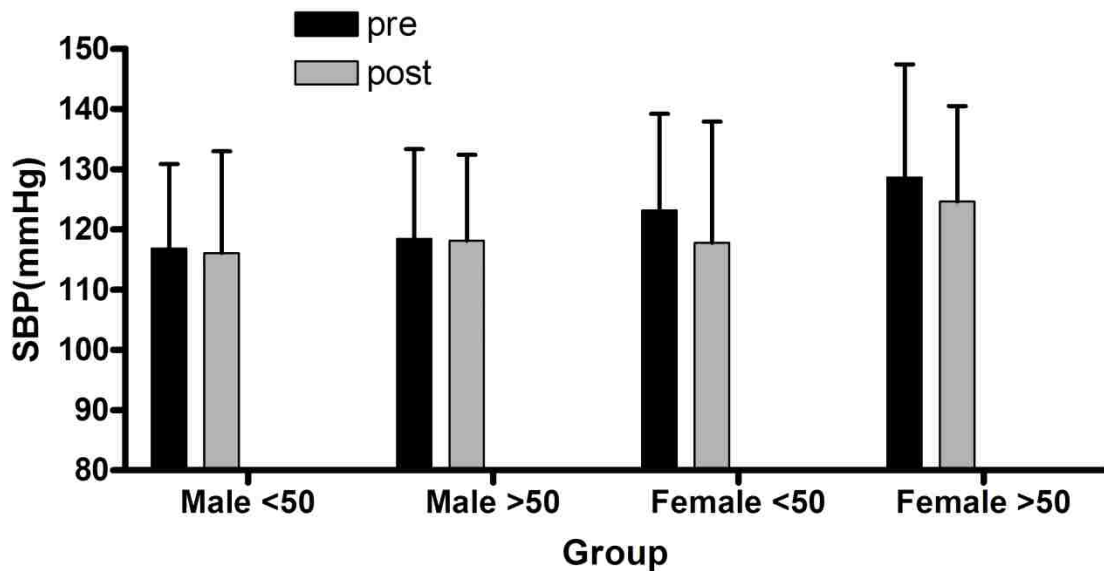


Figure 2. Within the variable there was statistically significant and the interaction of sex. However there was no statistical significance between the interaction of age and time, sex and time, and the interaction of sex and age in relationship to the variable.

Diastolic Blood Pressure

The difference between baseline diastolic blood pressure (N=198) and 12 week diastolic blood pressure (N=198) was statistically significant $F(1,193) = 10.20, p = .002$; partial eta squared = .050. The interaction between age and time was not significant $F(1,193) = .270, p = .604$; partial eta squared = .001. The interaction between time and sex was not statistically significant $F(1,193) = 2.069, p = .152$; partial eta squared = .011.

In the secondary analyses of sex and age differences, the main effect of sex is not statistically significant $F(1, 193) = .492, p = .484$; partial eta squared = .003. The main effect of age was statistically significant $F(1, 193) = 5.685, p = .018$; partial eta squared

= .028. The interaction of sex and age was not statistically significant $F(1, 193) = .047$, $p = .828$; partial eta squared = .000.

Figure 3. Comparison of baseline and post values for diastolic blood pressure

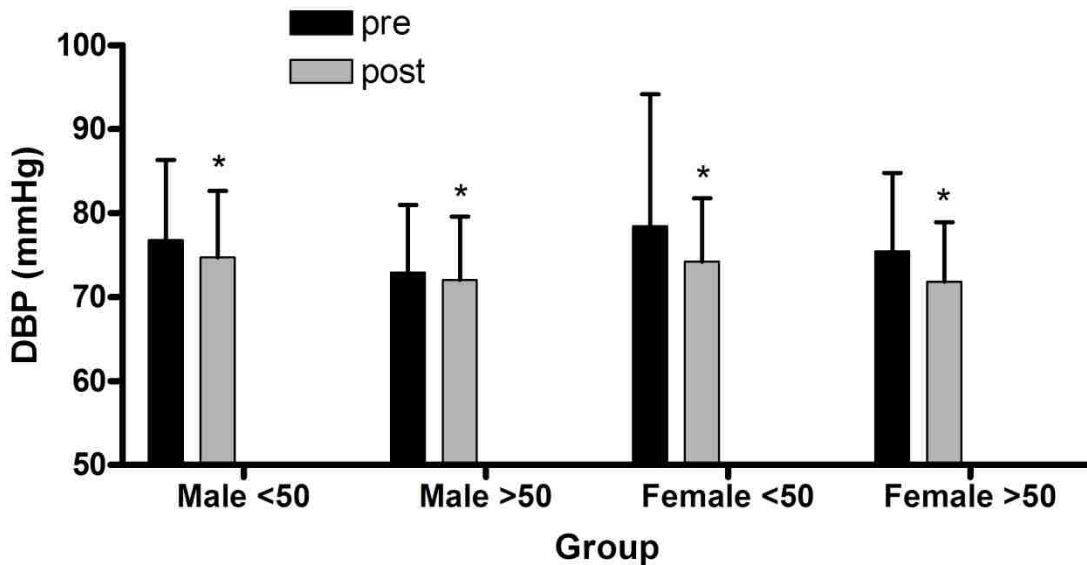


Figure 3. Within the variable there was statistical significance and the main effect of age was statistically significant.

Total Cholesterol

The difference between baseline total cholesterol (N=198) and 12 week total cholesterol (N=198) was statistically significant $F(1,193) = 19.777$, $p = .000$; partial eta squared = .093. The interaction between age and time was not significant $F(1,193) = .010$, $p = .921$; partial eta squared = .000. The interaction between time and sex was not statistically significant $F(1,193) = 1.086$, $p = .299$; partial eta squared = .006.

In the secondary analyses of sex and age differences, the main effect of sex was statistically significant $F(1, 193) = 13.093$, $p = .000$ and partial eta squared = .063. The main effect of age was not statistically significant $F(1, 193) = .328$, $p = .567$; partial eta squared = .002. The interaction of sex and age was not statistically significant $F(1, 193) = .227$, $p = .634$; partial eta squared = .001.

