

Effects of a One-Year Comprehensive Lifestyle Intervention Program on Cardiovascular
Disease Risk in At-Risk Adults

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This thesis titled
Effects of a One-Year Comprehensive Lifestyle Intervention Program on Cardiovascular
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ABSTRACT

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The Effects of a One-Year Comprehensive Lifestyle Intervention Program on
Cardiovascular Disease Risk Profiles in At-Risk Adults

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A comprehensive lifestyle intervention program was developed to decrease risk factors for cardiovascular disease in at-risk adults who were employees at a university in rural Ohio. This worksite wellness program included diet therapy, exercise, behavior modification, and stress management (yoga). Few studies have examined the impact of comprehensive interventions on cardiovascular disease risk factors. Each participant completed a physical assessment including body composition analysis, blood work for glucose and lipid levels, and an exercise stress test that determined maximal oxygen uptake (baseline, 100 days, 1 year). Measurements were obtained using standardized methods. Seventy-four adults (57 females [77.1%]; 17 males [22.9%]) were participants. Repeated measures ANOVAs were used to assess for changes at baseline, 100 days, and 1 year of treatment. Body weight ($p < .001$), % body fat ($p < .001$), fat mass ($p < .001$), BMI ($p < .001$), waist circumference ($p < .001$), LDL cholesterol ($p = .011$), HDL cholesterol ($p = .035$), triglycerides ($p = .019$), blood glucose ($p = .008$), blood pressure (systolic, $p = .028$; diastolic, $p = .001$), and VO_{2max} ($p < .001$) significantly decrease. Lean body mass and total cholesterol did not significantly change ($p > .05$). This comprehensive lifestyle intervention program was effective in reducing cardiovascular disease risk factors after 1 year of treatment, however the most significant changes

occurred during the first 100 days of treatment. Future studies should examine increasing program compliance after 100 days.

Approved: _____

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CHAPTER 1: INTRODUCTION

Cardiovascular disease has been the leading cause of death in American adults for the past century (National Center for Health Statistics [NCHS], 2010). Despite declining mortality rates, burden of disease due to cardiovascular disease continues to be a public health concern. In 2007, cardiovascular disease was the underlying cause of death in one out of every three deaths and was cited as a possible contributor in 55.4% of all deaths in that year (Xu, Kochanek, Murphy, & Tejada-Vera, 2010). It is estimated that cardiovascular disease claims more than 2,200 American lives each day, and more people die each year from cardiovascular disease than from accidents, cancer, and chronic lower respiratory disease combined (Xu et al., 2010). According to the 2007 estimates, the total economic burden of cardiovascular disease was \$286 billion, including \$119 billion due to lost productivity and early mortality and \$167 billion for healthcare services and prescription medication (Roger et al., 2011). This accounts for 15% of total health expenditures, making cardiovascular disease the most costly disease in the United States (Agency for Healthcare Research Quality [AHRQ], 2007; Kashihara & Carper, 2009).

Cardiovascular disease, or heart disease, encompasses several different diseases and conditions, including hypertension, coronary heart disease, myocardial infarction, angina pectoris, heart failure, and stroke. Coronary heart disease (CHD) is both the most common and the deadliest form of cardiovascular disease in the United States and accounts for one in every six deaths (Roger et al., 2011; Rosamond et al., 2008). CHD is generally caused by an underlying condition called atherosclerosis. Atherosclerosis is a disease process characterized by the thickening and narrowing of the arteries due to the

accumulation of plaque in the arterial wall. The development of atherosclerotic plaque can result in impaired blood flow that can lead to arrhythmias, myocardial infarctions, heart failure and death. The health of the arterial wall endothelium plays an important role in cardiovascular health. A healthy, normally functioning endothelium regulates vasoconstriction and vasodilation. Damage to the endothelium can interfere with both the integrity of the arterial wall and the ability of the endothelium to function properly (Davignon & Glanz, 2004). This endothelial dysfunction can initiate or exacerbate atherosclerotic processes.

CHD and its underlying causes make up more than half of all clinically significant events in men and women under the age of 75 (Thom, Kannel, Silbershatz, & D'Agostino, 2001). However, it has been estimated that 70% or more of cardiovascular disease could be prevented if certain risk factors were modified (Folsom, Yamagishi, Hozawa, & Chambless, 2009; Lloyd-Jones et al., 2006; Stamler et al., 1999). The modifiable risk factors that have been clinically proven to influence cardiovascular health include diabetes, high blood pressure, hypercholesterolemia, overweight and obesity, physical activity, and smoking/tobacco exposure (Roger et al., 2011; Yusef et al., 2004). Other nonmodifiable risk factors that can influence cardiovascular disease include age, gender, family history, and race or ethnicity. Studies have shown the presence of multiple risk factors and the severity of each risk factor can act synergistically to increase an individual's chance of developing cardiovascular disease.

A number of intervention approaches have been posited for the prevention and treatment of cardiovascular disease. Since cardiovascular risk factors can act

synergistically to increase the risk for developing heart disease, interventions that focus on modifying multiple risk factors may be more effective in reducing risk than those that focus on only one risk factor at a time. The most recent statement by American Heart Association/American College of Cardiology, Guidelines for Secondary Prevention for Patients with Coronary and Other Atherosclerotic Vascular Disease, supports the use of aggressive, comprehensive cardiovascular risk reduction strategies (Smith et al., 2006). A compelling body of evidence confirms that a multidisciplinary approach, one which combines diet, exercise, and behavior change can improve survival, prevent or reduce recurrent events and procedures, and improve quality of life (Smith et al., 2006). Similar comprehensive approaches are also suggested for the primary prevention of cardiovascular disease (Grundy et al., 1998).

Although cardiovascular mortality rates have been declining for the past 10 years, the number of individuals living with certain cardiovascular risk factors has remained relatively steady or increased. Despite the statements and guidelines being published by professional organizations such as the American Heart Association, many cardiovascular interventions continue to focus on singular risk factors such as physical activity or specific dietary intakes.

The Risk Reduction Program (RRP) at Ohio University combines an individually-tailored exercise program, personalized dietary education and counseling, and a stress management strategy in the form of yoga classes to reduce the prevalence of cardiovascular risk factors in program participants. The program was designed for individuals with: a) lifestyle diseases, including coronary heart disease, high blood

pressure, diabetes and/or obesity; b) individuals with stable cardiovascular disease who have not previously participated in any cardiac rehabilitation program and c) other individuals with medical conditions that require structure and clinical supervision.

Study Objectives, Research Questions and Hypotheses

The purpose of this study was to determine the effectiveness of a comprehensive lifestyle intervention program which integrates dietary modification, physical activity, stress management, and behavior modification counseling to reduce the risk of clinically significant cardiovascular events in at-risk adults. More specifically, the purpose of this study was to evaluate the efficacy of the RRP to reduce cardiovascular risk factors in an at-risk adult population after 1 year of program participation. Table 1 summarizes the specific research questions and hypotheses associated with this study.

Table 1

Research Questions and Hypotheses

Research Questions	Hypotheses
1. What is the effect of the RRP on body weight, % body fat, lean weight, fat weight, body mass index (BMI) after 1 year of program participation (baseline, 100days, 1year)?	The RRP will have an initial positive effect on body weight, % body fat, lean weight, fat weight, BMI from baseline to 100 days but will have an attenuated effect from 100 days to 1 year of participation.
2. What is the effect of the RRP on HDL, LDL and total cholesterol levels, triglycerides, blood pressure, fasting glucose levels and maximal oxygen uptake (VO ₂) max after 1 year of program participation (baseline, 100 days, 1year)?	The RRP will have an initial positive effect on HDL, LDL and total cholesterol levels, triglycerides, blood pressure, fasting glucose levels and VO ₂ max from baseline to 100 days but will have an attenuated effect from 100 days to 1 year of participation.

Limitations of the Study

This study had several limitations:

1. There was no control group or alternative intervention group in this study.

Without any comparison groups were unable to determine which portion of the intervention was most effective in impacting cardiovascular risk factors.

2. Dietary intakes were self-reported, and dietary education and instruction were done on an individual basis. Therefore, initial dietary intakes and adherence to subsequent nutrition prescriptions were not be controlled.

3. The physical activity prescriptions were based on each individual's medical conditions, needs, and abilities. While frequency and duration were prescribed, they were not controlled, thus were unable to determine whether any dose-response effect occurred in program participants.
4. Participants' compliance with the program were not measured in this study. Without compliance data were unable to determine how effective the RRP was with regard to the adoption of long term lifestyle modifications.
5. While stress management in the form of yoga was incorporated into the program, there were no psychological or psychosocial measures utilized to determine the effectiveness of yoga classes in reducing stress.

CHAPTER 2: LITERATURE REVIEW

The purpose of this study was to determine the effectiveness of a comprehensive lifestyle intervention program which integrated dietary modification, physical activity, stress management, and behavior modification counseling to reduce the risk of clinically significant cardiovascular events in at-risk adults. More specifically, the purpose of this study was to evaluate the efficacy of the RRP to reduce cardiovascular risk factors in an at-risk adult population after 1 year of program participation.

Cardiovascular Disease: Overview

Cardiovascular disease, or heart disease, encompasses several different diseases and conditions, including hypertension, CHD, myocardial infarction, angina pectoris, heart failure, and stroke. CHD is both the most common and the deadliest form of cardiovascular disease in the United States and accounts for one in every six deaths (Roger et al., 2011; Rosamond et al., 2008). CHD is generally caused by an underlying condition called atherosclerosis. Atherosclerosis is a disease process characterized by the thickening and narrowing of the arteries due to the accumulation of plaque in the arterial wall. The development of atherosclerotic plaques can result in impaired blood flow that can lead to arrhythmias, myocardial infarction, heart failure, and death (Roger et al., 2011).

Despite declining mortality rates, burden of disease due to cardiovascular disease continues to be a public health concern. The most recent update released by the American Heart Association estimates that 82,600,000 Americans have at least one type of cardiovascular disease (Roger et al., 2011). It is estimated that cardiovascular disease

claims more than 2,200 American lives each day or approximately one death every 39 seconds (Xu et al., 2010). Cardiovascular disease accounts for more deaths in U.S. adults over the age of 35 than any other major disease. With the exception of Asian-Americans, cardiovascular disease is the leading cause of death for men and women of all ethnicities, and, in general, cardiovascular disease mortality increases with age (NCHS, 2010).

Atherosclerosis and Cardiovascular Disease

Atherosclerosis is a disease process characterized by the thickening and narrowing of the arteries due to the accumulation of plaques in the arterial wall. A normal, healthy arterial wall endothelium plays a key role in cardiovascular health by promoting normal vasoconstriction and vasodilation of the artery (Britten, Zeiher, & Schachiger, 1999). The healthy endothelium also maintains arterial wall integrity by promoting anticoagulant and anti-inflammatory responses (Davignon & Ganz, 2004). Damage to the endothelium causes endothelial dysfunction. Endothelial dysfunction can disrupt the balance between constriction and dilation, which initiates or exacerbates atherosclerotic processes (Davignon & Ganz, 2004).

Atherosclerosis begins as a fatty streak in the intima of the artery and can appear as early as adolescence (Stary et al., 1994). In the presence of endothelial dysfunction, atherosclerosis promotes inflammatory responses within the arterial wall. As the disease progresses, atherosclerotic lesions, or plaque, begin to form inside the artery. Plaques are composed of connective tissue elements, inflammatory cells, smooth muscle cells and endothelial cells, and are responsible for the ischemia associated with coronary artery disease (Stary et al., 1995). The cellular components of the atherosclerotic plaque are

surrounded by a fibrous, collagen-rich cap. This cap often ruptures, exposing prothrombotic material from the core of the plaque to the blood, and can result in myocardial infarction, heart failure or sudden death (Hansson, 2005).

A causal relationship exists between endothelial dysfunction and cardiovascular disease risk factors. Risk factors for cardiovascular disease often promote endothelial dysfunction by further aggravating atherosclerosis (Chambless et al., 1997). Some studies have linked smoking and hypertension to the development of atherosclerosis (Talukder et al., 2010; Yusef et al., 2004). Results from the Multi-Ethnic Studies of Atherosclerosis (MESA) have shown that cardiovascular disease risk factors including high plasma cholesterol, high blood lipid levels, obesity, and poor dietary intake (e.g., decreased fruit, vegetable, whole grain and unsaturated fatty acid intake; increased sugar, sodium and saturated fat intake) are associated with increased atherosclerotic incidence (Nettleton et al., 2007).

Cardiovascular Disease Risk Factors

Certain medical conditions and lifestyle choices can increase the risk for developing cardiovascular disease. To be considered a risk factor, a medical condition or lifestyle choice must meet specific criteria identified by the American Heart Association, including having an independent and statistically significant association based on studies that have large numbers of outcome events (Greenland et al., 2010). The American Heart Association recognizes the following conditions and lifestyle choices as cardiovascular disease risk factors: high cholesterol; high blood pressure; impair fasting glucose or type 2 diabetes; overweight and obesity; physical inactivity; poor diet; and, cigarette smoking

(Lloyd-Jones et al., 2010). A number of large population-based studies including The Framingham Heart Study (1987, 2006), the Atherosclerosis Risk In Communities study (1997), and The INTERHEART study (2004) have established that risk factors can be used as predictive measures to estimate the risk of developing cardiovascular disease (Chambless et al., 1997; Fox et al., 2006; Wilson, Castelli, & Kannel, 1987; Yusef et al., 2004). Evidence from such studies has been used to help develop the generally accepted methods of assessing cardiovascular disease risk.

The American Heart Association Strategic Planning Task Force and Statistics Committee stated an ambitious goal: to improve the cardiovascular health of all Americans by 20%” by 2020 (Lloyd-Jones et al., 2010). To help achieve this goal, the committee defined the concept of ideal cardiovascular health. Ideal cardiovascular health includes seven metrics: current smoking habits, body mass index, physical activity habits, dietary patterns, total cholesterol, high blood pressure, and fasting plasma glucose (Lloyd-Jones et al., 2010). Together, these seven metrics are considered risk factors for cardiovascular disease. Achieving healthy behaviors (as measured by the seven metrics) is associated with decreased risk of developing cardiovascular disease, decreased morbidity from cardiovascular disease, decreased healthcare expenditures, and an increased quality of life (Lloyd-Jones et al., 2010).

In a recent survey, the Behavioral Risk Factor Surveillance System was used to estimate the prevalence of four of the ideal cardiovascular health metrics. Specifically, the survey examined the prevalence of nonsmoking, maintenance of a healthy body mass index, physical activity level, and dietary habits among American adults. According to

the results of the survey only 3% of the population met all four metrics (Reeves & Rafferty, 2005). Experts believe that avoiding or modifying certain risk factors could potentially eliminate $\geq 70\%$ of cardiovascular disease in the United States (Folsom, Yamagishi, Hozawa, & Chambless, 2009; Lloyd-Jones et al., 2006; Stamler et al., 1999). Table 2 summarizes the recommendations for ideal cardiovascular health as defined by the American Heart Association.

Table 2

Recommendations for Ideal Cardiovascular Health

Risk factor	Ideal cardiovascular health recommendations*
Smoking	Never smoked or quit >12 ago
Body mass index	< 25 kg/m ²
Physical activity	≥ 150 min/wk moderate intensity or ≥ 75 min/wk vigorous intensity or combination
Total cholesterol	< 200 mg/dL
Healthy diet	4-5 of the following: <ul style="list-style-type: none"> • ≥ 3, 1-oz equivalent servings of fiber-rich whole grains/day • ≥ 4.5 cups of fruits and vegetables/day • < 1500 mg sodium/day • 2, 3.5-oz servings of oily fish/week • ≤ 450 kcals of sugar sweetened beverages per week
Blood pressure	< 120/80 mmHg
Fasting plasma glucose	< 100 mg/dL

Note. From “Defining and Setting National Goals for Cardiovascular Health Promotion and Disease Reduction: The American Heart Association's Strategic Impact Goal Through 2020 and Beyond,” by D. M. Lloyd-Jones et al., 2010, *Circulation*, 121(4), p. 591. Copyright 2010 by Wolters Kluwer Health. Adapted with permission. * Recommendations are for adults ≥ 20 years of age.

Cholesterol

Elevated serum cholesterol levels (hypercholesterolemia) are strongly associated with coronary heart disease and an increased risk of adverse cardiovascular events

(Castelli, 1984; Greenland et al., 2003; Rosamond et al., 2008). The reduction of serum cholesterol levels is a critical step in the prevention and treatment of cardiovascular disease. The American Heart Association recommends a total cholesterol level of < 200 mg/dL for ideal cardiovascular health (Lloyd-Jones et al., 2010). For U.S. adults, the average cholesterol level is 200 mg/dL, and approximately 33.5 million adults have high cholesterol (Centers for Disease Control and Prevention [CDC], 2010). Other cardiovascular risk factors can negatively affect cholesterol level such as smoking, overweight and obesity, physical inactivity, poor dietary habit and impaired blood glucose (National Heart, Lung, and Blood Institute [NHLBI] Adult Treatment Panel III, 2002). It is important to first understand the normal biological functions of cholesterol within the human body before trying to examine the pathophysiology of hypercholesterolemia.

Cholesterol is type of lipid, and it has many essential physiological roles in the body. Cholesterol is a necessary component of cell membranes, and it is a precursor for steroid-based hormones such as estrogen and testosterone (Cox & Garcia-Palmieri, 1990; Maxfield & Tabas, 2005). Cholesterol is synthesized within the body; it is also absorbed from the diet via the ingestion of animal products (Nelms, Sucher, Lacey, & Long Roth, 2011). As a hydrophobic molecule, cholesterol must bind to protein and form a lipoprotein in order to be transported through the bloodstream. A lipoprotein is a compound comprised of a lipid core enveloped by a shell of cholesterol, phospholipids and protein. There are four major classes of lipoproteins: a) chylomicrons; b) very low-

density lipoproteins (VLDL); c) low-density lipoproteins (LDL); and d) high-density lipoproteins (HDL).

Chylomicrons are synthesized by intestinal cells after the consumption of dietary fat and are responsible for the packaging and transport of dietary fat. Chylomicrons are transported via the lymphatic system and eventually are transported to the liver, which uptakes the cholesterol they contain (NHLBI Adult Treatment Panel III, 2002). There is some evidence that partially degraded chylomicrons, or chylomicron remnants, may carry atherogenic potential (NHLBI Adult Treatment Panel III, 2002).

