Avondale College of Higher Education

School of Education

The effectiveness of a volunteer-delivered, community-based lifestyle modification program (CHIP) for reducing the risk factors associated with Metabolic Syndrome.

A Doctoral Thesis

Presented in Fulfillment

of the Requirements for the Award

of the Degree of

Doctor of Philosophy

by

Paul Rankin BA, MPH

2014

Student Declaration

I, Paul Meredith Rankin hereby declare that:

• this dissertation is my own work,

• all persons consulted, and all assistance rendered are fully acknowledged,

• all references used are indicated in the text and accurately reported in the list of references,

• the substance of this dissertation has not been presented, in whole, or part by me, to any other institution for a degree.

Signature  Date 10/10/13

Acknowledgements

To Sonia, Rebekah, Caleb and Rachel, my family and primary support mechanism, thank you for your support and patience not just with this Ph.D. but as we have pursued so many dreams. Without your love and support none of this would have happened.

Darren Morton, thanks mate. Not only have you been an extraordinary supervisor but you became a real friend as we embarked wholeheartedly on the CHIP adventure. The walk across Sydney Harbour Bridge in 2009 truly was a life changing event. Together we have flown!

The detailed statistical analysis contained in this dissertation would not have been possible without the support and coaching of Peter Morey who was my co-supervisor. Thank you for your patience and enthusiasm for this project and your encouragement to continue writing. Also to Esther Chang, thank you, for your passion for CHIP, encouragement and continued reminders to "tell the story" and that we are doing this in His service.

This project would not have been possible without the founder of CHIP: Hans Diehl. Thank you for your generosity and your passion to make a difference in people's lives. Thanks also to John Gobbles who collected the CHIP data used in this dissertation and generously made it available for analysis and to Dena Guthrie and Harold Burden for providing permission to utilise the dataset.

To Jerry Matthews, Peter Lynch and Graeme Drinkall, at the New Zealand Pacific Union Conference of the Seventh-day Adventist Church, thank you for providing fee assistance and release time from work for the initial three years of this project. Jerry,

thank you for your passion to see the departmental directors under your supervision pursue further study and encouraging us to make it a priority.

To my current boss, Kevin Price, Health Director for the South Pacific Division of the Seventh-day Adventist Church who insisted that I make the completion of this dissertation a priority even when other responsibilities were pressing, thank you. It's your turn now Kevin.

Kevin Jackson, Cathy MacDonald and Christina Hawkins at Sanitarium Health and Wellbeing and the Lifestyle Medicine Institute, thank you for your commitment to the vision of CHIP and your recognition of the importance of quality research in supporting this project.

A special thanks to Michelle Down, Richard Anderson and Elaine Lumsdaine the librarians at Avondale College of Higher Education who tracked down countless journal articles and patiently instructed me on how to use Endnote. Also to Dr Don Hanson who proofread this manuscript, I trust that all the apostrophes are now in their correct position.

Thank you to all of the CHIP directors and facilitators in both New Zealand and Australia; you have shown me that passionate volunteers really can make a difference in people's lives. Thanks also to the late Vic Gidley and his wife Jean Ward who brought CHIP to the South Pacific.

And above all thank you to my Creator and Saviour. The more I have learnt about the human body the more I have come to realise that we truly are fearfully and wonderfully made. Lord please show me where you want me to serve and give me the courage to follow your directions.

Abstract

In the latter half of the 20th Century increases in the availability of inexpensive, energy-dense foods combined with an estimated 60-70% reduction in daily energy expenditure, created an "obesogenic" environment. The epidemic of obesity that has emerged has been associated with the Metabolic Syndrome (MetS) which is a cluster of risk factors that are precursors to chronic diseases including cardiovascular disease (CVD), type II diabetes (T2D), stroke, and dementia. These chronic diseases are having a significant social and economic impact worldwide with their related morbidity and mortality.

Epidemiological and interventional studies have shown that lifestyle interventions based on a low-fat, plant-based diet are effective for the management of MetS and associated chronic diseases. To date, the research has focused on lifestyle interventions delivered by professional facilitators, mostly in clinical settings. The primary aim of this dissertation was to examine the effectiveness of a lifestyle intervention known as the Coronary Health Improvement Program (CHIP), when delivered by volunteer facilitators to free-living participants in their community. The secondary aim of the dissertation was to examine the impact of selected participant factors including age, gender, religious affiliation, marital status, previous history, family history and body mass index on the participants' responsiveness to the CHIP intervention.

This study adopted a pre-test post-test design involving, a large cohort of 5070 individuals who participated in one of 178 community-based CHIP interventions delivered by volunteers between 2006 to 2009 in the United States and Canada.

In 30 days, significant reductions ($p < 0.001$) were recorded in body mass index (3.2%), total cholesterol (11.0%), low-density lipoprotein cholesterol (13.0%), triglycerides (7.7%), fasting plasma glucose (6.1%) and systolic and diastolic blood pressure (4.9% and 5.3%, respectively). Stratification of the data revealed larger reductions in those participants who presented to the program with the greatest risk factor levels.

Regardless of age, gender, marital status, religious affiliation, previous history, family history and body weight, participants in the CHIP intervention achieved significant improvements in the risk factors associated with MetS however these factors influenced the responsiveness of the participants to the program. In general, male participants achieved better results than the females, and males tended to achieve their best outcomes at a younger age than the female participants. Married participants achieved better outcomes than single, divorced or widowed participants. Seventh-day Adventist (SDA) participants had better risk profiles at baseline, however, the non-SDA participants achieved better outcomes during the intervention. Participants who had the highest body mass index (BMI) at baseline achieved the greatest changes in BMI, fasting plasma glucose and blood pressure, however, those participants with a BMI greater than 35 kg/m^2 showed significantly less improvement in their lipid profile than those participants with a BMI between 25 and 35 kg/m^2.

It was concluded that significant reductions in the risk factors associated with MetS can be achieved in 30 days using the CHIP lifestyle intervention when delivered by volunteers to free-living participants in their local community. Significant reductions in risk factors associated with MetS can be achieved regardless of age, gender, marital

status, religious affiliation or baseline biometrics. Utilising volunteers may therefore provide an effective and cost-efficient mode of delivering lifestyle interventions targeting MetS.