Figure 4. Comparison of baseline and post values for total cholesterol

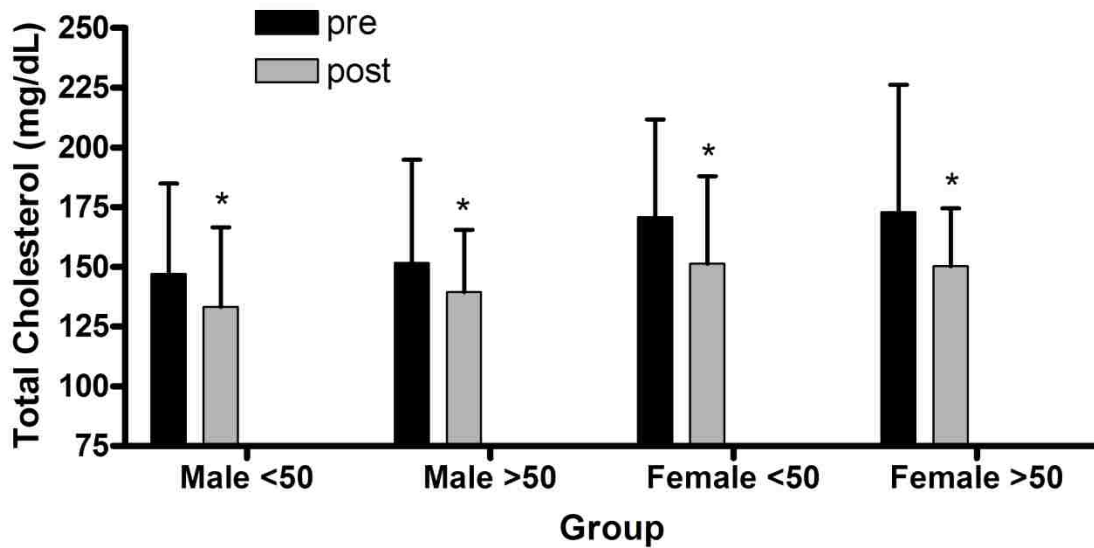


Figure 4. This graph shows that the cardiac rehabilitation program had an effect on the total cholesterol values. There was a main effect of sex on the variable but not on time or age.

LDL-C

The difference between baseline LDL-C (N=198) and 12 week LDL-C (N=198) was statistically significant $F(1,193) = 23.277, p = .000$; partial eta squared = .107. The interaction between age and time was not significant $F(1,193) = .300, p = .585$; partial eta squared = .002. The interaction between time and sex was not statistically significant $F(1,193) = .239, p = .626$; partial eta squared = .001.

In the secondary analyses of sex and age differences, the main effect of sex was not statistically significant $F(1, 193) = 3.809, p = .052$; partial eta squared = .019. The main effect of age was not statistically significant $F(1, 193) = .285, p = .594$; partial eta squared = .001. The interaction of sex and age was not statistically significant $F(1, 193) = .053, p = .819$; partial eta squared = .000.

Figure 5. Comparison of baseline and post values for LDL-C

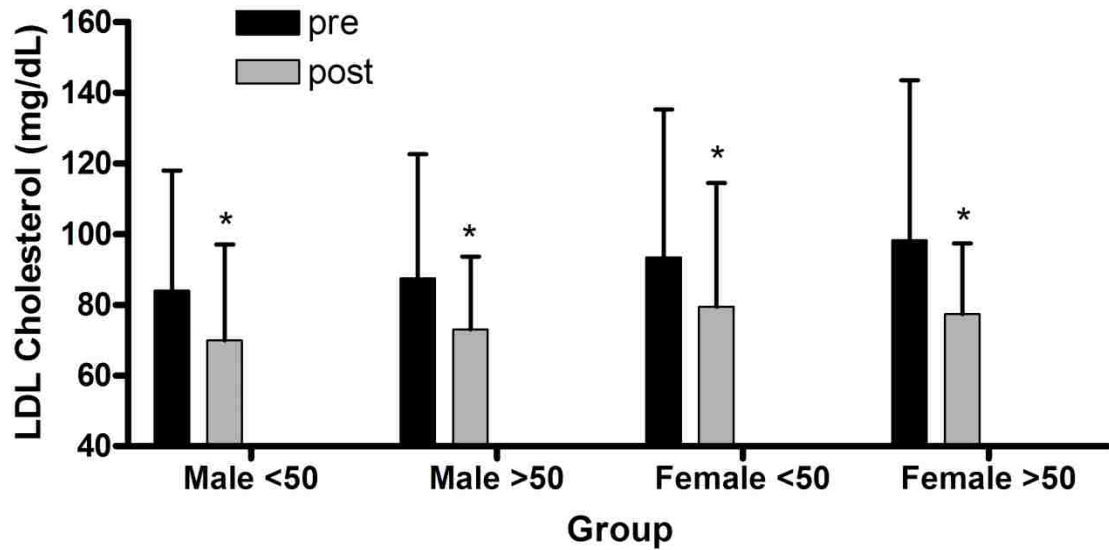


Figure 5. The cardiac rehabilitation program had an effect on the variable LDL-C. However there was no other interaction regarding this variable has statistically significant.

HDL-C

The difference between baseline HDL-C (N=198) and 12 week HDL-C (N=198) was statistically significant $F(1,193) = 8.347, p = .004$; partial eta squared = .041. The interaction between age and time was not significant $F(1,193) = .030, p = .862$; partial eta squared = .000. The interaction between time and sex was not statistically significant $F(1,193) = 2.027, p = .156$; partial eta squared = .010.

In the secondary analyses of sex and age differences, the main effect of sex was statistically significant $F(1, 193) = 11.060, p = .001$; partial eta squared = .054. The main effect of age was not statistically significant $F(1, 193) = 2.839, p = .094$; partial eta squared = .014. The interaction of sex and age was not statistically significant $F(1, 193) = 3.297, p = .071$; partial eta squared = .017.

Figure 6. Comparison of baseline and post values for HDL-C

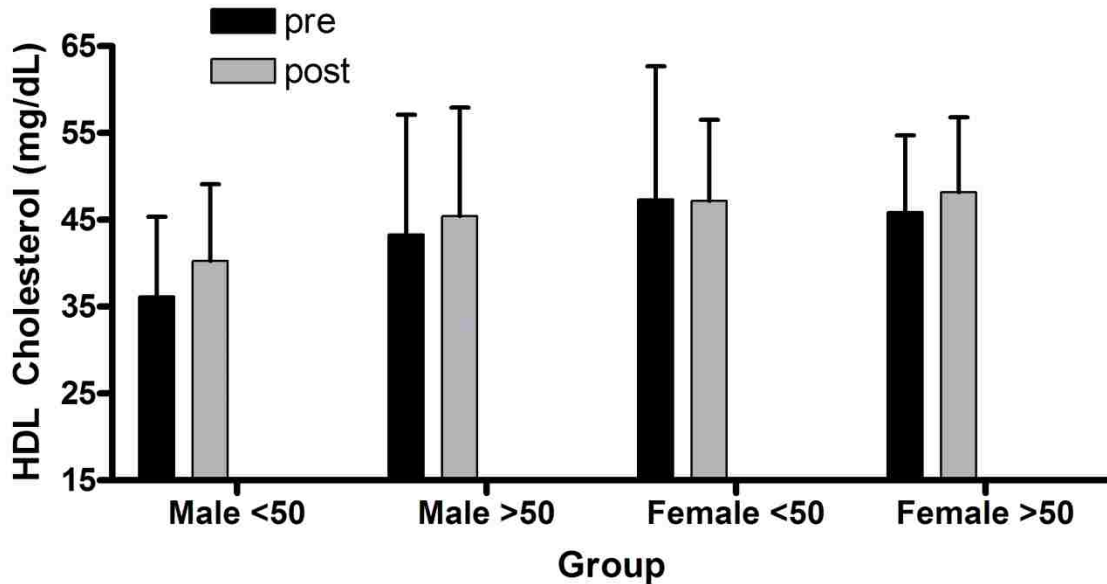


Figure 6. There was a within variable significance due to the cardiac rehabilitation program and there was a main effect of sex on the variable.