VLDL cholesterol is a triglyceride-rich molecule that is synthesized by the liver and makes up 10-15% of the total serum cholesterol (NHLBI Adult Treatment Panel III, 2002). Certain forms of VLDL, called VLDL remnants, appear to promote atherosclerosis by mechanisms similar to those of low density lipoproteins. The apolipoproteins associated with VLDL are apo B-100, Apo C I, II and II and apo E (NHLBI Adult Treatment Panel III, 2002). Apolipoproteins are the protein component of lipoproteins. While their main function is lipid transport, many apolipoproteins have specialized functions. Apo B-100, for example, plays an important role in cholesterol homeostasis where high plasmas levels of Apo B-100 have been strongly correlated with atherosclerosis (Li, Tanimura, Luo, Datta, & Chan, 1988).

LDL cholesterol makes up 60-70% of the total serum cholesterol and contains only one apolipoprotein, Apo B-100 (NHLBI Adult Treatment Panel III, 2002). LDL cholesterol transports cholesterol to cells or gets taken up and repackaged by the liver (Cox & Garcia-Palmieri, 1990). LDL cholesterol is the primary atherogenic lipoprotein.

A specific type of white blood cell, known as a scavenger cell, embedded within the arterial endothelium can absorb LDL cholesterol, particularly oxidized forms of LDL cholesterol. In the presence of elevated LDL levels, excess cholesterol accumulates inside the arterial cell wall and eventually leads to the formation of atherosclerotic plaques (Shashkin, Dragulev, & Ley, 2005). Several studies have found a direct relationship between the level of LDL cholesterol and the rate of new-onset coronary heart disease. LDL cholesterol levels ≥ 100 mg/dL appear to promote atherosclerosis (Lipid Research Clinics Coronary Primary Prevention Trial 1984; NHLBI Adult Treatment Panel III, 2002; Stamler, Wentworth, & Neaton, 1986).

The final class of lipoproteins is HDL cholesterol, which is synthesized by the liver and intestinal cells and comprises 20-30% of total serum cholesterol. Apo A-I and apo A-II are the major apolipoproteins in HDL cholesterol which together make up 90% of circulating HDL (Cox & Garcia-Palmieri, 1990; NHLBI Adult Treatment Panel III, 2002). HDL cholesterol functions to uptake excess cholesterol from the bloodstream and transports it back to the liver for excretion (Cox & Garcia-Palmieri, 1990). This lipoprotein also functions to remove cholesterol from the membranes of peripheral cells that are attached to the endothelial wall. Although this is a complex process that is still not clearly characterized, Apo A-I and A-II proteins are thought to be the major facilitators of this uptake (Kontush & Chapman, 2006). HDL cholesterol is considered a protective factor or negative risk factor; serum HDL cholesterol levels of ≥ 60 mg/dL are recommended for ideal cardiovascular health (NHLBI Adult Treatment Panel III, 2002).

Several studies have linked hypercholesterolemia and the development of endothelial dysfunction, a pivotal factor in the pathophysiology of atherosclerosis (Zeiber, Drexler, Wollschläger, & Just, 1991; Zeiber, Schächlinger, Hohnloser, Saurbier, & Just, 1994). Elevated serum levels of cholesterol appear to inhibit the ability of the endothelium to regulate blood flow through the coronary arteries. Specifically, the endothelium cannot properly dilate in response to normal hormonal stimuli that signal the need for increased blood flow through the coronary arteries (Zeiber et al., 1991). The extent of the endothelial dysfunction, i.e., the extent to which vasodilation is impeded, is directly proportional to total serum cholesterol levels. Hypercholesterolemia is determined by using a multivariate analysis of the total cholesterol present in the blood serum. Total cholesterol includes HDL and LDL cholesterol as well as an intermediate form of cholesterol (NHLBI Adult Treatment Panel III, 2002). Table 3 summarizes the definition of cholesterol level as outlined by the National Heart, Lung, and Blood Institute's National Cholesterol Education Program (NCEP) Adult Treatment Panel III.

Table 3

Adult Treatment Panel III Classification of Serum Cholesterol Levels

Cholesterol Type	Classification (mg/dL)		
Total cholesterol	Desirable <200 Borderline High 200-239 High \geq 240		
HDL Cholesterol	Desirable \geq 60 Low > 40		
LDL Cholesterol	CHD/CHD RE <100	(2+) RF <130	(0-1) RF <160

Note. From “Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adult (Adult Treatment Panel III) final report” by National Heart, Lung, and Blood Institute, Adult Treatment Panel III, 2002, *Circulation*, 106(25) p. 3143. Copyright 2002 by Wolters Kluwer Health. Reprinted with permission. HDL > 60mg/dL = Negative risk factor; CHD = Cardiovascular disease; RE = Risk Equivalents; RF = Risk Factors.

Desirable levels for LDL cholesterol are based on three categories of risk for cardiovascular disease: a) established CHD or CHD risk equivalents; b) multiple (2+) cardiovascular risk factors; and, c) 0-1 cardiovascular risk factors. Individuals with diagnosed CHD, other cardiovascular disease, type 2 diabetes and a Framingham 10-year risk score > 20% are considered to be at greatest risk. Risk factors assessed for the remaining categories included family history of premature CHD, hypertension, low HDL cholesterol levels, and smoking habits. It should be noted that HDL cholesterol is sometimes considered a negative risk factor, meaning that it is protective against cardiovascular disease. If an individual has an HDL cholesterol level \leq 60 mg/dL a risk factor is subtracted from the risk factor count (NHLBI Adult Treatment Panel III, 2002).

In contrast to the adverse health effects associated with elevated serum levels of total and LDL cholesterol, elevated levels of HDL cholesterol are associated with a decreased risk of cardiovascular disease (NHLBI Adult Treatment Panel III, 2002). Epidemiological studies have shown that a 1 mg/dL increase in HDL cholesterol is associated with a 2-3% decrease in risk for cardiovascular disease (Assmann & Schulte, 1987; Gordon, Kannel, Castelli, & Dawber, 1981). HDL exerts a protective effect against cardiovascular disease through reverse cholesterol transport, a process which removes cholesterol from the arterial wall thus preventing the build-up of atherosclerotic plaques (Linsel-Nitschke & Tall, 2005; Podrez, 2010). HDL and its associated apolipoproteins, Apo A-I, A-II, E and J, have anti-oxidant properties that help to prevent the oxidation of LDL cholesterol. The apolipoproteins, especially Apo A-I, are thought to maintain the structure of LDL cell walls through binding activities and the absorption of lipid-derived free-radical agents (Navab et al., 2001). These processes prevent oxidation and promote uptake of LDL cholesterol by scavenger cells within the arterial wall, thus preventing the formation of atherosclerotic plaque (Zerrad-Saadi, 2009).

Serum triglycerides have also been directly linked cardiovascular disease. Recent research has found that elevated serum triglycerides are an independent risk factor for cardiovascular disease, indicating that some triglyceride-rich lipoproteins may promote atherosclerotic events (Austin, Hokanson, & Edwards, 1998). Triglyceride-rich lipoproteins are small, cholesterol rich particles, such as remnant VLDL and intermediate density lipoproteins, which have properties similar to LDL cholesterol lipoprotein. Elevated triglycerides are generally the result of other cardiovascular risk factors and can

indicate the presence of one or more of these risk factors including overweight and obesity, physical inactivity, poor dietary habits, and smoking. Elevated serum triglyceride levels are also linked to low level of HDL cholesterol (NHLBI Adult Treatment Panel III, 2002). The recommended level for serum triglycerides is < 150mg/dL (NHLBI Adult Treatment Panel III, 2002).

Oxidative stress, also called chronic inflammation, is an inflammatory response that creates a pro-oxidative environment and plays a key role in the pathophysiology of atherosclerosis. Oxidative stress is associated with endothelial dysfunction. The presence of endothelial dysfunction causes enhanced platelet adhesion and increased permeability of the arterial wall (Kontush & Chapman, 2006). These physiological changes initiate the leukocyte adhesion cascade, a complicated process that ultimately leads to the accumulation of cholesterol in the arterial wall (Hulsmans & Holvoet, 2010). High serum concentrations of HDL cholesterol, specifically its associated apolipoproteins, Apo A-II, A-II and A-IV, work to inhibit adhesion and the cytokine-induced production of adhesion-specific molecules (Cockerill, Rye, Gamble, Vadas, & Barter, 1995; Kontush & Chapman, 2006). HDL cholesterol is also linked with anti-thrombotic properties due to its ability to stimulate the release of nitric oxide, an important endothelium-derived vasodilator (Arnal, Dinh-Xuan, Pueyo, Darblade, & Rami, 1999). Nitric oxide has also been shown to inhibit platelet aggregation and leukocyte adhesion within the arterial wall (Arnal et al., 1999). Low levels of circulating HDL cholesterol are often an indication of cardiovascular disease because the many anti-atherogenic effects associated with HDL are inhibited. Other cardiovascular risk factors

such as overweight and obesity, physical inactivity, poor dietary habits, poorly controlled type 2 diabetes, and smoking can contribute to low serum HDL cholesterol levels (NHLBI Adult Treatment Panel III, 2002). To achieve full benefit of the anti-atherogenic properties of HDL cholesterol, serum triglycerides levels need to be reduced to < 150 mg/dL, total cholesterol levels need to be reduced to 150 mg/dL and HDL cholesterol levels should not be below 60 mg/dL (NHLBI Adult Treatment Panel III, 2002).

Recommendations for lowering cholesterol target the underlying causes of high cholesterol such as overweight and obesity, physical inactivity, poor dietary habits, poor blood glucose control, and smoking. Most therapies are specifically targeted at lowering LDL cholesterol levels as this can slow or even reverse the atherosclerotic process, and thus reduces the risk of complications from cardiovascular disease (Hodis et al., 1994). The presence of excess body fat is associated with overweight, obesity and type 2 diabetes is directly linked to high serum triglyceride levels and low HDL cholesterol levels. Weight loss through physical activity and complimentary diet therapy is recommended in order to improve body composition and serum cholesterol levels (Fletcher et al., 2005; Schaefer et al., 1995). Participation in regular physical activity has been shown to have a positive overall effect on cholesterol level, specifically by reducing LDL cholesterol levels and increasing HDL cholesterol levels (Durstine & Thompson, 2001). Diets high in cholesterol and saturated fat coupled with low mono- and polyunsaturated fatty acid intake has also been linked to hypercholesterolemia (de Lorgeril et al., 1997). Dietary interventions that encourage increased intake of “heart healthy” foods, such as whole grains, fruit and vegetables, nuts, mono- and

polyunsaturated fatty acids (20% and 10% of total fat intake, respectively), as well as a total fat intake of less than 30% of total calories with < 10% from saturated fat and less than 200 mg cholesterol, can help reduce cholesterol levels. This dietary pattern has been shown to reduce total cholesterol, LDL cholesterol, and triglyceride levels and to increase HDL cholesterol levels (Fletcher et al., 2005; NHLBI Adult Treatment Panel III, 2002). Smoking has also been linked to poor blood lipid profiles, specifically with high triglyceride and low HDL levels. When combined with other cardiovascular risk factors, smoking appears to have a synergistic effect on the endothelial dysfunction. Smoking cessation is associated with the normalization of blood lipids and with improved endothelial function (Novello, 1990; Puranik & Celermajer, 2003).

The reduction and management of cholesterol levels is an important component of risk reduction and cardiovascular health. The overlap observed between hypercholesterolemia and other cardiovascular risk factors allows for concurrent treatment and reduction of risk. Engaging in regular physical activity, eating a heart healthy diet, maintaining a healthy body composition, abstaining from or quitting smoking, and properly controlling blood glucose levels can improve cholesterol and synergistically reduce the risk of cardiovascular disease (Lichtenstein et al., 2006; Lloyd-Jones et al., 2010).

Blood Pressure

Blood pressure is defined as the force exerted by blood against arterial walls as it is pumped through the body by the heart (NHLBI, 2011). Blood pressure is comprised of two different measures of pressure, systolic pressure and diastolic pressure. Systolic

pressure refers to the amount of force exerted on the arterial wall as the heart beats and pumps blood; diastolic pressure refers to the amount of force exerted on the arterial wall when the heart is at rest between beats. Fluctuation in blood pressure is normal; however, if blood pressure is chronically elevated it can have negative repercussions on cardiovascular and overall health (NHLBI, 2011).

Population level analyses have indicated that approximately one in three U.S. adults has high blood pressure, (hypertension), the single largest risk factor for cardiovascular mortality (Danaei et al., 2009; Fields et al., 2004; Lopez, Mathers, Ezzati, Jamison, & Murray, 2006). Hypertension is often referred to as the “silent killer” because the condition is generally asymptomatic. Elevated resting blood pressure, positive family history, central obesity, and physical inactivity serve as predictors for hypertension (Chobanian et al., 2003). Table 4 summarizes the classifications for blood pressure levels as outlined by the Seventh Report of the Joint National Committee of Prevention, Detection, Evaluation and Treatment of High Blood Pressure (Chobanian et al., 2003).

Table 4

Seventh Joint National Committee Classification of Blood Pressure Levels

Blood pressure classification	Systolic (mm Hg)	Diastolic (mm Hg)
Normal	< 120	And < 80
Pre-hypertension	120-139	Or 80-89
Hypertension, Stage 1	140-159	Or 90-99
Hypertension, Stage 2	≥ 160	Or ≥ 100

Note. From “the Seventh Report of the Joint National Committee of Prevention, Detection, Evaluation and Treatment of High Blood Pressure,” by A. Chobanian et al., 2003, *Hypertension*, 289(19), p. 1211. Copyright 2003 by Wolters Kluwer Health. Adapted with permission. Adults = ≥ 18 years of age.

A continuous, consistent, and independent relationship exists between blood pressure and cardiovascular risk, meaning that regardless of other risk factors, as blood pressure levels increase, so too does the risk for cardiovascular disease (Chobanian et al., 2003). Results from the Framingham Heart Study also indicate that individuals with only slightly higher than normal systolic or diastolic blood pressure (130-130 mmHg and 85-89 mmHg, respectively) are at greater risk for a cardiovascular event than an individual with optimal blood pressure (Vasan et al., 2002). Approximately 47% of cardiovascular events worldwide can be attributed to high blood pressure. However, roughly half of these events occur in individuals with systolic pressure below 145 mm Hg, indicating that even small elevations in blood pressure have the potential to negatively impact health (Lawes, Vander Hoorn, & Rodgers, 2008).

In the United States, an estimated 76.4 million adults over the age of 20 have high blood pressure, and 29% of those individuals are hypertensive (Ostchega, Yoon, Hughes,

& Louis, 2008; Roger et al., 2011). Risk factors for high blood pressure include age (51 years or older), excessive sodium intake, dyslipidemia, obesity, physical inactivity, and smoking (Rosendorff et al., 2007). Age is a non-modifiable risk factor for elevated systolic blood pressure. Between the ages of 1-18 years, systolic blood pressure increases, on average, by 1.7 mm Hg per year and 0.6 mm Hg every year after the age of 18 (Appel, 2008). Due to the strong relationship between blood pressure and age, it is estimated that 90% of adults will be hypertensive within their lifetime (Vasan, 2002).

Consumption of excessive amounts of sodium is a modifiable risk factor that has been linked to elevated blood pressure levels and endothelial dysfunction (Rosendorff et al., 2007). Sodium is usually consumed as sodium chloride and is an essential nutrient. Sodium functions in key roles to maintain fluid volume and osmolarity within the human body (Food and Nutrition Board, Institute of Medicine, 2006b). When consumed, sodium induces a dose-dependent rise in blood pressure. If consumed in excess this elevation of blood pressure contributes significantly to hypertension (Appel et al., 2011). Certain subsets of the population are more susceptible to sodium-induced elevation in blood pressure. The special populations include older individuals, individuals with hypertension, diabetes, or chronic kidney disease, and African Americans, who tend to exhibit greater sensitivity to sodium than the general population (Cappuccio & Markandu, 1997; Fields et al., 2004; Klag et al., 1996).

Studies have shown that increases in blood pressure due to sodium consumption are a factor in endothelial dysfunction. This process appears to be exacerbated by smoking, oxidative stress, and dyslipidemia, a condition characterized by elevated LDL

cholesterol and triglyceride levels and low HDL cholesterol levels (Rosendorff et al., 2007). Atherosclerotic plaque accumulates in the arteries over time disrupting normal vasodilation and constriction. As plaque accumulation continues it begins to impede the flow of blood through the artery, which often results in increased cardiac workload thus further exacerbating high blood pressure (Arnal et al., 1999).

Treatment options for hypertension often focus on lifestyle interventions. Weight reduction, the adoption of the Dietary Approaches to Stop Hypertension (DASH) eating plan, and regular physical activity have all been shown to independently decrease blood pressure and reduce the risk for cardiovascular disease (Chobanian et al., 2003).

Pharmacological therapy is another option for the treatment of hypertension, however the antihypertensive effects of pharmaceutical therapies are enhanced when coupled with diet therapy and physical activity. The synergistic effect observed by combining interventions elicits greater reductions in cardiovascular risk and thus the Joint National Committee of Prevention, Detection, Evaluation, and Treatment of High Blood Pressure recommends combining two or more lifestyles modifications (Chobanian et al., 2003).

Because excess sodium intake is directly related to increased blood pressure, limiting sodium consumption may be an effective therapy (Chobanian et al., 2003). The recommendation for sodium intake is < 2300 mg/day for the general population and < 1500 mg/day older adults (51 year and older), African Americans, and individuals with hypertension, diabetes, or chronic kidney disease (CDC, 2009). In a study of nonmedicated stage 1 and stage 2 hypertensive adults, adherence to these guidelines

alone results in a 12 mm Hg decrease of systolic blood pressure and a 6 mm Hg decrease of diastolic blood pressure (Kojuri & Rahimi, 2007).

Weight reduction in overweight and obese individuals is also associated with decreased blood pressure. While the overall goal of weight reduction should be obtaining a health body mass index (BMI; 18.4 -24.9 kg/m²), modest weight loss can achieve reduction in blood pressure (Appel et al., 2009; Trials of Hypertension Collaborative Research Group, 1997). Studies have shown that weight loss of 4.5 pounds can result in a 5-20 mm Hg reduction in systolic blood pressure and that greater weight loss results in further reductions of blood pressure (Appel et al., 2009; Trials of Hypertension Collaborative Research Group, 1997).

Participating in regular physical activity has also been shown to reduce blood pressure, even in individuals without hypertension. The reduction seen in nonhypertensive individuals suggests that regular physical activity may be the only therapy needed in order to reduce blood pressure in prehypertensive individuals (Thompson et al., 2003). The American College of Sport Medicine (2004) recommends a minimum of 30 minutes of aerobic activity 3-5 days per week in order to achieve blood pressure lowering effects (Pescatello et al., 2004).