Table of Contents

List of Tables

List of Figures

Glossary of abbreviations used in this dissertation

ACE/AACE	American College of Endocrinology / American Association of Clinical Endocrinologists
ADA	American Dietetic Association
AHS	Adventist Health Study
ATP III	National Cholesterol Education Program Adult Treatment Panel III
BMI	Body Mass Index
CHIP	Coronary Health Improvement Program
cm	centimetre
CVD	cardiovascular disease
DBP	diastolic blood pressure
EASD	European Association for the Study of Diabetes
EGIR	European Group for the Study of Insulin Resistance
FPG	fasting plasma glucose
FRS	Framingham Risk Score
g	grams
HDL	high density lipoprotein cholesterol
IDF	International Diabetes Federation
kg	kilogram
LDL	low density lipoprotein cholesterol
m	metre
MetS	Metabolic Syndrome
mg/dL	milligrams per decilitre
mmHG	millimetres of mercury
mmol/L	millimoles per litre
OGTT	oral glucose tolerance test
QALY	quality adjusted life years
SBP	systolic blood pressure
SDA	Seventh-day Adventist
T2D	type II diabetes
TC	total cholesterol
TG	triglycerides
TPB	Theory of Planned Behaviour
WC	waist circumference
WHO	World Health Organisation
WHR	waist- hip ratio
%	percentage
<	less than
>	greater than

Published papers and conference presentations arising from this dissertation

Published Papers

Rankin, P., Morton, D. P., Diehl, H., Gobble, J., Morey, P., & Chang, E. (2012). Effectiveness of a volunteer-delivered lifestyle modification program for reducing cardiovascular disease risk factors. *American Journal of Cardiology, 109*(1), 82-86.

Kent, L., Rankin, P., Morton, D. P., Ward, E. J., Grant, R., Gobble, J., et al. (2013). The effect of a low-fat, plant-based lifestyle intervention (CHIP) on serum HDL levels and the implications for Metabolic Syndrome status - a cohort study *Nutrition and Metabolism, 10*(1), 58

Conference presentations

Rankin, P., Morton, D. P., & Morey, P. Effectiveness of a volunteer delivered lifestyle modification program for reducing cardiovascular disease risk factors. Poster presentation at the Australian Disease Management Association Conference, Canberra, Australia. August, 2011

Rankin, P., Morton, D. P., & Morey, P. Gender and age influences on the outcomes achieved by a 30-day lifestyle modification program. Oral presentation at the Allied Health Professionals Association Conference, Canberra, Australia, April 2012

Kent, L, M., Rankin, P., Morton, D. P., Do men or women respond better to a lifestyle intervention program that emphasises a plant-based eating pattern? Poster presentation at the International Vegetarian Nutrition Conference, Loma Linda, Ca, USA, February 2013

Papers in preparation

Rankin, P., The effect of religious affiliation on the outcomes achieved by participants in a low-fat, plant-based lifestyle intervention (CHIP)

Rankin, P., Predictors of change for participants in lifestyle intervention emphasising a low-fat, plant-based eating pattern intervention (CHIP)

Rankin, P., The impact of weight loss on change achieved by participants in a lifestyle intervention emphasising low-fat, plant-based eating pattern.

Chapter 1. Introduction

1.1.Research rationale

Environmental and sociological changes arising in the latter half of the 20th century have created an "obesogenic" environment (Egger & Swinburn, 1997) . Increases in the availability of inexpensive, energy-dense foods, combined with an estimated 60-70% reduction in daily energy expenditure (Vogels, Egger, Plasqui, & Westerterp, 2004) has resulted in an epidemic of obesity (Agatston, 2012). The rise of obesity has been paralleled by a rise in a cluster of abnormalities that has been label the Metabolic Syndrome (MetS). MetS is defined as having three of the following five risk factors: increased body mass index (BMI), high triglyceride (TG) levels, low high density lipoprotein cholesterol (HDL) levels, hypertension, and raised fasting plasma glucose (FPG) levels (Alberti et al., 2009). The sequelae to MetS, including cardiovascular disease (CVD), type II diabetes (T2D), stroke, and dementia, with their related morbidity and mortality, have had a significant social and economic impact worldwide.

It has been suggested that the prevalence of MetS in the US adult population over the age of 20 is 42% in males and 38% in females (Alberti et al., 2009), and that health care costs increase by approximately 24% for each additional MetS component identified in an individual (Boudreau et al., 2009; Roger et al., 2012).

The medical establishment has a largely adopted a pharmaceutical and/or surgical approach for the management of MetS and its related symptoms. While

pharmaceutical and surgical interventions have benefits, the cost of these treatments has risen dramatically. In the United States it is estimated that in 2008 the direct medical cost of overweight and obesity alone was $113.9 billion which represents upwards of 10% of the total US health care spending (Tsai, Williamson, & Glick, 2011). The estimated fiscal burden of heart disease in the US was in excess of $500 billion in 2010 as a result of direct health care costs and lost productivity (Allen, 2009; Esselstyn, 2010). In 2010 the global health expenditure on diabetes was estimated to have been $USD 376 billion, representing 12% of the global health expenditure and this figure is forecast to rise to $490 billion annually by 2030 (Zhang et al., 2010).

As well as being costly, the pharmaceutical and/or surgical approach for the management of MetS does not treat the underlying cause of the syndrome. In fact, as they only target the symptoms of the condition, they have been referred to as "palliative care" (Esselstyn, 1999; Ornish, 2002). Indeed medication cannot compensate for, or neutralise, unhealthy lifestyle choices. Ferenczi, Asaria, Hughes, Chaturvedi, and Francis (2010) accurately assert that offering a free sashay of statins at fast food outlets to accompany the unhealthy meal will not remedy the growing burden of chronic disease.

Lifestyle intervention has been shown to be viable for the management of MetS and even superior to the prevailing medical treatment paradigm (Alberti, Zimmet, & Shaw, 2006). In a landmark study that compared the efficacy of lifestyle intervention to pharmaceuticals (Metformin) for preventing at-risk individuals progressing to develop T2D, the Diabetes Prevention Program (DPP) showed lifestyle intervention to be more than twice as effective (Herman et al., 2005). Similarly,

Orchard et al. (2005) found lifestyle intervention to reduce the incidence of MetS by 41% compared to a placebo while Metformin only reduced the incidence by 17%. Lifestyle intervention has been shown to be more cost effective than pharmaceutical intervention (Herman et al., 2005) as well as delivering significantly better outcomes overall and cardiovascular-event-free survival after first myocardial infarction than usual care (Tuttle et al., 2008).

In recent times, there has been an increasing interest in the lifestyle-centred approach for the management of chronic disease, and several lifestyle interventions have been developed. These interventions have been shown to be effective for the treatment of MetS when delivered by professional facilitators in residential settings (Barnard & Wen, 1994; Ornish et al., 1983), clinical settings and healthcare sites (Esselstyn, 1999; Silberman et al., 2010), workplace environments (Aldana, Greenlaw, Diehl, Englert, & Jackson, 2002), and in community settings (Diehl, 1998; Merrill & Aldana, 2008).