Multiple regression was used to identify whether there was statistical significance between the variable delta Framingham, the difference of baseline and post assessments, delta fat, the difference of baseline and post fat intake, and delta METs, the difference between baseline and post MET values participants reached completing an Atterbom walk test. There was mild statistical significance between delta fat and the delta Framingham scores $F(1, 182) = 8.556, p = .004$ and $R = .045$ (Figure 8). There was no statistical significance found in to between delta Framingham and delta MET $F(1, 192) = .054, p = .817$ and $R = .017$.

Figure 7. Comparison of the predicted delta Framingham risk score and actual delta Framingham risk score.

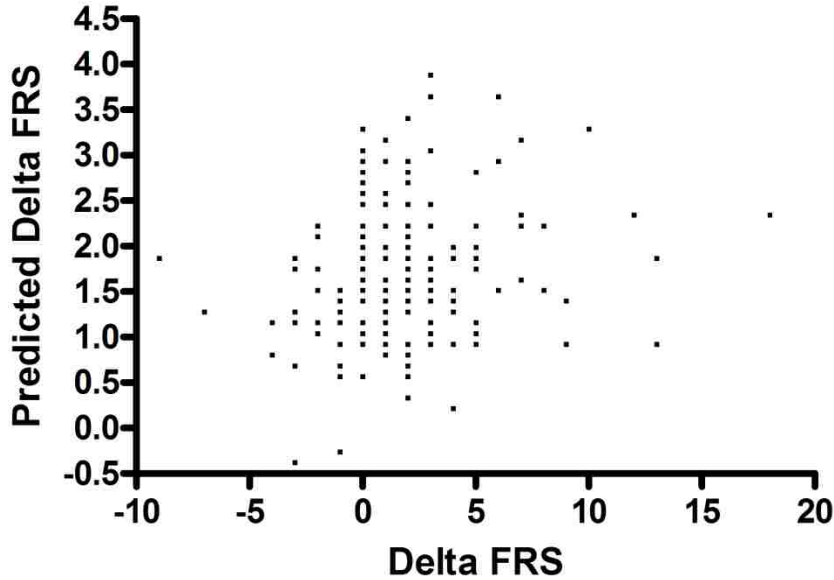


Figure 7. The delta FRS (Framingham risk score) was created from calculating the baseline and post values. The predicted values of the delta FRS is calculated by using the multiple regression equation ($Y = a + b_1 * X_1 + b_2 * X_2 + \dots + b_p * X_p$).

Figure 8. Delta Framingham risk score and delta fat intake

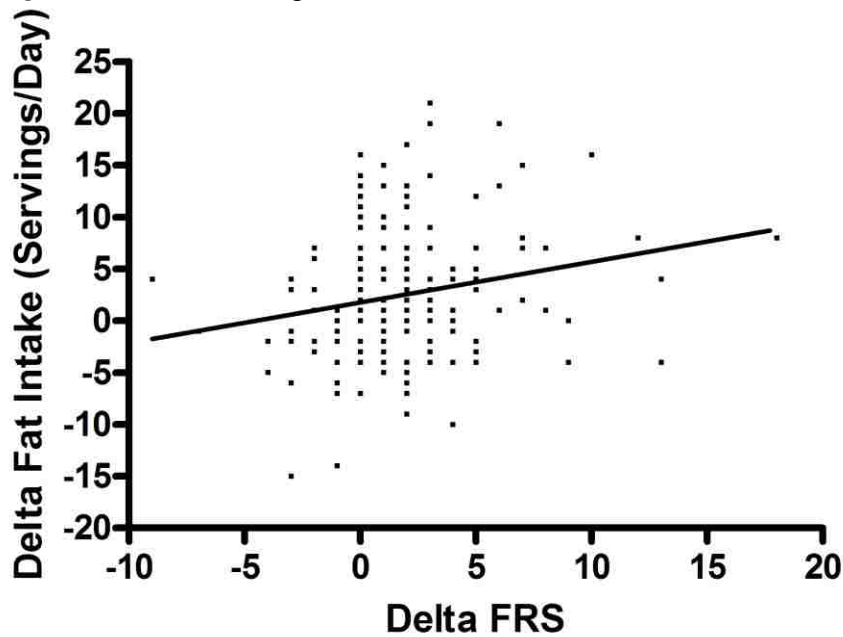


Figure 8. The delta was created from calculating the baseline and post values in both the Framingham risk score and their self-reported fat intake.

Figure 9. Comparison of residual and predicted delta Framingham risk score

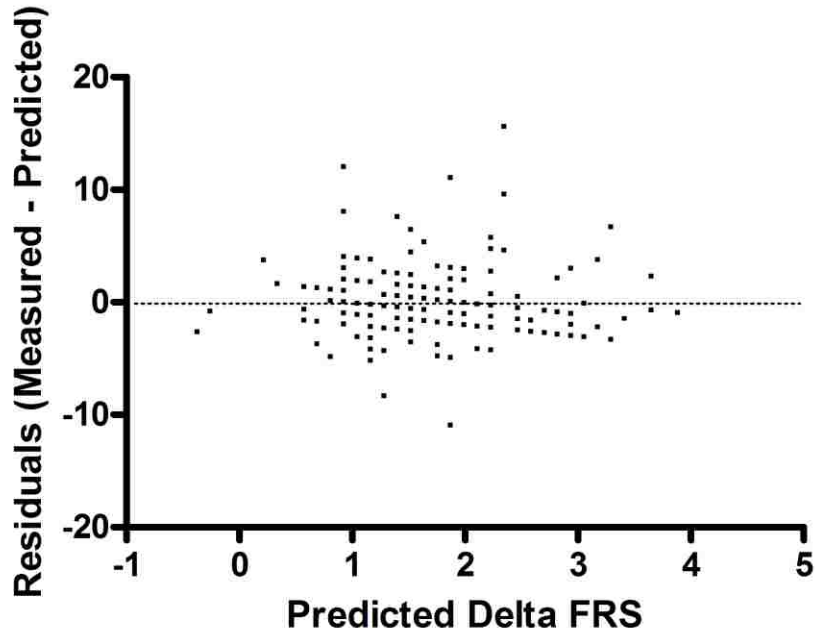


Figure 9. Residual scores of the Framingham risk score were calculated by subtracting the actual values from the predicted values.

Chapter V

Discussion

Coronary Heart disease is the leading killer of both men and women in the United States. Individuals that have developed any form of cardiovascular disease (coronary heart disease, coronary artery disease) and may have had an intervention (PTCI, PTCA, CABG, etc.) are eligible for cardiac rehabilitation. To a great extent research done validating cardiac rehabilitation programs focuses on graduation rate and occurrence of another cardiac event. The Framingham risk score seems to be a good instrument for evaluating cardiac rehabilitation programs. The purpose of this study was to examine the effectiveness of a cardiac rehabilitation program in modifying a patient's Framingham risk score after 36 sessions of cardiac rehabilitation.