High blood pressure is the single largest and most importantly, modifiable risk factor associated with cardiovascular mortality (Danaei et al., 2009). This makes hypertension a critical target for risk reduction. Once again, an overlap is seen between hypertension and other cardiovascular risk factors which allows for concurrent treatment and reduction of risk. Engaging in regular physical activity, eating a heart healthy diet

that limits sodium, and maintaining a healthy body composition can improve blood pressure levels and synergistically reduce the risk of cardiovascular disease (Lichtenstein et al., 2006; Lloyd-Jones et al., 2010).

Overweight and Obesity

According to the CDC, 68% of American adults, ages 20-74, are overweight or obese (Flegal, Carroll, Ogden, & Curtin, 2010). Overweight and obesity are the cause of death in approximately 1 out of every 10 deaths in the United States, and in individuals under the age of 70 these conditions are responsible for more deaths than high blood pressure (Danaei et al., 2009). The widespread prevalence of overweight and obesity has obtained the moniker of an “epidemic,” because these conditions are a serious threat to public health. All obese adults are considered at risk for developing cardiovascular. Obesity is an independent risk factor for cardiovascular disease, but it often presents with other cardiovascular risk factors including type 2 diabetes, hypertension, and hypercholesterolemia (Gregg et al., 2005; NHLBI, 1998).

Overweight and obesity are characterized by an excessive amount of adipose tissue in the body (Salans, Cushman, & Weismann, 1973). According to the NHLBI, obesity is a complex chronic disease that develops from the interactions of multiple influential factors such as genetic predisposition, behavioral factors, and environmental factors (NHLBI, 1998). The multifactorial nature of obesity is important because it highlight the challenges associated with treating this condition. It has been show that body composition is partially based on genetics and that some individuals are predisposed to excess weight gain. Recent studies have estimated that 25 to 40% of individual

difference in body composition can be explained by genetic differences (Bouchard, Pérusse, Leblanc, Tremblay, & Thériault, 1988; Vogler, Sørensen, Stunkard, Srinivasan, & Rao, 1995). However, the extent to which this genetic trait is expressed is not entirely clear and it can be influenced by the presence or absence of environmental factors (Farooqi & O'Rahilly, 2006; NHLBI, 1998).

Overweight and obesity can also be characterized as an imbalance between energy intake (kcal consumed) and energy output (kcal expended). This energy imbalance has been attributed to a number of factors including the increased availability of energy dense foods and decreased necessity for physical exertion in daily life (Farooqi & O'Rahilly, 2006). Increased energy intake should be coupled with an increase in physical activity in order to maintain the energy balance. However, according to the American Heart Association less than 1% of U.S. adults over the age of 20 adhere to an ideal healthy diet (Lloyd-Jones et al., 2010). Studies have also shown that the majority of the U.S. population does not participate in the amount of physical activity recommended to prevent weight gain (Hill & Wyatt, 2006). This imbalance of energy generally results in the excess fat deposition and eventually overweight and obesity.

Originally, fat tissue, or adipose tissue, was assumed to function as a site for energy storage in the body. However, the functions of adipose tissue are much more complex than simple energy storage. Adipose tissue appears to function much like an endocrine system with fat cells, also called adipocytes, secreting hormones to regulate several processes such as inflammation, energy balance, lipid metabolism, and insulin sensitivity (Gurevich-Panigrahi, Panigrahi, Wiechec, & Los, 2009; Rokling-Andersen et

al., 2007). The size and location of an adipocyte determines the type and amount of hormonal secretion that occurs. For example, the accumulation of adipose tissue in the abdominal region has a negative effect on metabolic function and increases the risk of cardiovascular disease (Klein et al., 2004). Abdominal adipose tissue accumulation is associated with abnormal lipid metabolism, impaired insulin function, and high blood pressure. The metabolic dysfunction associated with abdominal obesity can further contribute to the development of hypercholesterolemia, type 2 diabetes, hypertension, and, in turn, cardiovascular disease (Kissebah et al., 1982; Klein et al., 2004).

Overweight and obesity, particular abdominal or central adiposity, can be accompanied by a specific cluster of metabolic abnormalities that increase the risk of type 2 diabetes and cardiovascular disease. This particular characterization of overweight and obesity is known as the metabolic syndrome. Risk factors associated with the metabolic syndrome include abnormal body fat distribution in the form of central obesity, atherogenic dyslipidemia, insulin resistance, elevated blood pressure, presence of a pro-inflammatory state as evidenced by elevated C-reactive protein levels, and the presence of a prothrombotic state (Alberti, Zimmet, & Shaw, 2006). The use of the term “syndrome” implies that these factors tend to occur together more often than they occur individually. Table 5 summarizes the currently accepted diagnostic criteria for the metabolic syndrome. Although there is some controversy surrounding the exact definition and diagnostic criteria for the metabolic syndrome, according to the Joint Scientific Statement from the International Diabetes Federation Task Force on Epidemiology, NHLBI, American Heart Association, International Atherosclerotic

Society and International Association for the Study of Obesity, the presence of at least three of the five criteria outlined in Table 5 indicate a positive diagnosis of the metabolic syndrome (Alberti et al., 2009).

Table 5

Criteria for Diagnosis of the Metabolic Syndrome

Measure	Categorical cut points
Elevated waist circumference	≥ 102 cm for males; ≥ 88 cm for women
Elevated triglycerides	≥ 150 mg/dL or drug treatment for elevated triglycerides
Low HDL cholesterol levels	< 40 mg/dL in males; < 50 mg/dL in females; or drug treatment for reduced HDL levels
Elevated blood pressure	Systolic ≥ 130 and/or diastolic ≥ 85 mm Hg or use of antihypertensive medication
Elevated fasting glucose	≥ 100 mg/dL or drug treatment for elevated glucose

Note. From “Harmonizing the Metabolic Syndrome: A Joint Scientific Statement from the International Diabetes Federation Task Force on Epidemiology and Prevention, NHLBI, American Heart Association, World Heart Federation, International Atherosclerotic Society and International Association for the Study of Obesity,” K. Alberti et al., 2009, *Circulation*, 120(16), p. 1642. Copyright 2009 by Wolters Kluwer Health. Reprinted with permission. Categorical cut points for waist circumference determined by AHA/NHLBI; HDL = high density lipoprotein.

Body Composition

A direct relationship exists between the amount of abdominal visceral fat and waist circumference. Elevated waist circumference is also associated with potentially atherogenic metabolic abnormalities thus making it important to consider when assessing risk for cardiovascular disease (Ferland et al., 1989; Pouliot et al., 1994). Waist circumferences great than 102 cm (≥ 40 inches) in men and greater than 88 cm (≥ 35 inches) in women are associated with increased risk for developing cardiovascular diseases and other comorbidities associated with obesity (NHLBI, 1998). BMI is another measurement often used to assess overweight and obesity status. BMI is a measure of height and weight that is significantly correlated with total adiposity and it is the measurement used most frequently to assess and define body composition (Farooqi & O'Rahilly, 2006). BMI is generally calculated as weight (kg) divided by height² (m). Overweight is defined as a BMI between 25 and 22.9 kg/m² and obesity is defined as a BMI ≥ 30 kg/m² (NHLBI, 1998). Table 6 summarizes the complete list of BMI classifications as defined by the NHLBI.

Table 6

Classification of Overweight and Obesity in Adults

Weight classification	Obesity class	BMI (kg/m ²)
Underweight		< 18.5
Normal		18.5-24.9
Overweight		25.0-29.9
Obesity	I	30.0-34.9
Obesity	II	35.0-39.9
Extreme Obesity	III	≥ 40

Note. Adapted From “Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults,” by the Nation Heart, Lungs and Blood Institutes Expert Panel on the Identification, Evaluation and Treatment of Overweight in Adults, 1998, *American Journal of Clinical Nutrition*, 68(4), p. xiv. Copyright 1998 by American Society for Nutrition. Reprinted with permission.

BMI is the most frequently used measurement of weight status due to the inexpensive nature of collecting height and weight measurements and the overall ease of use (Farooqi & O’Rahilly, 2006). BMI classification and waist circumference can also be used monitor changes in body composition when assessing or treating cardiovascular disease and overweight and obesity (NHLBI, 1998). Greater BMI classification is generally associated with greater adiposity in the body and thus indicates that individuals may be at greater risk for cardiovascular disease. Population-based studies have indeed indicated a relationship between increased BMI classification and increased risk of cardiovascular mortality in both men and women (Flegal et al., 2010; Must et al., 2004).

BMI is an effective and inexpensive measure with which to assess body composition, however this measure cannot describe the exact amount or distribution of adipose tissue and both factors are important in order to assess health risks related to overweight and obesity accurately (NHLBI, 1998). BMI is an indirect measure and should be used in combination with other more direct measures of body composition to achieve more accurate assessments of overweight and obesity (NHLBI, 1998).

To accurately assess adiposity, percent body fat should be determined via body composition analysis. Percent body fat is particularly important when assessing weight loss or weight gain in overweight and obese individuals. Different methods of body composition analysis exist including hydrostatic weighing, dual energy X-ray absorptiometry, magnetic resonance imaging and air displacement plethysmography (Fields et al., 2004). These methods are commonly utilized and are considered accurate methods by which to determine body composition. It should be noted that while these are commonly used methods, few of these are appropriate for obese individuals with a BMI of $\geq 35 \text{ kg/m}^2$ (Das et al., 2003).

Hydrostatic weighing and air displacement plethysmography are two methods that are considered acceptable for assessing the body composition of individuals with a BMI of $\geq 35 \text{ kg/m}^2$ accurately. Hydrostatic weighing requires the complete submersion of an individual in a larger tank of water and this can be a physically demanding procedure (Biaggi et al., 1999). This method is not appropriate for individuals who cannot meet the physical demands of the weighing procedure. Air displacement plethysmography, however, is much less physically demanding and thus accessible to a wider range of

individuals. It has been shown to be a reliable and valid method for determining body composition (Biaggi et al., 1999; Fields et al., 2004; Ginde et al., 2005). In order to utilize air displacement plethysmography, a piece of equipment known commercially as the BOD POD is required (Life Measurement Inc., Concord, CA, USA). If BMI and body composition including percent body fat, fat mass, and lean mass are determined, body weight can be utilized as an intermediate measure to assess changes in body composition (NHLBI, 1998).

Defining and understanding measures of excess body fat are an important part of the health assessment process. Overweight and obesity have been directly linked with a higher prevalence of hypertension, type 2 diabetes, and hypercholesterolemia, all of which are risk factors for cardiovascular disease (Lloyd-Jones et al., 2010). Overweight and obesity generally present with multiple comorbidities and these comorbidities are often independent risk factors for cardiovascular disease. The presence of these multiple risk factors as well as the severity of each risk factor act synergistically to increase the risk for cardiovascular disease (Lloyd-Jones et al., 2010).

Overweight and obesity generally present with multiple cardiovascular risk factors. The treatment of overweight and obesity focuses on both weight loss and the reduction of any exhibited cardiovascular risk factors. Reducing these risk factors will reduce the risk for cardiovascular disease regardless of whether or not weight loss is achieved (NHLBI, 1998). However, studies have proven that treating cardiovascular risk factors and concurrent weight reduction can further improve cardiovascular health.

Specifically, weight loss can lower blood pressure, serum triglycerides, total cholesterol, and LDL cholesterol and can increase HDL cholesterol (Klein et al., 2004).

According to the NHLBI (1998), the general goals of weight reduction are to: a) reduce total body weight; b) maintain a lower body weight long term; and, c) prevent further weight gain (NHLBI, 1998). The initial aim of weight loss therapy should be to lose 1 to 2 pound per week in order to achieve a 10% reduction in body weight. Six months is considered an appropriate timeline for this initial weight loss goal. After 6 months, weight loss tends to plateau, and modifications of the weight loss regimen are generally required in order to maintain progress (NHLBI, 1998). The American Heart Association, the NHLBI, and other professional organizations recommend a multidisciplinary approach to treat overweight and obesity that combines diet therapy, physical activity regimens, and behavior modification therapy. This comprehensive approach is particularly important to prevent weight regain after the initial loss (Klein et al., 2004; NHLBI, 1998). The specific dietary, physical activity, and behavior modification intervention strategies used to treat overweight and obesity are outlined in Table 7.

Table 7

Multidisciplinary Intervention Strategies for Treatment of Overweight and Obesity

Weight loss intervention	Initial goal	Long term goal
Diet therapy	Weight loss of 1-2 lbs/week (kcal deficit of 500-1000 kcals/day); 10% reduction	Continued weight loss or weight loss maintenance
Physical activity	30-45 mins, moderate-intensity, 3-5 days/week	30 mins moderate-intensity, 5-7 days/week
Behavior modification	Identify and modify eating habits that negatively affect body weight Identify and eliminate barriers to physical activity	Weight loss maintenance through sustained behavior change

Note. Adapted from “Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults – The evidence report,” by National Heart Lung and Blood Institutes Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults, 1998, *American Journal of Clinical Nutrition*, 68(4), pp. 899-917. Copyright 1998 by American Society for Nutrition. Adapted with permission.

Utilizing a multidisciplinary intervention to treat overweight and obesity can be especially useful when targeting cardiovascular risk factors in addition to weight reduction. When diet therapy is combined with physical activity in weight reduction programs, increased amounts of abdominal fat are lost than compared to diet therapy alone, and cardiorespiratory fitness is improved (NHLBI, 1998). Studies have shown that weight reduction, particularly abdominal fat reduction, is associated with improved metabolic functioning including reduced cholesterol levels, lower blood pressure, and improved insulin function in overweight and obese individuals with hypertension,

hyperlipidemia, and type 2 diabetes (Goldstein, 1992). These improvements in various cardiovascular risk factors can be observed with only modest weight reduction and greater improvements occur as weight loss continues (Goldstein, 1992). It is important to note that reducing abdominal fat without adopting changes in diet and physical activity does not produce the same effects on cardiovascular risk factors. Liposuction and other surgical procedures can remove excess adipose tissue from the body. However, studies have shown the removal of excess abdominal fat through surgical means does not result in the metabolic improvements in blood pressure, serum cholesterol levels, and insulin function achieved with lifestyle interventions even when long term weight reduction was achieved (Klein et al., 2004; Mohammed, Cohen, Reeds, Young, & Klein, 2008).

Diabetes and Blood Glucose

As the prevalence of overweight and obesity in the United States has increased, so too has the prevalence of type 2 diabetes mellitus (Fox et al., 2006). In 2010, an estimated 25.8 million Americans were living with type 2 diabetes (CDC, 2011). Chronically elevated blood glucose levels and other complications associated with diabetes can negatively impact cardiovascular health. Adults with type 2 diabetes are at two to four times greater risk of mortality from cardiovascular disease than adults without type 2 diabetes (CDC, 2011). This makes type 2 diabetes a risk factor for cardiovascular disease (Grundy et al., 1999).

Diabetes mellitus is defined as a group of metabolic disease characterized by high blood glucose levels, also known as hyperglycemia, which results impaired insulin secretion and/or insulin action (American Diabetes Association [ADA], 1997). There are

two forms of diabetes that account for the majority of diagnosed cases of the disease.

Type 1 diabetes is characterized by the inability of the pancreas to produce insulin. This lack of insulin production is caused by an autoimmune process which attacks the insulin-producing β -cells of the pancreas. Also known as juvenile-onset diabetes or insulin-dependent diabetes, type 1 diabetes accounts for approximately 5-10% of the total prevalence of diabetes (ADA, 2011). Type 2 diabetes is characterized by insulin resistance and the defective secretion of insulin by the β -cells of the pancreas (ADA, 2011). Insulin resistance generally precedes the onset of type 2 diabetes and often presents with other cardiovascular risk factors including the presence of prothrombotic factors, hypertension, and atherogenic dyslipidemia (Gray et al., 1998; Hopkins, Hunt, Wu, Williams, & Williams, 1996).

Hyperglycemia, or elevated blood glucose levels, is the most commonly used diagnostic marker for type 2 diabetes. Blood glucose levels increase over time and can be asymptomatic until extremely high levels are reached (Grundy et al., 1999). The ADA (2011) accepts four methods for the diagnosis of diabetes: hemoglobin A1C, fasting plasma glucose (FPG), 2-hour plasma glucose oral glucose tolerance (OGTT), and a random plasma glucose reading in individuals who exhibit signs of hyperglycemia. Signs and symptoms of hyperglycemia and type 2 diabetes include extreme thirst, frequent urination, weight loss, tingling or numbness in the extremities, prolonged healing time of cuts or bruises, recurrent infections, and blurred vision (ADA, 1997).

Hemoglobin A1C indicates blood glucose levels over a 2- to 3- month period. A hemoglobin A1C reading $\geq 6.5\%$ reflects chronically high blood sugar levels; this cutoff

point is used to indicate and validate a diagnosis of type 2 diabetes (ADA, 2003).

Hemoglobin A1C is also used to track treatment adherence and progress because it reflects an individual's control of blood glucose levels over time. A fasting plasma glucose reading ≥ 126 mg/dl and a 2-hour plasma glucose reading ≥ 200 mg/dl during an OGTT both confirm a diagnosis of type 2 diabetes (ADA, 2003). In individuals presenting with typical signs and symptoms of hyperglycemia, a random plasma glucose reading ≥ 200 mg/dl is enough to confirm a diagnosis of type 2 diabetes (ADA, 2003).

The Expert Committee on Diagnosis and Classification of Diabetes (1997, 2003) also recognizes an intermediate metabolic stage, termed prediabetes, between normal glucose homeostasis and the diabetic state (ADA, 1997, 2003). The prediabetic stage is characterized by impaired fasting glucose (fasting plasma glucose ≥ 110 mg/dl and < 126 mg/dl) or impaired glucose tolerance (2-hour OGTT ≥ 140 mg/dl and < 200 mg/dL; ADA, 2011). While prediabetes is not a clinical entity in and of itself, individuals who fall within this metabolic stage are at increased risk for developing diabetes and cardiovascular disease (Fuller, Shipley, Rose, Jarrett, & Keen, 1980).

Many individuals diagnosed with impaired blood glucose are overweight, obese, and/or have excess abdominal adipose tissue (ADA, 2011). Individuals with impaired blood glucose typically exhibit concurrent cardiovascular risk factors such as dyslipidemia and hypertension (CDC, 2011). These conditions are often seen in individuals with prediabetes, as well. Impaired fasting glucose is generally the first indicator of insulin resistance; because pancreatic β -cells are capable of producing insulin once type 2 diabetes has set in, the presence of insulin resistance often remains

undetected for quite some time (Grundy et al., 1999). This clustering of abdominal obesity, insulin resistance, dyslipidemia, and hypertension is referred to as the metabolic syndrome and is exceedingly common in individuals with impaired blood glucose (Grundy et al., 1999).