One such lifestyle intervention is the Coronary Health Improvement Program (CHIP). The CHIP lifestyle intervention was designed by Dr Hans Diehl 27 years ago (Diehl, 1998; Gidley, 2008). CHIP builds off a number of behaviour change strategies (Aldana, Greenlaw, et al., 2006) but leans heavily on the Theory of Planned Behaviour (TPB) (Ajzen, 1985) as described in more detail in Chapter 4. CHIP has been shown to be effective when delivered using paid health professionals as facilitators in workplace and hospital settings (Aldana et al., 2002; Englert, Diehl, Greenlaw, Willich, & Aldana, 2007; Merrill & Aldana, 2009).

The primary aim of this study was to examine the effectiveness of CHIP interventions facilitated by volunteers, in a community setting. The study analysed a large cohort of 5046 individuals who participated in one of 178 CHIP programs delivered throughout North America between 2006 and 2009. The programs were community-based meaning that they were offered to free-living individuals in their own environment, as opposed to participants in residential lifestyle centres. The programs were volunteer-delivered in that the facilitators donated their time. If effective the use of volunteer facilitators means that lifestyle intervention can be delivered at a significantly reduced cost when compared with professionally facilitated programs.

To date, not a lot is known about the responsiveness of different individuals to lifestyle interventions. The secondary aim of the present study was therefore to identify the impact of selected participant factors, such as age, gender, marital status, religious affiliation, previous history, family history and weight at program entry on the outcome of the CHIP intervention.

1.1 Research questions

The aim of this research project was to examine the effectiveness of the Coronary Health Improvement Program (CHIP) lifestyle modification program, delivered by volunteers to free-living participants in their community, targeting the cluster of risk factors associated with the Metabolic Syndrome (MetS).

In particular this research was guided by the following specific questions:

1. How effective is the Coronary Health Improvement Program (CHIP) lifestyle intervention for reducing the selected risk factors of chronic disease that constitute the Metabolic Syndrome, when delivered by volunteers to free-living participants in their community?
2. What is the impact of selected participant factors, including age, gender, marital status, religious affiliation, previous history, family history and body weight on the outcomes achieved by participants in the CHIP intervention?

This dissertation is presented in six chapters. Chapter 1 provides an introduction to the topic and an overview of the research questions. Chapter 2 provides a review of the relevant literature. The methodology undertaken in the study are presented in Chapter 3. Chapter 4 presents the results relating to the first research question by examining the effectiveness of the CHIP intervention, as delivered by volunteers. Chapter 5 presents the results relating to the second research question exploring the impact of selected participant factors on the outcomes achieved by

participants in the CHIP intervention. Finally, conclusions, limitations and recommendations for further study are presented in Chapter 6.

Chapter 2 Review of related literature

2.1 Introduction

This dissertation examines the effectiveness of volunteer-delivered, community-based Coronary Health Improvement Program (CHIP) interventions. This review of the related literature is organised into six major sections. The first section examines the changing dietary and activity patterns in the Western world over the last several generations which have led to an epidemic of obesity and the rise of chronic diseases. The second section introduces the Metabolic Syndrome (MetS) which is a cluster of abnormalities seen as a precursor to a number of chronic diseases. The relevance, aetiology and treatment of MetS are presented. The third section examines the literature relating to the development of the emerging field of Lifestyle Medicine which underpins the CHIP intervention, focusing on epidemiological studies and the work of significant pioneers in this area who influenced development of CHIP. Limitations of Lifestyle Medicine are also presented. In section four an overview of the CHIP intervention is presented with a review of publications arising from the CHIP intervention to date. The fifth section examines the literature relating to the use of volunteers in health education settings. The final section presents a summary of this chapter.

2.2 The rise of chronic disease

Since the Industrial Revolution there has been a dramatic change in the primary causes of mortality and morbidity in the Western World (Caballero, 2007; Caldwell, 2001). Changes in public health and hygiene around the time of the Industrial Revolution to the late 19th century, combined with the development of medical

"miracles" such as vaccinations and antibiotics in the early 20th century, resulted in a dramatic reduction in morbidity and mortality caused by communicable diseases. By the 1960s, described as “the age of optimism”, it appeared that the battle against disease had been all but won (Le Fanu, 2000, p. 213). However, for the first time in human history, machines were beginning to carry out work previously done by humans. The mechanisation of food production and associated changes in farming practice combined with state subsidisation of the agricultural sector enabled a dramatic increase in the production of food, making what had been formally a relatively scarce resource now a common commodity (Pollan, 2007). The technological revolution of the late 20th century increased the processing potential for food. This enabling food manufacturers to artificially increase the caloric content, and thus the energy density, of the food but not the nutrient density (Egger, Binns, & Rossner, 2011, p. 12). This increase of cheap, readily available, energy-dense food, combined with an estimated 60-70% reduction in daily energy expenditure since the 1950s (Vogels et al., 2004), has resulted in a substantial portion of the population of the Western world now consuming more calories in their diet than they burn in their daily activity (Agatston, 2012). In the year 2000, for the first time in human history, more people were overweight than underweight (Caballero, 2007; Gardner, Halweil, & Peterson, 2000).

Obesity and inactivity are not new phenomena, Lucretius is reputed to have stated in 50 BC, "In primitive times, lack of food gave languishing bodies to death; now, on the other hand, it is abundance that buries them" (Potter, 2005). Some 3000 years ago Solomon admonished the slothful "Go to the ant, thou sluggard; consider her

ways, and be wise:" (Proverbs 6:6). However it is only very recently that the conditions in Western society have become “obesogenic”, leading to an epidemic in obesity (Hill & Peters, 1998). It appears that epidemic increased dramatically in the 1980s in the United States (Friedewald et al., 2007; Sturm, 2002; Taubes, 1998). While this study focuses on the United States a similar rise in obesity in the eighties has been observed in Australia, (Kent & Worsley, 2009) and is continuing to have a significant impact worldwide (Yanovski & Yanovski, 2011). This change in obesity levels is illustrated in Figure 1 which shows the dramatic rise in the percentage of the population classified as obese by state, in the United States since 1990. It has been estimated that during the 80s and 90s the average Australian adult was adding 1g in body weight per day (Egger & Swinburn, 1997). Further, the obesity epidemic is now impacting children and teens (Ogden, Carroll, Kit, & Flegal, 2012).