The Framingham risk score was used to evaluate a 36-session cardiac rehabilitation program. The Framingham risk score uses an individual's age, gender, HDL-C, LDL-C, total cholesterol, systolic and diastolic blood pressure, diabetes and smoking status to evaluate whether an individual will have a cardiac event within the next ten years. The retrospective data was collected from a cardiac rehabilitation program and a Framingham risk score was calculated. SPSS version 16 was used to evaluate the Framingham risk score through a multivariate analysis to identify whether the Framingham risk score and each factor that comprises the Framingham risk score are statistically significant between baseline and post tests. In the secondary research questions the Framingham risk score and the interaction between sex and age were statistically significant.

The results of the variables that were tested from baseline to 12 weeks showed that all of the variables were statistically significant, which is not surprising since the subjects were involved in exercise, taking prescribed medications and eating a heart healthy diet. In the secondary research questions the Framingham risk score was statistically significant in the interaction between sex and age and the main effect of age. The results stated above suggested that both age and sex play a role in the Framingham risk score.

Systolic blood pressure had a statistical significant interaction with sex, men had lower overall values in comparison to women from baseline to post test. However diastolic blood pressure had a statistical significant interaction with age, the exact reason why between age is unknown. Both total cholesterol and HDL-C had statistical significant interaction with sex from the research there appears to be an effect between sex and cholesterol values however there is no known reason. An interaction appeared between HDL-C and sex is not surprising since there are different clinical recommendations due to sex.

Individuals in cardiac rehabilitation have had life changing events that change their outlook on life. Cardiac rehabilitation programs accredited by AACVPR are designed to address a variety of areas: behavioral (education, tobacco cessation, stress management, adherence to diet, and exercise), clinical (functional capacity, lipids, MET levels, weight, blood pressure, and heart rate), health (quality of life and loss of work days), and service (patient satisfaction, access and utilization of service, and financial and economic concerns). There has been a great deal of research on the effects exercise has on the body. Exercise has proven to reduce the effects of diabetes, by reducing blood

sugar levels and the progression of the disease. Exercising over a longer period of time reduces the overall content of blood present in the circulatory system and reduces HbA1c (Hemoglobin A1c) HbA1c is a value found in the blood that gives a picture of blood glucose values over the last month. Besides improving the blood glucose regulation, exercise also improves cardiac function.

There have been various investigations in the last 50 years regarding the relationship of CHD and physical activity. Kohl (2001) has completed a literature review that evaluated this relationship. However there was a majority of research that showed a relationship between CHD and physical activity was inversely proportional and “U” shaped if physical activity levels are high then there is a higher risk for CHD (Wilund, 2007). The exact reasons between higher CHD and high levels of physical activity are unknown. Higher amounts of physical activity in individuals that already have CHD or CVD puts additional stress on the heart that may not be able to handle it. This thesis looked at the effect a 36-session cardiac rehabilitation program reduced the subjects Framingham risk score. The amount of the physical activity during this current body of research was not examined however, the risk factors that cause CHD were reduced overall.

In cardiac rehabilitation programs there are certain factors that are monitored closely including cholesterol levels, blood glucose, blood pressure and functional capacity. Richardson et al. (2008) investigated individuals who had no pre-existing cardiovascular disease to see if the Framingham risk score reduced from their baseline assessment to the one-year follow-up. Richardson (2008) gave the participants that needed in a dietician consultation, smoking cessation, and exercise guidelines. Since

Richardson's (2008) research did not examine patients with known cardiovascular disease it does not have direct bearing on this research. However the use of the Framingham risk score to evaluate the relationship it has with exercise, smoking and diet modifications due. The results of the study showed that there was a reduction of the Framingham risk score and improved levels of blood pressure and lipids and lower rates of the development of CHD among those you maintained the program. In the subjects that quit the program and a higher incidence of CHD and a higher Framingham risk score (Richardson, 2008).

Cardiac rehabilitation programs exist throughout the United States and Europe. However cardiac rehabilitation programs in these two regions differ in many ways including the time that is spent in the program. Nieuwland et al. (2000) investigated the difference between a low frequency and high frequency both programs that were 6-weeks long. The high frequency consisted of two sessions per day five days per week and the low frequency consists of 1 session per day twice a week (Nieuwland et al.). The cardiac rehabilitation program examined in this body of research had patients attend up to three times per week for one session per day. This protocol of three sessions per week sits right between the low and high frequency programs.

There were baseline and post program evaluations using a submaximal cycle ergometer test with blood pressure monitored and 12-lead EKG. Nieuwland's research protocol included a dietician consultation, and a meeting with a social worker or psychologist in addition to the exercise sessions. Spouses were also encouraged to participate in the exercise program. The New Heart Cardiac Rehabilitation program performs a baseline and post walk test using the Atterbom protocol, with the patient

attached to a 12-lead EKG and blood pressure monitoring. Spouses and family members were encouraged to join so that the subjects would have a support system in place

Many of the participants were men and had uncomplicated cardiac events and their cardiac events were relieved through surgery or medication (Nieuwland et al.). The results of this study showed that there was an improvement in both groups and at least a 10% improvement in all areas for the low frequency group. Nieuwland's research is similar to the cardiac rehabilitation programs used in this body of research however the submaximal tests are mainly walk tests and the cardiac rehabilitation programs last up to 36-sessions whereas this research lasted only 6 weeks. It was stated in the research done by Nieuwland et al. (2000) that the participants were highly motivated individuals and Caucasian, which makes the results difficult to apply to a larger mixed population.

Nieuwland, et al. study proves that a multifaceted approach to subjects that have had cardiac events improves the health and reduces a subject's risk for having another cardiac event. This research aligns and proves the primary questions that were asked. In both bodies of research the cardiac rehabilitation intervention improved lipid values.

Lavie et al. (1993) investigated the benefits of cardiac rehabilitation programs and exercise training in atherosclerosis prevention in the elderly. The researchers investigated plasma lipid levels, obesity, exercise capacity and compared it to elderly patients with diagnosed CHD. There were marked improvements in exercise capacity, obesity indices, and lipids levels from the baseline. Exercise improved all of the researched variables in not only the elderly but also the younger population. This research recommended that all participants in cardiac rehabilitation programs should have psychosocial, physical and risk factor evaluation besides exercise training.

Research investigated above looked at the effects cardiac rehabilitation programs have on their subjects. The research shows that there is an improvement in the subjects due to exercise and other interventions (Lavie, 1993). Limitations of this study are that the participants did not accurately reflect the general population, their subjects had cardiac events that were relieved with medical intervention and no secondary health issues. The cardiac rehabilitation program that was investigated for this research used the Framingham risk score as the variable to evaluate the program. Many of the research subjects may have had secondary health issues that may have complicated and influenced their progress in the cardiac rehabilitation program. All of the participants were on various kinds of medications to ensure that their lipid, blood pressure and blood glucose values, which may have an effect on their improvement on the variables investigated. The range of time that was investigated was from 6 weeks to 3 months.

