EFFECTS OF COCONUT OIL COMPARED TO MCT OIL ON CARDIOVASCULAR DISEASE RISK FACTORS

by
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ABSTRACT OF THESIS

OBJECTIVE: To critically analyze the current research on dietary intake of coconut oil compared to MCT oil and associated resulting cardiovascular risk factors and to determine whether there is a negative or positive association between coconut oil and CVD.

DESIGN: Academy of Nutrition and Dietetics (AND) Evidence Analysis Library (EAL) Project.

METHODS: AND's EAL Methodology, which is an objective and evidence based process of systematic reviews used to evaluate, synthesize, and grade the strength of current nutrition research. The process consists of five steps: 1) Formulate the Evidence Analysis Question, 2) Gather and Classify the Evidence, 3) Critically Appraise Each Article, 4) Summarize the Evidence and, 5) Write and Grade the Conclusion Statement.

RESULTS: Utilizing a search of the PubMed database, 25 articles were identified relating to Cardiovascular Disease risk factors and various types of fats, including coconut oil and MCT oil. From this search, seven studies were analyzed for this project that met inclusion criteria. Several studies were excluded for a variety of reasons including those that did not include coconut oil, only looked at blood pressure, sample size too small, study used infants, not adults, or not human subjects, or were excluded for other various reasons. Of the seven studies included, four were randomized controlled trials, one was nonrandomized and two were observational in design. None of the studies found any significant increase in CVD risk factors in subjects consuming coconut oil. Several studies showed weight loss, reduced BMI, decreases in WC, reduction in metabolic risk, reduced CRP, improved cardiac function, increased HDL, and no significant increase in LDL.

CONCLUSION: According to the current research reviewed here, coconut oil consumption does not have a negative effect on CVD risk factors. The evidence shows that coconut oil and/or MCT oil aides in weight loss, decreases WC, which reduces metabolic risk, reduces CRP, improves cardiac function, raises HDL, and does not significantly raise LDL. MCFA from virgin coconut oil does not increase CVD risk and may reduce risk. This conclusion was graded *Fair, II*.

ACKNOWLEDGMENTS

Primarily I would like to thank God for carrying me through the most difficult times of my life. Without my faith, I would be nothing. I would also like to thank my family, especially my daughters: Natasha, Samantha, and Leila for all of their encouragement and support throughout this process. I know it was not always easy putting up with my challenging schedule. Thanks for your understanding and love. In addition, a sincere thanks to all the faculty members who have provided assistance throughout the years of my entire educational progression, especially Dr. Megan Baumler and Dr. Tara LaRowe. This endeavor would not have been possible without your continued and unparalleled guidance throughout this project as well as my entire adventure through the Master of Science in Dietetics program.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1: INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death worldwide (Heart Foundation, 2017). CVD is a group of diseases that includes heart disease, myocardial infarction (MI), coronary artery disease (CAD), peripheral vascular disease (PVD), and cerebral vascular attack (CVA) among others. Globally, CVD is responsible for 17.3 million deaths annually and is expected to increase to 23.6 million deaths by the year 2030 according to the American Heart Association (AHA). In the United States, CVD claimed 787,000 lives in the year 2011 according to The Heart Foundation. In fact, CVD claims more lives each year than all forms of cancer and chronic lower respiratory disease combined. (AHA)

Risk factors for CVD include modifiable and non-modifiable elements. Age, gender, and genetic predisposition are examples of non-modifiable factors. Modifiable factors include lifestyle components such as diet, smoking, activity level, and alcohol and other drug use, obesity and hypertension (HTN). Traditional treatments for CVD include surgical, pharmacological, and lifestyle change interventions. Surgery includes cardiac catheterization, angioplasty, stenting, and coronary artery bypass grafting (CABG). Drug therapy includes blood pressure lowering medications and cholesterol lowering medications. Lifestyle change interventions include cardiac rehabilitation classes and changes aimed at reducing risk factors such as increased physical activity level and adopting a heart healthy diet.

Nutrition recommendations for a heart healthy diet include a diet low in total and saturated fatty acids (SFA) and high in polyunsaturated fatty acids (PUFA) (Babu et al., 2014). Coconut oil is high in SFA, but also is rich in medium chain fatty acids (MCFA). The lipid profile of coconut oil is different than pure MCT oil however, and many studies use MCT oil and not coconut oil. There is not a lot of research to make a definitive conclusion as to whether or not consumption of coconut oil in addition to or to replace other sources of SFA is harmful or beneficial in CVD risk. There is research using animals that suggests health benefits of coconut oil but few human trials. In cultures that predominantly use coconut products, there is a low incidence of CVD; however, more research is needed to determine effects on CVD outcomes.

Rationale

The rationale for conducting this research project is to critically analyze the evidence on dietary intake of coconut oil and associated resulting cardiovascular risk factors and to determine whether there is a negative or positive association between coconut oil and CVD.

Potential Significance

This project could potentially benefit the field of nutrition and dietetics by providing clinical practitioners with evidence-based information that can assist practitioners and patients in their health care decisions. This study may provide information on how coconut oil could potentially reduce risk for CVD. The addition of coconut oil to the diet may give people more options in their dietary choices and

may help improve the quality of life for patients. This study will benefit the ongoing research of CVD prevention and treatment of associated risk factors.

Research Question

What is the effect of coconut oil consumption compared to MCT oil on cardiovascular risk factors in adults with or without existing CVD?

Sub-problems

1. Does the refining process of coconut oil make a difference?
2. How does coconut oil compare to other oils in CVD risk?

Limitations

1. Available research studies that involve coconut oil consumption related to CVD risk.
2. Original studies conducted within the last 10 years.

Delimitations

1. Studies will include randomized and non-randomized controlled trials as well as case-control studies and prospective cohorts.
2. Adult men and women, ages 20-75 with and without existing CVD.

Assumptions

1. The research studies will be conducted with honesty and integrity and without causing harm to subjects.
2. Researchers will be without bias.

Definition of Terms

Angina: Chest pain caused by inadequate flow of blood to the heart.

Angiogram: An X-ray procedure in which a catheter is inserted near the groin or arm and threaded through the artery to the heart; used to evaluate for blockages in the arteries.

Anthropometric measures: Measurements including BMI, waist-to-hip ratio, waist circumference, and skin fold thickness, which are used to assess body size and composition.

Arachidonic acid (ARA): A polyunsaturated essential omega-6 fatty acid found in animals and humans.

Arrhythmias: Abnormal or irregular heartbeat.

Atherosclerosis: A condition in which fatty deposits build up in arteries, causing them to become hardened and narrowed.

Cardiovascular disease: A group of diseases that affect the heart and blood vessels; includes coronary artery disease.

Chylomicrons: Lipoprotein particles consisting of triglycerides, phospholipids, cholesterol, and proteins that transport dietary lipids from the intestines to adipose, skeletal, and cardiac tissue.

Coronary artery disease (CAD): A buildup of plaques that cause a blockage in the blood vessels leading to the heart.

C-reactive protein: An acute-phase protein produced by the liver in response to inflammation, caused by a wide variety of conditions, from infection to cancer.

Dyslipidemia: Abnormal level of lipids in the blood, including elevated cholesterol, triglycerides, and low HDL.

Echocardiogram: An ultrasound test used to create pictures of the heart to evaluate structure, valves and chambers.

Endarterectomy: A surgical procedure to remove plaque buildup or blockage from the lining of an artery, restoring blood flow.

Extra virgin coconut oil (VCO): Unrefined product made by cold pressing the liquid from the coconut meat, then separating the oil without heating or chemically treating.

Glycated hemoglobin: A measure of the amount of glucose attached to the hemoglobin over average plasma glucose concentration. Also called glycosylated hemoglobin, or HgbA1c.

HDL: High-density lipoprotein; transports cholesterol particles back to the liver to be removed; also called "good" cholesterol

Hypercholesterolemia: High level of cholesterol in the blood.

Hyperlipidemia: An abnormally high concentration of lipids in the blood.

Ischemia: An inadequate blood supply to an organ or part of the body, especially the heart muscles.

Lauric Acid: A medium chain saturated fatty acid consisting of 12 carbons and found in coconut oil.

LDL: Low-density lipoprotein; makes up most of the cholesterol found in the body; high levels are associated with increased risk of heart disease.

Lipid: A biological substance that is insoluble in water and includes fats, waxes, oils, sterols, and fat-soluble vitamins.

Lipid profile/lipid panel: Blood tests that screen for abnormalities of lipids including cholesterol and triglycerides to assess cardiovascular risk.

Lipoprotein: Soluble proteins that combine with and transport fat or other lipids in the blood plasma.

Medium chain triglyceride (MCT): Made up of fatty acid chains containing 6-12 carbon atoms; found in coconut oil, palm kernel oil, and dairy products.

Metabolic syndrome: A group of conditions that can indicate increased risk for CVD, including hypertension, hyperglycemia, abdominal obesity, high serum triglycerides, and low HDL levels.

Monounsaturated fat: Fatty acids that contain only one double bond (or unsaturated carbon) in the chain; found in meats, milk, nuts, olives, avocados; usually liquid at room temperature.

Myristic acid: A common saturated fatty acid found in plants and animals, including butter, coconut and palm oils.

Palmitic acid: The most common saturated fatty acid found in animals and plants.

Plaque: A sticky substance made up of fat, cholesterol, and calcium found in the blood, which can harden and cause narrowing of the arteries.

Polyunsaturated fat: Fatty acids that contain two or more double bonds; found in nuts, seeds, and fish; usually liquid at room temperature.

Statins: A class of lipid-lowering medications that have been found to reduce cardiovascular disease risk; includes Atorvastatin, Simvastatin, and Rosuvastatin.

Thrombosis: Local coagulation or clotting of the blood in a part of the circulatory system.

Visceral fat: Adipose tissue that is found deep in the abdominal area around the internal organs.

Viscosity: The state of being thick, sticky, and semifluid in consistency.

CHAPTER 2: LITERATURE REVIEW

Effects of Coconut Oil Compared to MCT Oil on Cardiovascular Disease Risk Factors

Introduction

Cardiovascular disease (CVD) is the number one cause of death worldwide. CVD is also known as heart disease and includes cerebrovascular accident (CVA) and other cardiovascular diseases such as heart failure, arrhythmia, and heart valve problems. CVD is the number one cause of death in the United States (U.S.), claiming nearly 787,000 lives in 2011 according to The Heart Foundation (Heart Foundation, 2017). Furthermore, the American Heart Association statistics for 2017 state that CVD accounts for nearly 801,000 deaths in the US, or about one of every three deaths in the US. Figure 1 shows deaths in the US in 2014 by percentage and type of CVD. (AHA 2017)

Globally CVD is the leading cause of death, responsible for 17.3 million deaths annually worldwide, and is expected to increase to more than 23.6 million deaths by the year 2030 according to the American Heart Association (AHA, 2017). CVD claims more lives each year than all forms of cancer and chronic lower respiratory disease combined. (Figure 2. AHA, 2017)

Figure 1. *Percentage breakdown of deaths attributable to cardiovascular disease (United States: 2014). Coronary heart disease includes International Classification of Diseases, 10th Revision (ICD-10) codes I20 to I25; stroke, I60 to I69; heart failure, I50; high blood pressure, I10 to I15; diseases of the arteries, I70 to I78; and other, all remaining ICD-I0 I categories.*
**Not a true underlying cause. With any-mention deaths, heart failure accounts for 36% of cardiovascular disease deaths.*
Source: National Heart, Lung, and Blood Institute from National Center for Health Statistics reports and data sets.

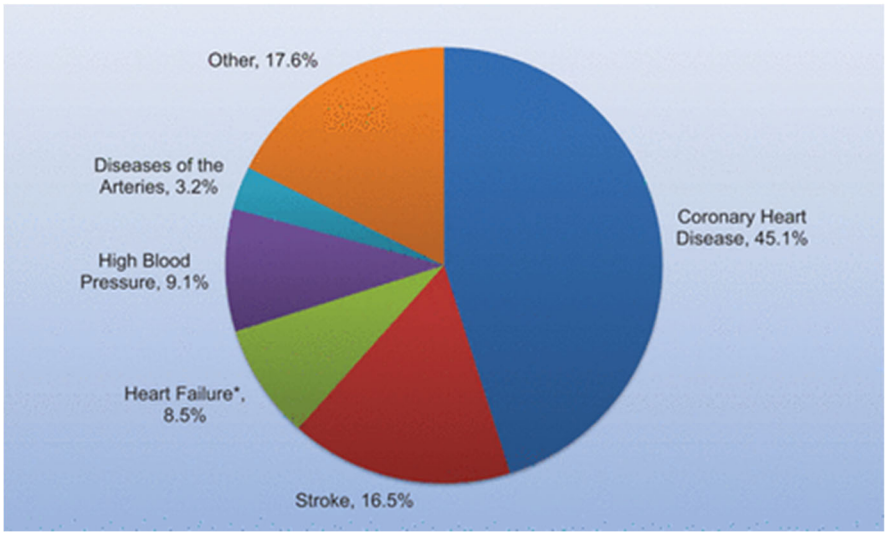
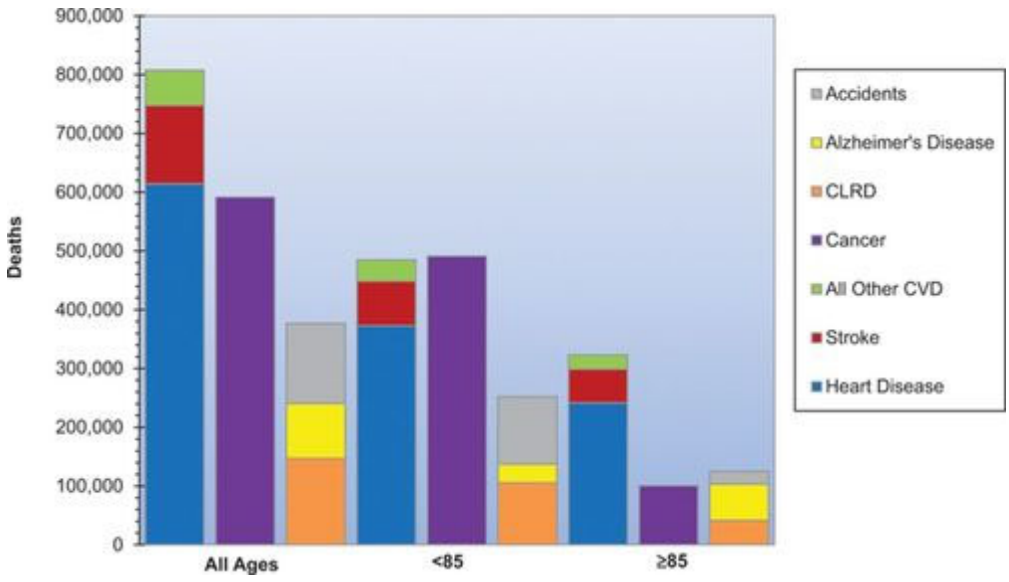


Figure 2. *Cardiovascular disease (CVD) and other major causes of death: total, <85 years of age, and ≥85 years of age.*
Deaths among both sexes, United States, 2014. Heart disease includes International Classification of Diseases, 10th Revision codes I00 to I09, I11, I13, and I20 to I51; stroke, I60 to I69; all other CVD, I10, I12, I15, and I70 to I99; cancer, C00 to C97; chronic lower respiratory disease (CLRD), J40 to J47; Alzheimer disease, G30; and accidents, V01 to X59 and Y85 and Y86.
Source: National Center for Health Statistics and National Heart, Lung, and Blood Institute.



There are many risk factors for CVD, including lifestyle, behavioral aspects, and genetic components that affect mortality and morbidity. Of these, nutrition plays a large part in the prevention and treatment associated with reducing risk factors and improving health outcomes. Research has shown that diets high in total fat, saturated fatty acid (SFA), and monounsaturated fatty acid (MUFA) intake are strong predictors of coronary heart disease (CHD) mortality (Babu et al., 2014). In particular, associations have been found between SFA intake and CVD. This information has resulted in nutrition recommendations designed to reduce cardiovascular risk by modifying dietary intake of fats and increasing intake of non-hydrogenated unsaturated fats, whole grains, fruits, vegetables, and omega-3 polyunsaturated fatty acids (ω-3 PUFAs). Studies have found these recommendations to be protective against CVD (Babu et al., 2014).

Coconut oil has been a controversial dietary fat due to its high SFA content. However, coconut oil is also rich in the medium chain fatty acids (MCFA), lauric acid and myristic acid, which may have numerous health benefits according to Babu et al., 2014. These MCFA are absorbed rapidly and taken up by the liver, where they are used as a source of energy production (Freeman et al., 2017). Lauric acid and myristic acid may increase total cholesterol as well as LDL-C, but they also raise HDL-C more than other fatty acids do. Freeman et al. (2017), state that there is little evidence supporting the reduction of CVD risk affiliated with the addition of virgin coconut oil (VCO) and available evidence is of low value. Evidence with regard to VCO and its cardiovascular benefits is at a very early stage and limited to animal studies and a few human trials (Babu et al., 2014). Ecological data reveals a low incidence of CVD in populations that currently consume coconut oil as a major part of their usual diet; however, there have not been any prospective studies that analyze coconut oil intake and effects on CVD outcomes (Freeman et al., 2017). There is some information from studies showing that replacing coconut oil with unsaturated fats resulted in a favorable impact on CVD risk by lowering total cholesterol and LDL-C. However, there is limited information on the use of coconut oil and VCO for cardiovascular benefits (Babu et al., 2014). The purpose of this literature review is to critically analyze the evidence on dietary intake of coconut oil and associated resulting cardiovascular risk factors.

Background

Cardiovascular Disease

Cardiovascular disease (CVD) is a group of diseases or conditions relating to the heart and its vessels. CVD is also known as heart disease or coronary artery disease (CAD) and includes myocardial infarction (MI) also known as heart attack or cardiac arrest, CVA or stroke, heart failure, angina (chest pain), peripheral artery or peripheral vascular disease (PAD, PVD), cardiomyopathy, and carditis among others. The pathology of the disease is related to atherosclerosis, which is a condition also known as hardening of the arteries, and is caused by the buildup of fatty plaques in the blood vessels. Plaques are sticky particles that may initially be deposited in response to an injury or damage to the lining of an artery caused by smoking, diabetes, HTN, or in response to inflammation caused by bacterial or viral infection. These plaques can eventually thicken and harden, causing a narrowing of the vessels, which restricts blood flow and can lead to a heart attack or stroke (American Heart Association). Figures 3-6 show the progression of atherosclerosis, starting with a normal healthy artery and ending with total occlusion of the blood vessel. MI happens when a blood clot forms in a blood vessel leading to the heart. The heart has two major arteries that transport oxygenated blood to it and if one becomes clogged, the supply of oxygen is cut off, leading to damage of the muscle and/or death of tissue. Cerebral vascular accident (CVA) or stroke happens when a blood vessel in the brain is blocked. This is also known as brain attack because it is similar to heart attack except the blockage occurs in the brain, cutting off the flow of oxygen to the brain

cells. This deprivation of oxygen causes damage to the brain cells and can lead to loss of memory and muscle control.