Many risk factors for impaired blood glucose are also independent risk factors for cardiovascular disease including obesity, glucose intolerance, hypertension, dyslipidemia, physical inactivity, and smoking. The American Heart Association (2010) recognizes that many of the health behaviors (see Table 2) indicated for the reduction of cardiovascular disease have also been shown to lower the risk for developing impaired blood glucose. Meeting the criteria for physical activity, healthy dietary habits, and healthy BMI can lower the risk of developing type 2 diabetes by approximately 88% (Buse et al., 2010). The fact that risk factors for impaired blood glucose and cardiovascular disease overlap has led to treatment guidelines for the concurrent treatment of diabetes and cardiovascular disease. The primary recommendation for the treatment of diabetes is weight reduction through improved dietary intake and an emphasis on increased physical activity (Grundy et al., 1999). While physical inactivity is associated with insulin resistance and the metabolic syndrome, participating in moderate intensity physical activity has been linked to decreased levels of abdominal obesity (Irving et al., 2008). Moderate physical activity has been shown to be most effective at lowering blood glucose levels during both the exercise and postexercise periods; therefore, it is the recommended form of physical activity for individuals with type 2 diabetes (Albright et al., 2000). The reduction of central obesity through physical

activity coupled with diet therapy has been shown to be the preferred method of improving blood sugar control and overall metabolic function.

Physical Activity

Participating in regular physical activity has been shown to reduce premature mortality, reduce the risk of developing chronic disease, and aid in the promotion and maintenance of overall health (Thompson et al., 2003). Engaging in regular physical activity has been shown specifically to reduce the risk of developing cardiovascular disease by improving overall body composition, blood lipid profiles, blood glucose levels, blood pressure, and endothelial function (Marwick et al., 2009). While the health benefits of engaging in regular physical activity have been well established, physical inactivity is much more common occurrence for Americans (Pleis, Ward, & Lucas, 2009). According to the most recent National Health Interview Survey, approximately one-third of U.S. adults are considered inactive, with inactivity being higher among females than males (Pleis et al., 2009). The prevalence of inactivity also appears to increase with age (Pleis et al., 2009). According to data from the Behavioral Risk Factor Surveillance System, of the 150,000 surveyed adults, only 22% reported engaging in regular physical activity (Reeves & Rafferty, 2005). The low prevalence of physical activity in the U.S. population may be related to advances in technology that limit the need for physical exertion, such as motor vehicles. The higher salaries associated with more sedentary jobs have also been cited as possible contributors to decreases in physical activity. In contrast, statistics show that individuals who hold at least a bachelor's degree

are more likely engage in the recommended amount of physical activity than individuals who hold less than a bachelor's degree (Haskell et al., 2007).

When physical activity is planned, structured, and repetitive and has the purpose of improving physical fitness and overall health, it is referred to as exercise (Maron et al., 1996). Factors such as the duration, frequency, and intensity of physical activity are important to consider when planning an exercise prescription to reduce the risk of cardiovascular disease (Albright et al., 2000). Duration refers to the length of time spent engaging in physical activity. Frequency refers to how often an individual engages in physical activity, specifically how many days out of the week. Intensity refers the rate of energy expended or amount of aerobic energy used during physical activity (Albright et al., 2000). A dose-response relationship exists between the duration, frequency, and intensity of physical activity and the beneficial health outcomes experienced. Individuals who engage in physical activity above the minimum recommendation tend to experience greater health benefits and higher levels of physical fitness (Thompson et al., 2003).

Intensity is expressed as a measure of maximal heart rate or as a percentage of maximal oxygen uptake (Thompson et al., 2003). Maximal oxygen uptake ($\text{VO}_{2\text{max}}$) is a measure of the maximum aerobic power of the cardiovascular system. As the intensity of physical activity increases, oxygen uptake by the cardiovascular system must also increase in order to accommodate the increased workload being place upon it and to deliver oxygenated blood to the muscles (King & Senn, 1996). A linear relationship exists between oxygen uptake (VO_2), work rate, and power output, indicating that as work rate and power output increase so too does oxygen uptake. Eventually, oxygen

uptake plateaus and is unable to accommodate any further increases in power output. This plateau is referred to as VO_2max and represents the maximal capacity of an individual's cardiorespiratory system (Nieman, 2003).

Maximal oxygen uptake indicates the relationship between cardiac output and the arteriovenous oxygen difference, or the exchange and uptake of oxygen by the muscles. Improvements in VO_2max may be due to increases in either of these variables, thus improvements in VO_2max are typically the result of exercise training (Howley, 2001; King & Senn, 1996). In general, a higher VO_2max indicates a higher level of physical fitness. An elite marathon runner, for example may have VO_2max values (ml/kg/min) in the upper 80's-90's, while a sedentary individual may demonstrate VO_2max values in the 20-30's (Nieman, 2003). The type of exercise training can affect VO_2max with endurance or aerobic training resulting in the largest improvements in VO_2max compared to other forms of training (Valkeinen, Aaltonen, & Kujala, 2010).

Determinations of VO_2max can be utilized to prescribe individualized exercise plans based upon intensity of physical activity. Moderate-intensity physical activity refers to energy expenditure at 40-60% of VO_2max and is equivalent to a brisk walk or light jog that noticeably increases heart rate. Vigorous intensity VO_2max refers to energy expenditure above 60% of VO_2max and is equivalent to a moderate jog or light run that further increases heart rate and results in labored breathing (Haskell et al., 2007). In general, adults can prevent weight gain or achieve modest weight reduction by engaging in moderate-intensity aerobic activity for at least 30 minutes 5 days each week (150-250 minutes of moderate-intensity aerobic activity every week) or engaging in

vigorous-intensity aerobic activity for at least 20 minutes 3 days each week (Lloyd-Jones et al., 2010; Thompson et al., 2003). Resistance training is recommended in addition to aerobic exercise. Resistance training involves performing weight-bearing activities that maintain or increase muscle mass. Resistance activities should include 8-10 repetitions of weight bearing exercises that target large muscle groups and should not be performed on consecutive days (Haskell et al., 2007).

Physical activity positively affects several cardiovascular risk factors including endothelial dysfunction, dyslipidemia, insulin resistance and type 2 diabetes, overweight and obesity, and blood pressure. The increased need for oxygen that occurs during moderate and vigorous intensity physical activity is accommodated by increased blood flow through the arteries. This increased blood flow leads to increased vasodilation, particularly in the coronary arteries. Although this is typically an acute response to exercise, regularly engaging in physical activity leads to the improved vasodilatory function of the endothelium over time (Britten et al., 1999). For this reason, physical activity is often used to improve the symptoms and slow the progression of atherosclerosis (Thompson et al., 2003). Physical activity produces favorable changes in endothelial function by improving serum cholesterol levels. Studies have shown that physical activity, when used in combination with the consumption of a low-fat diet, is associated with a reduction in size of atherosclerotic lesions and the reversal of plaque formation. This treatment also resulted in reductions in total serum cholesterol and triglyceride levels and increased HDL cholesterol (Schuler et al., 1992).

Physical activity is also recommended for those diagnosed with or at risk for developing type 2 diabetes. Several studies have shown that physical activity has beneficial effects on metabolic functioning and can improve insulin sensitivity (Baldi & Snowling, 2003; Mourier et al., 1996). Both aerobic physical activity and resistance training appear to play an equally beneficial role in improving metabolic functioning. Exercise programs that combined aerobic and resistance training resulted in greater improvements in glycemic control than either type of exercise alone (Sigal et al., 2007). After a bout of aerobic exercise, diabetic individuals experience acute improvements in glucose tolerance and insulin sensitivity; engaging in regular physical activity can sustain these benefits for longer periods of time (Albright et al., 2000; King et al., 1995). Regular resistance training can result in improved body composition which has been shown to promote positive metabolic changes in individuals with type 2 diabetes (Albright et al., 2000). Physical activity recommendations for individuals with type 2 diabetes who are also at risk for cardiovascular disease include accumulating at least 150 minutes/wk of moderate-intensity physical activity and/or 90 minutes/wk of vigorous-intensity physical activity. Additionally, these individuals should incorporate resistance training on 3 nonconsecutive days each week (Albright et al., 2000; King et al., 1995).

Physical activity can lead to favorable changes in body composition in overweight and obese individuals. Specifically, physical activity can result in reductions in waist circumference, decreases in central obesity, weight reduction, and, ultimately, a reduced risk for cardiovascular disease (Goldstein, 1992). Combining physical activity with diet therapy results in greater changes in body composition than either method alone, and

these changes appear to be sustained over longer periods of time. Specifically, combined therapy results in greater fat mobilization and greater losses in visceral adipose tissue (Albright et al., 2000). Physical activity and diet therapy are also important to maintain weight loss and prevent weight regain (Klein et al., 2004).

Participating in regular physical activity has also been shown to reduce blood pressure, even in individuals without hypertension. Studies of physical activity interventions to lower blood pressure levels indicate that approximately 30-60 minutes of moderate-intensity physical activity on most days of the week can effectively reduce both systolic and diastolic blood pressure (Fagard, 2001). The decrease in blood pressure after physical activity is seen in both hypertensive and nonhypertensive individuals with the greatest reductions experienced by hypertensive individuals. Systolic blood pressure was reduced by 1.8-2.6 mm Hg, and diastolic blood was reduced by 5.8-7.4 mm Hg in hypertensive individuals. The decrease in blood pressure observed after physical activity can continue for almost 24 hours after the exercise session has ended (Pescatello et al., 2004; Thompson et al., 2003). The reductions seen in nonhypertensive individuals also suggests that regular physical activity may be the only therapy needed in order to reduce blood pressure in prehypertensive individuals (Thompson et al., 2003).

Nutrition and Cardiovascular Disease

Several studies have shown that dietary habits can affect multiple cardiovascular risk factors including LDL cholesterol levels, HDL cholesterol levels, blood pressure, blood glucose levels, and overweight and obesity. Recently, the health care community has transitioned from looking at individual foods or nutrients to analyzing overall food

consumption patterns. Examining dietary patterns is advantageous because it takes into account the cumulative effects of multiple nutrients and the complex interactions between diet and disease (Hu, 2002). Three major diet patterns—the Therapeutic Lifestyle Changes Program, The DASH Diet, and a Mediterranean-like diet pattern—have been shown to be effective in reducing cardiovascular risk factors (Appel et al., 1997; de Lorgeril et al., 1999; NHLBI Adult Treatment Panel III, 2002).

The Therapeutic Lifestyle Changes (TLC) program is recommended by the NHLBI's Third Report of the NCEP Expert Panel for the reduction of cholesterol levels. Specifically, the program was designed to reduce the overall risk of cardiovascular disease and lower LDL cholesterol levels. The TLC program recommends consuming a diet rich in fruits, vegetables, and whole grains, while limiting intakes of cholesterol, saturated fat and *trans* fat. The TLC program also recognizes the relationship between dyslipidemia, overweight and obesity, and the metabolic syndrome and recommends increasing physical activity to expend at least 200 calories per day and decreasing energy intake to achieve a healthy BMI (NHLBI Adult Treatment Panel III, 2002).

Dietary patterns that emphasize increased intakes of fruits, vegetables, and whole grain and the replacement of cholesterol and saturated fats with unsaturated fats have been shown to decrease LDL cholesterol and overall cardiovascular risk (Mensink & Katan, 1992). Diets high in fruits, vegetables, and whole grains also tend to be high in vitamins, antioxidants, dietary fiber and phytochemicals. As previously summarized, dietary fiber has been shown to lower serum cholesterol levels by inhibiting the absorption of dietary fat and by aiding in the excretion of cholesterol-containing bile

acids (Food and Nutrition Board, Institute of Medicine, 2006a). Soluble fiber in particular binds to bile acids, a process which inhibits micelle formation by competing for micellar binding and causes the bile acids to be excreted instead of absorbed by the small intestine. The depletion of bile acids causes the body to mobilize cholesterol for the synthesis of new bile acids (Food and Nutrition Board, Institute of Medicine, 2006a). Soluble fiber appears to have the greatest effect on LDL cholesterol levels; an increase of 5-10g g of soluble fiber per day may decrease LDL cholesterol by up to 5% (NHLBI Adult Treatment Panel III, 2002).

Phytochemicals are biologically active, non-nutrient compounds that have health benefits. Plant-based foods such as vegetable oils, nuts, and seeds contain stanols and sterols, also called phytosterols, which are cholesterol-like compounds that have been shown to decrease cholesterol absorption in the body (Anderson & Hanna, 1999). Phytosterols help lower cholesterol levels by competitively inhibiting cholesterol absorptions (Genser et al., 2012). There is evidence that the consumption of plant sterols and stanols can reduce LDL cholesterol levels by approximately 11% (Katan et al., 2003). Food manufacturers have begun to add phytosterols to a number of spreads, yogurts, beverages, and other products. A dose-response relationship exists between the amount of phytosterols consumed and the LDL cholesterol-lowering effects. Maximum effects are observed when 2-3 g of phytosterols are consumed per day (Katan et al., 2003). However, amounts beyond this are not recommended because there are no additional effects observed on LDL cholesterol levels.

Elevated LDL cholesterol levels have been associated with the consumption of dietary cholesterol, saturated fat, and *trans* fat (NHLBI Adult Treatment Panel III, 2002). Dietary cholesterol is primarily found in animal products such as dairy products, eggs, fish, meat, and poultry. The intake of dietary cholesterol can result in an increase in total serum cholesterol. The consumption of 100 mg of cholesterol typically results in a 2 to 3 mg/dL increase in total serum cholesterol. This, in turn, results in an increase in LDL cholesterol levels, since LDL cholesterol makes up 60-80% of serum cholesterol (Food and Nutrition Board, Institute of Medicine, 2006a). While the consumption of dietary cholesterol is not essential, completely avoiding all sources of dietary cholesterol is not recommended. The human body is capable of synthesizing adequate amounts endogenous cholesterol, but the avoidance of all sources of dietary cholesterol would entail the adoption of a strict vegan diet pattern (Nelms et al., 2011). This type of diet pattern requires careful planning to ensure the appropriate intake of nutrients and is not feasible for the majority of the population. Monitoring cholesterol intake can allow for the continued consumption of animal proteins while still decreasing LDL cholesterol levels (Food and Nutrition Board, Institute of Medicine, 2006a).

Fat is an essential nutrient that functions as a major source of energy, aids in the absorption of fat-soluble vitamins, and plays a role in the structure of cell membranes. Similar to cholesterol, saturated fatty acids are synthesized in adequate amounts by the human body (Food and Nutrition Board, Institute of Medicine, 2006a). A linear relationship exists between total saturated fatty acid intake (in the form of saturated fat) and total and LDL cholesterol levels. A 1% increase in calories from saturated fat can

result in a 2% increase in LDL cholesterol levels (NHLBI Adult Treatment Panel III, 2002). The relationship between saturated fat intake and an increased risk for heart disease has led to very low intake recommendations. Like cholesterol, saturated fat generally comes from animal sources such as cheese, butter, milk, and meats. However, coconut and palm oils are two plant-based oils that are also very high in saturated fat (Food and Nutrition Board, Institute of Medicine, 2006a).

Trans fats also have a negative impact on cardiovascular health. Although it is classified as an unsaturated fatty acid, *trans* fat functions similar to saturated fat and increases LDL cholesterol levels. There is also evidence that increased consumption of *trans* fats may lower HDL cholesterol level, further increasing the risk for cardiovascular disease (de Roos, Schouten, & Katan, 2003; Sundram, French, & Clandinin, 2003). Unlike saturated fat and cholesterol, *trans* fat is not synthesized by the human body; it also has no known functional or health roles in the body (Food and Nutrition Board, Institute of Medicine, 2006a). *Trans* fat is formed when polyunsaturated oils are partially hydrogenated or treated with hydrogen. Sources of *trans* fat include prepackaged convenience foods, food fried in partially hydrogenated oils, and partially hydrogenated products like vegetable shortening (Emken, 1995).

Mono- and polyunsaturated fatty acids have been shown to decrease LDL cholesterol levels (Mensink & Katan, 1992). Monounsaturated fatty acids, such as oleic acid, are typically found in olive oil and have been associated with decreases in LDL cholesterol levels (NHLBI Adult Treatment Panel III, 2002). Polyunsaturated fatty acids, such as linoleic acid, are typically found in margarines, walnuts, sunflower seeds, and

liquid vegetable oil such as soybean oil and safflower oil. Fatty fish such as mackerel, salmon, and trout are also high in polyunsaturated fatty acids. Like monounsaturated fatty acids, polyunsaturated fatty acids have been linked to reductions in LDL cholesterol levels. Polyunsaturated fatty acids have also been shown to increase HDL cholesterol levels; however, overconsumption can lead to decreases in HDL levels and increases in triglyceride levels (NHLBI Adult Treatment Panel III, 2002). The TLC program recommendations for mono- and polyunsaturated fats are up to 20% and 10%, respectively, of total calories from fat (NHLBI Adult Treatment Panel III, 2002). The dietary recommendations of the TLC program and the approximate LDL cholesterol reductions associated with specified modifications are summarized in Table 8.

Table 8

Therapeutic Lifestyle Changes Program: Recommended Percent of Total Daily Calorie Intake to Reduce LDL Cholesterol

Dietary Component	Therapeutic Lifestyle Changes Recommendation	Approximate LDL Reductions
Carbohydrates	50-60%	-
Protein	~15%	-
Total Fat	25-35%	
Monounsaturated fatty acids	20% maximum	8-10%
Polyunsaturated fatty acids	10% maximum	
Saturated fat	<7%	
Cholesterol	< 200 mg/dL	3-5%
Dietary Fiber	20-30 g	3-5%
Soluble fiber	10-25 g	
Plant stanols and/or sterols	2 g	6-15%

Note. “Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adult (Adult Treatment Panel III) final report” by National Heart, Lung, and Blood Institute, Adult Treatment Panel III, 2002, *Circulation*, 106(25) p. 3143. Copyright 2002 by Wolters Kluwer Health. Reprinted with permission.

A second dietary pattern that decreases the risk of cardiovascular disease is the Mediterranean-type diet. This diet pattern emphasizes the consumption of fruits, vegetables, breads and cereals, beans, nuts, and seeds. It also includes olive and rapeseed oil, moderate wine consumption, low intakes of dairy products, fish, and poultry and very limited consumption of eggs and red meat (de Lorgeril et al., 1999). The Mediterranean-

type diet was studied in a randomized cardiovascular disease prevention trial called the Lyon Heart Diet Study. The Lyon Heart study was conducted from 1988-1992 and included 605 participants. Participants in the experimental (Mediterranean-type) diet saw 50-70% reduction in the risk of experiencing a clinically significant cardiovascular event. A 4-year follow up of the Lyon Heart indicated that significant reduction in cardiovascular disease risk and cardiovascular events was sustained in those participants who continued to follow a Mediterranean-type diet (de Lorgeril et al., 1999).