Recommendations

Research needs to investigate how much time and intensity benefits individuals with cardiac problems. Populations and research to encompass a variety of individuals with cardiac problems and secondary health concerns to more accurately represent the population. The Framingham risk score is a tool that is widely accepted to evaluate risk factors that lead to the development of coronary heart disease. However there was research that showed that it underestimated an individual's risk for developing coronary heart disease. The Framingham risk score needs to be modified to prevent the underestimation of the presence of disease that has been shown from prior research. Alternatively, a new tool needs to be developed that can better predict an individual's risk for developing coronary heart disease. The Framingham risk score does not look and

excess body fat as a factor, which has been shown to increase an individual's risk for developing coronary heart disease. Cardiac rehabilitation programs are an important part in improving an individual's health after a coronary event.

Appendix A



THE UNIVERSITY *of*
NEW MEXICO

Main Campus Institutional Review Board
Human Research Protections Office
1717 Roma NE, MSC05 3180
1 University of New Mexico~Albuquerque, NM 87131-0001
<http://hsc.unm.edu/som/research/HRRC/>

15-Apr-2009

Responsible Faculty: Robert Robergs
Investigator: Veronica K. Gonzales
Dept/College: Health Exercise & Sports Science

SUBJECT: IRB Approval of Research - Initial Review - Modification
Protocol #: 08-605
Project Title: The effects of a 36-session Cardiac Rehabilitation Program on the Framingham Risk Score
Type of Review: Expedited Review
Approval Date: 14-Apr-2009
Expiration Date: 13-Apr-2010

The Main Campus Institutional Review Board has reviewed and approved the above referenced protocol. It has been approved based on the review of the following:

1. IRB Application, revised, received 040209 & revised Attachment 8 received 040909
2. Data Collection Form (template) received 040209

Consent Decision:
Waived the requirement for informed consent
HIPAA Authorization Addendum waived

When consent is required, it is the responsibility of the Principal Investigator (PI) to ensure that ethical and legal informed consent has been obtained from all research participants. A date stamped original of the approved consent form(s) is attached, and copies should be used for consenting participants during the above noted approval period.

As the principal investigator of this study, you assume the following responsibilities:

Renewal: To comply with federal law, the IRB must conduct continuing review of this research before the expiration date noted above. It is the responsibility of the PI to submit a progress report to the IRB at least 30 days prior to the end of the approval period in order for this study to be considered for continuation.

Adverse Events: Any adverse events or reactions must be reported to the IRB immediately.

Modifications: Any changes to the protocol, such as procedures, consent/assent forms, addition of subjects, or study design must be submitted to the IRB as an Amendment for review and approval.

Completion: When the study is concluded and all data has been de-identified (with no link to identifiers), submit a Closure Report to close your study.

Please reference the protocol number and study title in all documents and correspondence related to this protocol.

Sincerely,

A handwritten signature in black ink, appearing to read "J. Scott Tonigan". The signature is fluid and cursive, with a long horizontal stroke at the end.

**J. Scott Tonigan, PhD
Chair
Main Campus IRB**

* Under the provisions of this institution's Federal Wide Assurance (FWA00004690), the Main Campus IRB has determined that this proposal provides adequate safeguards for protecting the rights and welfare of the subjects involved in the study and is in compliance with HHS Regulations (45 CFR 46).



THE UNIVERSITY *of*
NEW MEXICO

Main Campus Institutional Review Board
Human Research Protections Office
MSC08 4560

1 University of New Mexico-Albuquerque, NM 87131-0001
<http://hsc.unm.edu/som/research/HRRC/>

05-Feb-2010

Responsible Faculty: Robert Robergs
Investigator: Veronica K. Gonzales
Dept/College: Health Exercise & Sports Science

*SUBJECT: IRB Approval of Research - Amendment
Protocol #: 08-605*

Project Title: The effects of a 36-session Cardiac Rehabilitation Program on the Framingham Risk Score

Type of Review: Expedited Review

Approval Date: 05-Feb-2010

Expiration Date: 13-Apr-2010

The Main Campus Institutional Review Board has reviewed and approved the above referenced protocol. It has been approved based on the review of the following:

Request for Amendment form received 1/28/10

Amendment requesting minor protocol change to collect additional data fields in this retrospective data collection study. Additional fields are: dietary fat intake, MET values pre and post cardiac rehabilitation program.

Revised protocol version received 1/28/10

Consent Decision:
No changes.

When consent is required, it is the responsibility of the Principal Investigator (PI) to ensure that ethical and legal informed consent has been obtained from all research participants. A date stamped original of the approved consent form(s) is attached, and copies should be used for consenting participants during the above noted approval period.

As the principal investigator of this study, you assume the following responsibilities:

Renewal: Unless granted exemption, your protocol must be re-approved each year in order to continue the research. You must submit a Progress Report no later than 30 days prior to the expiration date noted above.

Adverse Events: Any adverse events or reactions must be reported to the IRB immediately.

Modifications: Any changes to the protocol, such as procedures, consent/assent forms, addition of subjects, or study design must be submitted to the IRB for review and approval.

Completion: When the study is concluded and all data has been de-identified (with no link to identifiers), submit a Final Report Form to close your study.

Please reference the protocol number and study title in all documents and correspondence related to this protocol.

Sincerely,

A handwritten signature in black ink, appearing to read "J. Scott Tonigan". The signature is fluid and cursive, with a long horizontal stroke at the end.

J. Scott Tonigan, PhD
Chair
Main Campus IRB

* Under the provisions of this institution's Federal Wide Assurance (FWA00004690), the Main Campus IRB has determined that this proposal provides adequate safeguards for protecting the rights and welfare of the subjects involved in the study and is in compliance with HHS Regulations (45 CFR 46).

Appendix B



To Whom It May Concern:

The purpose of the proposal is to evaluate the improvement in physical fitness of the individuals participating in the New Heart program

Patient confidentiality will be assured. All subjects signed a waiver that explained the risks of the initial walk test and the cardiac rehabilitation program. The waiver also had a section stating that all of their records may be used for statistical analyses or scientific purpose. We will be granted access to the records of their progress while at New Heart. Participants will not be at risk for exposure of any of their personal or outcomes data. All participants are aware of the study and are happy to be involved and are aware of the risks and benefits of the study.