Figure 3. *(AHA 2014)*

https://watchlearnlive.heart.org/CVML_Player.php?moduleSelect=athero

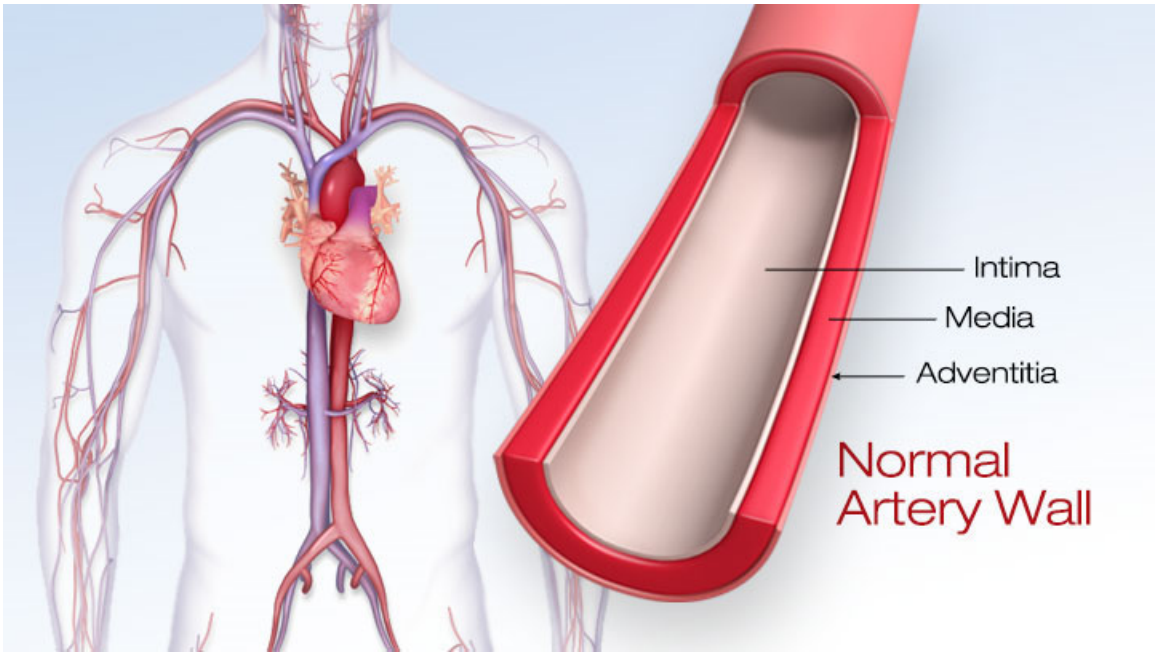


Figure 4. *(AHA 2014)*

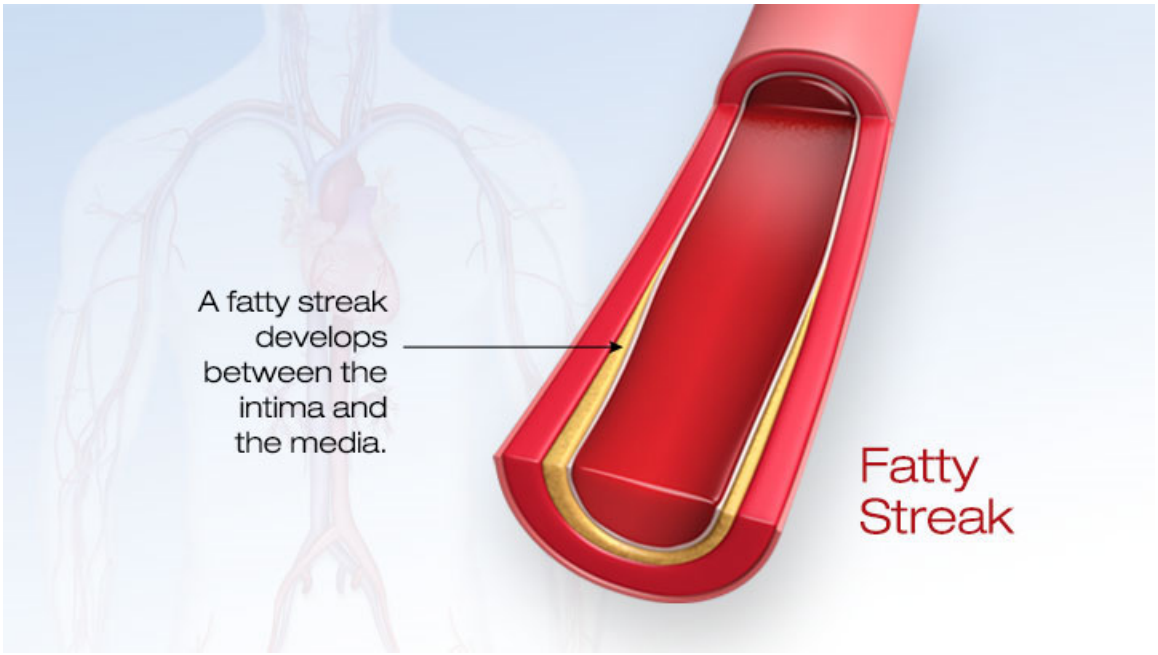


Figure 5. *(AHA 2014)*

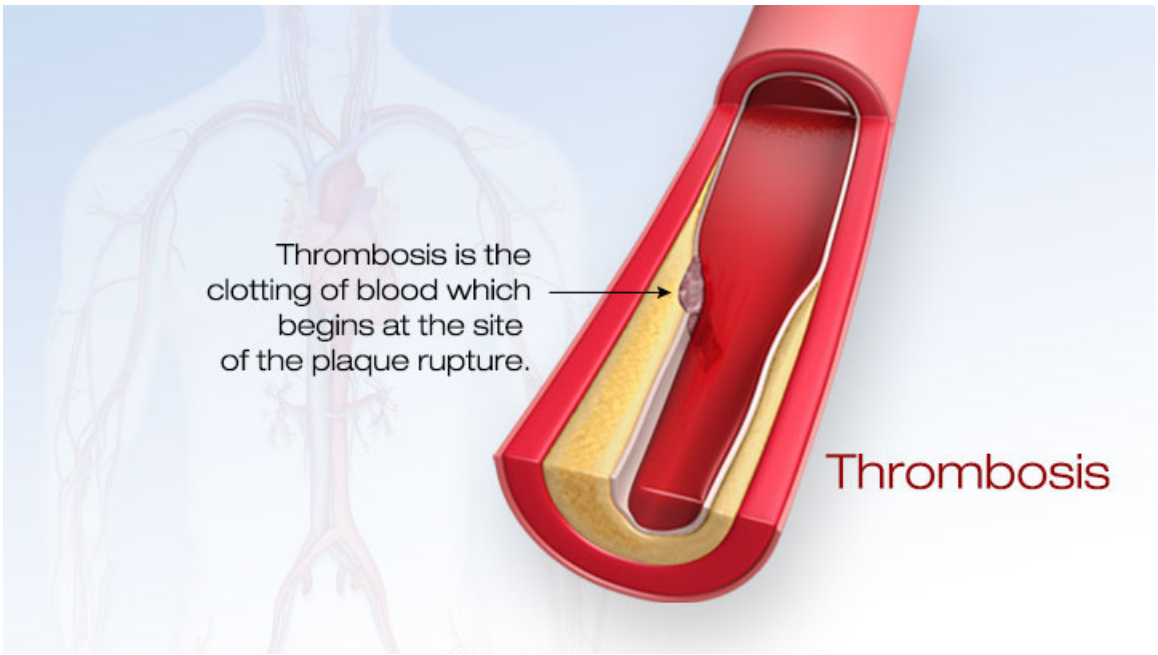
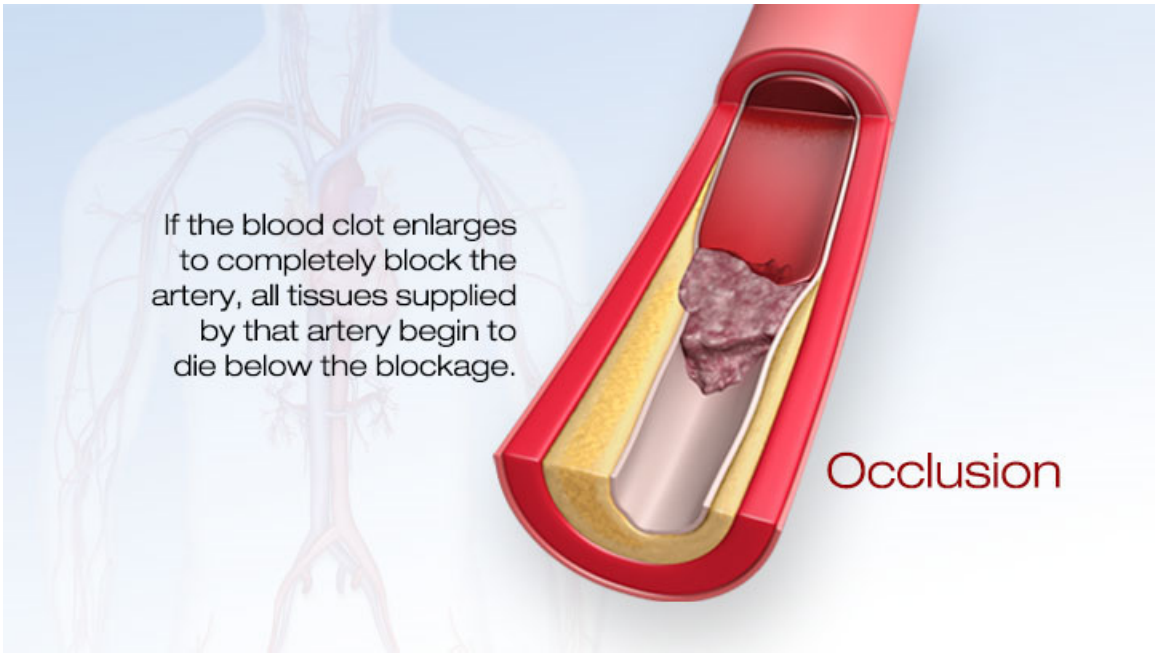


Figure 6. *(AHA 2014)*

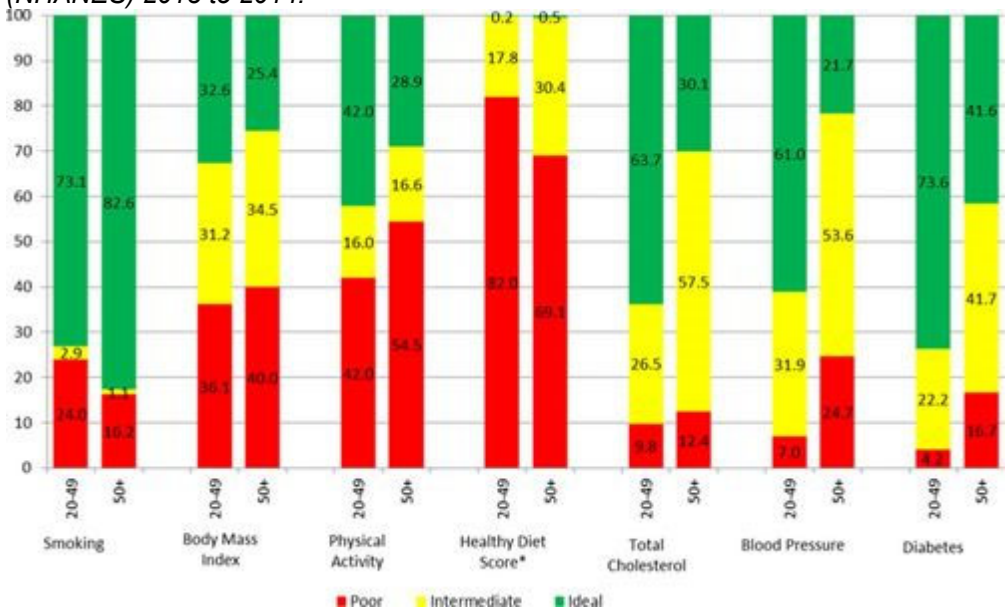


Risk Factors

Risk factors for CVD include modifiable as well as non-modifiable conditions. Non-modifiable risk factors include age, gender, inherited traits or genetic predisposition and ethnicity. Men are at higher risk for developing CVD and risk increases with age. People with certain genetic defects or who have a family history of CVD are at increased risk for CVD. Some ethnic groups are at higher risk, including those of African, Hispanic/Latino, Native American, Hawaiian, and Asian descent. Modifiable risk factors include smoking, diet, physical activity, high blood pressure, also known as hypertension (HTN), alcohol consumption, obesity, diabetes, and drug abuse. A diet high in total fat intake, particularly from saturated and trans fats increase the risk for developing CVD. It is estimated that up to 90% of CVD can be prevented through lifestyle modification

(McGill, et al). Figure 7 shows prevalence of seven of the modifiable risk factors for CVD in adults (AHA 2014).

Figure 7. *Prevalence (unadjusted) estimates of poor, intermediate, and ideal cardiovascular health for each of the seven metrics of cardiovascular health in the American Heart Association 2020 goals, among US adults aged 20 to 49 and ≥50 years.*
**Healthy Diet Score reflects 2011 to 2012 NHANES.*
Source: National Center for Health Statistics, National Health and Nutrition Examination Survey (NHANES) 2013 to 2014.



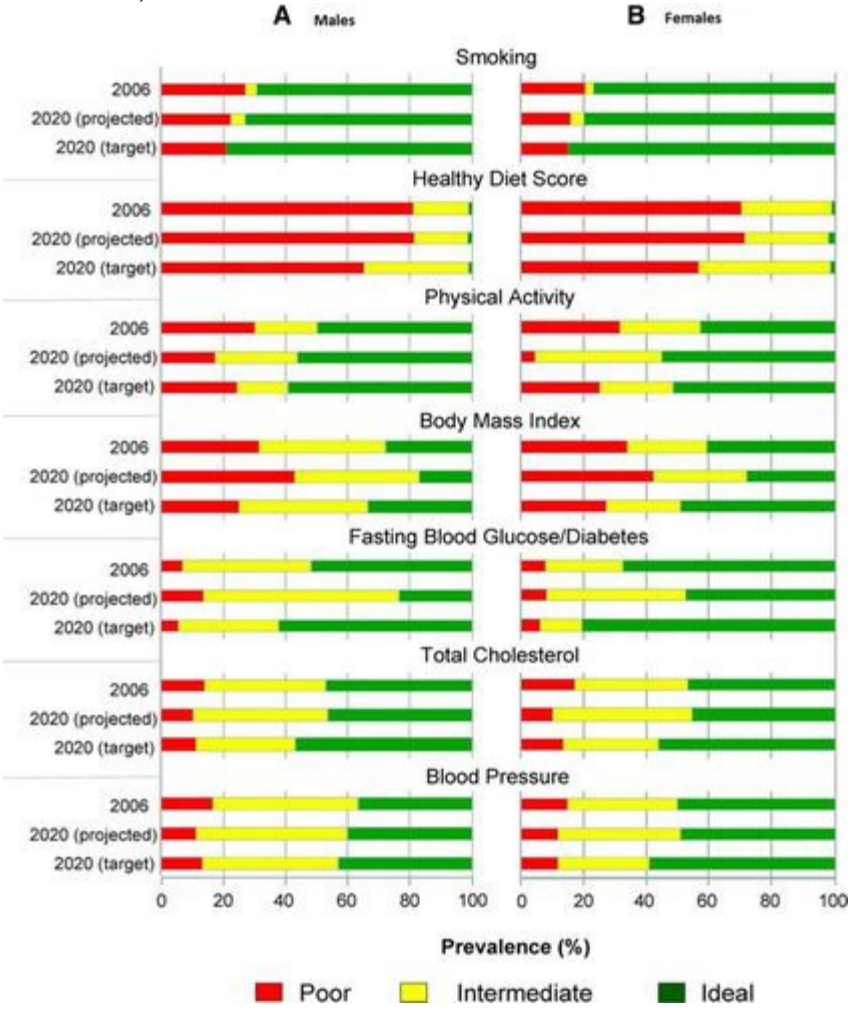
Treatment/Intervention

Traditional treatments for CVD include surgical, pharmacological, as well as lifestyle change interventions. Angioplasty, also known as percutaneous coronary intervention (PCI), is a common non-surgical technique used to open a clogged artery by placing a stent in the vessel. Coronary Artery Bypass Graft (CABG) is a surgical procedure that uses blood vessels taken from other parts of the body to go around, or bypass blocked arteries near the heart. This is performed after an MI, or before to prevent

one from happening. Cardiac rehab is often recommended after CABG, MI, and other CVD (NIH, 2014). This is a medically supervised program for survivors that uses a combination of educational strategies to teach exercise and other lifestyle interventions to improve health outcomes in CVD.

Recommended lifestyle changes include increasing physical activity, adopting a heart healthy diet, quitting smoking, reducing stress, lowering blood pressure, losing weight and/or avoiding obesity (NIH, 2014). Figure 8 shows prevalence of CVD risk factors in 2006 along with projections and targets for 2020. Of these, nutrition is of the most pertinent to this review. Traditionally, heart healthy diet recommendations include low fat, low sugar, low salt, low saturated and Trans fats, and increased fiber, fruits, vegetables, and whole grains.

Figure 8. *Prevalence of ideal, intermediate, and poor cardiovascular health metrics in 2006 (American Heart Association 2020 Impact Goals baseline year) and 2020 projections assuming current trends continue. The 2020 targets for each cardiovascular health metric assume a 20% relative increase in ideal cardiovascular health prevalence metrics and a 20% relative decrease in poor cardiovascular health prevalence metrics for males and females. American Heart Association, Inc. 2012*



Prevention

CVD includes HTN, coronary heart disease (CHD), heart failure (HF), congenital heart defects, and stroke. Common risk factors are often undertreated or under-controlled. Escott-Stump (2015) estimates that 70% of CVD cases can be prevented or delayed through changes in dietary choices or lifestyle modifications. Escott-Stump identifies 12 modifiable dietary/lifestyle and metabolic risk factors. These include high blood pressure,

high blood glucose levels, high LDL cholesterol levels, overweight/obesity, high intake of dietary trans-fatty acids and sodium, and low dietary intake of PUFA, omega-3 fatty acids, fruits/vegetables, physical inactivity, and alcohol use and smoking. Primary prevention is especially beneficial for people who have several of these risk factors. People at higher risk for CVD are encouraged to make lifestyle changes. Therapeutic lifestyle changes (TLC) is one method that is commonly used to counsel patients to make improvements in their cardiovascular health. Some may be able to reduce risk in as little as three months without the use of medications. The goals of TLC include physical and nutritional improvements; increased physical activity is encouraged and nutrition counseling is recommended. The behavior change model is often used as an appropriate intervention in counseling patients to help them achieve improvements in their health and reduce CVD risk.

Secondary prevention is beneficial for high-risk and low-risk patients. The Adult treatment panel (ATP III) report provides scientific evidence for dyslipidemia management. Dyslipidemia is an abnormal or elevated level of lipids in the blood. While small dense LDL particles are atherogenic, dietary cholesterol is only one of several factors that have a role in the etiology of CVD. Cholesterol is synthesized in all animal tissues and has many purposes in the body. Studies show links between the intake of fruits, vegetables, and whole grains providing protection against CVD due in part to the fiber, vitamins, minerals, and phytonutrient content. Adequate intake of folate, vitamin B6, B12, E, and C, as well as flavonoids, phytoestrogens, and a generally healthful dietary pattern may be protective against CVD.

Lipids

The term lipid refers to a variety of fats and oils and includes triglycerides, phospholipids and sterols from plants. Lipids have a variety of structures and functions and are essential in the body for optimal health. In addition to supplying energy, fats provide insulation and protection to organs and are necessary for absorption and transportation of some nutrients. All lipids are made up of carbon, hydrogen, and oxygen atoms and are insoluble in water, but will dissolve in organic solvents such as chloroform, benzene, and ether. Their insoluble properties are what sets them apart from protein and carbohydrates. Simple lipids are fatty acids that are neutral fats or esters of fatty acids, including monoglycerides, diglycerides, and triglycerides. Triglycerides are the most common form of lipid found in foods, comprising about 95% of dietary fat intake. Fat is stored in the body primarily as triglycerides, also accounting for approximately 95% of fat stores (Wardlaw et al., 2007). The triglyceride molecule is made from a glycerol backbone with three fatty acids attached to it. Free fatty acids are chains of carbons linked together and surrounded by hydrogens. There are many kinds of fatty acids (FA), but they all have a similar structure. FA have an acid (carboxyl) group at one end and a methyl group at the other end. FA chains vary in length, number of carbons, degree of saturation with hydrogen, and shape of the chain.

FA commonly contain from four to 24 Carbons. Long chain FA are made up of 14 or more carbons. These lipids are found in beef, pork, lamb, and most plant oils. Long chain FA require the most time to digest and are transported through the body in the lymphatic system. Medium chain FA consist of 6-12 carbons and are found in coconut

and palm kernel oils (Mahan et al., 2017). Coconut oil also contains saturated fatty acids, which causes it to be semiliquid at room temperature because the fatty acids are mostly short and medium chain, 8-14 carbons, according to Mahan et al. (2017). Fatty acids found in coconut oil are caprylic acid with eight carbons, capric acid with 10 carbons, lauric acid with 12 carbons, and myristic acid with 14 carbons (Mahan et al., 2017). Medium chain FA are digested almost as rapidly as glucose and are transported via the circulatory system. Short chain FA contain fewer than eight carbons, are usually liquid at room temperature, and are found in dairy products, butter, and whole milk. Short chain FA are rapidly digested and transported via the circulatory system (Wardlaw et al., 2007).

Medium chain triglycerides (MCT) are triglycerides with fatty acid chains of 6-12 carbon atoms. MCT oil may be extracted for commercial use from food sources including palm kernel oil and coconut oil. MCT oils are an important source of energy and have been used medically for a variety of treatments. They are used in infant formulas and in parenteral nutrition, for malabsorption problems and in the treatment of epilepsy. MCTs are metabolized and absorbed differently than LCFA. They are more rapidly absorbed through passive diffusion into the portal vein and transported directly to the liver, unlike LCFA which require more energy, are transported via the lymphatic system and require bile salts for digestion. There is controversy over whether coconut oil can truly be considered an MCT. It is mainly made up of lauric acid, which is 12 carbons in length. Pure MCT oil contains fatty acid chains of 8-10 carbons and no lauric acid. Only 20-30% of lauric acid is taken directly to the liver like shorter chain fatty acids (Clegg 2017).

FA can be fully saturated, monounsaturated, or polyunsaturated. The extent of saturation depends on available bonds with the carbon atom. The carbon atom has the ability to form four bonds. In SFA, every carbon has four bonds, meaning it is saturated. These fats tend to be solid at room temperature. Monounsaturated fatty acids (MUFA) have one carbon that is not saturated with hydrogen, leaving it with one double bond. Polyunsaturated fatty acids (PUFA) have at least two or more double bonds. PUFA tend to remain liquid at room temperature. FA with double bonds are vulnerable to oxidative damage. The shape of the FA can vary. Saturated and trans fatty acids have straight carbon chains. Unsaturated cis-fatty acids have chains that are bent or kinked. In the cis-form of fatty acids, the hydrogen attaches to the double bond of carbon on the same side, forming a bend in the chain. Trans-fats are created when hydrogen attaches at the double bond on opposite sides, resulting in a straight appearance, which causes the chain to resemble that of a SFA. Most unprocessed unsaturated cis-form FA come from oils that are pressed from nuts and seeds. Trans FA can be found naturally in some foods; however most trans-fats in the diet are artificially made by food manufacturers through a process called hydrogenation. This process is when hydrogen is added to a PUFA, modifying the structure to create a more saturated fat, which is solid at room temperature and results in a more shelf-stable product (Wardlaw et al., 2007).