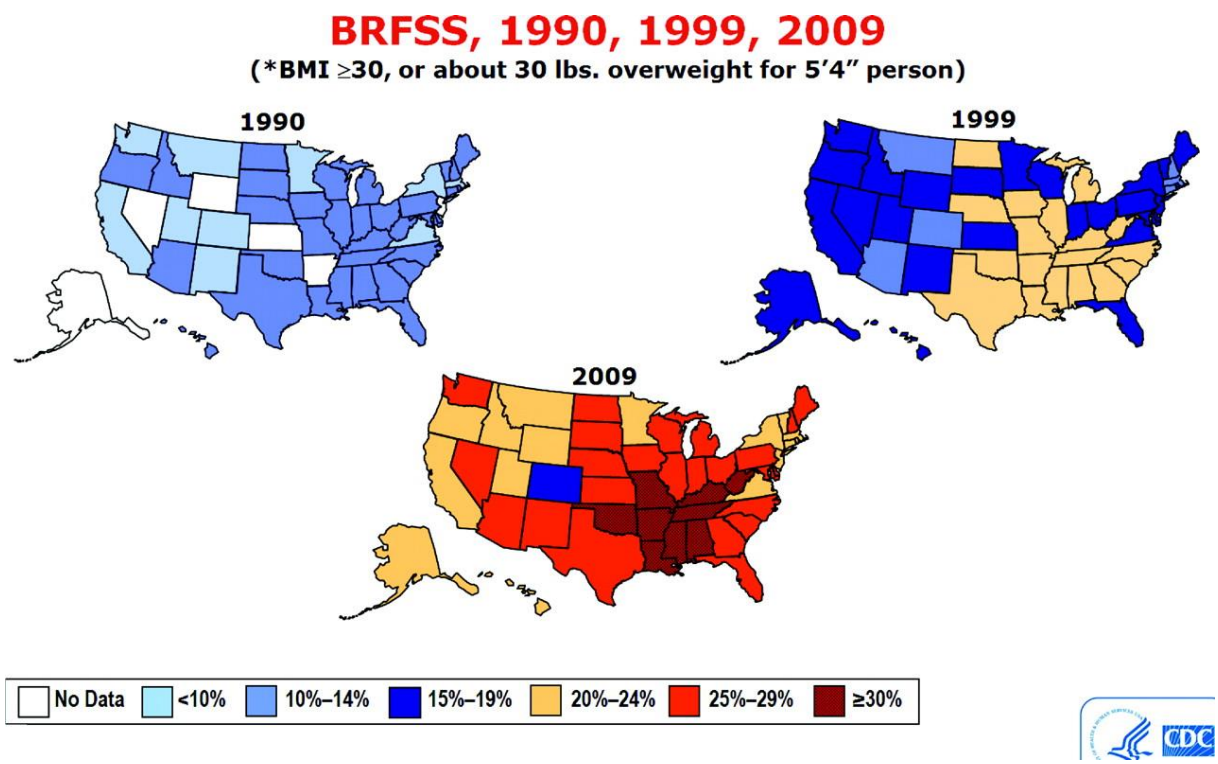


Figure 1 US obesity trends by state 1990 - 2009. (Agatston, 2012)

In recent decades the global epidemic of obesity has driven a dramatic increase in chronic diseases (Ford, Giles, & Mokdad, 2004). Using Quality Adjusted Life Years (QALY) to assess the overall burden of disease caused by smoking and obesity, from 1993 to 2008, Jia and Lubetkin (2010) showed that while the proportion of smokers in the US adult population declined by 18.5% over this period, the proportion of obese adults increased by 85%. The QALYs lost from smoking has remained relatively stable at 0.0438 QALYs lost per population, however, there has been a dramatic increase in the QALYs lost attributed to obesity from 0.0204 lost per population, in 1993, to 0.0464 in 2008. This represents a 127% increase over a 15 year period. It has been suggested that obesity is now a greater health risk than both smoking and the unhealthy consumption of alcohol (Sturm, 2002).

Lifestyle factors including smoking, an unhealthy diet, obesity and reduced physical activity have been linked to a number of chronic diseases including type II diabetes (T2D) (Freemantle, Holmes, Hockey, & Kumar, 2008), diseases of the circulatory system including cardiovascular disease (CVD), myocardial infarction (Yusuf et al., 2004) and stroke (Mokdad, Marks, Stroup, & Gerberding, 2004), depression (Pischke, Frenda, Ornish, & Weidner, 2010) (An Pan et al., 2012), the Metabolic Syndrome (MetS) (Eckel, Grundy, & Zimmet, 2005), some cancers (McNaughton, Marks, & Green, 2005) (Pan, Sun, & Bernstein, 2012) (Larsson & Wolk, 2012), kidney disease (Tang, Yan, & Zhuaig, 2012) and dementias and Alzheimer's disease (de la Monte & Wands, 2008).

This “toxic environment” (Yanovski & Yanovski, 2011) has created a situation where the incidence of T2D has risen to epidemic proportions (Wild, Roglic, Green, Sicree, & King, 2004) and cardiovascular disease (CVD) is the leading cause of premature death globally, accounting for almost one third of worldwide mortality (Ascunce, Berger, Weintraub, & Schwartzbard, 2012). Increasingly this burden of disease is being borne by developing countries (Alwan, 2011; Salehi, Hanifi, Khaleghparast, Ghadrdoost, & Zamari Nobari Shabnam, 2011) where it is estimated that 80% of all CVD mortality occurs (Perk et al., 2012).

For the first time we have a situation where more people are dying from causes related to how they live, that is their lifestyle, than from infectious disease or injury. In 1971, Omran applied the term "epidemiological transition" to describe this change. He proposed that there were three ages in relation to the primary causes of mortality: the *Age of Pestilence and Famine* followed by the *Age of Receding Pandemics* and then

finally the *Age of Degenerative and Man-Made Disease.* Modifications to Omran's theory have been suggested (Olshansky & Ault, 1986), however, it is well established that a clear transition has occurred over the past few decades from the primary cause of death being contagious diseases and injury to the current situation in the Western World where the primary cause of death is chronic diseases such as CVD, cancer and diabetes (Lopez & Mathers, 2006; Mackenbach, 1994; Magee, Henry, & Narayan, 2011).