A key aspect of the Mediterranean-type diet appears to be the increased consumption of omega-3 fatty acids. These polyunsaturated fatty acids are found in fatty fish, canola oil, and flaxseed and are consumed as part of this particular dietary pattern. Animal sources of omega-3 fatty acids include eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and demonstrate greater lipid lowering and cardiovascular protective effects than the alpha linolenic acid (ALA) found in plants (Dyerburg & Bang 1979; Lavie, Milani, Mehra, & Ventura, 2009). Emerging research has indicated that omega-3 fatty acids may reduce serum triglyceride levels. Omega-3 fatty acids appear to reduce serum triglycerides by inhibiting the synthesis of VLDL cholesterol which is a triglyceride-rich molecule (Leaf & Weber, 1988). The mechanisms by which DHA and EPA are not well characterized, but these fatty acids increase fat metabolism and also decrease triglyceride synthesis.

Omega-3 fatty acids may also play a role in reducing systolic and diastolic blood pressure, improving endothelial function; they may also have anti-inflammatory properties. The consumption of omega-3 fatty acids appears to increase the production of

nitric oxide, a compound that is critical for normal endothelial functioning. Increased levels of nitric oxide may diminish vasoconstrictive responses and enhance vasodilation (Chin, Gust, Nestel, & Dart, 1993; Leeson et al., 2002). The mechanism by which omega-3 fatty acids improve endothelial function to have effects on blood pressure and inflammation, as well (Mozaffarian & Wu, 2011). Omega-3 fatty acids could mediate inflammation through several biological pathways. Studies conducted with rheumatoid arthritis patients found that consuming high doses (1.7 to 9.6 g/day) of omega-3 fatty acids was associated with reduced morning stiffness and reduced joint pain (Goldberg & Katz, 2007). Although past studies examining the effects of omega-3 fatty acids on specific cardiovascular biomarkers of inflammation have been mixed, there is a growing body of evidence supporting the anti-inflammatory effects of omega-3 fatty acids.

Dietary patterns that emphasize fruits, vegetables, whole grains, and reduced intakes of total and saturated fat have also been shown to have beneficial effects on blood pressure (Appel et al., 1997). The Dietary Approaches to Stop Hypertension (DASH) diet emphasizes the increased consumption of whole grains, fruits, vegetables, nuts, seeds, legumes, and low-fat dairy products, as well as decreased consumption of dietary fat, red meats, sweets, and sugar-containing beverages. The DASH diet trial examined the effects of three dietary interventions on blood pressure in 459 participants with systolic blood pressure < 160 mm Hg and diastolic blood pressure between 80 and 95 mm Hg. Participants were randomly assigned to the control diet, the fruit and vegetable diet, or the combination diet. The control diet was supposed to mimic the typical American/Western dietary pattern including calcium, magnesium, and potassium levels at

approximately the 25th percentile of U.S. consumption and average macronutrient and fiber consumption. The fruit and vegetable diet provided magnesium and potassium levels near the 75th percentile of U.S. consumption and high amounts of fiber, but was otherwise similar to the control diet. The combination diet (DASH) was rich in fruits, vegetables, and low-fat dairy products and included reduced intake of total fat, saturated fat, and cholesterol. The combination diet also provided calcium, magnesium, and potassium levels at approximately the 75th percentile of U.S. consumption, plus high fiber and protein intakes (Appel et al., 1997). The sodium consumption of each diet pattern was similar at approximately 3 g/day. After 8 weeks of treatment, the combination diet group experienced a 5.5 mm Hg decrease in systolic blood pressure and a 3 mm Hg decrease in diastolic blood pressure. The fruit and vegetable diet also decreased blood pressure, but did so to a much lesser degree (Appel et al., 1997). It is notable that the combination diet reduced blood pressure in hypertensive and nonhypertensive patients. However, hypertensive patient demonstrated greater decreases in blood pressure compared to their nonhypertensive counterparts (Appel et al., 1997).

A second DASH diet trial examined the effects of the DASH diet pattern on blood pressure when combined with reduced sodium intake (Sacks et al., 2001). Participants were 412 adults with blood pressures > 120/80 mm Hg and were randomly assigned to either the control diet or the DASH diet. Each diet group was subdivided in three smaller groups based on sodium intake (high, intermediate and low). The high sodium intake consumed 150 mmol (~8.7g of sodium per day, approximately the amount the average American consumes; the intermediate group consumed 100 mmol (~5.8g) of sodium per

day; and the low group consumed 50 mmol (~2.9g) of sodium per day (Sacks et al., 2001). After 30 days of treatment, reduced sodium intake significantly lowered systolic and diastolic blood pressures in both the control and DASH study groups (Sacks et al., 2001). However, the reductions were greater in the DASH diet groups. The DASH diet resulted in significantly lower systolic blood pressures at every sodium level when compared to the control group. The combination of the DASH diet and the lowest sodium level resulted in systolic blood pressure reductions of 11.5 mm Hg, 12.6 mm Hg, and 9.5 mm Hg in hypertensive patients, hypertensive African-Americans, and other hypertensive patients, respectively. Nonhypertensive patients, nonhypertensive African-Americans, and other non-hypertensive patients experienced systolic blood pressure reductions of 7.1 mm Hg, 7.2 mm Hg, and 6.9 mm Hg, respectively (Sacks et al., 2001). The DASH diet also appeared to have a greater effect on systolic and diastolic blood pressure in the high sodium groups as compared to the groups consuming lesser amounts of sodium. The DASH diet also produced significantly lower diastolic blood pressures in the high and intermediate sodium groups (Sacks et al., 2001).

The DASH diet trials demonstrated that specific dietary factors play key roles in reducing blood pressure (Appel et al., 1997; Sacks et al., 2001). Decreased sodium intake and increased intake of calcium, magnesium and potassium, in combination with weight reduction, can effectively reduce blood pressure (Appel & American Society of Hypertension Writing Group, 2009). The DASH diet was shown to achieve even greater reductions in certain populations at greater risk for high blood pressure. In African Americans and hypertensive individuals, the DASH diet produced significantly greater

reductions in systolic and diastolic blood pressure as compared to the corresponding effects in non-African Americans and nonhypertensive individuals (6.9/3.7 mm Hg vs. 3.3/2.4 mm Hg; 11.6/5.3 mm Hg vs. 3.5/2.2 mm Hg, respectively; Appel & American Society of Hypertension Writing Group, 2009). The current recommendation for low sodium intake in the general population is to consume less than 2400 mg/day. In high-risk populations such as older adults (≥ 51 years), African Americans, hypertensive individuals, individuals with diabetes, and individuals with chronic kidney disease, sodium intake should not exceed 1500 mg per day (Chobanian et al., 2009).

In order to target all cardiovascular risk factors, the American Heart Association recommends following a DASH-like eating pattern that incorporates a plan for achieving or maintaining desirable cholesterol level (Lloyd-Jones et al., 2010). In addition, the American Heart Association recommends the following secondary dietary metrics: ≥ 4 servings of nuts, legumes, and seeds per week; ≤ 2 servings of processed meats per week; and $< 7\%$ of total calories from saturated fat (Lloyd-Jones et al., 2010). The increased consumption of mono- and polyunsaturated fatty acids in the form of nuts, seeds, and fatty fish is also recommended.

Because overweight and obesity are independent risk factors for cardiovascular disease and also linked to the pathophysiology of cardiovascular risk factors, dietary patterns should support the achievement and maintenance of a healthy BMI. A calorie-restricted diet is generally necessary in order to achieve this goal. The NHLBI's clinical guidelines on the identification, evaluation, and treatment of overweight and obesity recommend a deficit of 500 to 1000 calories per day in order to produce a weight

reduction of one to two pounds per week, with an overall goal of a 10% reduction of body weight within 6 months (NHLBI, 1998). As discussed earlier, weight reduction can also result in favorable changes in metabolic functioning. Dietary patterns that incorporate caloric restriction can aid in the treatment and prevention of insulin resistance and type 2 diabetes. Specifically, the reduction of central obesity through diet therapy, coupled with physical activity, has been shown to be the preferred method of improving blood sugar control and overall metabolic function (Albright et al., 2000).

Smoking

There is an extensive body of evidence documenting the harmful effects of smoking. Smoking has been linked to several leading causes of death in the United States, including cardiovascular disease, stroke and many forms of cancer (Ockene & Miller, 1997; U.S. Department of Health and Human Services [DHHS], 1990). The U.S. Surgeon General has recognized smoking major risk factor for several diseases since 1964, and the list of diseases continues to grow. The casual relationship between smoking and adverse health outcomes is so definitive that smoking is now considered the single most modifiable risk factor for morbidity and mortality in the United States (Ockene & Miller, 1997).

From 2000 to 2004, an estimated 443,000 American adults (≥ 35 years of age) died prematurely due to smoking-related illnesses. Approximately one-third of these deaths were associated with cardiovascular disease (CDC, 2008). Smoking contributes to atherogenesis by adversely affecting the normal functioning of the arterial endothelium. Specifically, smoking appears to promote endothelial dysfunction by disrupting the

normal endothelial secretion of nitric oxide, the compound that helps regulate normal vasoconstriction and dilation (Arnal et al., 1999; Pittilo, 1990). The relationship between smoking and endothelial dysfunction appears to be dose-dependent with endothelial-dependent vasodilation and constriction decreasing as the number of cigarettes and number of years spent smoking increases (Celermajer et al., 1993). Studies also suggest that smoking may contribute to a prothrombotic state within the arteries. Smoking is directly associated with damaging changes to the properties of platelets including platelet activation and adhesion, which increase the likelihood of aggregation within the arterial wall (Lakier, 1992; Lassila et al., 1988).

Additionally, certain chemical components within cigarettes can contribute to poorer overall functioning of the cardiovascular system with regard to the supply and demand of oxygen. Smoking introduces carbon monoxide into the bloodstream which interferes with the blood's ability to transport oxygen throughout the body. Carbon monoxide competes with oxygen to bind to hemoglobin thus decreasing oxygen levels in the blood. This may result in compensatory erythrocytosis, which can increase blood viscosity and further contribute to a prothrombotic state (Rampling, 1993). Reductions in the oxygen-carrying capacity of the blood is also associated with significant myocardial perfusion abnormalities, which in turn increase the risk for myocardial infarction, complications from coronary heart disease and sudden death in individuals who smoke (Quillen et al., 1993).

Exposure to secondhand smoke can also cause adverse health effects including an increased risk of developing cardiovascular disease and early mortality (CDC, 2008).

Studies have found a causal relationship between secondhand smoke exposure and an increased risk for coronary heart disease-related morbidity and mortality. Exposure to secondhand smoke is estimated to increase these risks by 25-30% (Barnoya and Glanz, 2005; U.S. DHHS, 2006). As with active smoking, exposure to secondhand smoke can cause endothelial dysfunction, impaired platelet functions, and increased risk of acute myocardial infarction. The relationship between secondhand smoke exposure and cardiovascular disease is also dose-dependent, with higher rates of exposure corresponding to a higher risk for cardiovascular disease (U.S. DHHS, 2006).

The health benefits of smoking cessation have been well documented. According to the U.S. DHHS's report on the Health Benefits of Smoking Cessation (1990), immediate benefits are experienced by men and women of all ages with or without diagnosed cardiovascular disease. These benefits include a significant decrease in morbidity and mortality related to cardiovascular disease, lung disease, certain forms of cancer, and stroke. There is evidence that the risk for coronary heart disease is substantially reduced within the first 2 years following smoking cessation (U.S. DHHS, 1990).

Stress

A growing body of evidence supports a relationship between psychological factors, particularly stress, and the progression of cardiovascular disease (Figuerdo, 2009). While the effects of stress and other psychological factors on cardiovascular disease remain largely unrecognized, professional organizations such as the NHLBI and the American Heart Association have adopted stress as a risk factor for cardiovascular

disease. Although the mechanism by which stress affects cardiovascular health is not well characterized, chronic stress appears to increase heart and blood pressure and also decrease heart rate variability (Chandola et al., 2008). Stress has been shown to contribute to an increased likelihood of participation in negative health behaviors such as smoking, poor dietary compliance, physical inactivity, and poor adherence to prescribed medical regimens or intervention programs (Rozanski, Blumenthal, & Kaplan, 1999). Interestingly, psychosocial risk factors, such as stress, and adverse health behaviors tend to occur in clusters similar to traditional cardiovascular risk such as diabetes and overweight or obesity (Rozanski et al., 1999). For these reasons, comprehensive lifestyle intervention programs incorporate stress management techniques in order to increase program adherence and help prevent relapse to poorer health behaviors (Byrd-Bredbenner, Abbot, & Cussler, 2011).

The practice of yoga, a form of exercise that includes mind and body relaxation, may be an effective method for reducing stress. Yoga utilizes meditation and diaphragmatic breathing, often referred to as mindfulness-based stress reduction techniques. These techniques have been implicated in the relaxation of the muscles and an overall sense of well-being (Jayasinghe, 2004). The slow, controlled breathing characteristic of yoga practice has been associated with increased heart rate variability and enhances cardiac rhythms. Yoga may play a role in reducing cardiovascular risk, particularly in overweight and obese individuals. While there is some evidence that yoga may be associated with significant reductions in cardiovascular risk, causal associations are limited, and, at this time, somewhat inconclusive (Jayasinghe, 2004).

Treatment Options

According to the 2007 estimates, the total economic burden of cardiovascular disease was \$286 billion, including \$119 billion due to lost productivity and early mortality, and \$167 billion for healthcare services and prescription medication (Roger et al., 2011). This accounts for 15% of total health expenditures, making cardiovascular disease the most costly disease in the United States (AHRQ, 2007; Kashihara & Carper, 2009). Population-wide reduction of cardiovascular risk factors could significantly reduce both the total cost and overall mortality associated with cardiovascular disease. Individuals who engage in behaviors that promote ideal cardiovascular health into middle age have lower health care expenditures than individuals who do not engage in such behaviors. The average annual expenditures for men and women engaging in ideal cardiovascular health behaviors into middle age were reduced by two-thirds (\$1,615) and one-half (\$1,885), respectively, when compared to individuals who do not engage in such heart-healthy behaviors (Daviglius et al., 1998).

It has been estimated that 70% or more of cardiovascular disease could be prevented if certain risk factors were modified (Folsom et al., 2009; Lloyd-Jones et al., 2006; Stamler et al., 1999). Additionally, the NHLBI estimates that by effectively treating cardiovascular risk factors, life expectancies could increase by up to 7 years (Anderson, Odell, Wilson, & Kannel, 1991). Comprehensive treatment plans that target multiple risk factors are necessary in order to accomplish such beneficial public health outcomes. The primary goal of a comprehensive intervention program should be to achieve the ideal cardiovascular health metrics outlined by the American Heart

Association (see Table 1). These types of interventions are often referred to as lifestyle treatment programs because they target the overall lifestyle behaviors that work synergistically to increase the risk of cardiovascular disease (Lichtenstein et al., 2002).

Behavior Modification

The NHLBI recommends the addition of behavior modification to physical activity and diet therapy, because this combination of treatments has been shown to be most effective method to achieve weight reduction, which influences almost every cardiovascular risk factor (NHLBI, 1998). Comprehensive lifestyle interventions target behaviors that contribute to the development of cardiovascular risk factors, and such programs require major lifestyle changes. Many individuals have difficulty trading current behaviors for new health behaviors. Thus, behavior modification therapy is often incorporated in order to increase the adherence to, and effectiveness of, risk reduction programs (NHLBI, 1998). Behavior modification therapy attempts to identify current behaviors that contribute to increased health and to modify these behaviors in order to decrease health risks. Typically, energy intake and physical activity patterns are targets of behavior modification therapy.

For behavior modification therapy to be successful, a collaborative relationship must be formed between the behavior modification counselor and the patient. The behavior modification therapist must establish a rapport with the patient and work to understand any potential barriers to change. Recognizing and understanding barriers to change is essential so that the behavior modification counselor can provide successful strategies for change and support to the patient (Miller & Rollnick, 1991). It is important

that the counselor is able to express empathy of the patient's condition and to allow the patient to feel that they can actively contribute to the treatment process (Miller & Rollnick, 1991). Patient-centered treatment strategies, such as motivational interviewing and self-monitoring, may be beneficial. Studies have indicated that motivational interviewing is an effective treatment strategy for overweight and obese patients, particularly those individuals who are unsure about making lifestyle changes. This technique also allows patients to engage in self-motivation and is often used in conjunction with behavior modification (Armstrong et al., 2011; Miller & Rollnick, 1991). Self-monitoring generally entails keeping detailed self-records related to a target behavior such as diet intake or exercise. This strategy increases an individual's self-awareness and is considered to be one of the most crucial factors in successful behavior change interventions (Fabricatore & Wadden, 2003). Studies have shown that the more consistent individuals are in their self-monitoring, the more successful they are at adopting their target behavior (Bouteille, Kirschenbaum, Baker, & Mitchell, 1999; Tate, Jackvony, & Wing, 2003)

Once the collaborative counselor-patient relationship has been established, specific behavioral strategies can be employed to effect changes associated with energy intake and physical activity (Foreyt & Poston, 1998). There are many different strategies that are used to reduce risk factors for cardiovascular disease. Strategies that employ goal-setting exercises help involve patients in their treatment process. Well-defined goals, particularly those that are challenging yet realistic, easily attainable and specific, have been shown to be effective (Hurn, Kneebone, & Cropley, 2006). In order to

formulate well-defined goals, health care practitioners may utilize the SMART goal evaluation method. SMART goals are specific, measurable, achievable, realistic and time-bound (Mogensen, Bowman, Lannin, Cook, & McCluskey, 2006). This method assures patient involvement in the treatment process and allows for the counselor and patient to engage in an open and honest dialogue regarding the expectations of therapy.

The processes that mediate true behavior change are extremely complex. Often behavior changes are contingent on patients' readiness to change. The Transtheoretical Model, a theory of behavior change, utilizes a construct term the "stages of change" (DiClemente & Prochaska, 1983). According to the stages of change construct, change is a process that happens over time and is realized in individuals' progression through five stages: precontemplation, contemplation, preparation, action and maintenance. Each stage is characterized by certain attitudes or tasks and involves different change processes. These five stages of change are outlined in Table 9.