Humans are able to synthesize a wide variety of FA in the body, but are unable to manufacture two PUFA. These are alpha-linolenic acid (major omega-3 FA) and linoleic acid (major omega-6 FA). These fatty acids are considered essential fatty acids (EFA) because we must get these from our food and are unable to synthesize them (Wardlaw et

al., 2007). Humans store fat predominantly as saturated palmitic FA (C 16:0) and stearic FA (C 18:0) (Mahan et al., 2017). Cell membranes must be stable and flexible. Membrane phospholipids contain one SFA and one highly PUFA, the most abundant of which is arachidonic acid (C 20:4) (Mahan et al., 2017). Dietary fat is fundamental in many bodily functions. It is essential in the digestion, absorption, and transportation of fat-soluble vitamins and phytochemicals, including carotenoids and lycopenes. Fat reduces gastric secretions, slows gastric emptying time, stimulates release of biliary and pancreatic enzymes, and promotes digestion (Mahan et al., 2017).

Phospholipids are similar in structure to triglycerides, except that one fatty acid is replaced with a phosphate compound that contains phosphorous and nitrogen. This creates a unique substance that acts as an emulsifier in the body. One end of the phospholipid is hydrophobic (water fearing) and the other is hydrophilic (water loving), which allows it to function in a variety of ways without clumping together. One important function is the formation of cell membranes. Sterols are another lipid in the body that are made up of many long chains of carbons that are arranged in rings. Sterols include steroids and cholesterol. Cholesterol is used in the body to synthesize hormones, bile, cell membranes, and lipoproteins, which are important for transporting lipids through the circulatory system. Chylomicrons are a type of lipoprotein that transport fats through the blood. They are primarily composed of triglycerides and their main function is to transport dietary fats from the small intestine to the cells. Very-low-density lipoproteins (VLDL) are also made up mainly of triglycerides and carry fats from the liver to the cells. Low-density lipoproteins (LDL) have the highest composition of

cholesterol, which is synthesized in the liver as well as from other sources and is transported to the cells. High-density lipoproteins (HDL) have the highest proportion of protein, which makes it the densest. HDL helps to remove excess cholesterol from the body by transporting it to the liver to be recycled or excreted. Low levels of HDL and/or high levels of LDL in the blood are associated with increased risk for CVD.

Preliminary Evidence

The purpose of a study conducted by Deol et al. (2015) was to research the effect of different types of dietary oils as well as intake of fructose on obesity and diabetes among other comorbidities, which are risk factors for CVD. This was a randomized controlled trial using male mice at the University of California, Riverside. The mice were assigned to five different diets with 12 in each group. The high fat diet (HFD) consisted of 36% kcals from coconut oil and 4% from soybean oil. A high soybean oil diet (SO-HFD) contained 19% kcals from soybean oil and 21% kcals from coconut oil. The diets with added fructose (F-HFD and F-SO-HFD) consisted of the same fatty acid composition as HFD and SO-HFD but with added fructose equaling 25.9% of kcals. The control diet was a low fat regular vivarium (Viv) chow (Purina Test Diet 5001), provided from Newco Distributors, Rancho Cucamonga, CA. All of the HFD diets had identical composition of macronutrients and were lower in protein, higher in carbohydrate and fats than the Viv diet. The intervention lasted up to 35 weeks, at which time subjects were euthanized according to NIH guidelines. Mice were measured for glucose tolerance (GTT) and insulin sensitivity (ITT) and they were weighed weekly. Tissue samples were

taken from various organs including liver, adipose, and kidneys and were preserved by freezing for storage.

Amount of food consumed did not differ significantly between the four HFDs. No significant difference in weights were found between mice consuming the high fructose diets, regardless of soybean oil content, although those receiving SO-HFD gained more weight, at a faster rate compared to the HFD group. The addition of fructose to the diet increased weight compared to HFD alone, though not as much as SO-HFD. Mice receiving the SO-HFD gained slightly more weight than mice fed the F-SO-HFD gained, although this was significantly different only between weeks 8 and 16. Significant differences were found in many parameters, including the amount of mesenteric and subcutaneous white adipose tissue (WAT), which was found to be significantly greater in SO-HFD than any other HFD mice. The GTT and ITT were examined to identify risk factors for diabetes. After 20 weeks, F-HFD did not show any increase in risk for diabetes, however the SO-HFD did. The mice on this diet showed extreme intolerance to glucose compared to F-HFD, which was only slightly less tolerant than Viv diet. The mice on the HFD did not develop diabetes or glucose intolerance after 20 weeks. The results of this study are important because of the fact that obesity and diabetes are risk factors for CVD. RCTs are difficult to conduct in human populations because of differences in many variables. That is why this study was of value because the research was conducted using laboratory animals under a tightly controlled situation to compare the effects of saturated fat from coconut oil and unsaturated fat from soybean oil in addition to fructose on risk factors for CVD. The authors concluded that soybean oil

contributed to obesity and diabetes more than coconut oil did (Deol et al., 2015). Strengths of this study include the study design, which was a randomized controlled trial conducted in a well-controlled clinical laboratory setting. Weaknesses include the difficulty of interpreting results and how they may apply to humans due to the use of laboratory animals.

Primary Prevention-Observational Studies

In a cross-sectional study conducted in the Samoan Islands in 2002-2003 and published in The American Journal of Clinical Nutrition by Dibello et al. (2009), researchers observed different dietary patterns and resulting effect on prevalence of metabolic syndrome. They state that metabolic syndrome is related to CVD risk factors and has reached epidemic proportions in Samoa and American Samoa. According to the NIH, metabolic syndrome is defined as having at least three of these five characteristics: abdominal obesity, high triglyceride level, low HDL cholesterol level, hypertension, and/or impaired fasting blood glucose. Study participants were recruited from those that had participated in a previous cross-sectional genetic study, the Samoan Family Study of Overweight and Diabetes in 2002–03. Subjects from American Samoa were randomly selected from individuals who had participated in a 1990–94 cohort study in American Samoa. Subjects were not chosen based on obesity or related co-morbidities. Adults age 18 and over were included and one was excluded from American Samoa due to missing a majority of information on the FFQ. The FFQ had to be altered to include commonly consumed food items in Samoa that had previously been omitted, resulting in excluding

106 subjects. This left a final sample size of 723 subjects in American Samoa and 785 participants in Samoa.

Anthropometric measurements including height, weight, abdominal/waist circumference (WC) and blood pressure were obtained. Fasting blood samples were collected and analyzed for total cholesterol (TC), triglycerides, and HDL. FFQ were administered by trained workers in participants' homes along with health history, and information regarding socioeconomic status (SES), and physical activity. Information pertaining to SES and health history included questions about smoking, diabetes and hypertension, and any current treatments for these conditions.

Samoan women were significantly more likely to meet the criteria for metabolic syndrome than Samoan men were ($P= 0.0006$). Prevalence of metabolic syndrome was higher in American Samoa (49.4%) than in Samoa (30.6%). Those with metabolic syndrome were significantly older ($P<0.001$); had a higher SES index ($P<0.03$), higher BMI ($P<0.001$), and larger WC ($P<0.001$); and had lower levels of physical activity than those without metabolic syndrome ($P=0.0004$). Intakes of saturated fat and fiber were not significantly different between the groups.

In American Samoa, the neo-traditional dietary pattern consisted of high intakes of fish, crab and lobster, coconut milk and coconut cream dishes, papaya soup, taro, yam, and papaya, and low intakes of sausage, egg, butter and margarine, rice, instant noodle soup, bread, potato chips, and Coca Cola. The neo-traditional pattern in Samoa was similar, with high intakes of crab and lobster, coconut cream and coconut cream dishes, papaya soup, and ripe coconut and low intakes of sausage, egg, cheese, rice, instant

noodle soup, soup with vegetables, chop suey, pancakes, cake, and potato chips. The modern dietary pattern in American Samoa was associated with high intakes of sausage, egg, milk, cheese, butter and margarine, coconut cream, rice, instant noodle soup, bread, pancakes, cereal, cake, and potato chips and low intakes of fish, crab and lobster, papaya soup, and bread fruit. In Samoa, the modern dietary pattern was characterized by high intakes of sausage, egg, ripe coconut, papaya, rice and rice dishes, instant noodle soup, soup with vegetables, chop suey, pancakes, cereal, cake, potato chips, and crackers, and low intakes of coconut milk and coconut cream dishes and taro.

There were associations between dietary patterns and prevalence of metabolic syndrome, though not always significant. The neo-traditional dietary pattern was associated with a significant increase in serum concentrations of HDL cholesterol in American Samoa ($P<0.02$) and a significant decrease in abdominal circumference in both groups ($P<0.03$). The modern dietary pattern showed an association with an increase in serum triglyceride levels in both American Samoa ($P=0.04$) and in Samoa, though not significant ($P=0.06$).

The researchers identified two main dietary patterns among these groups in Samoa and American Samoa. The neo-traditional pattern consisted primarily of high intakes of seafood and coconut products and low intakes of processed foods. This pattern showed a trend toward decreased prevalence of metabolic syndrome across both groups although this relationship did not reach statistical significance. In American Samoa and Samoa, the neo-traditional dietary pattern was significantly associated with decreased abdominal circumference, whereas in residents of American Samoa, it was associated

with increased serum concentrations of HDL cholesterol. The neo-traditional diet was also positively associated with high fiber and saturated fat intake. Interestingly, this pattern did not increase the risk of metabolic syndrome in this population, although increased consumption of saturated fat has been associated with both an increase in insulin resistance and development of CVD. The majority of saturated fat intake associated with the neo-traditional pattern is derived from coconut cream and coconut intake. Lauric acid found in coconut oil has been linked to increased levels of HDL cholesterol. This association may help explain the lack of association between high intake of coconut products and heart disease, as well as the positive association between the neo-traditional pattern and increased levels of HDL cholesterol seen in this study. Furthermore, the consumption of coconut products is associated with an overall dietary pattern high in fiber and seafood intakes and relatively low in meat products; the combined effects of the dietary exposures in this pattern may be protective for the occurrence of metabolic syndrome. The modern dietary pattern primarily characterized by high intake of sausage and eggs and processed foods rich in refined grains such as rice, potato chips, instant noodle soup, and pancakes was associated with increased prevalence of metabolic syndrome in both Samoa and American Samoa ($P = 0.05$ and 0.08, respectively). The modern pattern was significantly associated with increased levels of serum triglycerides.

Strengths of this study include the sample size, which was large enough to be representative of the populations of Samoa and American Samoa. The method of identifying dietary patterns and associations with disease risk was a strength of this study.

Partial least squares regression (PLS) was effective in identifying dietary patterns associated with disease risk. Limitations of this study include the study design, which was cross-sectional and therefore not showing causation. There may be confounding factors that could be better accounted for in a randomized controlled trial or prospective cohort study. There may also be some measurement errors of the individual components of the syndrome.

The results of this study indicate the potential protective effect of the neo-traditional diet (which includes high intake of coconut products), on the development of metabolic syndrome, which is a component of CVD risk, in American Samoa and Samoa. The modern dietary pattern was associated with increased risk of metabolic syndrome (Dibello et al., 2009).

Similarly, to the study by Dibello et al., a case-control study conducted in Indonesia, the authors Lipoeto et al. (2004) also investigated the difference in food patterns and risk factors for coronary heart disease (CHD) between the case subjects and the control subjects. The CHD cases were selected from the outpatient cardiovascular departments of five local hospitals. The control subjects consisted of healthy individuals randomly chosen from the same hospitals, similar to the cases according to gender and age. Study participants answered questionnaires pertaining to their general health and lifestyle status as well as a food frequency for intakes over the past year. Nutrient intakes were determined for macronutrients as well as for different types of lipids-monounsaturated, polyunsaturated and cholesterol. Subjects were divided into quartiles according to their intakes.

There were significantly higher intakes in g/day of meats and meat products (47.1 ± 40.2, P<0.01), eggs (37.7 ± 31.7, P<0.01), sugar (34.6 ± 28.8, P<0.05), and fruits (129.9 ± 73.7, P<0.01), among the case subjects; and significantly lower intakes of rice and cereals (382.3 ± 108.9, P<0.001), compared to the controls. Total animal foods consumption, which included fish, eggs, beef, chicken, and dairy foods, was significantly higher (247 g/d vs 187 g/d, P<0.0001) in the case group compared to the controls. No difference was found between the two groups for plant food intake, which includes coconut milk and grated coconut. The case group had significantly higher intakes of protein (92.0 g ± 33.5, P<0.01) and dietary cholesterol (296 mg ± 205, P<0.0001) but lower intake of carbohydrate (204.1 g ± 55.9, P<0.001) compared to controls. No significant differences were found for saturated or unsaturated fatty acid intake between the two groups, with the exception of arachidonic acid (0.06 ± 0.03, P<0.0001). The case group had a significantly higher intake of this fatty acid due to the higher intake of animal protein sources. No relationship was found for CHD risk and total dietary fat intake between the case and controls. In some studies, saturated fat intake has been linked to increased risk for CHD and coconut oil was suspected to contribute to CHD risk. However, this study did not find a relationship. In this study, subjects in both groups had a similar intake of SFA, about 27 g/day, which was estimated as the equivalent of 129 g of coconut milk or 31.5 g of coconut oil, which is commonly used for cooking in this population. The researchers concluded that the results did not indicate an association between total SFA intakes, and in particular SFA from coconut oil and other coconut products, and increased risk for CHD, at least in this population. Weaknesses of this study include the fact that this was a case-control, not a RCT, so no cause and effect can

be inferred. In addition, the use of questionnaires and FFQ may not be a reliable source of accurate information. A strength of this study was that the researchers were careful to match the control subjects with the case subjects according to age, gender, and total fat intake. This study indicates that more research is needed to determine associations between individual SFAs and CHD risk (Lipoeto et al., 2004).

Secondary Prevention of Cardiovascular Events

A non-randomized clinical trial published by Cardoso et al. (2015) examined the effect of a diet rich in coconut oil and its impact on cardiovascular risk factors. The study recruited subjects from an outpatient center of a cardiology hospital in Rio de Janeiro, Brazil. Eligibility criteria included hospital patients who had previously suffered a cardiovascular event, but had been stable for at least six months prior to intervention, and currently taking lipid-lowering medications. The first three months was a phase to standardize dietary intakes. The prescribed diet was designed according to a standard diet recommended for patients with dyslipidemia, the Dietary Reference Intakes (2005), and National Cholesterol Education Program - Adult Treatment Panel III (NCEP ATPIII) (2002). Compliance to the diets was assessed through 24-hour recalls at each visit that were compared to baseline recalls. For the second phase, subjects were assigned to one of two groups, the first remained on the standard diet (DG) and the second received extra virgin coconut oil (CODG) in addition to the standard diet. Demographics were obtained along with past medical histories; physical activity level and prescribed drug therapy were tracked with monthly visits to the hospital. At each visit, anthropometrics were

obtained along with collection of blood samples and blood pressure. All subjects received nutritional advice including the standard diet that they were instructed to adhere to. Blood samples were collected and analyzed for total cholesterol, triglycerides, HDL-C, LDL-C, ApoA-1, ApoB, fasting plasma glucose, and HgA1c. Baseline and monthly 24-hour diet recalls were administered to assess for compliance among subjects. Along with the standard diet treatment, the CODG group was given containers of extra virgin coconut oil. They were instructed to consume one packet per day, each containing 13mL of coconut oil, for the three-month intervention period.

There were no significant differences in demographic characteristics between groups at the beginning of the trial period. After the three month intervention period, significant decreases were observed for weight (-0.6 kg ±1.8, $p<0.01$), BMI (-0.2 ±0.7, $p<0.01$), waist circumference (WC) (-2.1 cm ±2.7, $p<0.01$), neck circumference (NC) (-0.4 cm ±0.9, $p<0.01$), and diastolic blood pressure (DBP) (-3.5 mmHg ±13.8, $p<0.01$), in the CODG group. There was also a significant difference between the groups for WC (-2.1 ± 2.7; $p < 0.01$). Subjects in the CODG group showed a significant increase in HDL-C (3.1 mg/dL ±7.4, $p<0.01$), compared to DG ($P<0.01$) as well as ApoA (4.7 mg/dL ±12.7, $p< 0.01$), and ApoB, (6.4 mg/dL ±17.6, $p<0.01$). Weaknesses of this study noted by the authors include the small sample size and the lack of randomization when assigning subjects to intervention groups. Other limitations include the use of lipid lowering medications and the exact type and amount of fat consumed in the DG was not clearly specified. Strengths include the fact that the CODG and DG groups were similar in relation to anthropometric and biochemical data at baseline. There were no significant

differences between the groups for dietary characteristics during the intervention. Both groups had a similar intake of energy in total kcals and percentage of kcals from protein, carbohydrates, lipids, SFA, monounsaturated fatty acids (MFA), and polyunsaturated fatty acids (PFA). The researchers concluded that including extra virgin coconut oil in the diet significantly increased the levels of HDL-C and reduced WC. This is important and more research is needed to investigate further treatments in prevention of CAD risk factors (Cardoso et al., 2015).

MCT Oil Compared to Olive Oil

The purpose of a study conducted by St-Onge et al. (2008) was to examine the effects of MCT oil consumption compared to olive oil, as part of a weight loss diet, on metabolic risk profile which are included as risk factors for CVD. This was a randomized controlled trial conducted during a 16-week weight loss program. Adults age 19-50 were recruited from Birmingham, Alabama through flyers placed in area newspapers. Inclusion criteria included stable weight, absence of chronic diseases, and having a normal score on a symptoms inventory questionnaire. Those that were on medications to control blood pressure, lipids or blood glucose were allowed to participate as long as they were stable and no changes were made for the duration of the study. Exclusions from the study included subjects who were using any weight loss medications, pregnant women or those planning to become pregnant, and those that had given birth within the past year or were currently breast-feeding.

Individuals were randomly assigned to two groups, one each to receive MCT oil or olive oil. Krusteaz, Seattle, WA, provided cranberry or blueberry muffins that

contained 10 gm of either olive oil or MCT oil. In addition, subjects were also expected to incorporate their assigned oil into their cooking. Women were instructed to add 8 g and men 14 g/day. Weekly counseling sessions were held to encourage weight loss. Total kcal intake was restricted to 1500 kcal/day for women and 1800 kcal/day for men. For both men and women, approximately 12% of total kcal intake was to come from the assigned oil. This was a double-blinded study in that neither the participants nor the Dietitian or Clinical Coordinator knew which oil they were consuming. Measurements included fasting blood samples and blood pressure, which were obtained at baseline, 8 and 16 weeks. Weights and waist circumference were measured weekly. Blood samples were analyzed for TC, LDL, HDL, TG, insulin, and blood glucose.

Of the 49 people who originally enrolled in this study, only 31 were able to complete it. MCT-16, olive oil-15. Those that dropped out included scheduling conflicts, complaints about the food, injury unrelated to the study, family emergency, one pregnancy, and two lost to follow-up. Overall, there was weight loss in both groups, although the MCT oil group had a higher weight loss. There was a significant effect of week ($P < 0.0001$) and a trend, though not significant, for a diet-by-week interaction on body weight ($P = 0.1043$). Weight loss was significant in the MCT group (-3.16 ± 0.49 kg; $P < 0.0001$) but not in the olive oil group (-1.41 ± 0.49 kg; $P = 0.117$). Body weight at the study endpoint was lower in the MCT group than in the olive oil group (unadjusted $P = 0.013$). There was a significant effect of week ($P = 0.0001$) and a diet-by-week interaction ($P = 0.045$) on body weight. Percentage change in body weight was analyzed, significant effect of week ($P < 0.0001$) and a diet-by-week interaction ($P = 0.0032$) in the

completers analysis. Both groups lost weight (MCT oil, $P < 0.0001$; olive oil, $P = 0.0030$); however, percentage change in body weight at week 16 was significantly greater in the MCT group than in the olive oil group ($P = 0.011$). Significant main effects of week ($P = 0.0282$) and diet ($P = 0.0428$) but no diet-by-week interaction ($P = 0.3770$) on percentage change in body weight. Significant effects were seen in weekly data on percentage total body fat ($P = 0.0037$), absolute fat mass ($P = 0.0013$), and absolute trunk fat mass ($P = 0.0036$). Trends were noted for endpoint percentage total body fat, which was lower in the MCT group than in the olive oil group (−0.88 ± 0.46%, unadjusted $P = 0.063$). Changes in fat mass were significant in the MCT oil group−2.232 ± 0.571 kg (unadjusted $P = 0.0005$), but not for olive oil. Endpoint absolute fat mass was lower in the MCT group than in the olive oil group (−1.542 ± 0.581 kg; unadjusted $P = 0.01$). Absolute trunk fat mass changed significantly in the MCT group by −1.203 ± 0.353 kg (unadjusted $P = 0.0012$) but not in the olive oil group. The difference between groups was not statistically significant ($P = 0.10$). However, endpoint trunk fat mass was lower in the MCT oil group than in the olive oil group (−0.875 ± 0.359 kg; unadjusted $P = 0.0179$). Changes in percentage trunk fat mass were not significant (olive oil: −0.49 ± 0.6%, $P = 0.42$; MCT oil: −1.23 ± 0.85%, $P = 0.17$). There was a trend toward a diet-by-week interaction on total body lean mass ($P = 0.0921$), with endpoint lean mass being lower in the MCT group than in the olive oil group (−0.929 ± 0.408 kg, unadjusted $P = 0.0267$).