The burgeoning rise in chronic disease has also become a major financial problem worldwide. In the United States it is estimated that the direct medical cost of overweight and obesity was $113.9 billion, in 2008 which representing upwards of 10% of the US health care spending (Tsai et al., 2011). The estimated cost of heart disease in the United States was in excess of $500 billion in 2010 as a result of health care costs and lost productivity (Allen, 2009; Esselstyn, 2010).The estimated healthcare related cost of CVD in the UK in 2004 was £17.4 billion representing 18% of the overall health expenditure in the UK for that year (Luengo-Fernández, Leal, Gray, Petersen, & Rayner, 2006). The global health expenditure on diabetes is estimated to have been $USD 376 billion in 2010 representing 12% of worldwide health expenditure (Zhang et al., 2010). This figure is projected to rise to $490 billion by 2030 (Zhang et al., 2010).

While the rise in the incidence of chronic diseases such as CVD and T2D has had a major impact on individuals and populations, there are a number of risk factors that are seen as precursors to these chronic diseases. A cluster of these abnormalities including obesity, dyslipidaemia, hypertension and hypoglycaemia has been labelled

the Metabolic Syndrome (MetS). The next section will review the development of MetS, followed by a discussion of the relevance, aetiology and treatment of Mets.

2.3 The Metabolic Syndrome

The Metabolic Syndrome (MetS) is the contemporary name given to a cluster of abnormalities that have increased substantially in modern times: obesity, hyperlipidaemia, hypertension and raised blood sugar (Eckel, Alberti, Grundy, & Zimmet, 2010). MetS is seen as a precursor to T2D and is associated with increased risk of CVD and stroke (Daskalopoulou, Athyros, Kolovou, Anagnostopoulou, & Mikhailidis, 2006).

There has been an evolution in both the naming of MetS and its defining characteristics. Historically, the condition has been labelled Syndrome X (Reaven, 1988), Reaven's Syndrome, Insulin Resistance Syndrome, Deadly Quartet, Dismetabolic Syndrome (Eckel et al., 2005) and the acronym CHAOS—Coronary artery disease, Hypertension, Atherosclerosis, Obesity, and Stroke (Gale, 1998). While the term 'Insulin Resistance Syndrome' is still used occasionally, 'Metabolic Syndrome' remains the most widely accepted label (K. G. Alberti et al., 2006) after being introduced to the Medical Subject Headings (MeSH) database in 2002.

MetS is an important concept in the present dissertation as it is used as the framework for evaluating the effectiveness of the lifestyle intervention studied. Accordingly, the evolution of the definition of MetS is presented in detail below.

2.4 Definitions of the Metabolic Syndrome

The Swedish physician Kylin first noted an association between hypertension, hyperglycaemia and gout in the 1920s (Levesque & Lamarche, 2008). In 1947, Vague noted that upper body adiposity was commonly associated with metabolic abnormalities found in T2D and CVD (Daskalopoulou et al., 2006). However, it was not until 1988 that Reaven published his Banting Medal Award Lecture where he described a condition that he referred to as 'Syndrome X' (Reaven, 1988). Reaven defined Syndrome X as a constellation of insulin resistance, hyperglycaemia, hypertension, low high-density lipoprotein cholesterol (HDL-C), increased very low density lipoprotein (VLDL) and raised triglyceride (TG) levels. Surprisingly, Reaven did not link obesity with Syndrome X (Alberti, Zimmet, & Shaw, 2005).

Since Reaven (1988), various groups and agencies have attempted to define and categorise MetS resulting in considerable discussion as to what should and should not be included in its definition. Clearly, the deliberations have been influenced by the different backgrounds of the groups involved in the discussion; groups with a primary focus on T2D have applied different emphases to groups with a primary interest on CVD.

The first official definition of MetS was published by the World Health Organisation (WHO) in 1998, as part of the document entitled "Definition Diagnosis and Classification of Diabetes Mellitus and Its Complications" (World Health Organisation, 1999). The report introduced MetS as a major classification and diagnostic tool to meet the therapeutic challenge of the person with hypertension, central (upper body) obesity, and dyslipidaemia, with or without hyperglycaemia.

While the WHO considered several other components of the MetS that had been proposed such as hyperuricaemia, coagulation disorders, and raised plasminogen activator inhibitor-1, they concluded that these were not necessary for the diagnosis of the condition. The WHO proposed definition of MetS is shown in Table 2-1. This definition required that insulin resistance be present for a diagnosis of MetS, plus any two of the other criteria.

The major limitation of the WHO definition was that it required measuring insulin resistance using the euglycaemic clamp method or oral glucose tolerance test (OGTT), as well as measurement of microalbuminuria, making it difficult to apply in clinical practice and epidemiological studies (Grundy, Brewer, Cleeman, Smith, & Lenfant, 2004). Another criticism of the WHO report was that it limited the ability to use the diagnosis for "pre-diabetic" or non-diabetic subjects (Daskalopoulou et al., 2006).

In the WHO definition of MetS normal levels for fasting plasma glucose (FPG) were defined as being less than 6.1 mmol/L which was a reduction from the formerly recommended level of 6.7mmol/L.

The WHO report, while having a major focus on diabetes and insulin abnormalities, noted the relationship between MetS and CVD (Beck-Nielsen, 1999). The WHO report suggested that insulin resistance was the underpinning aetiological factor for the individual components of MetS.

Table 2-1 Definitions of the Metabolic Syndrome

	World Health Organization(1999) (WHO)	European Group for the Study of Insulin Resistance (1999) (EGIR)	National Cholesterol Education Program Adult Treatment Panel III (2001) (ATPIII)	American College of Endocrinology / American Ass of Clinical Endocrinologists (2003) (ACE/AACE)	International Diabetes Federation (2006) (IDF)	Harmonized Definition (2009)
Criteria for Diagnosis	Insulin resistance (plus two other factors)	Insulin resistance or fasting hyperinsulinaemia (in non-diabetic people)	Three or more of the following five risk factors	No specific definition but relies on clinical judgement	Central obesity plus any two of the other factors or specific treatment for these factors	Three or more of the following five risk factors or specific treatment for these factors
Insulin Resistance	Glucose intolerance, IGT or diabetes and/or insulin resistance	Insulin resistance is defined as the top 25% of fasting insulin in the non-diabetic population				
Central Obesity	Waist-hip ratio Male >0.9 Female >0.85 and/or BMI >30kg/m^2	Waist circumference Male ≥0.94m Female ≥0.80m	Waist circumference Male ≥1.02m Female ≥0.88m		Waist circumference Ethnic Specific or a BMI of greater than 30 kg/m^2	Waist circumference Ethnic Specific
HDL*	Male ≤0.9 mmol/L Female ≤1 mmol/L	≤1 mmol/L	Male <1.0mmol/L Female<1.3mmol/L	Male <1.0mmol/L Female<1.3mmol/L	Male <1.0mmol/L Female<1.3mmol/L	Male <1.0mmol/L Female<1.3mmol/L
TG *	≥1.7 mmol/L	≥2.0 mmol/L	>1.7 mmol/L	>1.7 mmol/L	>1.7 mmol/L	>1.7 mmol/L
FPG		6.1mmol/L	≥6.1	6.1-6.9mmol/L	>5.6mmol/L	>5.6mmol/L
BP	Systolic>140mmHg Diastolic>90mmHg	Systolic>140mmHg Diastolic>90mmHg	Systolic>130mmHg Diastolic>85mmHg	Systolic>130mmHg Diastolic >85mmHg	Systolic>130mmHg Diastolic>85 mmHg	Systolic>130mmHg Diastolic>85 mmHg
Microalbuminuria	Urinary albumin excretion rate ≥ 20 µg/min or album in: creatinine ratio≥ 30 mg/g					
Reference	(World Health Organisation, 1999)	(Balkau & Charles, 1999)	(National Cholesterol Education Program, 2002)	(Grundy et al., 2004)	(K. G. Alberti et al., 2006)	(Alberti et al., 2009)