Table 9

Transtheretical Model: Stages of Change

Stage	Readiness to change	Description
Precontemplation	No intention to change in the near term (i.e., 6 months)	Patient is uninformed or underinformed about the consequences of their behavior(s); resistant or unmotivated
Contemplation	Contemplating change; intend to change within 6 months	Patient is aware of the consequences of their behavior; ambivalent or engaging in behavioral procrastination
Preparation	Planned change, typically within the next month	Patient has already take a significant step toward behavior change; has a plan of action
Action	Specific modifications made, within the past 6 months	Patient has made observable changes in behavior and has maintained new behavior for less than 6 months
Maintenance	Specific behavior change has occurred; relapse prevention	Patient consistently engages in new behavior (≥ 6 months); working to prevent relapse

Note. From "Stages of change ," by J. C. Norcross, P. M. Krebs, & J.O. Prochaska, 2011, *Journal of Clinical Psychology*, 67(2), p. 144. Copyright 2010 by John Wiley and Sons. Adapted with permission.

Social-cognitive theory is another theory of behavior change often used by health care practitioners. This theory emphasizes reciprocal determinism, the idea that interactions between a people and their environment determine behaviors (Bandura, 1997). In order for individuals to successfully change a behavior they must be motivated and must have the ability to create an environment that is conducive to behavior change. Social-cognitive theory also emphasizes the concept of self-efficacy, referring to a belief in the ability to perform behaviors and achieve a desired outcome (Glanz, Rimer, & Viswanath, 2008). This theory recognizes that the ability to effectively deal with environmental stressors is a critical component to successful behavior change (Bandura, 1997).

Utilizing the behavior modification therapies, such as motivational interviewing, transtheoretical stages of change, and social cognitive theory as part of a health intervention, can help create a personalized, patient-centered approach to risk factor reduction. Studies have shown that behavior therapy, in combination with diet therapy and physical activity interventions, results in greater success regarding an initial weight loss goal and better maintenance of weight loss over time (Wadden, Sarwer, & Berkowitz, 1999). Thus, to achieve the greatest reduction of cardiovascular disease risk, interventions to treat modifiable risk factors should combine diet therapy, physical activity and behavior therapy. Table 10 outlines the results of recent studies utilizing multidisciplinary lifestyle interventions for the reduction of cardiovascular risk factors.

Table 10

Results of Comprehensive Lifestyle Interventions to Treat Cardiovascular Risk Factors

Authors	Participants	Behavior modification	Exercise	Nutrition	Duration	Results
Eriksson, Westborg, & Eliasson, 2006	n=75 55.3±6.9 yoa HTN, T2DM, Obesity, Dyslipidemia	3 monthly physiotherapy meetings	40-60 mins/day, 3 days/wk	5 groups sessions with dietitian	12 months	Significant ↓ in weight, waist circumference, blood pressure, LDL-C, triglycerides
Kim et al., 2006	n=32 55±8.1 yoa T2DM	Nutrition Edu PA Education Health Coach	150 mins/wk 40-60% of VO ₂ max	Dietitian mediated	6 months	Significant ↓ in weight BMI, blood pressure, HR
Jordan et al., 2008	n=60 27.5 yoa (avg.) Overweight Obesity	Social Cognitive* Theory	Supervised: 30 mins, 1 day/wk Encouraged: 45 mins, ≥ 3 days/wk	Calorie-restricted diet, 1200-1500 kcal/day	6 months	Significant ↓ in weight, waist circumference, % body fat

(Table 10, continued)

Authors	Participants	Behavior modification	Exercise	Nutrition	Duration	Results
Lutes et al., 2008	n=59 39.9 yoa (avg.) Overweight Obesity	Didactic behavioral counseling 20 mins/wk	Supervised: 40-45 mins, 2 day/wk Encouraged: 30 mins, \geq 4 days/wk	1600-2000 kcal/day	16 weeks	Significant \downarrow in weight, waist circumference, abdominal fat
Aizawa, Shoemaker, Overend, & Petrella, 2009	n=63 55.9 \pm 8.7 yoa MetS, PreHTN, PreDM	Stages of Change** SNAC intervention	30 mins/day \geq 4 days/wk	Mediterranean- style diet, Kcal/day prescribed by physician	24 weeks	Significant \downarrow in blood pressure, fasting glucose, waist circumference
Chen et al., 2009	n=29 44 \pm 10.5 yoa Overweight Obesity	Nutrition Educ	60 min/day 2 days/wk Jogging 10,000 steps/day	Calorie- restricted diet, 1200 kcal/day	3 months	Significant \downarrow in blood pressure, fasting glucose, LDL-C total cholesterol, triglycerides, BMI waist circ., weight

(Table 10, continued)

Authors	Participants	Behavior modification	Exercise	Nutrition	Duration	Results
Blumenthal et al., 2010	n=49 52 yoa (avg.) Overweight Obesity, High BP	Weekly cognitive-behavioral therapy session – weight loss	45 mins/day 3 days/wk 70-85% max HR	DASH Diet Calorie restriction ↓ 500 kcal/day	4 months	Significant ↓ in blood pressure, weight
Goodpaster et al., 2010	n=130 46.1±6.5 yoa HTN, T2DM Obesity Dyslipidemia	1 st 6 months: 3 group meeting, 1 indiv/month 2 nd 6 months: 2 group meetings, 1 indiv/month	60 mins/day 5 days/wk 10,000 steps/day	1200-2100 kcal/day 50-55% CHO 20-20% PRO 20-30% FAT	12 months	Significant ↓ in abdominal fat, blood pressure, insulin resistance, waist circ., weight
Oh et al., 2010	n= 31 49-70 yoa MetS	TLM 1st 3 months: 3 meetings/month 2 nd 3 months: 2 meetings/months	1st 3 months: 40 mins/day, 3 days/week 2 nd 3 months: 40 mins/day, 2 days/wk	Calorie-restricted diet, (1500 kcal/day) 55-60% CHO	6 months	Significant ↓ in BMI, waist circumference, weight

(Table 10, continued)

Authors	Participants	Behavior modification	Exercise	Nutrition	Duration	Results
Arrebola et al., 2011	n=27 18-50 yoa Grade II overweight Grade I-II obesity	11 sessions 2 per month Nutrition Edu. PA Education Psychological Support	30 mins/day ≥ 3 days/wk	Calorie-restricted diet (↓ 500-1000 kcal/day) 50-55% CHO 15-20% PRO < 30-35% FAT	6 months	Significant ↓ in BMI, % body fat, waist circumference, weight
Cocco & Pandolfi, 2011	n=44 59±4 yoa HTN	Nutrition Edu.	20 mins twice/day 5 days/wk 80% VO ₂ max	Calorie-restricted diet 1500 kcal/day	6 months	Significant ↓ blood pressure, weight

Note. yoa= years of age; CHO, carbohydrates; PRO, protein; FAT, fat; mins, minutes; Edu, education; PA, physical activity; SNAC, Staged Nutrition and Activity Counseling; BMI, body mass index; TLM, therapeutic lifestyle modifications; VO₂max, maximal oxygen capacity; ↓, reduction; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; MetS, metabolic syndrome; HTN, hypertension; T2DM, type 2 diabetes; BP, blood pressure; HR, heart rate; indic, individual; * Social cognitive theory (Bandura, 1994). **Stages of change (Prochaska & DiClemente, 1983).

CHAPTER 3: METHODOLOGY

The purpose of this study was to evaluate the efficacy of the RRP to reduce cardiovascular risk factors in an at-risk adult population after 1 year of program participation. Table 1 outlines the specific research questions and hypotheses associated with this study. Prior to data collection, approval for this study was obtained from the Institutional Review Board of Ohio University, Athens, Ohio. This study examined a subset of data pooled from 12 cohorts of individuals participating in the RRP between 2006 and 2010.

Participants

This study represents a convenience sample of Ohio University faculty, staff and their dependents participating in the RRP and classified as at-risk for cardiovascular disease. To be classified as at-risk, individuals must have presented with either lifestyle diseases or risk factors for cardiovascular disease such as coronary artery disease, cigarette smoking, diabetes, dyslipidemia, obesity, physical inactivity, and/or stress. Participants were referred by a physician or recruited by material distributed by the office of Human Resources at Ohio University. Participants were treated using a comprehensive approach that integrated personalized nutrition education and counseling, an individualized exercise regimen and stress reduction strategies in the form of yoga classes to reduce existing cardiovascular disease risk factors. The RRP was a 1-year program with an intensive 100 day “kick-start” during which an interdisciplinary team consisting of a certified personal trainer, certified yoga instructor, exercise physiologist, health coordinator, and registered dietitian worked with participants individually.

Participants were required to partake in all exercise sessions, nutrition education meetings, yoga and group support classes schedules by the interdisciplinary team in order to remain active in the program. The total cost of the program was \$3000; participants were responsible for a \$300 participation fee and the office of Human Resource paid the remaining \$2700. In addition, participants could earn back up to \$75 of their participation fee each quarter by meeting attendance and health goals. If all \$300 was earned back, the participant was also eligible for a second membership to WellWorks, Ohio University's employee wellness facility in the College of Health Sciences and Professions.

Structure of the Risk Reduction Program

The RRP was a structured program designed to enhance the lives of individuals with risk factors for cardiovascular disease or significant lifestyle diseases (i.e., coronary heart disease, high blood pressure, diabetes, and/or obesity). The program is a 1-year program with four phases. Phase 1 includes the intensive 100 day kick-start and lasts from week 1 to week 15 of the program. During the first week of the program participants completed a physical assessment including measurements of body composition, blood pressure, blood work for glucose and lipid levels, and an exercise stress test to determine maximal oxygen uptake (VO_{2max}). A written medical history form was also completed at this time. Other written instruments used to collect personal information and objective data during this study included the RRP Demographic Information Form, a 3-day food recall sheet, and medical outcome survey.

Body composition, which includes percent body fat, lean weight, and fat weight, was measured by air displacement plethysmography (ADP) using the BodPod Gold Standard Body Composition Tracking System (Life Measurement Inc., Concord, CA) according to standard methods (Dempster & Aitkens, 1995). Prior to use, the machine was calibrated using a 2-point calibration system by taking a measurement at baseline with the chamber empty and then again after placing a 50-liter calibration cylinder in the chamber. Participants wore a tightly fitting swimsuit, a swim cap, and a nose clip while being measured. Participants were first weighed on a calibrated scale, and they were then asked to enter the BodPod chamber where they were required to sit upright. The door was then closed so that an initial measurement could be obtained. During the initial measurement, participants were asked to relax and breathe normally in the chamber for 20 seconds. This process was repeated twice, and the measurement was accepted if the two body volume measurements agreed within 150 mL. After the base volume measurement was obtained, the door was opened, participants were asked to breathe through a long hose connected to the BodPod's breathing circuit, and the door was closed again. This second measurement was performed in order to measure functional residual capacity (FRC), the amount of air that remains in the lungs after normal exhalation. The system then calculated participants' body composition (percent fat, fat mass, and fat-free mass) from the data obtained.

A certified exercise physiologist measured waist circumference using a calibrated self-retractable, no stretch measuring tape (The Gulick II, model 67020; Gay Mills, WI) with a 4-oz tension indicator. Participants stood with feet slightly apart and arms hanging

loosely for the waist measurement. Waist circumference (in) was measured at the narrowest point between the iliac crest and the lowest rib with clothing displaced to reveal the area. The measuring tape was placed horizontally around participants' waists and the measurement was taken after exhalation. The tension indicator was used to ensure the measuring tape adhered to participants' skin surface and that tension was consistent between measures.

Blood pressure measurements were recorded in millimeters of mercury (mm Hg) and were obtained using a Welch Allyn sphygmomanometer (part number 5098-02; Skaneateles Falls, NY) and a Welch Allyn stethoscope (part number 5079-73; Skaneateles Falls, NY). Certified physical trainers and exercise physiologists obtained resting blood pressure measurements. Participants were asked to sit quietly for 10 minutes with feet on the floor, back supported by the chair and arm resting at heart level. The blood pressure cuff was placed approximately one inch above the antecubital fossa of the elbow. After the cuff was inflated, both systolic and diastolic blood pressure measurements were obtained using standard methods as outline by the American Heart Association (Bordley, Connor, Hamilton, Kerr, & Wiggers, 1951).

Maximal oxygen uptake, or $\text{VO}_{2\text{max}}$ (mL/kg/min), was predicted from a maximal treadmill stress test using the Bruce protocol (Bruce, Kusumi, & Hosmer, 1973). According to this protocol, the treadmill began at 1.7 mph and 10% grade (4 METs). The speed and grade of the treadmill increased at 3-minute intervals (a 3 MET increase per stage) until participants reached volitional fatigue. Participants were allowed to place one or two fingers on the treadmill handrail for balance purposes; however, using

handrails for body weight support was not permitted. The study physician used a standard 12 lead electrocardiogram (ECG) to monitor cardiac function (heart rate and rhythm) throughout the test. At the end of each stage participants were asked to their perceived exertion using the 6-20 Borg scale. The scale was properly grounded prior to the start of the exercise test. Blood pressures measures were also obtained at each stage of the Bruce protocol during the exercise stress test to ensure proper hemodynamic response to the exercise test. During this measurement, participants were asked to rest their arm on the exercise physiologist's shoulder. Participants were asked to exercise to maximum volitional exhaustion, at which time maximal heart rate and rating of perceived exertion (RPE) were measured and recorded. A regression equation was then used to predict $\text{VO}_{2\text{max}}$ (mL/kg/min), which took into account total time accomplished during the test and maximum RPE.

Blood lipid and blood glucose levels were measured after a 12-hour overnight fast and were obtained by intravenous blood sample. Blood samples were drawn by a certified phlebotomist via venipuncture. Blood samples were sent to the Athens Medical Lab in Athens, OH, where they were analyzed for total, LDL and HDL cholesterol levels, serum triglyceride levels, and fasting blood glucose levels using a Johnson and Johnson Ortho-Clinical Diagnostic Virtos® 350 Chemistry System.

The results of the baseline medical tests were reviewed and initial physical activity goals for weeks 2-15 were established. Participants met with a case manager to review their needs, set SMART (Specific, Measurable, Action-Orientated, Realistic, and Time-Bound) goals, and to design an appropriate and individualized exercise program.

Each program was modified to meet the participants' individual goals. Participants engaged in assisted exercise with a certified personal trainer twice weekly for 1-1.5 hours of aerobic and resistance training. Participants were also encouraged to exercise on their own to meet their goals. Participants were also required to attend the following sessions during the first 100 days: 1 hour of a facilitated support group per week, 2 hours stress management/yoga classes with a certified yoga instructor per week, and 3 1-hour nutrition education groups with a registered dietitian. Table 11 summarizes the content of the three group nutritional counseling sessions.

Table 11

RRP Group Nutrition Counseling Session Content

Session	Theme	Content
Session 1	Introduction to DASH-like diet	<ul style="list-style-type: none"> • Whole grain, fruits, vegetables, legumes, nuts, low-fat dairy
	Identifying and choosing fats wisely	<ul style="list-style-type: none"> • Ca⁺, Mg, K⁺, Fiber • Type and sources of fat • Low-fat alternatives • How to decrease discretionary kcals
Session 2	Mediterranean meal planning for heart health	<ul style="list-style-type: none"> • Omega-3 fatty acid sources
	Increasing protein and decreasing fat	<ul style="list-style-type: none"> • Nutrition label reading • Decreasing sodium • Plant based protein sources • Meal planning techniques • Vegetarian meal option
Session 3	Portion control	<ul style="list-style-type: none"> • Eating out techniques
	Holiday/Special Occasion Eating	<ul style="list-style-type: none"> • Identifying high risk situations • Relapse prevention • Portion Management • Staying Active

The group nutritional education sessions focused on heart healthy DASH-like and Mediterranean-like dietary recommendations. Behavioral modification therapy

techniques were used, including motivational interviewing, the stages of change, and cognitive behavioral therapy, throughout the group and individual counseling sessions. A registered dietitian provided participants with dietary intake goals based on individual needs, focusing on decreased caloric intake (-500 to -1000 kcals) for weight loss and specific macro- and micronutrient intake goals based on DASH, Mediterranean diet recommendations and age-specific RDAs. Participants were asked, but not required, to keep 24-hour food record for three days (two weekdays and one weekend day) after each group and individual session to assess intakes. All food records were reviewed by the registered dietitian and entered in Nutritionist Pro™ (First Data Bank, San Bruno, CA, USA) for analysis. Participants also made SMART goals based on nutrient intakes after each session. General dietary guidelines were to: a) incorporate at least 4-5 servings of vegetables; b) include 2-4 serving of fruit; c) consume 3 servings of low-fat dairy; d) make at least half of your grains whole grains; e) decrease saturated and *trans* fat intake; f) increase mono- and polyunsaturated fatty acid intake; and, g) increase vegetarian meals made with legumes, beans, nuts, and seeds.

Personalized nutrition counseling was also available during the first 100 days and consisted of an initial 1-hour intake session to determine individual goal and eating plans. This initial session was followed up 7 to 14 days later with a ½-hour session to gauge progress. The registered dietitian determined how often additional ½-hour sessions were needed and these sessions were scheduled on a weekly or bi-weekly basis. Weight was used to track progress. Dietary intake patterns were analyzed using Nutritionist Pro™ (First Data Bank, San Bruno, CA, USA). Behavior modification, motivational

interviewing, the stages of change, and cognitive behavioral therapy techniques were applied in the individual sessions, as well. Participants were “staged” using the stages of change construct prior to beginning the RRP and then periodically throughout the program. A variety of tools were utilized based on participants’ individual stages. For example, participants who were in the preparation stage may have received menu patterns and tracking tools, as well as information related to meal planning. After 2-3 weeks, the registered dietitian assessed participants’ progress and modified their diet therapy appropriately. Self-monitoring techniques were strongly encouraged, though not a required part of the intervention. Self-monitoring tools utilized included computer and smartphone applications, such as LoseIt!, FitDay, MyPlate.gov and sparkpeople.com, food and activity logs, and individual journaling. During week 15, the last week of the 100 day kick-start, all tests from week 1 were repeated; these samples were collected and analyzed in the same manner as week 1.

Phase II began after the 100 day kick-start and lasted from month 3 through month 6. During phase II participants met with program staff in order to reset SMART goals for the next 90 days. Supervised exercise sessions continued with a certified personal trainer, however, training sessions were increased to three times a week and focused on advanced resistance exercises. Participants continued to be encouraged to exercise on their own to meet new individual goals. During this time yoga/stress management were no longer mandatory; however, attendance was strongly encouraged. Participants attended monthly meetings with a case manager and monthly group support sessions. Participants also met with the registered dietitian at least once during this phase

to review nutritional goals. At the end of Phase II, participants completed exercise stress tests and were measured for blood lipids and blood glucose levels, waist circumference, and body composition. All samples were collected and tests were completed in the same manner as week 1.