Limitations of this study include high rate of dropout which resulted in small sample size (n=31). The power may have been too low; researchers set the confidence

interval at 95% with a power of only 80%; there were few men participants, and a lack of ethnic diversity. The use of olive oil is a weakness because it is not entirely comparable to MCT oil. It is composed mainly of monounsaturated fats and it is low in SFA. MCT oil may not be entirely comparable to coconut oil as well. Coconut oil is comprised of 47.7% lauric acid (C 12), 5.5% capric acid (C10), 7.6% caprylic acid (C8), and .52% caproic acid (C6) (Clegg, M. E., 2017). MCT oil does not contain any lauric acid, instead it is composed of 55% caprylic acid (C8) and 45% capric acid (C10) (St-Onge et al., 2008). This study shows that consumption of MCT oil did not have adverse effects on CVD risk factors and had a similar effect on CVD risk factors compared to olive oil. Subjects consuming MCT oil lost more weight than subjects who consumed olive oil. It is important to note that long chain SFA have been shown to raise TC and LDL compared to unsaturated fats. Results in this study suggest that consumption of saturated fats in the form of MCT may be comparable to olive oil, which has been considered protective in terms of reducing CVD risk factors (St-Onge et al., 2008).

Coconut Oil Compared to Sunflower Oil

In an article published in The Indian Heart Journal, the authors Vijayakumar et al., (2016) investigated the effect of different types of cooking oils (coconut oil and sunflower oil) on known risk factors for CVD, including lipid profile, antioxidant mechanism, and endothelial function in patients previously diagnosed with CAD. This pilot study conducted in India was a randomized single blinded clinical trial. This population traditionally uses coconut oil in cooking. The subjects were selected from the hospital outpatient center, had established CAD and were receiving standard medical

care, including statin medications. They were randomly assigned to two groups, each consisting of 100 patients. Anthropometric measurements obtained included BMI, body fat percentage, and waist to hip ratio. One group was given coconut oil, and the other group was given sunflower oil to use for cooking over a period of two years. Although the exact dosage of oils was not specified, study participants submitted 24-hour diet recalls prior to initiating the study to assess their usual intake and determine energy needs. These recalls were used to calculate the amount of oil needed to supply 15% of total calories daily. Assigned oils were then provided to the subjects as well as family members in order to ensure compliance. Diet recalls, and diaries were used periodically during the study to monitor adherence to the diet. Biomarkers that were assessed included lipid profiles, total cholesterol, HDL, LDL, and VLDL, triglycerides, and lipoproteins, as well as Glycosylated hemoglobin, and the inflammatory marker C-reactive protein.

No statistically significant differences were found in any of the measurements of anthropometrics, biochemical criteria, vascular function, or cardiovascular events after 2 years between the coconut and sunflower oil groups. Lipids profiles were assessed at baseline, 3 months, 12 months, and 24 months. Adherence to the dietary interventions were assessed using 24-hour recalls, 7-day recalls and diet diaries. The authors concluded that cooking with coconut oil for the intervention period of two years did not alter any of the cardiovascular risk factors compared with sunflower oil. Limitations identified by the authors in the study included small sample in relation to total population, the use of medications, and duration of the study may not be enough to assess long-term outcomes.

Funding was provided in part by the Coconut Development Board of India, although they had no role in study design or data analysis (Vijayakumar et al., 2016).

Summary and Conclusions

CVD is prevalent throughout the world and incidence is predicted to be increasing. Traditional lifestyle modification recommendations to improve or reduce risk include reducing intake of SFA in the diet. There has been much controversy over the use of coconut oil due to its SFA content. Results of these studies did not show any increased risk for CVD associated with coconut oil or MCT oil consumption. Three of the studies were conducted with patients previously diagnosed with CVD and patients in two of those studies were on prescribed lipid-lowering medications. The same three studies also took place in countries that traditionally use coconut oil and other coconut products in their cooking and cuisine. None of the studies showed any association between coconut oil consumption and increased risk factors for CVD. The results may not be comparable to the U.S. with our traditional western, or standard American diet, however. It has been commonly believed that saturated fats contribute to CVD risk. The recommendations for consumption of coconut oil has been controversial, however as these studies suggest, the fact that coconut oil is made up of medium chain triglycerides (MCT) may indicate a cardio protective ability. However, it is important to note that coconut oil may not be comparable to MCT oil since coconut oil is made up predominantly of lauric acid which may not truly be considered a MCFA. True MCT oil is predominantly caprylic and capric acid, which are shorter chains of 8-10 carbons. There is controversy over whether lauric acid should really be considered to be an MCT or not. This is because only 20-30% of

lauric acid is transported directly to the liver via the portal vein, unlike the shorter chain fatty acids in MCT oil (Clegg, 2017). In fact, according to Clegg (2017), this means that only approximately 23.16% of coconut oil contains MCT that is absorbed and metabolized like pure MCT oil. As was shown in the animal study by Deol et al. (2015), soybean oil contributed to an increase in CVD risk factors more than the coconut oil did. This is contrary to what is usually recommended in the U.S. where the polyunsaturated fatty acids (PUFA) in soybean and other vegetable oils have long been promoted as being "heart healthy" oils for use in cooking. There was no significant difference found between coconut oil and sunflower oil on CVD risk factors in the RCT in India. Similarly, MCT oil did not increase CVD risk factors compared to olive oil.

There were some limitations noted, including sample size and duration of studies as well as the location of the sample population and subjects (human vs animal), but the overall potential for future dietetic practice is optimistic. Before coconut oil can be promoted as a beneficial fat though, more research is needed to study the effects of coconut oil in the diet of the U.S. population and how it relates to CVD risk factors including obesity and diabetes, particularly compared to MCT oil.

CHAPTER 3: METHODS

The Academy of Nutrition and Dietetics (AND) created the Evidence Analysis Manual to assist expert work groups in analyzing and understanding research. The Evidence Analysis Library (EAL) is an online resource that was started in 2004 to assist registered dietitian nutritionists in their practice by providing up to date evidence-based practice guidelines. The process consists of five steps: Step 1: Formulate the Evidence Analysis Question; Step 2: Gather and Classify the Evidence; Step 3: Critically Appraise Each Article; Step 4: Summarize the Evidence; and Step 5: Write and Grade the Conclusion Statement. These five steps are outlined here.

Step 1: Formulate the Evidence Analysis Question

The process begins by first identifying the question that will be researched. This step uses the Nutrition Care Process (NCP), which includes nutrition assessment, nutrition diagnosis, nutrition intervention, and nutrition monitoring, and evaluation. The NCP provides a systematic structure for the dietetics professional to use in assessing and meeting goals for patient care. Questions should be formulated using PICO format if possible. PICO stands for population, intervention, comparison intervention, and outcome of interest. Table 1 shows the PICO format for this question. Population or problem in this case is people with or without CVD. Intervention is the intake of coconut oil in the diet. Comparison will be to other oils or types of fatty acids including MCT oil, soybean oil, sunflower oil, etc. Outcome of interest will be effect on CVD risk factors, including waist circumference, BMI, blood pressure, and cholesterol levels. The EAL question needs to be well thought out and clear, not too narrow or vague. As stated earlier, the

question is: What is the effect of coconut oil consumption compared to MCT oil on cardiovascular risk factors in adults with or without existing CVD?

Table 1. *Evidence Analysis Question using PICO Format*

Population (Patient or Problem)	**Intervention** (cause, treatment or prognostic factor)	**Comparison** Intervention (if necessary)	**Outcomes**
Adults with and without existing CVD.	Coconut oil consumption.	Other types of lipids.	Effects on CVD risk factors.

Step 2: Gather and Classify the Evidence

Once the question has been identified, a plan must be developed to gather evidence using a detailed search of available literature. Inclusion and exclusion criteria should be specified and key terms identified. The best quality available resources will be used from Mount Mary University Library databases, including PubMed, Medline, and EBSCO. Studies are classified by type of report as identified in Table 2. Search terms will include coconut oil, MCFA, MCT, cardiovascular disease, and heart disease among others. Inclusion criteria will be a variety of study designs such as non-randomized and RCT, observational case-control studies and prospective cohorts; male and female adults with or without existing but stable CVD. Exclusion criteria will be articles that are not published in a peer-reviewed journal, any that are not written in the English language, studies conducted on animals, meta-analysis, reviews, and those that are published before the year 2000. After conducting the search and reviewing articles, results will be

documented including a list of included and excluded articles along with the reason for exclusion.

Table 2. *Hierarchy and Classification of Studies (Academy of Nutrition and Dietetics Evidence Analysis Library, 2012)*

Primary Reports		**Secondary Reports**	
A	Randomized Controlled Trial Cluster Randomized Trial Randomized Crossover Trial	**M**	Meta-analysis or Systematic review Decision analysis Cost-benefit analysis Cost-effectiveness study
B	Prospective Cohort Study Retrospective Cohort Study		
C	Non-Randomized Controlled Trial Non-Randomized Crossover Trial Case-Control Study Time Series Study Diagnostic, Validity or Reliability Study	**R**	Narrative review (Review article) Consensus statement Consensus report
D	Non-Controlled Trial Case Study or Case Series Other Descriptive Study Cross-Sectional Study Trend Study Before-After Study	**X**	Medical opinion

Example of Search Plan and Results

Question: What is the effect of coconut oil consumption on cardiovascular risk factors in adults with or without existing CVD?

Date of Literature Review for the Evidence Analysis: September 2018

Inclusion Criteria:

- Age: Adults (20 years and older)

- Setting: Outpatient and ambulatory care
- Health Status: Healthy or stable CVD
- Nutrition Related Problem/Condition: Healthy adults without co-morbid conditions or with the following co-morbid conditions: overweight, obesity, diabetes mellitus (types 1 & 2), hyperlipidemia, hypertension
- Study Design Preference: 1) RCT or Clinical Controlled Studies, 2) Large randomized observational studies, 3) Cohort
- Size of Study Groups: The sample size will be greater than or equal to 10 individuals
- Study Drop Out Rate: <20%
- Year Range: 2000-2018
- Language: English

Exclusion Criteria:

- Age: Young adults less than 20 years of age, infants, children, and adolescents
- Setting: Inpatient or acute care
- Health Status: Patients with poor prognosis
- Nutrition Related Problem/Condition: Critical illness and other diseases and conditions
- Study Design Preference:
- Size of Study Groups: <10 individuals for each study group. For example, this would include 10 patients in the intervention group and 10 patients in the control group.

- Study Drop Out Rate: >20%
- Year Range: Prior to 2000
- Authorship: Studies by same author similar in content
- Language: Articles not published in English

Search Terms: Search Vocabulary

Health Condition: Cardiovascular disease, heart disease, coronary artery disease

Intervention: Coconut oil, MCFA, MCT, olive oil, other types of oils

Type of Study Design: RCT, Clinical Studies, Observational Studies, Cohort and Case-Control Studies

Electronic Databases:

Database: PubMed/MEDLINE

Search Terms: (CVD) or (HD) or (CAD) and (coconut oil or MCFA or oils) (RCT)

Hits: 297

Articles to Review: 16

Total articles identified to review from electronic databases: 7

Inclusion List:

List of Articles Included from Electronic Databases:

Airhart, S., Cade, W. T., Jiang, H., Coggan, A. R., Racette, S. B., Korenblat, K., . . . Peterson, L. R. (2016). A Diet Rich in Medium-Chain Fatty Acids Improves Systolic Function and Alters the Lipidomic Profile in Patients with Type 2 Diabetes: A Pilot Study. *The Journal of Clinical Endocrinology & Metabolism, 101*(2), 504-512. doi:10.1210/jc.2015-3292

Cardoso, D. A., Moreira, A. B., De Oliveira, G. M., Luiz, R. R., & Rosa, G. (2015). A coconut extra virgin oil-rich diet increases HDL cholesterol and decreases waist circumference and body mass in coronary artery disease patients. *Nutricion Hospitalaria, 32*(5), 2144-2152.

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Vijayakumar, M., Vasudevan, D., Sundaram, K., Krishnan, S., Vaidyanathan, K., Nandakumar, S., . . . Mathew, N. (2016). A randomized study of coconut oil versus sunflower oil on cardiovascular risk factors in patients with stable coronary heart disease. *Indian Heart Journal, 68*(4), 498-506. doi:10.1016/j.ihj.2015.10.384

List of Excluded Articles with Reason:

Connor, W. (1988). Effects of Omega-3 Fatty Acids in Hypertriglyceridemic States. Seminars in Thrombosis and Hemostasis, 14(03), 271-284. doi:10.1055/s-2007 1002789
Reason: Year of publication, did not use coconut oil

Covas, M., Torre, R. D., & Fitó, M. (2015). Virgin olive oil: A key food for cardiovascular risk protection. *British Journal of Nutrition, 113*(S2). doi:10.1017/s0007114515000136
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Ferrara, F., Tedin, L., Pieper, R., Meyer, W., & Zentek, J. (2016). Influence of medium chain fatty acids and short-chain organic acids on jejunal morphology and intra epithelial immune cells in weaned piglets. *Journal of Animal Physiology and Animal Nutrition*, 101(3), 531-540. doi:10.1111/jpn.12490
Reason: Not human subjects

Fuchs GJ1, Farris RP, DeWier M, Hutchinson S, Strada R, Suskind RM. (1994). Effect of dietary fat on cardiovascular risk factors in infancy. *Pediatrics*, 93 (5), 756-63.
Reason: Year of publication and used infants

Gradek, W., Harris, M. T., Yahia, N., Davis, W. W., Le, N., & Brown, W. (2004). Polyunsaturated fatty acids acutely suppress antibodies to malondialdehyde modified lipoproteins in patients with vascular disease. *The American Journal of Cardiology*, 93(7), 881-885. doi:10.1016/j.amjcard.2003.12.028
Reason: Sample size too small, did not use coconut oil

Harper, C. R., & Jacobson, T. A. (2005). Usefulness of Omega-3 Fatty Acids and the Prevention of Coronary Heart Disease. *The American Journal of Cardiology*, 96(11), 1521-1529. doi:10.1016/j.amjcard.2005.07.071
Reason: Did not use coconut oil

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Reason: Did not use coconut oil

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Reason: Year of publication, sample size too small, did not use coconut oil

Rasmussen, B. M., Vessby, B., Uusitupa, M., Berglund, L., Pedersen, E., Riccardi, G., Hermansen, K. (2006). Effects of dietary saturated, monounsaturated, and n−3 fatty acids on blood pressure in healthy subjects. *The American Journal of Clinical Nutrition*, 83(2), 221-226. doi:10.1093/ajcn/83.2.221
Reason: Only looked at blood pressure

Summary of Articles Identified to Review:
Number of Primary Articles Identified: 16

Number of Review Articles Identified: 0

Total Number of Articles Included: 7

Number of Articles Reviewed but Excluded: 9

Step 3: Critically Appraise Each Article

Each article will be critically reviewed and analyzed using the EAL worksheet or Data Extraction Template (DET) to extract and record important details that will be used for the final step. Both the DET and the Quality Criteria Checklist (QCC) are used to abstract key information from articles and assess the quality of the study design. The DET is used to collect information on study design, sample, intervention/exposure/test, outcomes reported, and results. The QCC is used to assess research design through a series of yes/no questions, and to determine a rating for the article, which will be positive, neutral or negative.

Step 4: Summarize the Evidence

This step involves two parts; first, the creation of overview tables of the evidence that has been gathered so that all the research can be compared easily. Second, from this overview table, a review summary of the evidence can be written. The worksheet overview table template is a tool that helps organize information from the studies so that data can be easily viewed and compared at a glance. The evidence can then be extracted from each study into a brief summary statement. The result of this phase is called the Evidence Summary.

Step 5: Write and Grade the Conclusion Statement

The final step is to write a conclusion statement from all the evidence that has been gathered. This statement should answer the EAL question clearly. A grade that represents the strength of the evidence will then be assigned according to the EAL Grade Definition and Conclusion Grading Table (Table 3). Grades I through V are described here:

Grade I: Good--The evidence consists of results from studies with a strong design and consistent results that are free of bias and flaws in research design.

Grade II: Fair-- The evidence consists of either results from studies of strong design with inconsistencies among the results from different studies; or the evidence consists of results from weaker designs with results that have been confirmed in other studies and are consistent.

Grade III: Limited-- The evidence consists of results from a limited number of studies of weak design.

Grade IV: Expert Opinion Only-- Based on experience of the medical experts without any actual research study results.

Grade V: Not Assignable-- There is no evidence available to make a conclusion.

Table 3. *AND's Grade Definition and Conclusion Grading Table (Academy of Nutrition and Dietetics Evidence Analysis Library, 2012)*

Table 5.0 Conclusion Grading Table					
Strength of Evidence Elements	**Grades**				
	I Good/Strong	**II Fair**	**III Limited/Weak**	**IV Expert Opinion Only**	**V Grade Not Assignable**
Quality • Scientific rigor/validity • Considers design and execution	Studies of strong design for question Free from design flaws, bias and execution problems	Studies of strong design for question with minor methodological concerns, OR Only studies of weaker study design for question	Studies of weak design for answering the question OR Inconclusive findings due to design flaws, bias or execution problems	No studies available Conclusion based on usual practice, expert consensus, clinical experience, opinion, or extrapolation from basic research	No evidence that pertains to question being addressed
Consistency Of findings across studies	Findings generally consistent in direction and size of effect or degree of association, and statistical significance with minor exceptions at most	Inconsistency among results of studies with strong design, OR Consistency with minor exceptions across studies of weaker design	Unexplained inconsistency among results from different studies OR single study unconfirmed by other studies	Conclusion supported solely by statements of informed nutrition or medical commentators	NA
Quantity • Number of studies • Number of subjects in studies	One to several good quality studies Large number of subjects studied Studies with negative results have sufficiently large sample size for adequate statistical power	Several studies by independent investigators Doubts about adequacy of sample size to avoid Type I and Type II error	Limited number of studies Low number of subjects studied and/or inadequate sample size within studies	Unsubstantiated by published research studies	Relevant studies have not been done
Clinical impact • Importance of studied outcomes • Magnitude of effect	Studied outcome relates directly to the question Size of effect is clinically meaningful Significant (statistical) difference is large	Some doubt about the statistical or clinical significance of the effect	Studied outcome is an intermediate outcome or surrogate for the true outcome of interest OR Size of effect is small or lacks statistical and/or clinical significance	Objective data unavailable	Indicates area for future research
Generalizability To population of interest	Studied population, intervention and outcomes are free from serious doubts about generalizability	Minor doubts about generalizability	Serious doubts about generalizability due to narrow or different study population, intervention or outcomes studied	Generalizability limited to scope of experience	NA

CHAPTER 4: RESULTS

There is limited current research available regarding the effects of coconut oil consumption on CVD risk factors, and none comparing coconut oil to MCT oil. For this project, seven studies that took place from 2004-2018 were critically analyzed using the Data Extraction Template worksheets and Quality Criteria Checklists according to the EAL Manual. Four of the seven studies were randomized controlled trials, two were observational studies and one was a nonrandomized-controlled design. All the studies assessed effects on CVD risk factors either by comparing different types of fats or by observing usual food patterns in populations that consume coconut products. Following is a brief narrative summary of each of these studies including relevant findings. A summary of the evidence can also be found in the Overview Table in the Appendix. The conclusion statement, which answers the EAL question, is included with a grade that represents the overall strength of the evidence.

St-Onge et al., (2008) Study design: Randomized Controlled Trial; Class: A; Quality Rating: Positive (+)

The RCT conducted by St-Onge et al., 2008 found that MCT oil could be used as part of a weight loss program without adverse effects on CVD risk factors. They compared MCT oil to olive oil as part of a weight loss program. This was a small study of only 31 men and women, aged 19-50 with BMI of 27-33 who were free from chronic diseases. Subjects were randomized to receive muffins that contained either MCT oil or olive oil. Participants also received group counseling sessions for a period of 16 weeks. Both groups lost weight, but weight loss was greater in the MCT group compared to the

olive oil group. There was a significant decrease in fat mass in the MCT oil group compared to olive oil, which is one of the risk factors for CVD.

Although researchers concluded that MCT oil aided in weight loss without adverse effects on metabolic risk, this was a small sample of unequal numbers of men and women and there was a high dropout rate, indicating that larger studies need to be done in the future. It is important to note that coconut oil, which was not used in this study, is not the same as pure MCT oil since the first is largely made up of fatty acid chains of 12 carbons in length and the latter consists of 8-10 carbon chains.

Lipoeto et al., (2004) Study design: Case Control Study; Class: C; Quality Rating: Neutral (⦸)

Lipoeto et al., 2004 found that there was no association between SFA intake from coconut oil and increased risk for CVD. The authors of this case control study examined the difference in food patterns and CVD risk among the coconut consuming people in West Sumatra, Indonesia. The cases were outpatients previously diagnosed with CVD selected from local hospitals and matched with controls who were disease free. This was a larger study of a total of 282 subjects who were randomly selected to match the cases according to gender and age. This observational study used food frequency questionnaires among other tools to assess usual intakes and determine dietary patterns of participants. Results showed no association between SFA intake from coconut oil and increased risk for CVD. Table 4 shows the odds ratio and 95% CI of CVD events by food and nutrient intake. However, since this was not an RCT, no causation can be implied. Since this was a case control study, the likelihood of bias is common. As a result of being

diagnosed with the disease, the cases may have made changes to their diet. There may be recall bias, due to trouble remembering past experiences. There is a potential for selection bias especially when subjects are recruited from hospitals, since they may not be representative of the general population.