*Abnormal HDL and triglycerides were combined as one criteria, dyslipidaemia, in the WHO and EGIR definitions

The European Group for the Study of Insulin Resistance (EGIR) modified the WHO criteria in 1999. This report expanded the definition of MetS to include people who did not have diabetes but required hyperinsulinaemia to be present along with any two of the following: central obesity, dyslipidaemia, hypertension and/or raised fasting plasma glucose (FPG).

For the EGIR report waist circumference was used as a measure of central obesity as opposed to hip-waist ratio or body mass index (BMI). The cut-off level for triglycerides (TG) was raised to greater than 2.0 mmol/L and a fasting plasma glucose (FPG) greater than or equal to 6.1 mmol/L was included as an independent criteria (Balkau & Charles, 1999). Consistent with the WHO report, dyslipidaemia was defined by either low HDL or raised triglycerides and these two factors were not itemised as separate criteria, unlike later definitions of MetS (Table 2-1). As with the WHO report, the EGIR definition of MetS primarily focused on diabetes and its associated conditions.

In their third report the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) (ATP III), primarily focused on lowering blood cholesterol and thus their definition of MetS focused on CVD rather than diabetes (National Cholesterol Education Program, 2002). Further, while the WHO and EGIR definitions focused on international and European populations, the ATP III report justified each defining criteria by its prevalence in the American population (Daskalopoulou et al., 2006). The ATP III defined MetS as having at any three of abdominal obesity, atherogenic dyslipidaemia (elevated triglyceride (TG), small (low density lipoprotein (LDL) particles, low HDL cholesterol), raised blood pressure, insulin resistance (with or without glucose

intolerance) or prothrombotic and proinflammatory states (Table 2-1). Abdominal obesity was defined by waist circumference but more generous limits were applied than the EGIR criteria probably as a concession to the American norms, which caused some criticism.

The Adult Treatment Panel Third Report was welcomed for its use of Fasting Plasma Glucose (FPG) as a measure of the insulin resistant characteristic, as it provided a measurement that was relatively easy-to-use in a clinical setting. However, the inclusion of FPG in the definition of MetS received criticism due to a perception that there is a lack of clinical and experimental evidence supporting its inclusion (Kahn, Buse, Ferrannini, & Stern, 2005). The ATP III definition failed to consider patients who were undergoing specific treatment for hypertension or dyslipidaemia as having these components of the definition.

The American College of Endocrinology and American Association of Clinical Endocrinologist 2003 (ACE/AACE) definition of MetS (Grundy et al., 2004) used the same cut-off points as the ATP III definitions however, significantly, they modified the ATP III definition by including patients who were undergoing treatment for dyslipidaemia or hypertension but did not apply this criteria to abnormalities related to insulin resistance (Daskalopoulou et al., 2006). The ACE/AACE statement deliberately did not provide a specific definition of a syndrome but allowed the diagnosis to rely on clinical judgement (K. G. Alberti et al., 2006).

In 2006 the International Diabetes Federation (IDF) published a new MetS definition that aimed to be globally applicable for the identification of people at high risk of T2D and vascular events (Alberti, Zimmet, Shaw, & Grundy, 2006). The consensus group who prepared the International Diabetes Federation definition intended the

definition to be easy to apply in clinical practice by being based on clinical end-points and avoiding the need for measurements usually only available in research settings (K. G. Alberti et al., 2006). This definition attempted to incorporate the risk factors related to both CVD and T2D (Alberti et al., 2005). The International Diabetes Federation criteria for the clinic identification of MetS are shown in Table 2-1.

The International Diabetes Federation definition defined obesity by using ethnic specific waist circumference values as shown in Table 2-2. The ethnic specific cut-off values for waist circumference include values for a number of ethnic groups, but the tables do not cater well for some ethnic groups including the Polynesian or other Pacific Island communities. It also states that if BMI is >30 then central obesity can be assumed and waist circumference does not have to be taken. This definition required a waist girth of 94 cm for men and 80 cm for women (for Europids) which equates roughly to a BMI of 25 compared to the more liberal ATP III waist circumference measurements of 102 cm for men, and 85 cm for women which equates to a BMI of approximately 30.

Table 2-2 Ethnic specific values for waist circumference

Ethnic group	**Waist circumference (as measure of central obesity)**
Europids	
Men	≥ 94 cm
Women	≥ 80 cm
South Asian	
Men	≥ 90 cm
Women	≥ 80 cm
Chinese	
Men	≥ 90 cm
Women	≥ 80 cm
Japanese	
Men	≥ 90 cm
Women	≥ 80 cm
Ethnic South and Central Americans	Use European data until more specific data are available
Sub-Saharan Africans	Use European data until more specific data are available
Eastern Mediterranean and Middle East (Arab populations)	Use European data until more specific they are available

(K.G. Alberti et al., 2006)

The International Diabetes Federation definition differed from the ATP III definition in that it required central obesity as one of the three factors needed for a diagnosis of MetS criteria and it allowed treatment for dyslipidaemia, diabetes or hypertension to be used as a diagnostic factor for MetS.

Despite the publication of the International Diabetes Federation definition in 2006, controversy remained as to the definition of MetS. The ATP III (2001) and International Diabetes Federation (2006) definitions were the most commonly used, however the fact that the IDF definition made it mandatory to include a threshold value for waist circumference while the ATP III definition did not, presented a major difference. In 2009 an initiative of the International Diabetic Federation and the American Heart Association/National Heart, Lung and Blood Institute was joined by the World Heart

Federation, the International Atherosclerosis Society and the International Association for the Study of Obesity to develop a Harmonized Definition of MetS.