Sincerely,

Richard D Lueker, MD, FACC
Medical Director

Appendix C

Patient : _____

DOB: ____/____/____

BL 12-wk 6-mo Yrly

Physician Evaluation and Comments

Hemodynamics

- Normal Response
- Hypertensive
- Flat BP
- Blunted HR
- Rapid Chronotropic
- Other: _____

Exercise Capacity

- Markedly Impaired
- Impaired
- Average
- Above Average
- Excellent

Change from Previous Test:

- No change
- Increase
- Decrease

Resting EKG

Normal

Other: _____

Exercise EKG

Normal

No Change

Accent ST-T

Minor J pt

_____ mm ST depression in _____ leads

Other _____

Conclusion _____

Read by _____

Patient: _____ Age: ___yrs Date: __/__/__ Tech: ____

Anthropometric Data

Ht : ____ in
Wt : ____ lb
BMI : ____ kg/m²
Waist: ____ in

Blood Sugar

CBG_{pre} : ____ mg/dl
CBG_{post} : ____ mg/dl
Not applicable: ____

Ankle Brachial Index

Pre-Exer : R L ankle : ____
brachial: ____ ABI_{pre} : ____
Post-Exer: R L ankle : ____
brachial : ____ ABI_{post} : ____
Not applicable: ____ Reason: _____

Exercise Test Data

HR_{rest} : ____ bpm
HR_{75%} : ____ bpm

BP_{rest} : ____ / ____ mmHg
BP_{max} : ____ / ____ mmHg

SaO_{2pre} : ____ %
SaO_{2post} : ____ %

O₂ : ____ L

Exer _{min}	MPH	%	METS	RPE	HR	BP	SaO ₂	Symptoms
	1.0		1.77			/		
1	1.5		2.15			/		
2	2.0		2.53			/		
3	2.5		2.92			/		
4	3.0		3.3			/		
5	3.0	2	4.13			/		
6	3.0	5	5.37			/		
7	3.0	7	6.19			/		
8	3.5	7	7.06			/		
9	3.5	9	8.03			/		
10	3.7	9	8.43			/		
11	3.7	11	9.45			/		
12	4.0	11	10.13			/		
13	4.0	13	11.24			/		
14	4.2	13	11.75			/		
15	4.2	15	12.91			/		

Recovery								
1						/		
2						/		
3						/		

Test Results

MET_{mod} : ____
MET_{max} : ____

HR_{max} : ____ bpm
HR_{target} :
____ - ____ bpm

Symptoms

- Chest Discomfort
- Dizziness
- Dyspnea
- Fatigue
- None
- Other _____

Comments: _____

Patient : _____

DOB: ____/____/____

BL 12-wk 6-mo Yrly

Physician Evaluation and Comments

Hemodynamics

- Normal Response
- Hypertensive
- Flat BP
- Blunted HR
- Rapid Chronotropic
- Other: _____

Exercise Capacity

- Markedly Impaired
- Impaired
- Average
- Above Average
- Excellent

Change from Previous Test:

- No change
- Increase
- Decrease

Resting EKG

Normal

Other: _____

Exercise EKG

Normal

No Change

Accent ST-T

Minor J pt

_____ mm ST depression in _____ leads

Other _____

Conclusion _____

Read by _____

Patient: _____ Age: ____ yrs Date: __/__/__ Tech: ____

Anthropometric Data

Ht : _____ in
Wt : _____ lb
BMI : _____ kg/m²
Waist: _____ in

Blood Sugar

CBG_{pre}: _____ mg/dl
CBG_{post}: _____ mg/dl
Not applicable: ____

Ankle Brachial Index

Pre-Exer : R L ankle : _____
brachial: _____ ABI_{pre} : _____
Post-Exer: R L ankle : _____
brachial : _____ ABI_{post}: _____
Not applicable: ____ Reason: _____

Exercise Test Data

HR_{rest}: _____ bpm
HR_{75%}: _____ bpm

BP_{rest}: _____/_____ mmHg
BP_{max}: _____/_____ mmHg

SaO_{2pre}: _____ %
SaO_{2post}: _____ %

O₂: _____ L

Exer _{min}	MPH	%	METS	RPE	HR	BP	SaO ₂	Symptoms
1	.5		1.38			/		
2	.8		1.61			/		
3	1.0		1.77			/		
4	1.3		2.00			/		
5	1.3	2	2.35			/		
6	1.3	5	2.89			/		
7	1.3	7	3.25			/		
8	1.5	7	3.6			/		
9	2.0	9	4.46			/		
						/		
						/		
						/		
						/		
Recovery								
1						/		
2						/		
3						/		

Test Results

MET_{mod}: _____
MET_{max}: _____

HR_{max}: _____ bpm
HR_{target}:
_____ - _____ bpm

Symptoms

- Chest Discomfort
- Dizziness
- Dyspnea
- Fatigue
- None
- Other _____

Comments: _____

Appendix D

Men's Framingham Risk Score

Step 1

Age			
Years	LDL Pts	Chol Pts	
30-34	-1	[-1]	
35-39	0	[0]	
40-44	1	[1]	
45-49	2	[2]	
50-54	3	[3]	
55-59	4	[4]	
60-64	5	[5]	
65-69	6	[6]	
70-74	7	[7]	

Step 2

LDL - C		
(mg/dl)	(mmol/L)	LDL Pts
<100	<2.59	-3
100-129	2.60-3.36	0
130-159	3.37-4.14	0
160-190	4.15-4.92	1
≥190	≥4.92	2

Cholesterol		
(mg/dl)	(mmol/L)	Chol Pts
<160	<4.14	[-3]
160-199	4.15-5.17	[0]
200-239	5.18-6.21	[1]
240-279	6.22-7.24	[2]
≥280	≥7.25	[3]

Step 3

HDL - C			
(mg/dl)	(mmol/L)	LDL Pts	Chol Pts
<35	<0.90	2	[2]
35-44	0.91-1.16	1	[1]
45-49	1.17-1.29	0	[0]
50-59	1.30-1.55	0	[0]
≥60	≥1.56	-1	[-2]

Step 4

Blood Pressure		
Systolic (mm Hg)	Diastolic (mm Hg)	
<120	<80	0 [0] pts
120-129	80-84	0 [0] pts
130-139	85-89	1 [1] pts
140-159	90-99	2 [2] pts
≥160	≥100	3 [3] pts

Note: When systolic and diastolic pressures provide different estimates for point scores, use the higher number.