Table 4. *Odds Ratio (95% confidence interval) of coronary events by food and nutrient variables (Lipoeto et al., 2004)*

	Total	Men	Women
Total Carbohydrate	0.7	NA	0.98 **
(highest vs lowest quartile)	*(0.36 – 1.47)*		*(0.97 – 0.99)*
Animal food intake	4.8 ****	5.6 ***	4.7 *
(highest vs lowest quartile)	*(2.25 – 10.30)*	*(1.99 – 16.89)*	*(1.28 – 16.98)*
Physical activity	0.4 **	0.3 *	NA
(highest vs lowest quartile)	*(0.2 – 0.8)*	*(0.1 – 0.7)*	
Stress level	2.9 **	2.8 *	3.6 **
(highest vs lowest quartile)	*(1.6–6.5)*	*(1.2 – 6.3)*	*(1.3 – 10.3)*
Smoke	NA	NA	0.2 **
(highest vs lowest quartile)			*(0.04 – 0.7)*

Variables entered into model include the intakes of animal food, carbohydrate, protein, cholesterol, saturated fat, physical activity, stress level and smoking status; NA: data not available. The variable was removed from the model (the significance level did not reach 0.15).
*Significantly different from the odds ratio 1.0: *, P<0.05; **, P<0.01; ***, P<0.001; ****, P<0.0001.*

Dibello et al., (2009) Study design: Cross-sectional; Class: D; Quality Rating: Positive (+)

Dibello et al., 2009 observed dietary patterns in Samoa and American Samoa to determine a relationship between food patterns and CVD risk. A neo-traditional pattern, which consists of higher intakes of coconut products, was found to have a protective effect on CVD risk compared to the modern pattern, which is more westernized. This was a large cross-sectional study of 1,508 randomly selected men and women in Samoa and

American Samoa. Dietary patterns were assessed through FFQ and interviews and a neo-traditional pattern and a more modern pattern were identified as a result. Subjects consuming the neo-traditional pattern were associated with significantly increased HDL and decreased abdominal circumference among other CVD risk factors. The authors concluded that there might be evidence for a potential protective effect on CVD risk with the consumption of the neo-traditional diet, which includes a high intake of coconut products among other factors in this population. This study was not a RCT and therefore only associations can be made.

Cardoso et al., (2015) Study design: Longitudinal Non-Randomized Clinical Trial; Class: C; Quality Rating: Positive (+)

In a longitudinal non-randomized controlled trial, Cardoso et al., 2015 evaluated the effect of consumption of extra virgin coconut oil on CVD risk factors. Subjects were men and women ages 45-85 years old who had been previously diagnosed with CAD who were prescribed lipid lowering medications as part of a standard medical care regimen. A total of 116 patients were divided into two groups, one was given 13 ml of coconut oil (CODG) to consume daily, the other continued to consume a standard diet (DG). Researchers found that coconut oil increased HDL and decreased waist circumference in patients with established CVD. There were significant decreases in weight, BMI, WC, NC and BP in the CODG and a significant increase in HDL in the CODG compared to DG. Limitations include this was a relatively small sample and the

study lacked randomization. Results are further limited by the fact that subjects were also taking statins.

Vijayakumar et al., (2016) Study design: Randomized Controlled Trial; Class: A; Quality Rating: Positive (+)

The Vijayakumar et al., 2016 study of 200 CAD patients receiving standard care, including lipid-lowering medications showed no significant differences between coconut oil and sunflower oil consumption over a 2-year period. This RCT sought to compare coconut oil to sunflower oil used for cooking among CAD patients in India. The study included men and women over the age of 18 who had stable CAD. Subjects were randomly assigned to two groups, one received coconut oil, the other sunflower oil. After two years, no statistically significant differences were found for any of the measured risk factors of CVD in either group (Table 5). Researchers concluded that coconut oil did not affect CVD risk compared to sunflower oil. The fact that this was a relatively small sample in relation to total population and subjects were using lipid-lowering medications were notable limitations of this study. Additionally, this study was partially funded by the Coconut Board of India, which may imply bias.

Table 5 *– Lipid profile. (Vijayakumar et al., 2016)*

Group I (coconut oil)			Group II (sunflower oil)		
Visits	Mean	SD	Mean	SD	P Value
Total cholesterol, mg/100 ml					
Baseline	149.81	29.92	146.79	26.55	0.45
3 Months	151.19	30.15	143.43	30.22	0.07
1 Year	144.58	30.92	139.80	33.75	0.30
2 Years	149.28	28.57	151.63	44.54	0.66
LDL, mg/100 ml					
Baseline	90.29	24.38	86.10	19.62	0.18
3 Months	89.31	24.75	84.12	22.16	0.12
1 Year	91.02	20.66	87.56	25.31	0.29
2 Years	91.04	21.82	89.62	28.91	0.70

Triglycerides, mg/100 ml					
Baseline	114.96	54.22	111.17	48.85	0.60
3 Months	111.23	37.26	108.90	39.28	0.67
1 Year	112.00	50.19	114.52	64.83	0.76
2 Years	109.32	47.06	112.20	45.15	0.66
HDL, mg/100 ml					
Baseline	40.80	9.16	40.74	9.95	0.96
3 Months	40.82	10.92	39.57	9.68	0.39
1 Year	42.41	9.48	40.10	11.10	0.11
2 Years	43.22	10.77	44.36	16.35	0.56
VLDL, mg/100 ml					
Baseline	21.82	8.00	23.27	16.76	0.44
3 Months	22.31	7.43	21.61	7.74	0.52
1 Year	21.27	9.58	22.43	15.41	0.44
2 Years	21.77	9.37	22.53	9.72	0.58
NEFA, nmol/L					
Baseline	0.44	0.32	0.45	0.28	0.97
3 Months	0.61	0.32	0.6	0.35	0.95
1 Year	0.57	0.31	0.72	0.45	0.01
2 Years	0.58	0.35	0.54	0.36	0.45

Airhart et al., (2016) Study design: Randomized Controlled Trial; Class: A; Quality Rating: Positive (+)

Airhart et al., 2016 studied patients with type 2 diabetes to assess the effects of a diet rich in medium-chain fatty acids (MCFA, 6-12 carbons long) vs long-chain fatty acids (LCFA) on CVD risk factors. This was a prospective, double-blinded RCT of ambulatory patients in the general community. Subjects were randomly assigned to receive either MCFA or LCFA as part of their diet. All meals were prepared for and provided to study participants and were similar in macronutrient distribution. Cognis Corporation provided a commercially available MCFA triglyceride Delios S. Although the specific source and fatty acid profile of the MCFA was not specified, coconut oil does consist primarily of MCFA that are 6-12 carbons long and was noted to be a common food source of MCFA. However, it is important to note that pure MCT oil consists of

fatty acid chains that are 8-10 carbons long, while coconut oil is mostly made up of fatty acid chains that are 12 carbons in length. CVD risk factors that were measured included various cardiac function tests as well as lipid profiles. The authors found that the MCFA diet did not harm and may benefit cardiac function and fasting insulin levels. Fasting lipid levels significantly decreased in the MCFA but not LCFA group (Table 6). This was a very small sample, consisting of only 16 subjects and the intervention period lasted only two weeks. Larger and longer-term studies are needed to further investigate the effects of MCFA on CVD risk.

Table 6. *Body Composition and Metabolic Responses (Airhart et al., 2016)*

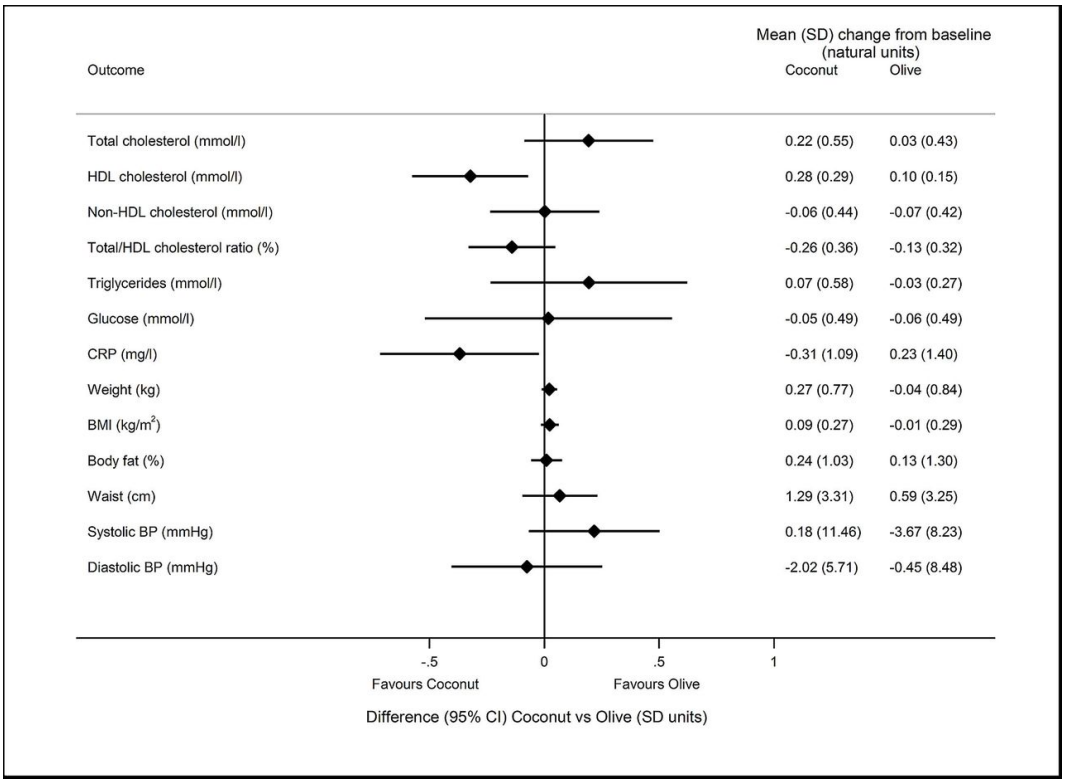
	LCFA			MCFA		
	Pre	Post	P value LCFA Pre-post	Pre	Post	P value MCFA Pre-post
Age (y)	52 ± 3			48 ± 3		
Sex (number M/F)	3 / 4			1/8		
Race (AA, Caucasian)	4,3			1,8		
Height (cm)	69 ± 1			66 ± 1		
Weight (kg)	101.0 ± 5.9	99.8 ± 5.9	<0.01	95.6 ± 5.2	93.8 ± 5.2	<0.001
Body mass index (kg/m^2)	33.2 ± 1.9	32.8 ± 1.9	<0.01	34.1 ± 1.7	33.5 ± 1.7	<0.0001
Waist/hip	0.97 ± 0.03	1.00 ± 0.03	NS	0.96 ± 0.02	0.91 ± 0.03	NS
Fat-free mass (kg)	59.6 ± 3.2			52.3 ± 3.1		
% Fat	39.4 ± 2.9			44.9 ± 1.7		
ALT (units/liter)	78 ± 18	68 ± 18	NS	42 ± 15	41 ± 16	NS
AST (units/liter)	48 ± 8	40 ± 9	<0.05	36 ± 7	29 ± 8†	<0.05
Glucose (mg/dL)	169 ± 25	146 ± 25	<0.05	143 ± 22	130 ± 22	NS
Insulin (μU/mL)	15 ± 6	12 ± 6	NS	22 ± 5	20 ± 5	NS
HbA1c (%)	7.7 ± 0.7	7.9 ± 0.7	NS	7.7 ± 0.6	7.4 ± 0.6	NS
HbA1c (mmol/mol)	61 ± 20	63 ± 20	NS	61 ± 17	57 ± 17	NS
TG (mg/dL)	162 ± 46	162 ± 47	NS	149 ± 40	182 ± 41	NS
Total cholesterol (mg/dL)	156 ± 10	142 ± 10	NS	187 ± 9*	176 ± 9	<0.05
HDL (mg/dL)	49 ± 6	48 ± 6	NS	50 ± 5	46 ± 5	NS
LDL (mg/dL)	75 ± 9	67 ± 10	NS	108 ± 8*	98 ± 9	<0.05
hsCRP (mg/dL)	2.7 ± 2.0	2.3 ± 2.0	NS	7.1 ± 1.8	5.7 ± 1.8	NS
Hepatic TG (%)	19.5 ± 4.5	17.6 ± 4.5	NS	23.9 ± 3.9	23.9 ± 3.9	NS
Cardiac TG (%)	1.5 ± 0.3	1.5 ± 0.4	NS	1.8 ± 0.3	1.4 ± 0.3	NS
Whole body lipid oxidation(g/kg/day)	1.5 ± 0.2	1.6 ± 0.2	NS	1.6 ± 0.2	1.4 ± 0.2	NS
Whole body carbohydrate oxidation (g/kg/day)	1.1 ± 0.4	0.7 ± 0.4	NS	1.1 ± 0.4	1.4 ± 0.4	NS

Khaw et al., (2018) Study design: Randomized Controlled Trial; Class: A; Quality Rating: Positive (+)

In a most recent RCT by Khaw et al., 2018, researchers found that even though coconut oil and butter are saturated fats, they have different effects on blood lipids compared to olive oil. Three different dietary fats were compared to assess changes in lipid profile and other CVD risk factors. Study participants were healthy men and women aged 50-75 years with no previous history of CVD or diabetes. They were not taking any cholesterol lowering medications. Subjects were randomly assigned to receive 50 grams

daily of either extra virgin coconut oil, extra virgin olive oil, or butter for the duration of the intervention, which lasted four weeks. Butter increased LDL significantly compared to coconut oil and olive oil, but there was no significant increase in LDL in coconut oil compared to olive oil (Figure 9). HDL was significantly increased in those consuming coconut oil and TC/HDL did not differ significantly from olive oil. This was a relatively short-term intervention and blinding was not used, which are important limitations to this study. In addition, dietary intake was not controlled for. However, subjects were from the general population and there was good compliance. This was a randomized controlled study and outcome measures were objective, which minimized confounding and bias.

Figure 9. *(Khaw et al., 2018)*



Question to be Answered:

What is the effect of coconut oil consumption compared to MCT oil on cardiovascular risk factors in adults with or without existing CVD?

Conclusion Statement

According to the available research reviewed here, coconut oil consumption does not have a negative effect on CVD risk factors. Coconut oil is largely made up of fatty acids that are 8-12 carbons in length, which are digested and metabolized differently than other SFA and LCFA. In addition, coconut oil is not the same as pure MCT oil, which does not contain any lauric acid which is a 12-chain carbon fatty acid. The evidence presented here suggests that MCT oil may aide in weight loss, improve cardiac function, reduce TC and LDL and had no adverse effects on CVD risk. Studies that used coconut oil showed decreased WC, NC, BMI, reduced CRP, which reduces metabolic risk, also raised HDL, and did not significantly raise LDL. The method of processing of the coconut oil may play a role however, with emphasis on virgin or extra virgin coconut oil showing a more favorable outcome with regards to reducing risk for CVD. MCFA from virgin coconut oil does not increase CVD risk and may reduce risk.

Seven studies were reviewed, four of which were of strong design, using the gold standard of randomized controlled trials. One was a non-randomized controlled trial. Two studies used MCT oil and five used coconut oil. Two of the studies examined secondary prevention for subjects who had already experienced a cardiac event and were receiving standard medical care, including the use of lipid-lowering medications. This is an important limitation, as it is impossible to make a recommendation regarding coconut oil

because of the use of statins. All but one of the studies received positive ratings. However, some studies had a very small sample size and two were observational in design. Therefore, this project was given the following grade.

Grade: II, Fair

CHAPTER 5: DISCUSSION AND CONCLUSION

Evidence Summary

There is a limited amount of recent evidence available related to this research topic, and no available studies that specifically compared coconut oil to MCT oil. The seven articles reviewed have some similarities and some differences. They all explore CVD in some way, whether directly or indirectly. Dibello et al., 2009 and Lipoeto et al., 2004 observed differences in food patterns in groups of people and their relative risk of CVD. Lipoeto compared the diets of cases with existing CVD with controls that were free from disease. No relationship was found between SFA intake and CVD risk. Dibello also found no increase to CVD risk among the subjects with a high intake of coconut products. Populations in both of these studies consume a large amount of coconut products in their usual diet, which is high in SFA.

Cardoso et al., 2015 and Vijayakumar et al., 2016 both looked at secondary prevention of CVD using patients with stable CAD who were also receiving standard medical care. Results of both studies showed there was no association with consumption of coconut oil and increased risk for CVD. These were both small samples and subjects were using statins or other lipid lowering medications. No definitive conclusion can be made regarding the effectiveness or safety of coconut oil when statins are also being used.

The four RCT that were reviewed included Khaw et al., 2018, St-Onge et al., 2008, Vijayakumar et al., 2016 and Airhart et al., 2016, compared different types of fats

including coconut oil, olive oil, butter, sunflower oil, as well as MCT oil, MCFA and LCFA. The St-Onge study was a weight loss study, this is important because obesity is a risk factor for CVD. Small samples and short intervention periods were limiting factors, although the Vijayakumar trial lasted for 2 years. All the studies used adult men and women. Three of the studies used patients with pre-existing CAD. Three studies used subjects with risk factors including overweight, type 2 diabetes or metabolic syndrome, while one RCT used relatively healthy individuals with no known risk factors for CVD. Two studies were observational in design and therefore no cause and effect can be assumed. These two studies had relatively large samples of people with traditional diets that include coconut products. Both of these studies did identify food patterns and showed no association between coconut intake and increased risk for CVD. None of the studies showed an increase in LDL with coconut oil and most if not all showed increase in HDL as well as weight loss, lowered BP, WC, and no association with increased CVD risk.

Application to Practice

Nutrition guidelines have been well established in the prevention of CVD risk factors. Traditionally, a diet low in total fat and saturated fat intake is recommended due to associations between dietary fat intake and increased risk for CVD. These recommendations were based on evidence from past research and the process of scientific research is constantly changing and evolving. The results of this research project have indicated that the type of fatty acid found in virgin coconut oil, although a saturated fat, may actually have a cardioprotective quality due to the chain length of the SFA. This

information could potentially impact nutrition guidelines and change recommendations given to patients in dietetic practice, particularly cardiac patients. Interest in coconut oil has been growing in recent years and this latest research can lead to interventions in combination with other factors such as a general healthful diet and increased exercise to help improve quality of life and improved health outcomes for populations with and without existing CVD. However, caution must be observed when making recommendations regarding coconut oil because coconut oil is not the same as MCT oil and more research is needed before promoting its use as safe and effective.

Recommendations for Future Research

The current available research is still very limited in this topic. Although the evidence presented here received a fair grade II, there is opportunity for future studies that would include larger samples and longer-term prospective research designs. Strong study designs utilizing double-blinded randomization should be done to further investigate the effects of coconut oil in comparison with other fats, particularly regarding MCT oil, and in a variety of diets in various cultures. According to this preliminary evidence, there may be health benefits to coconut oil, especially virgin coconut oil, but it is still too early to make broad recommendations. Further research should compare coconut oil to MCT oil in particular and should examine primary prevention in a long-term prospective study as well as effects in patients with existing CVD. The incidence and prevalence of CVD has grown, and it is important to continue research into causes and prevention of this group of diseases. There is not yet enough evidence based research to make a definitive conclusion as to whether or not to recommend consuming coconut

oil as a safe and advantageous addition to the usual diet for the general population. There is certainly interest however and as shown in this project, there is evidence to suggest a health benefit associated with the use of virgin coconut oil.

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Appendix A: Evidence Worksheets and Quality Criteria Checklists

Academy of Nutrition and Dietetics
Evidence Analysis Library® Worksheet Template

Citation	Airhart, S., Cade, W. T., Jiang, H., Coggan, A. R., Racette, S. B., Korenblat, K., . . . Peterson, L. R. (2016). A Diet Rich in Medium-Chain Fatty Acids Improves Systolic Function and Alters the Lipidomic Profile in Patients With Type 2 Diabetes: A Pilot Study. *The Journal of Clinical Endocrinology & Metabolism, 101*(2), 504-512. doi:10.1210/jc.2015-3292
Study Design	Double-blind Randomized Controlled Trial
Class	A
Quality Rating	**+ (Positive) +**
Research Purpose	To assess the effects of a diet rich in MCFA vs LCFA on CVD risk factors in type 2 diabetes patients.
Inclusion Criteria	Ambulatory patients in the general community. Men and women ages 37-65 years with type 2 diabetes. Ejection fraction >45% Not taking insulin. No other systemic diseases.
Exclusion Criteria	Ejection fraction <45% MI/ischemia, atrial fibrillation. Pregnant or lactating. Smokers
Description of Study Protocol	Recruitment: Ambulatory patients in the general community. Design: This was a prospective, double blind, randomized 2-week matched –feeding study. 16 subjects with type 2 diabetes underwent anthropometric measurements before randomization. Subjects provided 24-hour dietary recalls and fasting blood samples were drawn at each visit. Blinding used (if applicable): Double-blinded Intervention (if applicable): Subjects randomly assigned to receive either MCFA or LCFA as part of their diet. Both diets consisted of similar macronutrient distributions and were typical of a western diet. Subjects were provided with meals every 2-3 days. Dietitians monitored dietary compliance.