A report on this new definition was published in October 2009 (Alberti et al., 2009). One of the most significant characteristics of the consensus document was the removal of the mandate for obesity to be present, thus allowing the diagnosis of MetS to be used with non-obese patients who met three of the other criteria. The criteria for the Harmonized Definition are shown in Table 2-1.

A revised National Cholesterol Education Program Adult Treatment Panel (ATP IV) was expected to be published sometime in 2012 (Martin et al., 2012) which may include revisions to the definition of MetS. At the time of writing, this new definition has not yet been published. Unless otherwise noted, the Harmonized Definition of MetS is used in this dissertation.

2.4.1 Relevance of the Metabolic Syndrome

There is compelling evidence that MetS has reached epidemic proportions in the Western world. Using 1999-2004 National Health and Nutrition Examination Survey Data, the age adjusted prevalence of MetS in adults was 35% based on the ATP III (2001) definition and 39% using the IDF definition (Cornier et al., 2008). Alberti et al (2009) estimated the unadjusted prevalence of MetS in the US adult population 20 years and over, using the IDF definition, to be 42% in males and 38% in females.

The clinical relevance of MetS is that it identifies people who are at an increased long-term risk of CVD, stroke and T2D (Alberti et al., 2005; Chew, Gan, & Watts, 2006). All-cause mortality has been shown to be higher in individuals with MetS (Hu, Qiao, & Tuomilehto, 2004).

The American Diabetics Association and the European Association for the Study of Diabetes (ADA/EASD) have raised a number of concerns regarding MetS. These concerns include perceived ambiguity and incompleteness of criteria for MetS, the fact that the rationales for the criteria are ill-defined and that there is no clear basis for the inclusion or exclusion of other CVD risk factors. They question the value of including diabetes in the definition of MetS and whether insulin resistance can be identified as a unifying aetiology. They also speculate that the risk of CVD associated with MetS as a syndrome is no greater than the risk associated with the individual components of MetS and that the treatment of the syndrome "MetS" is no different than the treatment of the individual criteria. These concerns led them to conclude that the medical value of diagnosing the syndrome is unclear (Kahn et al., 2005).

Numerous studies have shown that the relative risk of diabetes is significantly higher for individuals with MetS. The relative risk of diabetes for those with MetS is 3.5 - 5.2 in the general population and as high as 33.6 if a population with zero abnormalities is used as reference (Bloomgarden, 2009; Ford, Li, & Sattar, 2008). Studies have also shown that patients diagnosed with MetS are at greater risk of developing CVD. The increase of risk of CVD ranges from 30 to 400%.(Kahn et al., 2005). Cornier et al (2008) concluded MetS approximately doubles CVD risk and the risk of T2D is more than five times higher for patients who have MetS than the general population that does not have MetS. It has been suggested that MetS accounts for the majority of CVD risk in the US population (Potenza & Mechanick, 2009). In addition MetS is associated with a number of other comorbidities such as non-alcoholic fatty liver disease, sleep disorders, reproductive tract disorders and microvascular disease (Cornier et al., 2008).

In response to the claim that the risk of CVD associated with MetS is no greater than the risk associated with the individual components of MetS, Grundy (2006) and others adamantly argue that the risk of CVD associated with the MetS is greater than the sum of the components (Alberti & Zimmet, 2006; Grundy, 2006b).

Some of the concerns raised above have been addressed by later definitions of MetS with the clarification of the criteria in the IDF and Harmonized Definition. The later definitions also allow the use of the diagnosis of MetS in non-diabetic patients.

While there is ongoing discussion as to whether there is a unifying aetiology for MetS, as discussed in the next section, Pladevall et al. (2006) concluded, using confirmatory factor analysis, that the concept of a single underlying factor is not only

plausible but best explains the observed correlations between the core components of MetS.

There is a growing consensus that MetS has value in determining CVD and diabetic risk (Eckel et al., 2010). While there continues to be ongoing discussions as to exactly what risk factors should be included in the definition of MetS, a number of groups support MetS as a valuable tool (Alberti et al., 2005). Even the American Diabetics Association and the European Association for the Study of Diabetes statement in Diabetes Care concluded by acknowledging MetS has been a useful paradigm (Kahn et al., 2005).

Overall, MetS appears to be a practical and useful means to identify individuals at increased risk of CVD and T2D (Alberti et al., 2009; Alberti & Zimmet, 2006).

2.4.2 Aetiology of the Metabolic Syndrome

When MetS was first proposed, insulin resistance was seen to be the root cause of the syndrome. Obesity, particularly central obesity, was viewed as a possible contributing factor. As mentioned earlier, Reaven (1988) did not include obesity in the original description of his "Syndrome X". However, over time, a shift from the traditional glucocentric understanding of MetS to an increasingly lipocentric viewpoint has occurred (Savage et al., 2005). This paradigm shift in the understanding of the causation of MetS is illustrated by two articles authored 12 years apart by the same authors. In 1998, Alberti and Zimmet, in their report for the WHO consultation, stated that "evidence is accumulating that insulin resistance could be the common aetiological factor for the individual components of the Metabolic Syndrome"(Alberti & Zimmet, 1998). In sharp

contrast, the same authors recently reported that "evidence now indicates that the metabolic syndrome all begins with excess central adiposity" (Eckel et al., 2010).

Grundy (2006a) suggested that MetS begins insidiously with abnormal obesity. Initially the increased risk is only marginal however as obesity increases and other exacerbating factors become involved the risk factors increase significantly. This can eventually leads to diabetic complications. Meanwhile atherogenesis is also taking place causing atherosclerotic cardiovascular disease. Grundy (2006b) explains that increasing age can also act as a precursor to abdominal obesity.

The pathophysiology of MetS has been summarised by Potenza and Mechanick (2009), with the undergirding of genetic predisposition, physical inactivity and an atherogenic western diet leading to increased central adiposity. The model proposes that visceral adipose tissue secretes inflammatory cytokines, adipokines and nonesterified fatty acids. These factors create insulin resistance at the level of skeletal muscles and the liver leading to hyperinsulinaemia that provokes atherogenesis, both directly and through compromised endothelial integrity. The increased blood fatty acid concentration also alters hepatic lipid production resulting in low high density lipoprotein (HDL), low-density library protein (LDL) changes and elevated triglycerides (TG).