Step 5

Diabetes		
	LDL Pts	Chol Pts
No	0	[0]
Yes	2	[2]

Step 6

Smoker		
	LDL Pts	Chol Pts
No	0	[0]
Yes	2	[2]

(sum from steps 1-6)

Step 7

Adding up the points	
Age	_____
LDL-C or Chol	_____
HDL - C	_____
Blood Pressure	_____
Diabetes	_____
Smoker	_____
Point total	_____

(determine CHD risk from point total)

Step 8

CHD Risk			
LDL Pts	10 Yr	Chol Pts	10 Yr
Total	CHD Risk	Total	CHD Risk
<-3	1%		
-2	2%		
-1	2%	[-1]	[2%]
0	3%	[0]	[3%]
1	4%	[1]	[3%]
2	4%	[2]	[4%]
3	6%	[3]	[5%]
4	7%	[4]	[7%]
5	9%	[5]	[8%]
6	11%	[6]	[10%]
7	14%	[7]	[13%]
8	18%	[8]	[16%]
9	22%	[9]	[20%]
10	27%	[10]	[25%]
11	33%	[11]	[31%]
12	40%	[12]	[37%]
13	47%	[13]	[45%]
≥14	≥56%	≥14	≥53%

(compare to average person your age)

Step 9

Comparative Risk			
Age (years)	Average 10 Yr CHD Risk	Average 10 Yr Hard* CHD Risk	Low** CHD Risk
30-34	3%	1%	2%
35-39	5%	4%	3%
40-44	7%	4%	4%
45-49	11%	8%	4%
50-54	14%	10%	6%
55-59	16%	13%	7%
60-64	21%	20%	9%
65-69	25%	22%	11%
70-74	30%	25%	14%

* Hard CHD events exclude angina pectoris

** Low risk was calculated for a person the same age, optimal blood pressure, LDL-C 100-129 mg/dL or cholesterol 160-199 mg/dL, HDL-C 45 mg/dL for men or 55 mg/dL for women, non-smoker, no diabetes

Risk estimates were derived from the experience of the Framingham Heart Study, a predominantly Caucasian population in Massachusetts, USA

Key	
Color	Relative Risk
green	Very low
white	Low
yellow	Moderate
rose	High
red	Very high

Women's Framingham Risk Score

Step 1

Age			
Years	LDL Pts	Chol Pts	
30-34	-9	[-9]	
35-39	-4	[-4]	
40-44	0	[0]	
45-49	3	[3]	
50-54	6	[6]	
55-59	7	[7]	
60-64	8	[8]	
65-69	8	[8]	
70-74	8	[8]	

Step 2

LDL - C		
(mg/dl)	(mmol/L)	LDL Pts
<100	<2.59	-2
100-129	2.60-3.36	0
130-159	3.37-4.14	0
160-190	4.15-4.92	2
≥190	≥4.92	2

Cholesterol		
(mg/dl)	(mmol/L)	Chol Pts
<160	<4.14	[-2]
160-199	4.15-5.17	[0]
200-239	5.18-6.21	[1]
240-279	6.22-7.24	[1]
≥280	≥7.25	[3]

Step 3

HDL - C			
(mg/dl)	(mmol/L)	LDL Pts	Chol Pts
<35	<0.90	5	[5]
35-44	0.91-1.16	2	[2]
45-49	1.17-1.29	1	[1]
50-59	1.30-1.55	0	[0]
>60	>1.56	-2	[-3]

Step 4

Blood Pressure				
Systolic (mm Hg)	Diastolic (mm Hg)			
	<80	80-84	85-89	90-99 >100
<120	-3 [-3] pts			
120-129		0 [0] pts		
130-139			0 [0] pts	
140-159				2 [2] pts
≥160				3 [3] pts

* Note: When systolic and diastolic pressures provide different estimates for point scores, use the higher number

Step 5

Diabetes		
	LDL Pts	Chol Pts
No	0	[0]
Yes	4	[4]

Step 6

Smoker		
	LDL Pts	Chol Pts
No	0	[0]
Yes	2	[2]

(sum from steps 1-6)

Step 7

Adding up the points

Age _____

LDL-C or Chol _____

HDL - C _____

Blood Pressure _____

Diabetes _____

Smoker _____

Point total _____

(determine CHD risk from point total)

Step 8

CHD Risk			
LDL Pts	10 Yr CHD Risk	Chol Pts	10 Yr CHD Risk
Total	Total	Total	Total
≤-2	1%	[-2]	[1%]
-1	2%	[-1]	[2%]
0	2%	[0]	[2%]
1	2%	[1]	[2%]
2	3%	[2]	[3%]
3	3%	[3]	[3%]
4	4%	[4]	[4%]
5	5%	[5]	[4%]
6	6%	[6]	[5%]
7	7%	[7]	[6%]
8	8%	[8]	[7%]
9	9%	[9]	[8%]
10	11%	[10]	[10%]
11	13%	[11]	[11%]
12	15%	[12]	[13%]
13	17%	[13]	[15%]
14	20%	[14]	[18%]
15	24%	[15]	[20%]
16	27%	[16]	[24%]
≥17	≥32%	≥[17]	≥[27%]

(compare to average person your age)

Step 9

Comparative Risk			
Age (years)	Average 10 Yr CHD Risk	Average 10 Yr Hard* CHD Risk	Low** 10 Yr CHD Risk
30-34	<1%	<1%	<1%
35-39	<1%	<1%	1%
40-44	2%	1%	2%
45-49	5%	2%	3%
50-54	8%	3%	5%
55-59	12%	7%	7%
60-64	12%	8%	8%
65-69	13%	8%	8%
70-74	14%	11%	8%

Key

Color	Relative Risk
green	Very low
white	Low
yellow	Moderate
rose	High
red	Very high

* Hard CHD events exclude angina pectoris

** Low risk was calculated for a person the same age, optimal blood pressure, LDL-C 100-129 mg/dL or cholesterol 160-199 mg/dL, HDL-C 45 mg/dL for men or 55 mg/dL for women, non-smoker, no diabetes

Risk estimates were derived from the experience of the Framingham Heart Study, a predominantly Caucasian population in Massachusetts, USA

References

- Addia, P.B., Carr T.P., Hassel C.A., Huang Z.Z., and Warner, G.J. Atherogenic and anti-atherogenic factors in the human diet. *Biochemical Society Symposium* (1995) 61:259-71.
- American Association of Diabetes Educators (AADE). AADE Position Statement: Diabetes and Exercise. *American Association of Diabetes Educators* (2007) 34(1): 37-40
- Angina. Retrieved from Mayo Clinic, 2009
<http://www.mayoclinic.com/health/angina/DS00994/DSECTION=symptoms>
- Anselmino, M., Wallander, M., Norhammar, A., Mellbin, L., and Ryden, L. Implications of abnormal glucose metabolism in patients with coronary artery disease. *Diabetes and Vascular Disease Research* (2008) 5(4): 285-290
- Benfante R., Reed, D., and Frank, J. Does cigarette smoking have an independent effect on coronary heart disease incidence in the elderly? *American Journal of Public Health* (1991) 81(7): 897-899
- Bianchi, C., Miccoli, R., Penno, G., and Del Prato, S. Primary prevention of cardiovascular disease in people with dysglycemia. *Diabetes Care* (2008) 31(2): S208-S214.
- Boule, N.G., Haddad, E., Kenny, G.P., Wells, G.A., and Sigal, R.J. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. *Journal of American Medical Association* (2001) 286(10):1218-1227
- Cardiovascular Disease Statistics. Retrieved from The American Heart Association, January 6, 2009. <http://www.americanheart.org/presenter.jhtml?identifier=4478>
- Choi, K.M., Lee, J., Lee, K.W., Seo, J.A., Oh, J.H., Kim, S.G., Kim, N.H., Choi, D.S., and Baik, S.H. Serum adiponectin concentrations predict the developments of type 2 diabetes and the metabolic syndrome in elderly Koreans. *Clinical Endocrinology* (2004) 61: 75-80
- Dawber, T.R., and Moore, F.E. (1952), "Longitudinal Study of Heart Disease in Framingham, Massachusetts: An Interim Report," in *Research in Public Health, Papers presented at the 1951 Annual Conference of the Milbank Memorial Fund*, 241-247.
- Dawber, T.R. (1980), *The Framingham Study: The Epidemiology of Atherosclerotic Disease*, Cambridge, Mass.: Harvard University Press.
- Diabetes. Retrieved from the American Diabetes Association, January 5, 2009.
<http://www.diabetes.org/diabetes-heart-disease-stroke.jsp>