	Statistical Analysis: SAS v9.3 (SAS Institute Inc.) was used to conduct all analyses. Means of subject characteristics at baseline compared using Student's t test or chi-squared test. ANOVA used to analyze within group and between group changes in heart function and lipidomic measures. Significance was determined to be $P < .05$ for all analyses. Pearson's correlation coefficient was used to assess linear relationship between change in heart function (S') and change in lipidomic species in MCFA group.
Data Collection Summary	Timing of Measurements: Body composition measures were taken at baseline and at the end of the 2-week intervention. All metabolic panels, lipids, cardiac function tests were also performed pre- and post-intervention. Dependent Variables: Body composition measurements, cardiac steatosis and function, lipidomic changes. Independent Variables: time, group, interaction between the two. Control Variables: Subjects were given a 300-calorie smoothie prior to magnetic resonance spectroscopy (MRS) study containing LCFA pre-diet and LCFA or MCFA post diet treatment to minimize effect of prolonged fasting.
Description of Actual Data Sample	Initial: 22 (4 Males 12 Females) Attrition (final N): 16 Age: 45-55 Ethnicity: African American, Caucasian Other relevant demographics: no significant differences between the groups Anthropometrics: BMI 31.3-35.8 Location: Washington University School of Medicine, St. Louis, MO
Summary of Results	Key Findings: Measure of cardiac contractility, S', improved in MCFA group ($P < .05$). Weight-adjusted stroke volume and cardiac output

	decreased in LCFA group (P < .05). Lipid measures significantly decreased after MCFA diet, but not after LCFA diet (all P < .05 or less). Other Findings: Fasting insulin trended down, though not significant (P < .06) in MCFA group but not LCFA group. Fasting plasma glucose tended to decrease in LCFA group but was not significant compared to MCFA group.
Author Conclusion	MCFA-rich diet does not harm and may benefit cardiac function and fasting insulin levels in patients with type 2 diabetes by altering the plasma lipidome.
Reviewer Comments	*Strengths: double blind, RCT. MCFA diet had higher percentage of SFA, did not result in significantly worse cholesterol profile; consistent with epidemiological studies of Tokelau islanders.* *Weaknesses: Very small sample. Relatively short term. Did not control for effects of sex hormones on diet and/or lipidome. Larger, longer-term studies needed to confirm effects of MCFA vs LCFA on CVD risk factors.*
Funding Source	Grants from the Diabetic Cardiovascular Disease Center and the National Institutes of Health.

***Quality Criteria Checklist*: Primary Research**

RELEVANCE QUESTIONS

1.	Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (NA for some Epi studies)	Yes
2.	Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3.	Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes
4.	Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
If the answers to all of the above relevance questions are "Yes," the report is eligible for designation with a plus (+) on the Evidence Quality Worksheet, depending on answers to the following validity questions.		
VALIDITY QUESTIONS		
1.	**Was the research question clearly stated?**	Yes
1.1	Was the specific intervention(s) or procedure (independent variable(s)) identified?	
1.2	Was the outcome(s) (dependent variable(s)) clearly indicated?	
1.3	Were the target population and setting specified?	
2.	**Was the selection of study subjects/patients free from bias?**	Yes
2.1	Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study?	
2.2	Were criteria applied equally to all study groups?	
2.3	Were health, demographics, and other characteristics of subjects described?	
2.4	Were the subjects/patients a representative sample of the relevant population?	
3.	**Were study groups comparable?**	Yes
3.1	Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)	
3.2	Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?	
3.3	Were concurrent controls used? (Concurrent preferred over historical controls.)	
3.4	If cohort study or cross-sectional study, were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis?	
3.5	If case control study, were potential confounding factors comparable for cases and controls? (If case series or trial with subjects serving as own control, this criterion is not applicable. Criterion may not be applicable in some cross-sectional studies.)	
3.6	If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	
4.	**Was method of handling withdrawals described?**	Unclear
4.1	Were follow up methods described and the same for all groups?	
4.2	Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.)	
4.3	Were all enrolled subjects/patients (in the original sample) accounted for?	
4.4	Were reasons for withdrawals similar across groups?	
4.5	If diagnostic test, was decision to perform reference test not dependent on results of test under study?	
5.	**Was blinding used to prevent introduction of bias?**	Yes
5.1	In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate?	
5.2	Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.)	
5.3	In cohort study or cross-sectional study, were measurements of outcomes and risk factors blinded?	
5.4	In case control study, was case definition explicit and case ascertainment not influenced by exposure status?	
5.5	In diagnostic study, were test results blinded to patient history and other test results?	

6.	**Were <u>intervention</u>/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were <u>intervening factors</u> described?**	Yes
6.1	In RCT or other intervention trial, were protocols described for all regimens studied?	
6.2	n observational study, were interventions, study settings, and clinicians/provider described?	
6.3	Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect?	
6.4	Was the amount of exposure and, if relevant, subject/patient compliance measured?	
6.5	Were co-interventions (e.g., ancillary treatments, other therapies) described?	
6.6	Were extra or unplanned treatments described?	
6.7	Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups?	
6.8	In diagnostic study, were details of test administration and replication sufficient?	
7.	**Were <u>outcomes</u> clearly defined and the <u>measurements valid and reliable</u>?**	Yes
7.1	Were primary and secondary endpoints described and relevant to the question?	
7.2	Were nutrition measures appropriate to question and outcomes of concern?	
7.3	Was the period of follow-up long enough for important outcome(s) to occur?	
7.4	Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures?	
7.5	Was the measurement of effect at an appropriate level of precision?	
7.6	Were other factors accounted for (measured) that could affect outcomes?	
7.7	Were the measurements conducted consistently across groups?	
8.	**Was the <u>statistical analysis</u> appropriate for the study design and type of outcome indicators?**	Yes
8.1	Were statistical analyses adequately described the results reported appropriately?	
8.2	Were correct statistical tests used and assumptions of test not violated?	
8.3	Were statistics reported with levels of significance and/or confidence intervals?	
8.4	Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)?	
8.5	Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)?	
8.6	Was clinical significance as well as statistical significance reported?	
8.7	If negative findings, was a power calculation reported to address type 2 error?	
9.	**Are <u>conclusions supported by results</u> with biases and limitations taken into consideration?**	Yes
9.1	Is there a discussion of findings?	
9.2	Are biases and study limitations identified and discussed?	
10.	**Is bias due to study's <u>funding or sponsorship</u> unlikely?**	Yes
10.1	Were sources of funding and investigators' affiliations described?	
10.2	Was there no apparent conflict of interest?	
MINUS/NEGATIVE (-)		
If most (six or more) of the answers to the above validity questions are "No," the report should be designated with a minus (-) symbol on the Evidence Worksheet.		
NEUTRAL (∅)		
If the answers to validity criteria questions 2, 3, 6, and 7 do not indicate that the study is exceptionally strong, the report should be designated with a neutral (∅) symbol on the Evidence Worksheet.		
PLUS/POSITIVE (+)		
If most of the answers to the above validity questions are "Yes" (including criteria 2, 3, 6, 7 and at least one additional "Yes"), the report should be designated with a plus symbol (+) on the Evidence Worksheet.		

Airhart, S., Cade, W. T., Jiang, H., Coggan, A. R., Racette, S. B., Korenblat, K., . . . Peterson, L. R. (2016). A Diet Rich in Medium-Chain Fatty Acids Improves Systolic Function and Alters the Lipidomic Profile in Patients With Type 2 Diabetes: A Pilot Study. *The Journal of Clinical Endocrinology & Metabolism, 101*(2), 504-512. doi:10.1210/jc.2015-3292

Academy of Nutrition and Dietetics
Evidence Analysis Library® Worksheet Template

Academy of Nutrition and Dietetics

Citation	Cardoso, D. A., Moreira, A. B., De Oliveira, G. M., Luiz, R. R., & Rosa, G. (2015). A coconut extra virgin oil-rich diet increases HDL cholesterol and decreases waist circumference and body mass in coronary artery disease patients. *Nutricion Hospitalaria, 32*(5), 2144-2152.
Study Design	Longitudinal non-randomized clinical trial
Class	C
Quality Rating	**+ (Positive) +**
Research Purpose	To examine the effect of consumption of extra virgin coconut oil on CAD risk factors.
Inclusion Criteria	Male and female, ages 45-85 years old. On secondary prevention of CAD (using lipid-lowering drugs). Previous MI and/or stable angina >6 months.
Exclusion Criteria	Subjects who had CABG and previous CV event within <6 months. Chronic renal failure with creatinine >1.2 mg/dL. Patients using coconut oil or other food supplements. Liver disease.
Description of Study Protocol	Recruitment: Patients screened from outpatient department of a specialized cardiology hospital during January- September 2012. Design: Three-month run-in phase to standardize food intake. Two intervention groups: diet group (DG) continued same diet; another group that received extra virgin coconut oil in addition to the standard diet (CODG). Diet prescribed during run-in according to dietary habits of volunteers and nutritional recommendations for individuals with dyslipidemia. CODG received extra virgin coconut oil in 13 ml sachets (30 per month). Patients seen monthly and received intensive dietary treatment with periodic phone calls to assess compliance. Fasting blood samples, 24-hour diet recalls, anthropometrics and BP obtained at each visit. Blinding used (if applicable): none Intervention (if applicable): CODG received 30 sachets/month containing 13 ml extra virgin coconut oil; instructed to consume one sachet per day, alone or added to fruit, without heating it.

	Statistical Analysis: SPSS, version 20.0 used to analyze data. Chi-square test used to compare categorical variables between groups; Kolmogorov-Smirnov adhesion test used. Paired student's t-test or Wilcoxen Signed Ranks used to measure changes in anthropometric and biochemical variables. Mann-Whitney U test used to evaluate effect of intervention groups. Student's t-test performed to evaluate differences between DG and CODG.
Data Collection Summary	Timing of Measurements: Monthly anthropometric measurements, BP, 12-hour fasting blood samples drawn, 24-hour diet recalls obtained. Dependent Variables: Body mass, height, waist circumference, neck circumference, BP, TG, TC, HDL, LDL, ApoA-1, ApoB, fasting plasma glucose, HgA1c. Independent Variables: coconut oil Control Variables: standard diet group
Description of Actual Data Sample	Initial: 360 – did not specify numbers of male/female; stated 63.2% of population male. Attrition (final N): 114 Age: 51-75 Ethnicity: did not specify, although the main ethnicity of Rio de Janeiro is Portuguese. Other relevant demographics: No significant difference between groups at the beginning. Anthropometrics: BMI 24-35.7 Location: Rio de Janeiro, Brazil
Summary of Results	Key Findings: Significant difference in weight (-0.6 kg +-1.8, $P<0.01$), BMI (-0.2 +-0.7, $P<0.01$), WC (-2.1 cm +-2.7, $P<0.01$), NC (-0.4 cm +-0.9, $P<0.01$) and diastolic blood pressure (DBP) (-3.5 mmHg +-13.8, $P<0.01$) in the CODG.

	Other Findings: Significant increase in HDL (3.1 mg/dl +-7.4, P<0.01) for CODG compared to DG (P<0.01).
Author Conclusion	Results show that including 13 ml of extra virgin coconut oil in the diet increases HDL and decreases WC, which helps in secondary prevention of CAD.
Reviewer Comments	*Strengths of this study include the similarity between the groups at baseline. Weaknesses include small sample size, lack of randomization, use of lipid-lowering drugs.*
Funding Source	Institute of National Cardiology

***Quality Criteria Checklist*: Primary Research**

RELEVANCE QUESTIONS

1. Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (NA for some Epi studies)	Yes
2. Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3. Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes
4. Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
If the answers to all of the above relevance questions are "Yes," the report is eligible for designation with a plus (+) on the Evidence Quality Worksheet, depending on answers to the following validity questions.	
VALIDITY QUESTIONS	
1. Was the research question clearly stated?	Yes
1.1 Was the specific intervention(s) or procedure (independent variable(s)) identified?	
1.2 Was the outcome(s) (dependent variable(s)) clearly indicated?	
1.3 Were the target population and setting specified?	
2. Was the selection of study subjects/patients free from bias?	Yes
2.1 Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study?	
2.2 Were criteria applied equally to all study groups?	
2.3 Were health, demographics, and other characteristics of subjects described?	
2.4 Were the subjects/patients a representative sample of the relevant population?	
3. Were study groups comparable?	Yes
3.1 Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)	
3.2 Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?	
3.3 Were concurrent controls used? (Concurrent preferred over historical controls.)	
3.4 If cohort study or cross-sectional study, were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis?	
3.5 If case control study, were potential confounding factors comparable for cases and controls? (If case series or trial with subjects serving as own control, this criterion is not applicable. Criterion may not be applicable in some cross-sectional studies.)	
3.6 If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	
4. Was method of handling withdrawals described?	Yes
4.1 Were follow up methods described and the same for all groups?	
4.2 Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.)	
4.3 Were all enrolled subjects/patients (in the original sample) accounted for?	
4.4 Were reasons for withdrawals similar across groups?	
4.5 If diagnostic test, was decision to perform reference test not dependent on results of test under study?	
5. Was blinding used to prevent introduction of bias?	No
5.1 In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate?	
5.2 Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.)	
5.3 In cohort study or cross-sectional study, were measurements of outcomes and risk factors blinded?	
5.4 In case control study, was case definition explicit and case ascertainment not influenced by exposure status?	
5.5 In diagnostic study, were test results blinded to patient history and other test results?	

6. Were intervention/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were intervening factors described? 6.1 In RCT or other intervention trial, were protocols described for all regimens studied? 6.2 n observational study, were interventions, study settings, and clinicians/provider described? 6.3 Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect? 6.4 Was the amount of exposure and, if relevant, subject/patient compliance measured? 6.5 Were co-interventions (e.g., ancillary treatments, other therapies) described? 6.6 Were extra or unplanned treatments described? 6.7 Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups? 6.8 In diagnostic study, were details of test administration and replication sufficient?	Yes
7. Were outcomes clearly defined and the measurements valid and reliable? 7.1 Were primary and secondary endpoints described and relevant to the question? 7.2 Were nutrition measures appropriate to question and outcomes of concern? 7.3 Was the period of follow-up long enough for important outcome(s) to occur? 7.4 Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures? 7.5 Was the measurement of effect at an appropriate level of precision? 7.6 Were other factors accounted for (measured) that could affect outcomes? 7.7 Were the measurements conducted consistently across groups?	Yes
8. Was the statistical analysis appropriate for the study design and type of outcome indicators? 8.1 Were statistical analyses adequately described the results reported appropriately? 8.2 Were correct statistical tests used and assumptions of test not violated? 8.3 Were statistics reported with levels of significance and/or confidence intervals? 8.4 Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)? 8.5 Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)? 8.6 Was clinical significance as well as statistical significance reported? 8.7 If negative findings, was a power calculation reported to address type 2 error.	Yes
9. Are conclusions supported by results with biases and limitations taken into consideration? 9.1 Is there a discussion of findings? 9.2 Are biases and study limitations identified and discussed?	Yes
10. Is bias due to study's funding or sponsorship unlikely? 10.1 Were sources of funding and investigators' affiliations described? 10.2 Was there no apparent conflict of interest?	Yes
MINUS/NEGATIVE (-) *If most (six or more) of the answers to the above validity questions are "No," the report should be designated with a minus (-) symbol on the Evidence Worksheet.*	
NEUTRAL (∅) *If the answers to validity criteria questions 2, 3, 6, and 7 do not indicate that the study is exceptionally strong, the report should be designated with a neutral (∅) symbol on the Evidence Worksheet.*	
PLUS/POSITIVE (+) *If most of the answers to the above validity questions are "Yes" (including criteria 2, 3, 6, 7 and at least one additional "Yes"), the report should be designated with a plus symbol (+) on the Evidence Worksheet.*	

Cardoso, D. A., Moreira, A. B., De Oliveira, G. M., Luiz, R. R., & Rosa, G. (2015). A coconut extra virgin oil-rich diet increases HDL cholesterol and decreases waist circumference and body mass in coronary artery disease patients. *Nutricion Hospitalaria, 32*(5), 2144-2152.

Academy of Nutrition and Dietetics
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Citation	Dibello, J. R., Mcgarvey, S. T., Kraft, P., Goldberg, R., Campos, H., Quested, C., . . . Baylin, A. (2009). Dietary Patterns Are Associated with Metabolic Syndrome in Adult Samoans. The Journal of Nutrition, 139(10), 1933-1943. doi:10.3945/jn.109.107888
Study Design	Cross-sectional
Class	D
Quality Rating	**+ (Positive) +**
Research Purpose	To observe differences in dietary patterns related to presence of metabolic syndrome in the Samoan Islands.
Inclusion Criteria	Adults over age 18 years.
Exclusion Criteria	Missing information on FFQ
Description of Study Protocol	Recruitment: from Samoa, subjects who took part in Samoan Family Study of Overweight and Diabetes in 2002-2003 and members of American Samoa pedigrees. Recruitment from American Samoa in 2002 randomly selected from individuals who participated in a 1990-94 cohort study in American Samoa. Design: Anthropometrics taken, blood samples obtained. Dietary intakes assessed during interviews and FFQ administered; had to be altered to include commonly consumed food items in Samoa. Blinding used (if applicable): N/A Intervention (if applicable): N/A Statistical Analysis: ANOVA used to measure within- and between-subject variation for FFQ validity and reliability. Chi-square tests used to measure categorical variables; t tests used for continuous variables.
Data Collection Summary	Timing of Measurements: N/A Dependent Variables: food intake, BMI, abdominal circumference, TC, TG, HDL, LDL, VLDL, BP, fasting glucose. Independent Variables: N/A Control Variables: N/A

Description of Actual Data Sample	Initial: (672 Males 836 Females) Attrition (final N): 1508 Age: 22-65 Ethnicity: Samoan and American Samoan Other relevant demographics: Anthropometrics: BMI 23.8-44.8 Location: Samoan Islands
Summary of Results	Key Findings: Neo-traditional and modern eating patterns were identified; neo-traditional associated with significant increase in HDL in American Samoa (P<0.02) and significant decrease in abdominal circumference in both groups (P<0.03). Other Findings:
Author Conclusion	Results provide evidence for potential protective effect of neo-traditional eating pattern on CVD risk in Samoa and American Samoa.
Reviewer Comments	*Strengths include large sample size; statistical methods thorough in identifying dietary patterns and associations with disease risk. Weaknesses include cross-sectional, not a RCT, unable to show causation.*
Funding Source	NIH grants

***Quality Criteria Checklist*: Primary Research**

RELEVANCE QUESTIONS

1. Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (NA for some Epi studies)	Yes
2. Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3. Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes
4. Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
If the answers to all of the above relevance questions are "Yes," the report is eligible for designation with a plus (+) on the Evidence Quality Worksheet, depending on answers to the following validity questions.	
VALIDITY QUESTIONS	
1. Was the research question clearly stated?	Yes
1.1 Was the specific intervention(s) or procedure (independent variable(s)) identified?	
1.2 Was the outcome(s) (dependent variable(s)) clearly indicated?	
1.3 Were the target population and setting specified?	
2. Was the selection of study subjects/patients free from bias?	Yes
2.1 Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study?	
2.2 Were criteria applied equally to all study groups?	
2.3 Were health, demographics, and other characteristics of subjects described?	
2.4 Were the subjects/patients a representative sample of the relevant population?	
3. Were study groups comparable?	Yes
3.1 Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)	
3.2 Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?	
3.3 Were concurrent controls used? (Concurrent preferred over historical controls.)	
3.4 If cohort study or cross-sectional study, were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis?	
3.5 If case control study, were potential confounding factors comparable for cases and controls? (If case series or trial with subjects serving as own control, this criterion is not applicable. Criterion may not be applicable in some cross-sectional studies.)	
3.6 If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	
4. Was method of handling withdrawals described?	Yes
4.1 Were follow up methods described and the same for all groups?	
4.2 Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.)	
4.3 Were all enrolled subjects/patients (in the original sample) accounted for?	
4.4 Were reasons for withdrawals similar across groups?	
4.5 If diagnostic test, was decision to perform reference test not dependent on results of test under study?	
5. Was blinding used to prevent introduction of bias?	Unclear N/A
5.1 In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate?	
5.2 Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.)	
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6. Were intervention/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were intervening factors described? 6.1 In RCT or other intervention trial, were protocols described for all regimens studied? 6.2 In observational study, were interventions, study settings, and clinicians/provider described? 6.3 Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect? 6.4 Was the amount of exposure and, if relevant, subject/patient compliance measured? 6.5 Were co-interventions (e.g., ancillary treatments, other therapies) described? 6.6 Were extra or unplanned treatments described? 6.7 Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups? 6.8 In diagnostic study, were details of test administration and replication sufficient?	Yes
7. Were outcomes clearly defined and the measurements valid and reliable? 7.1 Were primary and secondary endpoints described and relevant to the question? 7.2 Were nutrition measures appropriate to question and outcomes of concern? 7.3 Was the period of follow-up long enough for important outcome(s) to occur? 7.4 Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures? 7.5 Was the measurement of effect at an appropriate level of precision? 7.6 Were other factors accounted for (measured) that could affect outcomes? 7.7 Were the measurements conducted consistently across groups?	Yes
8. Was the statistical analysis appropriate for the study design and type of outcome indicators? 8.1 Were statistical analyses adequately described the results reported appropriately? 8.2 Were correct statistical tests used and assumptions of test not violated? 8.3 Were statistics reported with levels of significance and/or confidence intervals? 8.4 Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)? 8.5 Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)? 8.6 Was clinical significance as well as statistical significance reported? 8.7 If negative findings, was a power calculation reported to address type 2 error?	Yes
9. Are conclusions supported by results with biases and limitations taken into consideration? 9.1 Is there a discussion of findings? 9.2 Are biases and study limitations identified and discussed?	Yes
10. Is bias due to study's funding or sponsorship unlikely? 10.1 Were sources of funding and investigators' affiliations described? 10.2 Was there no apparent conflict of interest?	Yes
MINUS/NEGATIVE (-) *If most (six or more) of the answers to the above validity questions are "No," the report should be designated with a minus (-) symbol on the Evidence Worksheet.*	
NEUTRAL (∅) *If the answers to validity criteria questions 2, 3, 6, and 7 do not indicate that the study is exceptionally strong, the report should be designated with a neutral (∅) symbol on the Evidence Worksheet.*	
PLUS/POSITIVE (+) *If most of the answers to the above validity questions are "Yes" (including criteria 2, 3, 6, 7 and at least one additional "Yes"), the report should be designated with a plus symbol (+) on the Evidence Worksheet.*	