Phase III lasted from month 7 through month 9. During this time participants continued to engage in lifestyle changes but did so independently. They no longer met with their personal trainer, however new SMART goals were set and participants were encouraged to work out on their own to meet their new goals. Monthly check-in meetings occurred with the case manager and the registered dietitian, as needed. While group support meetings were no longer offered through the RRP, participants were encouraged to continue the meetings on their own. At the end of this period participants were again measured for blood lipids and blood glucose, waist circumference, and body composition and completed an exercise stress test. All samples were collected and tests were completed in the same manner as week 1.

Phase IV last from month 10 through month 12. As in Phase III, participants continued to engage in lifestyle changes but did so independently. They no longer met with their personal trainer, however new SMART goals were set and participants were encourage to work out on their own to meet their new goals. Monthly check-in meetings occurred with the case manager and the registered dietitian, as needed. While group support meetings were no longer offered through the RRP, participants were encouraged to continue the meetings on their own. During the last week of the program participants, completed another written medical history form, a physical assessment including

measurements body composition, blood work for glucose and lipid levels, and an exercise stress test that determined maximal oxygen uptake ($\text{VO}_{2\text{max}}$). All samples were collected and tests were completed in the same manner was week 1.

Data Collection

The measurements obtained during week 1 were compared with the measurements obtained at 100 days and at 1 year to determine the effect of the program after 100 days and after one year of participation. Specifically, data were pooled from 12 cohorts that completed the program during 2006 to 2010. The study consisted of 93 adult (53 females, 19 males) who completed the program. Repeated measures analyses of variance (ANOVA) were conducted to compare the aforementioned anthropometric and physiological variables at baseline, 100 days and 1 year. Table 12 summarizes the research questions and the corresponding statistical method for the analyses.

Table 12

Statistical Analyses Used to Answer Research Questions

Research questions	Hypotheses
1. What is the effect of the RRP on body weight, % body fat, lean weight, fat weight, BMI after 1 year of program participation (baseline, 100 days, 1 year)?	Repeated Measures Analysis of Variance (ANOVA)
2. What is the effect of the RRP on HDL, LDL and total cholesterol levels, triglycerides, blood pressure, fasting glucose levels and VO ₂ max after 1 year of program participation (baseline, 100 days, 1 year)?	Repeated Measures Analysis of Variance (ANOVA)

CHAPTER 4: RESULTS

The purpose of this study was to determine the effectiveness of a comprehensive lifestyle intervention program which integrated dietary modification, physical activity, stress management, and behavior modification counseling to reduce the risk of clinically-significant cardiovascular events in at-risk adults. More specifically, the purpose of this study was to evaluate the efficacy of the RRP to reduce cardiovascular risk factors in an at-risk adult population after one year of program participation.

Participant Demographics

This study utilized a pooled convenience sample of 97 adults who participated in the RRP from 2006 through 2011. A total of 72 females (74.2%) and 25 males (25.8%) were participants. Of the 97 individuals who began the program, 74 participants (76.3 %; 57 females [77.1%]; 17 males [22.9%])) completed it. Participants ranged in age from 28 to 72 years old, with a mean age of 50.1 ± 8.8 years. Figure 1 summarizes the gender and number of participants from each of the 12 cohorts utilized in this study. No other demographic information (e.g., race/ethnicity, education level) was collected during the study.

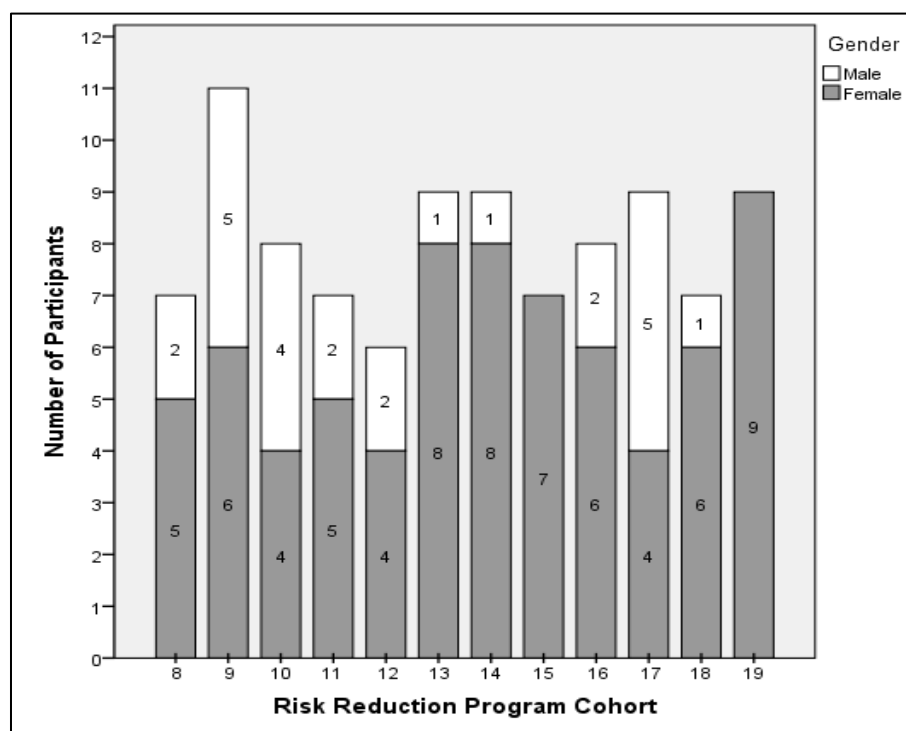


Figure 1. Number and gender of participants in the RRP from 2006-2010, grouped by cohort.

As noted in Chapter 3, repeated measures ANOVAs were used to determine whether the study variables differed significantly from baseline to 100 days of program participation, from 100 days to 1 year of participations, and from baseline to 1 year of participation. Table 13 summarizes the baseline characteristics of participants and the effects of the RRP on body composition, histological, and cardiorespiratory parameters after 100 days. Blood glucose ($p = .093$) and HDL cholesterol ($p = 1.0$) were the only measures that did not change significantly during the first 100 days of treatment. From 100 days to 1 year of treatment only HDL cholesterol improved significantly ($p < .001$). No other statistically significant changes occurred during this phase in the treatment process.

Table 13

Baseline Characteristics of RRP Participants and Effects of the RRP After 100 Days

Characteristic	Baseline		100 Days		p^a	p^b
	n	Value	n	Value		
Weight (lbs)	97	209.8 ± 49.85	94	200.94 ± 48.03	< .001	.911
Body Composition						
% Body fat	97	43.71 ± 8.11	94	40.84 ± 8.26	< .001	1.00
Lean mass (lbs)	97	117.05 ± 24.88	94	116.18 ± 24.78	< .001	1.00
Fat mass (lbs)	97	93.17 ± 32.16	94	83.97 ± 31.59	< .001	1.00
BMI (kg/m ²)	97	33.50 ± 7.55	94	32.01 ± 7.42	< .001	1.00
Waist Circumference (in)	96	40.37 ± 7.28	93	38.12 ± 7.72	< .001	1.00
Cholesterol (mg/dL)						
Total	97	191.21 ± 35.81	92	179.60 ± 34.63	< .001	.434

(Table 13, continued)

Characteristic	Baseline		100 Days		p^a	p^b
	n	Value	n	Value		
HDL	97	49.61 ± 13.42	92	49.48±11.82	1.00	.001
LDL	97	114.89 ± 33.85	92	108.29 ± 31.77	.005	1.00
Triglycerides (mg/dL)	97	138.28 ± 87.09	92	114.14 ± 53.77	.004	.389
Blood glucose (mg/dL)	95	97.96 ± 30.07	92	93.42 ± 16.19	.093	1.00
Blood Pressure (mm Hg)						
Systolic	97	134.82 ± 16.54	92	128.63 ± 15.32	< .001	1.00
Diastolic	97	83.46 ± 9.03	92	78.66 ± 9.89	< .001	1.00
VO ₂ max (mL/kg/min)	97	28.32 ± 5.10	92	32.17 ± 5.44	< .001	1.00

Note. Data are means ± standard deviations; BMI = body mass index; HDL = high-density lipoprotein; LDL= low-density lipoprotein; VO₂ max = maximal oxygen consumption; a = baseline to 100 days; b = 100 days to one year; c = baseline to 1 year.

Table 14 summarizes the effects of the program on body composition, histological, and cardiorespiratory parameters of those participants who completed the program. In the completers analysis, lean body mass did not obtain significance during the study period. Blood glucose ($p = .085$) and HDL cholesterol ($p = 1.00$) were the only measures that did not change significantly during the first 100 days of treatment. From 100 days to one year of treatment only HDL cholesterol improved significantly ($p < .001$). No other statistically significant changes occurred during this phase in the treatment process. At 1 year, total cholesterol (.053) was no longer significant.

Table 14

Results of a Multidisciplinary Intervention Program on Body Composition and Histological Parameters During 1 Year of Treatment

Characteristic	Baseline		100 Days		1 Year		p^a	p^b	p^c
	n	Value	n	Value	n	Value			
Weight (lbs)	74	204.64 ± 5.86	73	195.64 ± 5.60	73	195.35 ± 5.50	< .001	.938	< .001
Body Composition									
% Body fat	74	43.47 ± 1.00	73	40.41 ± 1.01	72	40.62 ± 1.04	< .001	1.00	< .001
Lean mass (lbs)	74	114.74 ± 2.95	73	114.69 ± 2.87	73	113.99 ± 2.74	1.00	.084	.111
Fat mass (lbs)	74	90.45 ± 0.91	73	81.06 ± 3.17	73	81.36 ± 3.77	< .001	1.00	< .001
BMI (kg/m ²)	74	32.87 ± 7.83	73	31.31 ± 0.89	73	31.33 ± 0.88	< .001	1.00	< .001
Waist Circumference (in)	73	39.68 ± 0.87	73	37.33 ± 0.92	70	37.80 ± 0.87	< .001	1.00	< .001
Cholesterol (mg/dL)									
Total	74	193.45 ± 4.24	73	181.59 ± 4.28	70	183.83 ± 5.18	< .001	.461	.053

(Table 14, continued)

Characteristic	Baseline		100 Days		1 Year		p^a	p^b	p^c
	n	Value	n	Value	n	Value			
HDL	74	49.20 ± 1.42	73	49.48 ± 1.30	70	53.01 ± 1.56	1.00	< .001	.011
LDL	74	116.77 ± 4.14	73	108.70 ± 3.78	70	108.53 ± 4.55	< .001	1.00	.035
Triglycerides (mg/dL)	74	143.68 ± 11.10	73	117.40 ± 6.68	70	118.51 ± 8.04	.004	.596	.019
Blood glucose (mg/dL)	72	99.00 ± 3.69	73	93.48 ± 1.92	70	93.07 ± 2.58	.085	1.00	.008
Blood Pressure (mm Hg)									
Systolic	74	135.11 ± 1.95	71	128.87 ± 1.90	72	130.57 ± 1.97	.001	1.00	.028
Diastolic	74	83.81 ± 1.06	71	78.87 ± 1.08	72	79.99 ± 1.02	< .001	1.00	.001
VO ₂ max (mL/kg/min)	74	28.55 ± 0.61	71	32.61 ± 0.68	72	32.82 ± 0.70	< .001	1.00	< .001

Note. Data are means ± standard errors; BMI = body mass index; HDL = high-density lipoprotein; LDL = low-density lipoprotein; VO₂ max = maximal oxygen consumption; a = baseline to 100 days; b = 100 days to one year; c = baseline to 1 year.

CHAPTER 5: DISCUSSION

The purpose of this study was to evaluate the efficacy of the RRP to reduce cardiovascular risk factors in an at-risk adult population after 1 year of program participation. The results demonstrate that this multidisciplinary lifestyle intervention program was effective in reducing cardiovascular disease risk factors in an at-risk adult population after 1 year of treatment. The results of this study are similar to those of other lifestyle interventions, indicating that a multidisciplinary approach to reducing cardiovascular disease risk factors is an effective means of treatment.

Effect of the RRP from Baseline to 100 Days

Overall, the majority of change observed in the study variables occurred from baseline to 100 days of program participation. With the exception of HDL cholesterol and blood glucose levels, weight, body composition parameters (% body fat, fat mass, lean body weight), histological parameters (total cholesterol, LDL cholesterol and triglycerides), and cardiorespiratory parameters (systolic and diastolic blood pressure, VO_{2max}) obtained significance at the completion of the first 100 day of the RRP. These results are reported elsewhere and will be discussed only briefly in this study (Knight, 2011).

Effects of the RRP after 100 Days of Treatment

The first aim of this study was to determine the effect of the RRP on body composition parameters including body weight, percent body fat, fat weight, BMI, and waist circumference after 1 year of program participation. Body composition was assessed three times: prior to starting the program, after 100 days, and after 1 year. It

was hypothesized that the RRP would have an initial positive effect on these variables from baseline to 100 days and would have an attenuated effect from 100 days to 1 year of program participation. Because each of these variables is related to cardiovascular health, significant improvement would be considered improved cardiovascular health, and ultimately, decreased risk for cardiovascular disease. During the initial 100 days of the program, significant reductions were seen in all body composition parameters. However, from 100 days to 1 year of program participation, no further significant reductions were observed, thus supporting our hypothesis. Interestingly, the changes in body composition parameters observed during the first 100 days of the RRP were robust enough to persist after 1 year of program participation.

The RRP participants demonstrated a significant reduction in body weight ($p < .001$) after 1 year of treatment. The average reduction in total body weight was -11.23 ± 0.05 lbs. This equates to an average reduction of $5.30 \pm 0.71\%$. The NHLBI (1998) recommends an initial weight loss goal of a 10% reduction in body weight. Six months is considered an appropriate timeline for which to meet this initial goal (NHLBI, 1998). On average, the RRP participants did not meet this goal. It should be noted, however, that participants demonstrated a significant reduction in body weight ($p < .001$) after the initial 100 days of the program. During the first 100 days, the average change in total body weight was -10.17 ± 0.89 lbs, or an average reduction of $4.87 \pm 0.41\%$. This indicates that at approximately 3 months (100 days), participants appeared to be on track to meet the initial 10% reduction outlined by the NHLBI. The NHLBI acknowledges that after the initial 6 months, weight loss tends to plateau and modifications of the weight

loss regime are generally required. This may be the case with the RRP. While the RRP was not successful in achieving a 10% weight reduction, it appears that the program was successful in preventing weight regain. Participants continued to lose weight over the course of the program, and although later body weight reductions were smaller, they were still statistically significant after the 1-year study period.

After 1 year of treatment, RRP participants demonstrated significant improvements in body composition parameters including significant decreases in percent body fat ($-7.13 \pm 1.28\%$; $p < .001$); fat mass (-9.94 ± 1.30 lbs; $p < .001$); BMI (-1.74 ± 0.23 kg/m²; $p < .001$); and waist circumference (-3.09 ± 1.11 inches; $p < .001$). The significant reductions in these parameters suggest a decrease in overall adiposity, as well as abdominal adiposity. It is well known that BMI is an indirect measure and cannot be used to characterize the amount or distribution of adipose tissue (Frankenfield, Rowe, Cooney, Smith, & Becker, 2001; NHLBI, 1998). Population-based studies indicate a relationship between increased BMI category and increased risk of cardiovascular mortality (Flegal et al., 2010; Must et al., 2004). BMI is often utilized as a monitoring tool because it is an effective, inexpensive measure. Waist circumference, a measure of abdominal adiposity, is more strongly associated with percentage of body fat (Yusef et al., 2004). However, when the above measures are viewed together, they give rise to a more complete, accurate representation of the amount and distribution of adipose tissue. The improvement of overweight or obesity, and particularly abdominal adiposity, is associated with improved metabolic function including reduced blood cholesterol, lower

blood pressure, improved insulin function, a reduced risk for type 2 diabetes, and ultimately, a reduced risk for cardiovascular disease (Goldstein, 1992; Klein et al., 2004).

A number of other multidisciplinary studies observed similar improvements in body weight and body composition parameters, further supporting that the RRP improves cardiovascular disease risk (Eriksson et al., 2006; Goodpaster et al., 2010; Oh et al., 2010). Eriksson et al. (2006) found statistically significant improvements occurred in anthropometric and clinical variables during the first 3 months of a lifestyle intervention program consisting of supervised physical activity, diet counseling, and regular follow-up meeting for patients in a primary care setting. Specifically, the observed reduction in body weight persisted as statistically significant after 1 year of program participation.

Goodpaster et al. (2010) assessed the effectiveness of a dietary intervention with delayed onset of physical activity compared to an intervention that combined diet therapy and initial physical activity. They observed significant reductions in body weight, waist circumference, BMI, and percent body fat in both groups at 6 months. This reduction persisted as statistically significant at 1 year of the intervention program, as well. Notably the diet therapy plus exercise group demonstrated significantly greater reductions in these body composition parameters compared with the diet only group at 6 months, although the reductions were not statistically different between groups after 1 year of treatment. Oh et al. (2010) examined the effectiveness of a 6-month, community-level lifestyle modification program that consisted of health education, physical activity, and diet therapy, with a follow-up period 6 months after the intervention ended. They also observed significant reductions in body weight, waist circumference and BMI at 6

months, and these parameters remained significant 6 months after the intervention ended. These results are consistent with the results of the present study.

A second aim of this study was to determine the effect of the RRP on HDL, LDL and total cholesterol levels, triglycerides, blood pressure, fasting glucose levels, and VO_2 max after one year of program participation. It was hypothesized that the RRP would have an initial positive effect on these variables from baseline to 100 days, followed by an attenuated effect from 100 days to 1 year of program participation. Because each of these variables is related to cardiovascular health, significant improvement would be considered improved cardiovascular health and, ultimately, a decreased risk for cardiovascular disease.

From baseline to 100 days of the program, significant reductions were seen in all histological parameters with the exception of HDL cholesterol and blood glucose levels. As seen with the body composition parameters, the changes in histological parameters observed from baseline to 100 days of the RRP were robust enough to persist after 1 year of program participation. HDL cholesterol did obtain statistical significance during the period from 100 days to 1 year of program participation; it was also significantly higher at 1 year. Significant improvement in blood glucose was only observed after 1 year of treatment. Notably, no significant improvements were observed in the any parameters other than HDL cholesterol during the period from 100 day to 1 year, supporting our hypothesis.