Diabetes. Retrieved from the World Health Organization, January 6, 2009.
<http://www.who.int/diabetes/en/>

Haskell, W.L., Lee, I., Pate, R.R., Powell, K.E., Blair, S.N., Franklin, B.A., Macera, C.A., Heath, G.W., Thompson, P.D. and Bauman, A. Physical Activity and Public Health: Updated Recommendation for Adults from the American College of Sports Medicine and American Heart Association. *Medicine and Science in Sports and Exercise* (2007) 123-1434

Hayes, C. Case Study: Exercise-related hypoglycemia in type 2 diabetes treated with oral glucose-lowering medications. *Clinical Diabetes* (2007) 25:150-151

Heart Disease. Retrieved from the Center for Disease Control, September 21, 2007.
<http://www.cdc.gov/nchs/fastats/heart.htm>

History of the Framingham Heart Study. Retrieved from Framingham Heart Study, December 16, 2008. <http://www.framinghamheartstudy.org/about/history.html>

Johnstone, M.T., and Kinzfohl, G.P. (2005) Diabetes Mellitus and heart disease. In Johnstone, M.T. and Veves, A. *Diabetes and Cardiovascular Disease Second Edition* (pp. 579-628) Totowa, NJ: Humana Press Inc.

Kohl III, H.W. Physical activity and cardiovascular disease: evidence for a dose response. *Medicine and Science in Sports and Exercise* (2001) 33:S472-483

Kuller, L.H., Ockene, J.K., Meilahn, E., Wentworth, D.N., Svendsen, K.H., and Neaton, J.D. Cigarette smoking and mortality. *Preventative Medicine* (1991) 20:638-654

Lavie, C.J., Milani, R.V., and Littman, A.B. Benefits of cardiac rehabilitation and exercise training in secondary coronary prevention in the elderly. *Journal of the American College of Cardiology* (1993) 22:678-683.

Lu, H., Raptis, M., Black, E., Stan, M., Amar, S., Graves, D.T. Influence of Diabetes on the exacerbation of an inflammatory response in cardiovascular tissue. *Endocrinology* (2004) 145(11): 4934-4939

Millen, B.E., Quatromoni, P.A., Nam, B., Pencina, M.J., Polak, J.F., Kimokoti, R.W., Ordovas, J.M., and D'Agostino, R.B. Compliance with expert population-based dietary guidelines and lower odds of carotid atherosclerosis in women: the Framingham Nutrition Studies. *American Journal of Clinical Nutrition* (2005) 82:174-180.

Moller DE., Potential Role of TNF- α in the pathogenesis of insulin resistance and type 2 diabetes. *Trends in Endocrinology and Metabolism*. (2000) 11(6): 212-217.

National Diabetes Fact Sheet, 2007. Retrieved from the Center for Disease, January 12, 2008. <http://apps.nccd.cdc.gov/ddtstrs/FactSheet.aspx>

Nieuwland, W., Berkhuisen, M.A., van Veldhuisen, D.J., Brügemann, J., Landsman, M.L.J., van Sonderen, E., Lie, K.I., Crijns, H.J.G.M., and Rispens, P. Differential effects of high frequency versus low frequency exercise training in rehabilitation of patients with coronary artery disease. *Journal of the American College of Cardiology* (2000) 36:202-207.

Ockene J.K., Kuller, L.H., Svendsen, K.H., and Meilahn, E. The relationship of smoking cessation to coronary heart disease and lung cancer in the multiple risk factor intervention trial. *American Journal of Public Health* (1990) 80(8):954-958

Powell, J.T. Vascular damage from smoking: Disease mechanisms at the arterial wall. *Vascular Medicine* (1998) 3(1):21-28

Richardson, G., van Woerden, H.C., Morgan, L., Edwards, R., Harries, M., Hancock, E., Sroczynski, S., and Bowley, M. Healthy Hearts- A community-based primary prevention programme to reduce coronary heart disease. *BMC Cardiovascular Disorders* (2008) 8:18

Rozanski, A., and Blumenthal, J. Cardiac rehabilitation, exercise training and psychosocial risk factors: Reply. *Journal of the American College of Cardiology* (2006) 47:212-213

Segal, S.S. (2009) Treating Coronary Artery Disease. In Boron, W.F., Boulpaep, E.L. *Medical Physiology Second Edition* (pp.585). Philadelphia, PA: Saunders.

Shaten B.J., Kuller, L.H., and Neaton, J.D. Association between baseline risk factors, cigarette smoking, and CHD mortality after 10.5 years. *Preventative Medicine* (1991) 20:655-669

Silander, K., Alanne, M., Kristiansson, K., Saarela, O., Ripatti, S., Auro, K., Karvanen, J., Kulathinal, S., Niemelä, M., Ellonen, P., Vartiainen, E., Jousilahti, P., Saarela, J., Kuulasmaa, K., Evans, A., Perola, M., Salomaa, V., and Peltonen, L., Gender differences in genetic risk profiles for cardiovascular disease. *PLOS One* (2008) 3(10):e3615.

Taylor A.J., Burke, A.P., O'Malley, P.G., Farb, A., Malcom, G.T., Smialek, J., and Virmani, R. A comparison of the Framingham risk index, coronary artery calcification and culprit plaque morphology in sudden cardiac death. *Circulation* (2000) 101:1243-1248

Thomas, R.J., King, M., Lui, K., Oldridge, N., Piña, I.L., Spertus, J., and ACC/AHA Task Force Members. AACVPR/ACC/AHA 2007 Performance measures on cardiac rehabilitation for referral to and delivery of cardiac rehabilitation/secondary prevention services. *Journal of Cardiopulmonary Rehabilitation and Prevention* (2007) 27:260-290

Vasan, R.S., Sullivan, L.M., Wilson, P.W.F., Sempos, C.T., Sundström, J., Kannel, W.B., Levy, D., and D'Agostino, R.B. Relative importance of borderline and elevated levels of coronary heart disease risk factors. *Annals of Internal Medicine* (2005) 142:393-402

Wilund, K.R. Is the anti-inflammatory effect of regular exercise responsible for reduced cardiovascular disease? *Clinical Science* (2007) 112:543-555

Woodside JV, McKinley MC, and Young IS. Saturated and trans fatty acids and coronary heart disease. *Current Atherosclerosis Rep.* 2008 Dec;10(6):460-6. Links World Health Organization, 2008