Dibello, J. R., Mcgarvey, S. T., Kraft, P., Goldberg, R., Campos, H., Quested, C., . . . Baylin, A. (2009). Dietary Patterns Are Associated with Metabolic Syndrome in Adult Samoans. The Journal of Nutrition, 139(10), 1933-1943. doi:10.3945/jn.109.107888

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Citation	Khaw, K., Sharp, S. J., Finikarides, L., Afzal, I., Lentjes, M., Luben, R., & Forouhi, N. G. (2018). Randomised trial of coconut oil, olive oil or butter on blood lipids and other cardiovascular risk factors in healthy men and women. *BMJ Open, 8*(3). doi:10.1136/bmjopen-2017-020167
Study Design	Randomized Controlled Trial
Class	A
Quality Rating	**+ (Positive) +**
Research Purpose	To assess changes in lipid profile, weight and other metabolic markers after consumption of either coconut oil, olive oil or butter.
Inclusion Criteria	Men and women aged 50-75 years. No known history of cancer, CVD or diabetes. Not on lipid lowering medication. No contraindication to high fat diet. Willingness to participate.
Exclusion Criteria	History of heart disease, stroke, cancer or diabetes. Taking statins or other cholesterol lowering medication. Gall bladder or bowel problems.
Description of Study Protocol	Recruitment: Recruited by the British Broadcasting Corporation (BBC) via website. Design: RCT conducted from June through July 2017. Subjects had two assessments, at baseline and again after 4 weeks of intervention. Participants answered lifestyle, diet and physical activities questionnaires. DietWebQ, a 24-hour diet assessment that included estimated nutrient intake, and was developed in Oxford was conducted. Blinding used (if applicable): None Intervention (if applicable): Subjects randomly assigned to one of three groups, extra virgin coconut oil, butter or extra virgin olive oil. They were to consume 50 g of their assigned fat daily for 4

	weeks. Participants continued to consume their usual diet, incorporating their assigned fat as a supplement or substitute for other fats or oils. Statistical Analysis: 30 subjects in each group provided approximately 80% power to detect a difference in mean within-person change in LDL, comparing pairs of randomized groups. Primary analysis used intention-to-treat (ITT), whether or not individuals complied with the intervention. Secondary analysis used a per protocol (PP) population, a subset of ITT, consisting of subjects that were compliant with the intervention. P value calculated to compare three groups using linear regression; change from baseline was the outcome. Difference between pairs and 95% CI were estimated.
Data Collection Summary	Timing of Measurements: Assessment conducted at baseline including anthropometric measurements, BP and fasting blood samples. Follow-up assessments at 4 weeks post intervention period, anthropometrics and fasting blood samples were taken again. Dependent Variables: LDL, TC, HDL, TG, fasting blood glucose, C reactive protein, weight, BMI, body fat percentage, WC and BP. Independent Variables: coconut oil, butter, olive oil Control Variables: N/A
Description of Actual Data Sample	Initial: 160 (31 Males 63 Females) Attrition (final N): 94 Age: 52-67 Ethnicity: 98% European Caucasian Other relevant demographics: Anthropometrics: BMI 20.5-30

	Location: Cambridgeshire, UK
Summary of Results	Key Findings: LDL significantly increased on butter compared to coconut oil (+0.42, 95% CI 0.19 to 0.65 mmol/L, P<0.0001) and with olive oil (+0.38, 95% CI 0.16 to 0.60 mmol/L, P<0.0001). Coconut oil significantly increased HDL compared to butter (+0.18, 95% CI 0.06 to 0.30 mmol/L) Other Findings: Butter significantly increased TC/HDL ratio and non-HDL compared to coconut oil, which did not differ significantly from olive oil. There were no significant differences in weight, BMI, fasting blood glucose, or BP in any of the groups.
Author Conclusion	The two different saturated fats appeared to have different effects on blood lipids compared to olive oil. This may be related to chain length of fatty acids, processing methods, or even in relation to dietary patterns.
Reviewer Comments	*Strengths of this study include randomization, high completion rate, self-reported compliance. Large sample, not on any medications to lower cholesterol.* *Weaknesses include relatively short trial period, blinding was not used, dietary intake was not controlled.*
Funding Source	British Broadcasting Corporation, National Institute of Health Research Senior Investigator Award to K-TK and core MRC Epidemiology support.

***Quality Criteria Checklist*: Primary Research**

RELEVANCE QUESTIONS

1. Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (NA for some Epi studies)	Yes
2. Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3. Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes
4. Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
If the answers to all of the above relevance questions are "Yes," the report is eligible for designation with a plus (+) on the Evidence Quality Worksheet, depending on answers to the following validity questions.	
VALIDITY QUESTIONS	
1. Was the research question clearly stated?	Yes
1.1 Was the specific intervention(s) or procedure (independent variable(s)) identified?	
1.2 Was the outcome(s) (dependent variable(s)) clearly indicated?	
1.3 Were the target population and setting specified?	
2. Was the selection of study subjects/patients free from bias?	Yes
2.1 Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study?	
2.2 Were criteria applied equally to all study groups?	
2.3 Were health, demographics, and other characteristics of subjects described?	
2.4 Were the subjects/patients a representative sample of the relevant population?	
3. Were study groups comparable?	Yes
3.1 Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)	
3.2 Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?	
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3.6 If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	
4. Was method of handling withdrawals described?	Yes
4.1 Were follow up methods described and the same for all groups?	
4.2 Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.)	
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4.4 Were reasons for withdrawals similar across groups?	
4.5 If diagnostic test, was decision to perform reference test not dependent on results of test under study?	
5. Was blinding used to prevent introduction of bias?	No
5.1 In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate?	
5.2 Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.)	
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7. Were outcomes clearly defined and the measurements valid and reliable? 7.1 Were primary and secondary endpoints described and relevant to the question? 7.2 Were nutrition measures appropriate to question and outcomes of concern? 7.3 Was the period of follow-up long enough for important outcome(s) to occur? 7.4 Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures? 7.5 Was the measurement of effect at an appropriate level of precision? 7.6 Were other factors accounted for (measured) that could affect outcomes? 7.7 Were the measurements conducted consistently across groups?	Yes
8. Was the statistical analysis appropriate for the study design and type of outcome indicators? 8.1 Were statistical analyses adequately described the results reported appropriately? 8.2 Were correct statistical tests used and assumptions of test not violated? 8.3 Were statistics reported with levels of significance and/or confidence intervals? 8.4 Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)? 8.5 Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)? 8.6 Was clinical significance as well as statistical significance reported? 8.7 If negative findings, was a power calculation reported to address type 2 error?	Yes
9. Are conclusions supported by results with biases and limitations taken into consideration? 9.1 Is there a discussion of findings? 9.2 Are biases and study limitations identified and discussed?	Yes
10. Is bias due to study's funding or sponsorship unlikely? 10.1 Were sources of funding and investigators' affiliations described? 10.2 Was there no apparent conflict of interest?	Yes
MINUS/NEGATIVE (-) *If most (six or more) of the answers to the above validity questions are "No," the report should be designated with a minus (-) symbol on the Evidence Worksheet.*	
NEUTRAL (∅) *If the answers to validity criteria questions 2, 3, 6, and 7 do not indicate that the study is exceptionally strong, the report should be designated with a neutral (∅) symbol on the Evidence Worksheet.*	
PLUS/POSITIVE (+) *If most of the answers to the above validity questions are "Yes" (including criteria 2, 3, 6, 7 and at least one additional "Yes"), the report should be designated with a plus symbol (+) on the Evidence Worksheet.*	

Khaw, K., Sharp, S. J., Finikarides, L., Afzal, I., Lentjes, M., Luben, R., & Forouhi, N. G. (2018). Randomised trial of coconut oil, olive oil or butter on blood lipids and other cardiovascular risk factors in healthy men and women. *BMJ Open, 8*(3). doi:10.1136/bmjopen-2017-020167

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Evidence Analysis Library® Worksheet Template

Academy of Nutrition and Dietetics

Citation	Lipoeto, N. I., Agus, Z., Oenzil, F., Wahlqvist, M. L., & Wattanapenpaiboon, N. (2004). Dietary intake and the risk of coronary heart disease among the coconut-consuming Minangkabau in West Sumatra, Indonesia. *Asia Pacific Journal of Clinical Nutrition, 13*(4), 377-384.
Study Design	Case-control study
Class	C
Quality Rating	☐ ⊘ **(Neutral)**
Research Purpose	To examine the difference in food patterns and CHD risk between cases and controls.
Inclusion Criteria	Subjects with CHD from 5 local hospitals outpatient; diagnosed <6 months prior by a cardiologist. Controls from outpatient Ear Nose Throat Eyes clinic from same hospitals.
Exclusion Criteria	Subjects in control group with health problems related to CVD, HTN, and diabetes. Pregnant women.
Description of Study Protocol	Recruitment: Cases selected from outpatient clinic of cardiovascular unit of 5 local hospitals in 2 cities in West Sumatra. Control subjects recruited from outpatient Ear Nose Throat Eyes clinics from same hospitals. Design: Controls randomly selected to match cases in age and gender. Questionnaires on demographics, health status, lifestyle, food habits and food frequency administered. Information on nutrient content and composition of foods obtained from Nutrient Composition of Malaysian/Indonesian foods as well as USDA Nutrient Database. Blinding used (if applicable): N/A Intervention (if applicable): N/A Statistical Analysis: SAS software version 6.12 for Windows. Descriptive statistics used for sample distributions and attributes for confounders and antecedent factors. Mean, standard deviations, percentiles used for continuous variables. Frequency and percentage used for discrete variables. Subjects divided into quartiles to compare CHD events with food/nutrient variables. Odds ratio (OR) also calculated.
Data Collection Summary	

	Timing of Measurements: Information obtained regarding intake over the previous 12 months. Dependent Variables: food patterns/intake Independent Variables: N/A Control Variables: Healthy individuals
Description of Actual Data Sample	Initial: (175 Males 107 Females) Attrition (final N): N/A Age: did not specify Ethnicity: Indonesian Other relevant demographics: Anthropometrics: not specified Location: West Sumatra
Summary of Results	Key Findings: Significantly higher intakes of meats, eggs, sugar in case subjects. No difference in plant food intake, including coconut milk and grated coconut; no statistical difference for saturated or unsaturated fatty acid intake between groups. No relationship found for CHD risk and total dietary fat intake. Other Findings:
Author Conclusion	Results did not indicate an association between SFA intake (especially from coconut oil) and increased risk for CHD.
Reviewer Comments	*Not a RCT; FFQ may not be reliable; cases matched to controls, although specific demographics not noted; intake of Western foods was minimal.*
Funding Source	Not indicated.

***Quality Criteria Checklist*: Primary Research**

RELEVANCE QUESTIONS

1. Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (NA for some Epi studies)	Yes
2. Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3. Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes
4. Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
If the answers to all of the above relevance questions are "Yes," the report is eligible for designation with a plus (+) on the Evidence Quality Worksheet, depending on answers to the following validity questions.	
VALIDITY QUESTIONS	
1. Was the research question clearly stated?	Yes
1.1 Was the specific intervention(s) or procedure (independent variable(s)) identified?	
1.2 Was the outcome(s) (dependent variable(s)) clearly indicated?	
1.3 Were the target population and setting specified?	
2. Was the selection of study subjects/patients free from bias?	Unclear
2.1 Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study?	
2.2 Were criteria applied equally to all study groups?	
2.3 Were health, demographics, and other characteristics of subjects described?	
2.4 Were the subjects/patients a representative sample of the relevant population?	
3. Were study groups comparable?	Yes
3.1 Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)	
3.2 Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?	
3.3 Were concurrent controls used? (Concurrent preferred over historical controls.)	
3.4 If cohort study or cross-sectional study, were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis?	
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3.6 If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	
4. Was method of handling withdrawals described?	No
4.1 Were follow up methods described and the same for all groups?	
4.2 Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.)	
4.3 Were all enrolled subjects/patients (in the original sample) accounted for?	
4.4 Were reasons for withdrawals similar across groups?	
4.5 If diagnostic test, was decision to perform reference test not dependent on results of test under study?	
5. Was blinding used to prevent introduction of bias?	No
5.1 In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate?	
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7. Were outcomes clearly defined and the measurements valid and reliable? 7.1 Were primary and secondary endpoints described and relevant to the question? 7.2 Were nutrition measures appropriate to question and outcomes of concern? 7.3 Was the period of follow-up long enough for important outcome(s) to occur? 7.4 Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures? 7.5 Was the measurement of effect at an appropriate level of precision? 7.6 Were other factors accounted for (measured) that could affect outcomes? 7.7 Were the measurements conducted consistently across groups?	Yes
8. Was the statistical analysis appropriate for the study design and type of outcome indicators? 8.1 Were statistical analyses adequately described the results reported appropriately? 8.2 Were correct statistical tests used and assumptions of test not violated? 8.3 Were statistics reported with levels of significance and/or confidence intervals? 8.4 Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)? 8.5 Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)? 8.6 Was clinical significance as well as statistical significance reported? 8.7 If negative findings, was a power calculation reported to address type 2 error?	Yes
9. Are conclusions supported by results with biases and limitations taken into consideration? 9.1 Is there a discussion of findings? 9.2 Are biases and study limitations identified and discussed?	Yes
10. Is bias due to study's funding or sponsorship unlikely? 10.1 Were sources of funding and investigators' affiliations described? 10.2 Was there no apparent conflict of interest?	Yes
MINUS/NEGATIVE (-) *If most (six or more) of the answers to the above validity questions are "No," the report should be designated with a minus (-) symbol on the Evidence Worksheet.*	
NEUTRAL (∅) *If the answers to validity criteria questions 2, 3, 6, and 7 do not indicate that the study is exceptionally strong, the report should be designated with a neutral (∅) symbol on the Evidence Worksheet.*	
PLUS/POSITIVE (+) *If most of the answers to the above validity questions are "Yes" (including criteria 2, 3, 6, 7 and at least one additional "Yes"), the report should be designated with a plus symbol (+) on the Evidence Worksheet.*	

Lipoeto, N. I., Agus, Z., Oenzil, F., Wahlqvist, M. L., & Wattanapenpaiboon, N. (2004). Dietary intake and the risk of coronary heart disease among the coconut-consuming Minangkabau in West Sumatra, Indonesia. *Asia Pacific Journal of Clinical Nutrition, 13*(4), 377-384.

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Citation	St-Onge, M., Bosarge, A., Goree, L. L., & Darnell, B. (2008). Medium Chain Triglyceride Oil Consumption as Part of a Weight Loss Diet Does Not Lead to an Adverse Metabolic Profile When Compared to Olive Oil. Journal of the American College of Nutrition, 27(5), 547-552. doi:10.1080/07315724.2008.10719737
Study Design	Randomized Controlled Trial
Class	A
Quality Rating	**+ (Positive) +**
Research Purpose	To examine effects of MCT oil compared to olive oil consumption as part of a weight loss diet on cardiovascular disease risk.
Inclusion Criteria	Men and women age 19-50 BMI of 27-33 Stable weight for >= 6 months Free from chronic disease
Exclusion Criteria	Individuals using any weight loss medications Pregnant women, planning to become pregnant, <1 year postpartum, or breastfeeding
Description of Study Protocol	Recruitment: From Birmingham, AL greater metropolitan area through newspaper advertisements and flyers. Design: Subjects randomized to one of two weight loss groups: consumed either 18-24 g/day MCT oil or olive oil, provided in muffins. Weekly group counseling sessions for 16 weeks. Caloric intakes of 1500-1800 kcal/day. Weight and waist circumference measured weekly. Blood samples and blood pressure measured at baseline, 8 weeks and 16 weeks. Blinding used (if applicable): double-blinded Intervention (if applicable): MCT oil or olive oil consumption in a weight loss program Statistical Analysis: SAS Softwear for Windows version 9.1 ANOVA used to analyze random variable: subject; fixed variables: race, time, diet. Quantitative variables: change in body weight Change between groups analyzed by unpaired t tests

Data Collection Summary	Timing of Measurements: Baseline, 8 weeks and 16 weeks; weekly weight and waist circumference Dependent Variables: Body weight, waist circumference, TC, LDL, HDL, TG, insulin, glucose, BP Independent Variables: MCT oil Control Variables: Olive oil
Description of Actual Data Sample	Initial: 49 (3 Males 28 Females) Attrition (final N): 31 Age: 33-40 Ethnicity: AA, C, H Other relevant demographics: 76.7-84.5 kg Anthropometrics: BMI 28.9-30.6 Location: Birmingham AL
Summary of Results	Key Findings: Weight loss in both groups; MCT oil had lower weight at conclusion: (-1.67 +- 0.67 kg, P=0.013) Other Findings: Trend toward greater fat mass loss (P=0.071) with MCT oil.
Author Conclusion	Results suggest that MCT oil can be used as part of a weight loss program without adverse effects on metabolic risk.
Reviewer Comments	*Strengths of this study include randomization and double-blinded design. Limitations include dropout rate and small sample size; lack of ethnic diversity; unequal gender representation; olive oil not entirely comparable to MCT oil.*
Funding Source	International Life Sciences Institute, North America and GCRC grant M01 RR-00032 from the National Center for Research Resources. MCT oil donated by Stepan Company.

***Quality Criteria Checklist*: Primary Research**

RELEVANCE QUESTIONS

1. Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (NA for some Epi studies)	Yes
2. Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3. Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes
4. Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
If the answers to all of the above relevance questions are "Yes," the report is eligible for designation with a plus (+) on the Evidence Quality Worksheet, depending on answers to the following validity questions.	
VALIDITY QUESTIONS	
1. Was the research question clearly stated?	Yes
1.1 Was the specific intervention(s) or procedure (independent variable(s)) identified?	
1.2 Was the outcome(s) (dependent variable(s)) clearly indicated?	
1.3 Were the target population and setting specified?	
2. Was the selection of study subjects/patients free from bias?	Yes
2.1 Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study?	
2.2 Were criteria applied equally to all study groups?	
2.3 Were health, demographics, and other characteristics of subjects described?	
2.4 Were the subjects/patients a representative sample of the relevant population?	
3. Were study groups comparable?	Yes
3.1 Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT)	
3.2 Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline?	
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4. Was method of handling withdrawals described?	Yes
4.1 Were follow up methods described and the same for all groups?	
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5. Was blinding used to prevent introduction of bias?	Yes
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5.4 In case control study, was case definition explicit and case ascertainment not influenced by exposure status?	
5.5 In diagnostic study, were test results blinded to patient history and other test results?	

6. Were intervention/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were intervening factors described? 6.1 In RCT or other intervention trial, were protocols described for all regimens studied? 6.2 n observational study, were interventions, study settings, and clinicians/provider described? 6.3 Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect? 6.4 Was the amount of exposure and, if relevant, subject/patient compliance measured? 6.5 Were co-interventions (e.g., ancillary treatments, other therapies) described? 6.6 Were extra or unplanned treatments described? 6.7 Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups? 6.8 In diagnostic study, were details of test administration and replication sufficient?	Yes
7. Were outcomes clearly defined and the measurements valid and reliable? 7.1 Were primary and secondary endpoints described and relevant to the question? 7.2 Were nutrition measures appropriate to question and outcomes of concern? 7.3 Was the period of follow-up long enough for important outcome(s) to occur? 7.4 Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures? 7.5 Was the measurement of effect at an appropriate level of precision? 7.6 Were other factors accounted for (measured) that could affect outcomes? 7.7 Were the measurements conducted consistently across groups?	Yes
8. Was the statistical analysis appropriate for the study design and type of outcome indicators? 8.1 Were statistical analyses adequately described the results reported appropriately? 8.2 Were correct statistical tests used and assumptions of test not violated? 8.3 Were statistics reported with levels of significance and/or confidence intervals? 8.4 Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)? 8.5 Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)? 8.6 Was clinical significance as well as statistical significance reported? 8.7 If negative findings, was a power calculation reported to address type 2 error?	Yes
9. Are conclusions supported by results with biases and limitations taken into consideration? 9.1 Is there a discussion of findings? 9.2 Are biases and study limitations identified and discussed?	Yes
10. Is bias due to study's funding or sponsorship unlikely? 10.1 Were sources of funding and investigators' affiliations described? 10.2 Was there no apparent conflict of interest?	Yes
MINUS/NEGATIVE (-) *If most (six or more) of the answers to the above validity questions are "No," the report should be designated with a minus (-) symbol on the Evidence Worksheet.*	
NEUTRAL (∅) *If the answers to validity criteria questions 2, 3, 6, and 7 do not indicate that the study is exceptionally strong, the report should be designated with a neutral (∅) symbol on the Evidence Worksheet.*	
PLUS/POSITIVE (+) *If most of the answers to the above validity questions are "Yes" (including criteria 2, 3, 6, 7 and at least one additional "Yes"), the report should be designated with a plus symbol (+) on the Evidence Worksheet.*	

St-Onge, M., Bosarge, A., Goree, L. L., & Darnell, B. (2008). Medium Chain Triglyceride Oil Consumption as Part of a Weight Loss Diet Does Not Lead to an Adverse Metabolic Profile When Compared to Olive Oil. Journal of the American College of Nutrition, 27(5), 547-552. doi:10.1080/07315724.2008.10719737

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Evidence Analysis Library® Worksheet Template

Citation	Vijayakumar, M., Vasudevan, D., Sundaram, K., Krishnan, S., Vaidyanathan, K., Nandakumar, S., . . . Mathew, N. (2016). A randomized study of coconut oil versus sunflower oil on cardiovascular risk factors in patients with stable coronary heart disease. *Indian Heart Journal, 68*(4), 498-506. doi:10.1016/j.ihj.2015.10.384
Study Design	Randomized Controlled Trial
Class	A
Quality Rating	**+ (Positive) +**
Research Purpose	To evaluate coconut oil and sunflower oil on CVD risk factors in stable CAD patients receiving standard care.
Inclusion Criteria	Male/female >18 years old Diagnosed with CAD Achieved target lipid levels per adult treatment panel III (ATP-III) Good glycemic control (HbA1c <7mg %) Willing to comply with follow-up visits Signed informed consent form
Exclusion Criteria	Untreated or uncontrolled hypothyroidism or diabetes mellitus Severe CHF Pre-existing malabsorption syndrome Dietary pattern unsuitable for trial design Abnormal renal function, creatinine >2.0 mg/dl Abnormal hepatic enzymes
Description of Study Protocol	Recruitment: Subjects selected from patients at outpatient center of a hospital in India. Patients were previously diagnosed with CAD by coronary angiogram, echocardiography, or ECG evidence of MI. Design: Patients randomly assigned to two groups, one received coconut oil (Group I), and the other received sunflower oil (Group II) to cook with for a period of two years. Subjects were also receiving standard medical care. Blinding used (if applicable): Single blinded; did not provide details. Intervention (if applicable): Two groups, one provided with coconut oil, the other received sunflower oil. Oils given to subjects as well as family members. 24-hour diet recalls taken at start to test dietary patterns. Recalls were used to calculate the amount of oil needed to supply 15% of

	daily Calories. 7-day recalls and diet diaries used to monitor for adherence throughout the duration of study. Statistical Analysis: Student's t-test used to compare difference in mean values of all variables. Wilcoxen rank sum test used when sample size was relatively small for some variables. Chi-square test used for categorical variables.
Data Collection Summary	Timing of Measurements: 3 months, 6 months, 1 year and 2 years. Dependent Variables: BMI, percentage body fat, waist hip ratio, flow-mediated vasodilation, lipid profiles, HgA1c, antioxidant enzymes. Independent Variables: coconut oil, sunflower oil Control Variables: standard medical care
Description of Actual Data Sample	Initial: 420 (187 Males 13 Females) Attrition (final N): 200 Age: 50-68 Ethnicity: Indian Other relevant demographics: groups were similar in active lifestyle, smoking and occupation. Anthropometrics: BMI 19-31 Location: India
Summary of Results	Key Findings: No statistically significant differences were found for any of the measurements in 2 years from either group. Other Findings: There was a reduction in inflammatory marker CRP in Group I, though not significant.
Author Conclusion	Cooking with coconut oil over a period of 2 years did not affect CVD risk factors compared to sunflower oil.
Reviewer Comments	*Small sample in relation to total population. Subjects receiving statins. Source of funding may have played a role.*
Funding Source	Coconut Development Board of India and Amrita Institute of Medical Sciences, Kocho, India.