From baseline to 1 year, significant improvements were observed significant reductions in LDL cholesterol (-10.26 ± 3.61 mg/dL, $p = .035$), and triglycerides (-22.97

± 8.26 mg/dL, $p = .019$) and a significant increase in HDL cholesterol (2.85 ± 1.36 mg/dL, $p = .007$). The INTERHEART study, a study of 52 countries, has identified dyslipidemia as one of the most important risk factors for developing cardiovascular disease (Yusuf et al., 2004). Surprisingly, total cholesterol was no longer statistically significant after 1 year of treatment, contradicting the results of other similar studies. The observed reductions in LDL cholesterol, and triglycerides are consistent with the results from other multidisciplinary risk reduction program (Chen et al., 2009; Goodpaster et al., 2010; Kuller et al., 2006). Chen et al. (2009) found that triglycerides, total cholesterol and LDL cholesterol were significantly improved after a 3-month lifestyle intervention. Goodpaster et al. (2010) observed that blood lipid profiles were significantly improved after 6 months of treatment in a lifestyle intervention program. The improvements observed in blood lipid profiles persisted as statistically significant after one year of treatment. Kuller et al. (2006) also found that cholesterol, LDL cholesterol, and triglycerides were significantly reduced in a female study population after 6 months of treatment. Notably, these parameters persisted as statistically significant after 18 months of treatment.

The reduction of other cardiometabolic risk factors including overweight and obesity and waist circumference have also been associated with improve blood lipid profiles (NHLBI 1998). A number of studies have reported decreases in HDL cholesterol during active weight loss. A meta-analysis by Dattilo and Kris-Etherton (1992) found that during active weight loss, small but significant decreases in HDL cholesterol can occur. However, this decrease in HDL cholesterol appears to resolve once weight

reduction has stabilized (Dattilo & Kris-Etherton, 1992). The initial decrease in HDL cholesterol may be related to lipoprotein lipase (LPL) activity. During caloric restriction, the concentration of tissue LPL can decrease by 50-80% which may impair both triglyceride-rich lipoprotein synthesis and VLDL cholesterol catabolism (Taskinen & Nikkila, 1979). This impairment in lipid metabolism can, in turn, limit the transfer of lipid to HDL cholesterol and result in an initial decrease in HDL cholesterol concentration in response to weight loss. Once weight reduction is stabilized lipid metabolism normalizes and small increases (0.14 mmol/L) in HDL cholesterol have been observed (Dattilo & Kris-Etherton, 1992). Since RRP participants experienced the majority of their weight loss during the first 100 days of the program, this may account for the lack of change in HDL cholesterol during that time. The significant increases in HDL cholesterol demonstrated from 100 days to 1 year may be a result of the study population being a prolonged state of weight maintenance.

Physical activity and diet therapy have been independently shown to raise HDL cholesterol (Durstine & Thompson, 2001; Fletcher et al., 2005). Specifically, a diet pattern that encourages the increased intake of “heart-healthy” foods, such as whole grains, fruit and vegetables, nuts, mono- and polyunsaturated fatty acids, as well as a total fat intake of less than 30% of total calories with < 10% from saturated fat and less than 200 mg cholesterol, can help reduce cholesterol levels. This dietary pattern has been shown to reduce total cholesterol, LDL cholesterol, and triglyceride levels and to increase HDL cholesterol levels (Fletcher et al., 2005). While we cannot determine the individual

effects of increasing physical activity or following the above dietary pattern on HDL cholesterol, the intervention appears to have an overall positive effect.

After 1 year of treatment, the RRP participants demonstrated a significant decrease in blood glucose levels ($p = .008$). An average decrease of 4.46 ± 3.11 mg/dL, or $4.53 \pm 1.33\%$, was observed. Somewhat surprisingly, there was no significant decrease in blood glucose during the initial 100 days of treatment which contradicts the results of similar, although shorter, multidisciplinary studies (Aizawa et al., 2009; Goodpaster et al., 2010; Kim et al., 2006; Oh et al., 2010). Aizawa et al. (2009), Kim et al. (2006) and Oh et al. (2010) reported significant reductions in fasting blood glucose levels after 6 months. Notably, Goodpaster et al. (2010) reported results similar to our study, with blood glucose levels reaching a statistically significant reduction only after 1 year of treatment.

It is well documented that individuals with type 2 diabetes and impaired insulin function or prediabetes are often overweight or obese and/or present with abdominal adiposity (ADA, 2011). Our results support the guidelines of the American Colleges of Sports Medicine, the American Diabetes Association, and the American Heart Association that recommend concurrent diet therapy and physical activity in order to improve blood glucose levels (Albright et al., 2000; Grundy et al., 1999). The improvement of other cardiometabolic risk factors such as dyslipidemia, hypertension, overweight and obesity, and waist circumference have also been associated with improvements in blood glucose levels (Chobanian et al., 2003; Grundy et al., 1999; NHLBI, Adult Treatment Panel III, 2002). Each of these risk factors was improved

during the RRP and could also account for the observed improvements in blood glucose levels.

RRP participants demonstrated significant reductions in blood pressure (SBP, $p = .028$; DBP, $p = .001$) after 1 year of treatment. Weight reduction, the restriction of sodium, and regular physical activity has all been shown to independently decrease blood pressure and reduce the risk for cardiovascular disease. Studies have demonstrated that weight loss of 4.5 pounds can result in a 5-20 mm Hg reduction in systolic blood pressure (Appel et al., 2009; Trials of Hypertension Collaborative Research Group, 1997). The RRP supports these results with participant losing an average of -11.23 ± 1.47 pounds and demonstrating an average reduction in systolic blood pressure of 5.15 ± 1.89 mm Hg. The RRP results are also consistent with those of the Trials of Hypertension and the recommendation of the Seventh National Joint Committee (JNC-7) on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure which demonstrate the effectiveness of diet therapy, physical activity, and weight reduction to reduce both systolic and diastolic blood pressure (Chobanain et al., 2003; Trials of Hypertension Collaborative Research Group, 1997).

The Trials of Hypertension Prevention phase II, study demonstrated that weight loss alone, a reduced sodium diet alone, and a combination of weight loss and diet therapy each effectively reduced systolic and diastolic blood pressure in overweight adults. However, at 6 months of treatment, the combination of weight loss and diet therapy results in the greatest reductions in both systolic and diastolic blood pressure (Trials of Hypertension Collaborative Research Group, 1997). The JNC-7 recommends

the implementation of physical activity and dietary interventions that promote weight loss in order to reduce blood pressure (Chobanain et al., 2003).

After 1 year of treatment, RRP participants demonstrated significant improvements in VO_2 max ($p < .001$). VO_2 max is considered the gold standard for determining cardiorespiratory fitness. Improvements in this measure are a result of exercise training and reflect improvements in both physical fitness and cardiovascular health. In this study, VO_2 max increased from 28.55 ± 5.24 to 32.82 ± 5.95 ml/kg/min after one year of treatment. This is approximately a 15.72% increase and is similar to other multidisciplinary programs (Blumenthal et al., 2010; Eriksson et al., 2006).

Eriksson et al. (2006) observed significant increases in VO_2 max after a 6-month lifestyle intervention which persisted as statistically significant after 1 year. Blumenthal et al. (2010) reported a 19% increase in VO_2 max after only 4 months of treatment in a multidisciplinary lifestyle modification program. A meta-analysis by Kodama et al. (2009) reported that a 1 MET increase in VO_2 max is associated with a 13% decrease in the risk of all-cause mortality and a 15% decrease in the risk of coronary heart disease. Other studies have shown that aerobic exercise intervention can lead to increases in VO_2 max on the magnitude of 16.3-22.5% (approximately 3.5-3.8 mL/kg/min), which is also consistent with the results the of the present study (Green & Crouse, 1995; Huang, Gibson, Tran, & Osness, 2005). Studies have shown that improvement in cardiorespiratory fitness, without concomitant improvement in BMI or body composition, is associated with improvements in cardiometabolic health and decreases in cardiovascular and all-cause mortality (Lee, Blair, & Jackson, 1999; Ross & Janiszewski,

2007; Sojung et al., 2005). Improvements in cardiorespiratory fitness as small as a 1 minute increase in treadmill time have been related to positive improvements in cardiometabolic health (DiPietro, Kohl, Barlow, & Blair, 1998). Genetics and exercise training can both play a significant role in the determination of VO_2 max. According to results from the HERITAGE Family Study, which examined the role of genotype in response to aerobic exercise training, changes in VO_2 max are approximately 50% genetically influenced in sedentary adults (Bouchard et al., 1999). The greatest increases in VO_2 max occur in individuals with low pretraining measures, deconditioned individuals and in response to aerobic training (Nieman, 2003; Valkeinen, Aaltonen, & Kujala, 2010).

Conclusions and Recommendations

This study was designed to determine effectiveness of the comprehensive lifestyle intervention program that combined diet therapy and an exercise regimen, as recommended by the American Heart Association, plus stress management techniques and behavior modification counseling, in order to reduce the prevalence of clinically-significant cardiovascular events. Specifically, the RRP integrated an individually-tailored exercise program, personalized dietary education and counseling, and a stress management strategy in the form of yoga classes. The purpose of the study was to evaluate the efficacy of the RRP to reduce cardiovascular risk factors in an at-risk adult population after 1 year of program participation. Although a number of studies have been published that include the treatment variables outlined by the American Heart Association, the integration of a stress management strategy (via yoga), the duration of

the intervention program and the measurement of an extensive array of histological, cardiometabolic, and cardiorespiratory parameters reported in the present study make the RRP unique among lifestyle modification programs.

After 1 year of treatment the RRP demonstrated significant improvements in body composition (body weight, percent body fat, fat mass, BMI, waist circumference) histological parameters (blood lipid profile, blood glucose levels) and significant increases in VO_2 max. The physical activity component of this intervention effectively improved cardiorespiratory fitness, as demonstrated by increases in VO_2 max. The significant reductions in total body weight, percent body fat, fat mass, BMI, waist circumference support the RRP as an effective intervention program. The results of this study are consistent with NHLBI's and the American Heart Association's recommendations utilizing multidisciplinary programs to reduce overweight and obesity and achieve ideal cardiovascular health, respectively.

The combination of diet therapy and physical activity in this program were effective in improving blood lipid profiles (HDL, LDL and total cholesterol) and blood glucose levels. The reduction in triglycerides and LDL and total cholesterol levels observed in the RRP supports recommendations from the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol, specifically the adoption of the Therapeutic Lifestyle Changes program (NHLBI, ATP III, 2002). The improvements in blood lipid profile components demonstrated by RRP participants also appears to support the results from Lyon Heart Mediterranean diet study and the positive effects of mono- and

polyunsaturated fatty acids on blood lipid profile (de Logreril et al., 1997). The significant improvements observed in systolic and diastolic blood pressure, especially in the presence of physical activity and weight maintenance are supported by results from studies utilizing DASH-like dietary interventions (Appel et al., 1997; Blumenthal et al, 2010; Sacks et al., 2001). The blood pressure reductions observed in RRP participants are also consistent with recommendations from JNC-7 on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. The significant improvements on the histological, cardiometabolic and body composition parameters in this study, when examined as a whole, indicate that the RRP was indeed successful in improving overall cardiovascular health by reducing the prevalence and severity of specific cardiovascular risk factors.

Surprisingly, the most robust improvements occurred during the initial 100 days of program. However, these changes were significant enough to persist through 1 year of treatment. It is important not to overlook what the results of this study indicate in terms of physiological significance. The improvements observed in body weight, percent body fat, fat mass, waist circumference, BMI, LDL and HDL cholesterol, blood glucose levels, systolic and diastolic blood pressure and VO_2 max are indicative of synergistic reductions in the risk for cardiovascular disease because each parameter is used as a measure for cardiovascular disease risk. Improvements in all parameters translate to an overall reduction in the risk for cardiovascular disease even if some parameters do not yet meet definitions for ideal cardiovascular health. For example, professional organizations recommend striving for HDL cholesterol levels ≥ 60 mg/dL. While the RRP participants

did not achieve this level, they did demonstrate, on average, approximately a 3 mg/dL increase in HDL cholesterol. Studies have shown that each 1 mg/dL increase in HDL cholesterol is associated with a 2-3% decrease in overall risk for cardiovascular. Based on HDL cholesterol alone, the RRP participants have decreased their risk for cardiovascular disease by 6-9%. While it is difficult to put an exact number the magnitude by which participants have reduced their risk for cardiovascular disease, the improvements observed all parameters related to cardiovascular disease coupled with the persistence of those improvements at 1 year indicates that the RRP was extremely successful.

This study had several limitations. First, there was no control group or alternative intervention group in this study. The lack of a comparison groups limits our ability to determine whether an individual portion of the RRP was more or less effective in impacting cardiovascular risk factors. That being said, there appears to be a general consensus in the literature on this topic that diet therapy plus exercise is the preferred method of treatment. Second, although group nutrition education and personalized nutrition prescription were provided in this intervention, dietary intakes were self-reported. Therefore, initial dietary intake and subsequent adherence to nutrition prescriptions could not be accurately measured or controlled for, limiting our interpretation of the results. Third, physical activity prescriptions were based on the needs and abilities of each individual RRP participant. While frequency and duration of exercise were prescribed they could not be controlled. Records related to exercise compliance were not recorded but should be included in subsequent studies. This limits

our ability to interpret correlations between exercise dose and risk factor improvements. Fourth, stress management was included in the RRP in the form of yoga classes. However, there were no psychological or psychosocial measures used in this study, limiting our ability to comment on the effectiveness of this component of the RRP. Finally, compliance was not measured in this study. Without compliance data we are unable to determine how effective the RRP in affecting the adoption of long term lifestyle modifications.

In light of the result from this study and the associated limitations, future studies should examine whether the lifestyle modifications stressed in the RRP program are feasible long-term strategies to the reduction of cardiovascular disease. The most robust improvements occurred during the initial 100 days (3 months) of the RRP. Although the improvements were strong enough to persist after 1 year of treatment, the value of some parameters were on the rise indicating that participants may not have been as successful at maintaining the observed improvement, and by association, the overall lifestyle changes.

The plateau effect observed in the RRP participants may be related to several factors. During the first 100 days, participants had access and intense support from the multidisciplinary team; this access and support was actually in place during the first 6 months of the program. After 6 months, participants were expected to execute their newly adopted lifestyle changes on their own. No longer having access to a personal trainer may have been particularly detrimental. The personal trainers spent the most time with participants out of any member of the multidisciplinary team and may have been a

greater source of motivation for participants than the other members of the team. The sudden lack of a support system may account for the lack of significant change in the study parameters during the second 6 months of the program. The lack of a support system may also have played a role in the nearly 25% drop-out rate in this study.

Analyzing participants' progress at 6 months, in addition to the existing time points in this study may yield a more complete characterization of the study population.

Implementing some type of trainer weaning schedule may also be useful. The current structure of the RRP means that participants go from working out with a personal trainer 2-3 times per week for 6 months to having no access to their trainer. Slowly transitioning participants from 3 weekly sessions to 1 weekly session and finally to no sessions may improve program outcomes.

It is also possible that trying to make so many different lifestyle changes was overwhelming for many participants and this caused them to either drop out of the study or to plateau. It may be prudent to train multidisciplinary team members to look for warning signs related to being overwhelmed so that they may intervene to keep participants motivated and in the program. If necessary, the multidisciplinary team should consider slowing the program down and focusing on fewer lifestyle changes simultaneously. In order to help jumpstart participants' motivation, member of the team should focus on the positive changes participants have already made and encourage the continuation of changes already adopted before adding any new goals.

The cost of RRP may have been vital to its success. The total cost of participation in the RRP was \$3,000 per person. Participants were only responsible for 10% of the

total cost of the program and could earn back the initial \$300 fee if they completed the program. The Department of Human Resources at Ohio University covered the remaining cost of the program. The personal investment may have improved compliance, and could potentially improve insurance claims over time, particularly if participants are successful in adopting the lifestyle changes in the long-term. As an employer, Ohio University may also see returns on their investment (\$2,700-\$3,000 per participant) if participants are successful in adopting long-term lifestyle change. Studies have shown that well-designed, well-implemented worksite wellness program can result in positive clinical and cost outcomes. A meta-analysis by Chapman (2012) concluded that worksite wellness programs can result in savings up to 25% each on absenteeism, healthcare costs, workers' compensation costs and disability costs for participants during a 2- to 5-year period (Chapman, 2012). Studies have also shown that for each dollar spent, comprehensive worksite wellness or risk reduction programs can see a \$3-\$6 return on their investment (ROI; Aldana, 2001; Goetzal, Juday & Ozminkowski, 1999; Pelletier, 2009). Based on the results of this study, Ohio University spent \$222,000 for the 74 completers to participate in the program. If a \$3 ROI for each dollar was achieved, Ohio University could potentially earn back \$666,000 on just the 74 completers from this study. If a \$6 ROI for each dollar was achieved, Ohio University would earn back over \$1.3 million dollars. Future studies should track insurance claims, healthcare costs and other related variable to determine the cost-benefits of implementing a risk reduction program.

Future studies should also include compliance measures and self-efficacy measures, particularly with the diet therapy and exercise components of the intervention, in order to determine the effectiveness of adoption of these behaviors. In the current RRP food and exercise journals are not required, only strongly encouraged. It has been well documented that individuals who keep food and activity logs tend to be more successful in their adoption of health behaviors. Developing a program-specific food and activity log may increase compliance and greater success for participants. The addition of such an instrument, especially if it was well received, more also provide specific information related to how each component of the RRP may reduce cardiovascular disease risk factors. If RRP participants lack self-efficacy, they will not be as successful at adopting lifestyle changes, and ultimately, will be less compliant with the RRP. The inclusion of self-efficacy measures may also allow the members of the multidisciplinary team to individualize strategies that build or promote self-efficacy for RRP participants.

Psychological or psychosocial measures should also be included in future studies in order to assess whether or not the stress management component of the intervention is effective in modifying perceived stress. The American Heart Association and the NHLBI have adopted stress as a cardiovascular risk factor; however there is a dearth of evidence on effective methods of mediating stress. A psychosocial instrument that includes items related perceived barrier to change, perceived susceptibility to cardiovascular disease, self-efficacy, perceived stress and quality of life should be incorporated in the RRP. This instrument should be completed each time participants complete their physiologic testing (blood samples, body composition assessment, and exercise stress test).

Future studies should increase the duration of the RRP, or similar programs.

Longitudinal studies that examine the effects of the intervention at 3 months, 6 months, 1 year, 2 year and 5 years would result in greater information about program compliance and long term adoption of the lifestyle changes promoted by the RRP. The RRP should also be adapted to other populations, particularly in youths/adolescents or younger adult populations, in order to examine and clarify the effects of a comprehensive lifestyle program on health outcomes in these populations. It is also suggested that a program similar to the RRP be utilized for primary and primordial prevention, as well as secondary prevention, in an effort to promote the adoption of long-term lifestyle changes.

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