Quality Criteria Checklist: Primary Research

RELEVANCE QUESTIONS	
1. Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group? (NA for some Epi studies)	Yes
2. Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes
3. Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes
4. Is the intervention or procedure feasible? (NA for some epidemiological studies)	Yes
If the answers to all of the above relevance questions are "Yes," the report is eligible for designation with a plus (+) on the Evidence Quality Worksheet, depending on answers to the following validity questions.	
VALIDITY QUESTIONS	
1. Was the research question clearly stated? 1.1 Was the specific intervention(s) or procedure (independent variable(s)) identified? 1.2 Was the outcome(s) (dependent variable(s)) clearly indicated? 1.3 Were the target population and setting specified?	Yes
2. Was the selection of study subjects/patients free from bias? 2.1 Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient detail and without omitting criteria critical to the study? 2.2 Were criteria applied equally to all study groups? 2.3 Were health, demographics, and other characteristics of subjects described? 2.4 Were the subjects/patients a representative sample of the relevant population?	Yes
3. Were study groups comparable? 3.1 Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT) 3.2 Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline? 3.3 Were concurrent controls used? (Concurrent preferred over historical controls.) 3.4 If cohort study or cross-sectional study were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis? 3.5 If case control study, were potential confounding factors comparable for cases and controls? (If case series or trial with subjects serving as own control, this criterion is not applicable. Criterion may not be applicable in some cross-sectional studies.) 3.6 If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	Yes
4. Was method of handling withdrawals described? 4.1 Were follow up methods described and the same for all groups? 4.2 Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80 %.) 4.3 Were all enrolled subjects/patients (in the original sample) accounted for? 4.4 Were reasons for withdrawals similar across groups? 4.5 If diagnostic test, was decision to perform reference test not dependent on results of test under study?	Yes
5. Was blinding used to prevent introduction of bias? 5.1 In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate? 5.2 Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.) 5.3 In cohort study or cross-sectional study, were measurements of outcomes and risk factors blinded?	Yes

5.4	In case control study, was case definition explicit and case ascertainment not influenced by exposure status?	
5.5	In diagnostic study, were test results blinded to patient history and other test results?	
6.	**Were intervention/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were intervening factors described?**	Yes
6.1	In RCT or other intervention trial, were protocols described for all regimens studied?	
6.2	n observational study, were interventions, study settings, and clinicians/provider described?	
6.3	Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect?	
6.4	Was the amount of exposure and, if relevant, subject/patient compliance measured?	
6.5	Were co-interventions (e.g., ancillary treatments, other therapies) described?	
6.6	Were extra or unplanned treatments described?	
6.7	Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups?	
6.8	In diagnostic study, were details of test administration and replication sufficient?	
7.	**Were outcomes clearly defined and the measurements valid and reliable?**	Yes
7.1	Were primary and secondary endpoints described and relevant to the question?	
7.2	Were nutrition measures appropriate to question and outcomes of concern?	
7.3	Was the period of follow-up long enough for important outcome(s) to occur?	
7.4	Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures?	
7.5	Was the measurement of effect at an appropriate level of precision?	
7.6	Were other factors accounted for (measured) that could affect outcomes?	
7.7	Were the measurements conducted consistently across groups?	
8.	**Was the statistical analysis appropriate for the study design and type of outcome indicators?**	Yes
8.1	Were statistical analyses adequately described the results reported appropriately?	
8.2	Were correct statistical tests used and assumptions of test not violated?	
8.3	Were statistics reported with levels of significance and/or confidence intervals?	
8.4	Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)?	
8.5	Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)?	
8.6	Was clinical significance as well as statistical significance reported?	
8.7	If negative findings, was a power calculation reported to address type 2 error?	
9.	**Are conclusions supported by results with biases and limitations taken into consideration?**	Yes
9.1	Is there a discussion of findings?	
9.2	Are biases and study limitations identified and discussed?	
10.	**Is bias due to study's funding or sponsorship unlikely?**	Unclear
10.1	Were sources of funding and investigators' affiliations described?	
10.2	Was there no apparent conflict of interest?	

MINUS/NEGATIVE (-)

If most (six or more) of the answers to the above validity questions are "No," the report should be designated with a minus (-) symbol on the Evidence Worksheet.

NEUTRAL (∅)

If the answers to validity criteria questions 2, 3, 6, and 7 do not indicate that the study is exceptionally strong, the report should be designated with a neutral (∅) symbol on the Evidence Worksheet.

PLUS/POSITIVE (+)

If most of the answers to the above validity questions are "Yes" (including criteria 2, 3, 6, 7 and at least one additional "Yes"), the report should be designated with a plus symbol (+) on the Evidence Worksheet.

Vijayakumar, M., Vasudevan, D., Sundaram, K., Krishnan, S., Vaidyanathan, K., Nandakumar, S., . . . Mathew, N. (2016). A randomized study of coconut oil

versus sunflower oil on cardiovascular risk factors in patients with stable coronary heart disease. *Indian Heart Journal, 68*(4), 498-506. doi:10.1016/j.ihj.2015.10.384

Appendix B: Overview Table

Author, Year, Study Design, Class Rating	Study Type/Purpose	Study Population	Intervention	Outcomes	Conclusion	Limitations
St-Onge et al., 2008 Study Design: RCT Class: A Rating: +	To examine effects if MCT oil compared to olive oil consumption as part of a weight loss diet on CVD risk.	31 Men and women aged 19-50 with BMI 27-33, stable weight for >= 6 months, free from chronic disease.	Subjects randomized to 2 groups: each consumed 18-24 g/day either MCT oil or olive oil, provided in muffins. Weekly group counseling sessions for 16 weeks.	Weight loss in both groups, MCT oil group had lower weight, trend toward greater fat mass loss.	MCT oil can be used as part of weight loss program without adverse effects on metabolic risk.	High dropout rate, small sample size; lack of ethnic diversity; unequal gender represented; olive oil not entirely comparable to MCT oil
Lipoeto et al., 2004 Study Design: Case-control study Class: C Rating: +	To examine the difference in food patterns and CHD risk between cases and controls.	282 total subjects; Cases with CHD from 5 local hospitals outpatient; diagnosed <6 months prior by a cardiologist. Controls from outpatient Ear Nose Throat Eyes clinic from same hospitals.	62 men and 31 women in the Case group; 113 men and 76 women in the Control group. Controls randomly selected to match cases in age and gender. Questionnaires on demographics, health status, lifestyle, food habits and food frequency administered. Information on nutrient	Significantly higher intakes of meats, eggs, sugar in case subjects. No difference in plant food intake, including coconut milk and grated coconut; no statistical difference	Results did not indicate an association between SFA intake (especially from coconut oil) and increased risk for CHD.	Not a RCT; FFQ may not be reliable; cases matched to controls, although specific demographics not noted; intake of Western foods was minimal.

			content and composition of foods obtained from Nutrient Composition of Malaysian/Indonesian foods as well as USDA Nutrient Database.	for saturated or unsaturated fatty acid intake between groups. No relationship found for CHD risk and total dietary fat intake.		
Dibello et al., 2009 Study Design: Cross-sectional Class: D Rating: +	To observe differences in dietary patterns related to presence of metabolic syndrome in the Samoan Islands.	1,508 adult men and women from Samoa, subjects who took part in Samoan Family Study of Overweight and Diabetes in 2002-2003 and members of American Samoa pedigrees. Recruitment from American Samoa in 2002 randomly selected from individuals who participate	Anthropometrics taken, blood samples obtained. Dietary intakes assessed during interviews and FFQ administered; had to be altered to include commonly consumed food items in Samoa.	Neo-traditional and modern eating patterns were identified; neo-traditional associated with significant increase in HDL in American Samoa ($P<0.02$) and significant decrease in abdominal circumference in both groups ($P<0.03$).	Results provide evidence for potential protective effect of neo-traditional eating pattern on CVD risk in Samoa and American Samoa.	Weaknesses include study design was cross-sectional, not a RCT, unable to show causation.

		d in a 1990-94 cohort study in American Samoa.				
Cardoso et al., 2015 Study Design: Longitudinal non-randomized clinical trial Class: C Rating: +	To examine the effect of consumption of extra virgin coconut oil on CAD risk factors.	Male and female, ages 45-85 years old. On secondary prevention of CAD (using lipid-lowering drugs). Previous MI and/or stable angina >6 months.	Three-month run-in phase to standardize food intake. Two intervention groups: diet group (DG) continued same diet; another group that received extra virgin coconut oil in addition to the standard diet (CODG). Diet prescribed during run-in according to dietary habits of volunteers and nutritional recommendations for individuals with dyslipidemia. CODG received extra virgin coconut oil in 13 ml sachets (30 per month). Patients seen monthly and received intensive dietary treatment with periodic phone calls to assess compliance. Fasting blood	Significant difference in weight (-0.6 kg +-1.8, P<0.01), BMI (-0.2 +-0.7, P<0.01), WC (-2.1 cm +-2.7, P<0.01), NC (-0.4 cm +-0.9, P<0.01) and diastolic blood pressure (DBP) (-3.5 mmHg +-13.8, P<0.01) in the CODG. Significant increase in HDL (3.1 mg/dl +-7.4, P<0.01) for CODG compared to DG (P<0.01).	Results show that including 13 ml of extra virgin coconut oil in the diet increases HDL and decreases WC, which helps in secondary prevention of CAD.	Weaknesses include small sample size, lack of randomization.

			samples, 24-hour diet recalls, anthropometrics and BP obtained at each visit.			
Vijayakumar et al., 2016 Study Design: Randomized Controlled Trial Class: A Rating: +	To evaluate coconut oil and sunflower oil on CVD risk factors in stable CAD patients receiving standard care.	Male/female >18 years old, diagnosed with CAD and achieved target lipid levels per adult treatment panel III (ATP-III); good glycemic control (HbA1c <7mg %), willing to comply with follow-up visits and signed informed consent form.	Patients randomly assigned to two groups, one received coconut oil (Group I), and the other received sunflower oil (Group II) to cook with for a period of two years. Subjects were also receiving standard medical care.	No statistically significant differences were found for any of the measurements in 2 years from either group. There was a reduction in inflammatory marker CRP in Group I, though not significant.	Cooking with coconut oil over a period of 2 years did not affect CVD risk factors compared to sunflower oil.	Small sample in relation to total population. Subjects receiving statins. Source of funding may have played a role.
Airhart et al., 2016 Study Design: Double-blind Randomized Controlled Trial Class: A Rating: +	To assess the effects of a diet rich in MCFA vs LCFA on CVD risk factors in type 2 diabetes patients.	Ambulatory patients in the general community. Men and women ages 37-65 years with type 2 diabetes. Ejection fraction >45%	This was a prospective, double blind, randomized 2-week matched –feeding study. 16 subjects with type 2 diabetes underwent anthropometric measurements before randomization.	Measure of cardiac contractility, S', improved in MCFA group (P < .05). Weight-adjusted stroke volume and	MCFA-rich diet does not harm and may benefit cardiac function and fasting insulin	Very small sample. Relatively short term. Did not control for effects of sex hormones on diet and/or lipidome. Larger,

		Not taking insulin. No other systemic diseases.	Subjects provided 24-hour dietary recalls and fasting blood samples were drawn at each visit. Subjects randomly assigned to receive either MCFA or LCFA as part of their diet. Both diets consisted of similar macronutrient distributions and were typical of a western diet. Subjects were provided with meals every 2-3 days. Dietitians monitored dietary compliance.	cardiac output decreased in LCFA group ($P < .05$). Lipid measures significantly decreased after MCFA diet, but not after LCFA diet (all $P < .05$ or less). Fasting insulin trended down, though not significant ($P < .06$) in MCFA group but not LCFA group. Fasting plasma glucose tended to decrease in LCFA group but was not significant compared to MCFA group.	levels in patients with type 2 diabetes by altering the plasma lipidome.	longer-term studies needed to confirm effects of MCFA vs LCFA on CVD risk factors.
Khaw et al., 2018 Study Design: Randomi	To assess changes in lipid profile, weight	Men and women aged 50-75 years.	Subjects randomly assigned to one of three groups, extra virgin	LDL significantly increased on butter	The two different saturated fats	Weaknesses include relatively short trial period,

zed Controlled Trial Class: A Rating: +	and other metabolic markers after consumption of either coconut oil, olive oil or butter.	No known history of cancer, CVD or diabetes. Not on lipid lowering medication. No contraindication to high fat diet. Willingness to participate.	coconut oil, butter or extra virgin olive oil. They were to consume 50 g of their assigned fat daily for 4 weeks. Participants continued to consume their usual diet, incorporating their assigned fat as a supplement or substitute for other fats or oils.	compared to coconut oil (+0.42, 95% CI 0.19 to 0.65 mmol/L, P<0.0001) and with olive oil (+0.38, 95% CI 0.16 to 0.60 mmol/L, P<0.0001). Coconut oil significantly increased HDL compared to butter (+0.18, 95% CI 0.06 to 0.30 mmol/L) Butter significantly increased TC/HDL ratio and non-HDL compared to coconut oil, which did not differ significantly from olive oil. There were no	appeared to have different effects on blood lipids compared to olive oil. This may be related to chain length of fatty acids, processing methods, or even in relation to dietary patterns.	blinding was not used, dietary intake was not controlled.

				significant differences in weight, BMI, fasting blood glucose, or BP in any of the groups.		

Appendix C: Quality Criteria Summary

Questions	Airhart et al., 2016	Cardoso et al., 2015	Dibello et al., 2009	Khaw et al., 2018	Lipoeto et al., 2004	St-Onge et al. 2008	Vijayakumar et al., 2016
Relevance Questions							
1. Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
2. Did the authors study an outcome (dependent variable) or topic that the patients/clients/population group would care about?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
3. Is the focus of the intervention or procedure (independent variable) or topic of study a common issue of concern to dietetics practice?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
4. Is the intervention or procedure feasible?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Validity Questions							
1. Was the research question clearly stated? 1.1 Was the specific intervention(s) or procedure (independent variable(s)) identified? 1.2 Was the outcome(s) (dependent variable(s)) clearly indicated? 1.3 Were the target population and setting specified?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
2. Was the selection of study subjects/patients free from bias? 2.1 Were inclusion/exclusion criteria specified (e.g., risk, point in disease progression, diagnostic or prognosis criteria), and with sufficient	Yes	Yes	Yes	Yes	Unclear	Yes	Yes

detail and without omitting criteria critical to the study? 2.2 Were criteria applied equally to all study groups? 2.3 Were health, demographics, and other characteristics of subjects described? 2.4 Were the subjects/patients a representative sample of the relevant population?							
3. Were <u>study groups comparable</u>? 3.1 Was the method of assigning subjects/patients to groups described and unbiased? (Method of randomization identified if RCT) 3.2 Were distribution of disease status, prognostic factors, and other factors (e.g., demographics) similar across study groups at baseline? 3.3 Were concurrent controls used? (Concurrent preferred over historical controls.) 3.4 If cohort study or cross-sectional study, were groups comparable on important confounding factors and/or were preexisting differences accounted for by using appropriate adjustments in statistical analysis? 3.5 If case control study, were potential confounding factors comparable for cases and controls? (If case series or trial with subjects serving as own control, this criterion is not applicable. Criterion may not be applicable in some cross-sectional studies.) 3.6 If diagnostic test, was there an independent blind comparison with an appropriate reference standard (e.g., "gold standard")?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
4. Was method of handling <u>withdrawals</u> described? 4.1 Were follow up methods described and the same for all groups? 4.2 Was the number, characteristics of withdrawals (i.e., dropouts, lost to follow up, attrition rate) and/or response rate (cross-sectional studies) described for each group? (Follow up goal for a strong study is 80%.) 4.3 Were all enrolled subjects/patients (in the original sample) accounted for?	Unclear	Yes	Yes	Yes	No	Yes	Yes

4.4 Were reasons for withdrawals similar across groups? 4.5 If diagnostic test, was decision to perform reference test not dependent on results of test under study?							
5. Was <u>blinding</u> used to prevent introduction of bias? 5.1 In intervention study, were subjects, clinicians/practitioners, and investigators blinded to treatment group, as appropriate? 5.2 Were data collectors blinded for outcomes assessment? (If outcome is measured using an objective test, such as a lab value, this criterion is assumed to be met.) 5.3 In cohort study or cross-sectional study, were measurements of outcomes and risk factors blinded? 5.4 In case control study, was case definition explicit and case ascertainment not influenced by exposure status? 5.5 In diagnostic study, were test results blinded to patient history and other test results?	Yes	No	Unclear	No	No	Yes	Yes
6. Were <u>intervention</u>/therapeutic regimens/exposure factor or procedure and any comparison(s) described in detail? Were <u>intervening factors</u> described? 6.1 In RCT or other intervention trial, were protocols described for all regimens studied? 6.2 n observational study, were interventions, study settings, and clinicians/provider described? 6.3 Was the intensity and duration of the intervention or exposure factor sufficient to produce a meaningful effect? 6.4 Was the amount of exposure and, if relevant, subject/patient compliance measured? 6.5 Were co-interventions (e.g., ancillary treatments, other therapies) described? 6.6 Were extra or unplanned treatments described? 6.7 Was the information for 6.4, 6.5, and 6.6 assessed the same way for all groups?	Yes	Yes	Yes	Yes	Yes	Yes	Yes

6.8 In diagnostic study, were details of test administration and replication sufficient?							
7. Were <u>outcomes</u> clearly defined and the <u>measurements valid and reliable</u>? 7.1 Were primary and secondary endpoints described and relevant to the question? 7.2 Were nutrition measures appropriate to question and outcomes of concern? 7.3 Was the period of follow-up long enough for important outcome(s) to occur? 7.4 Were the observations and measurements based on standard, valid, and reliable data collection instruments/tests/procedures? 7.5 Was the measurement of effect at an appropriate level of precision? 7.6 Were other factors accounted for (measured) that could affect outcomes? 7.7 Were the measurements conducted consistently across groups?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
8. Was the <u>statistical analysis</u> appropriate for the study design and type of outcome indicators? 8.1 Were statistical analyses adequately described the results reported appropriately? 8.2 Were correct statistical tests used and assumptions of test not violated? 8.3 Were statistics reported with levels of significance and/or confidence intervals? 8.4 Was "intent to treat" analysis of outcomes done (and as appropriate, was there an analysis of outcomes for those maximally exposed or a dose-response analysis)? 8.5 Were adequate adjustments made for effects of confounding factors that might have affected the outcomes (e.g., multivariate analyses)? 8.6 Was clinical significance as well as statistical significance reported? 8.7 If negative findings, was a power calculation reported to address type 2 error?	Yes	Yes	Yes	Yes	Yes	Yes	Yes

9. Are <u>conclusions supported by results</u> with biases and limitations taken into consideration? 9.1 Is there a discussion of findings? 9.2 Are biases and study limitations identified and discussed?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
10. Is bias due to study's <u>funding or sponsorship</u> unlikely? 10.1 Were sources of funding and investigators' affiliations described? 10.2 Was there no apparent conflict of interest?	Yes	Yes	Yes	Yes	Yes	Yes	Unclear