A limitation of this model is that not all people with MetS are overweight or obese (St-Onge, Janssen, & Heymsfield, 2004), and not all overweight and obese individuals have MetS. This would suggest that MetS has a multifactorial aetiology, involving complex interactions between factors including genetics, hormones and nutrition. Further, there is evidence to suggest an epigenetic component to MetS. Bruce and Hanson (2010) suggest that maternal obesity during pregnancy and gestational diabetes can often result in foetal

overgrowth which can lead to increased MetS susceptibility in adulthood. However, they point out that while this epigenetic 'priming' may be present, it requires the appropriate stimulus, such as an atherogenic diet combined with a sedentary lifestyle, to trigger MetS in the susceptible adult.

Bays et al (2008) discussed a pathogenic form of fat which causes the symptoms of MetS. The pathogenic potential of adipose tissue appears to be effected by acquired and/or environmental factors. This pathogenic fat has been referred to as "sick fat" (Bays, 2009) and can affect individuals regardless of body weight. In the underweight patient with lipodystrophy there is a lack of adipose tissue that results in impaired adipose tissue functionality. Inadequate adipose tissue stores may compromise the storage of free fatty acid which results in increased circulating free fatty acids causing lipotoxicity. This lipotoxicity is characterised by the depositing of ectopic fat in muscle, the liver and the pancreas, all of which can contribute to T2D and MetS (Bays et al., 2008).

In the normal weight individual, during positive caloric balance where adipogenesis (Gregoire, Smas, & Sul, 1998; Roche, 1981) is impaired, the existing fat cells (adipocytes) must undergo hypertrophy (lipodystrophy), to store the excess energy (Gregoire, 2001). The hypertrophy of the adipocytes decreases their ability to function effectively leading to increased circulating levels of adiponectin and free fatty acids causing lipotoxicity.

Regardless of whether an individual is underweight or overweight, during times of positive caloric balance the excess energy may be stored through fat cell hypertrophy rather than adipogenesis, leading to pathologic adipose tissue responses and MetS (Bays et al., 2008).

2.4.3 Treatment of the Metabolic Syndrome

Arising from the above discussion, it appears that central obesity, exacerbated by sedentary lifestyle and the Western diet, are the primary cause of MetS (Hoerger & Ahmann, 2008; Pladevall et al., 2006). Accordingly, the treatment of choice for MetS involves lifestyle interventions targeting weight loss (Bays & Dujovne, 2006; Eckel et al., 2010; Horton, 2009; Pereira et al., 2009) with appropriate drug treatment only used to treat the "residual" CVD risk once lifestyle treatment has been utilised (Eckel et al., 2010).

Yet while lifestyle intervention is the preferred treatment strategy, pharmacological treatment of the individual abnormalities associated with MetS such as dyslipidaemia, hypertension and blood glucose abnormalities are mostly employed (Grundy, 2006a), despite being costly and often less effective than lifestyle intervention. For example, the projected Australian expenditure on statins, used to treat dyslipidaemia, for the period January 2009 to December 2019 exceeds $13 billion (Clarke & Fitzgerald, 2010). This presents an average cost in excess of one billion dollars per year and while substantial savings could be made by the use of generic statins, it is still represents an enormous economic burden. Furthermore, statin therapy does not eliminate cardiovascular risk (Nabel & Braunwald, 2012).

As well as being relatively expensive, pharmaceutical approaches for the treatment of MetS are often less effective than lifestyle modification. Ratner et al. (2005) found that in a three year follow up of MetS patients in the Diabetes Prevention Program, pharmacological treatment was 28% less effective for hypertension and 25% less effective for hyperlipidaemia than lifestyle intervention. The "Portfolio" diet that included cholesterol-lowering foods such as oatmeal, nuts, and soy was found to be as effective as

statins in the management of dyslipidaemia (D. J. Jenkins et al., 2003; Jenkins, Kendall, Marchie, Faulkner, Wong, et al., 2005). In reducing the absolute incidence of diabetes, lifestyle intervention was shown to "dominate" the pharmaceutical approach, with lifestyle intervention shown to be 20% more effective than a placebo in reducing the absolute incidence of diabetes compared to only 8% for Metformin (Herman et al., 2005). Lifestyle intervention was shown to reduce the incidence of MetS by 41% when compared to a placebo, while Metformin was shown to reduce the incidence of MetS by only 17% (Orchard et al., 2005).

Morbidity and mortality from just CVD can be reduced by at least 50% by healthy lifestyle change (Slavicek et al., 2008). Lifestyle intervention using either a Mediterranean-style diet or a low-fat diet have been shown to deliver significantly better overall health outcomes and cardiovascular-event-free survival after first myocardial infarction than usual care. Patients receiving dietary intervention had better primary-outcome-free survival after 24 months (85 of 101) than the usual-care control (61 of 101) ($p < 0.001$) (Tuttle et al., 2008). Lifestyle treatment has been shown to compare favourably with pharmaceutical treatment of hyperlipidaemia and hypertension (Liberopoulos, Tsouli, Mikhailidis, & Elisaf, 2006; Sacks & Katan, 2002). This was demonstrated in the Health Professionals Follow-up Study involving 42,847 men, leading to the researchers concluding "cardiovascular medication should be used as an adjunct, and not just a replacement for healthy lifestyle practices, especially in the setting of primary prevention"(Chiuve, McCullough, Sacks, & Rimm, 2006, p. 164).

Despite the clear benefits of lifestyle modification for the management of MetS, a perception persists, in the medical community, that it is easier to prescribe drugs than to

change dietary habits of patients and a belief that patients will not change their lifestyle habits. However several studies have shown that significant change in lifestyle patterns is achievable and sustainable long-term. In the Lyon Diet Heart Study, researchers found that several years after randomisation most of the patients in the experimental group were still closely following the recommended diet. They suggest that provided the instruction and supervision of the diet was delivered in a professional manner and that the new dietary habits were financially achievable and acceptable from a taste perspective, then long-term adherence to the new diet was achievable for most of the study participants (de Lorgeril et al., 1999). Esselstyn had three quarters of his experimental group stay with a very low fat, plant based diet for 20 years (Esselstyn, 2008). Furthermore, it is often overlooked that there are major problems with adherence to pharmaceutical treatments targeting the symptoms associated with MetS. Chapman et al., (2005) found that the percentage of patients adherent to both antihypertensive and lipid-lowering pharmacological therapy dropped rapidly following treatment initiation, with only 45% patients adherent at three months and 36% after six months.

A key reason why lifestyle interventions are preferable to pharmaceutical interventions for the treatment of chronic disease is that lifestyle interventions treat the underlying cause of the disease (Esselstyn, 1999; Ornish, 2002). As a consequence, lifestyle interventions address all of the symptoms of MetS as compared to medication that typically only treats its individual components. For example, statins can be used for treating dyslipidaemia but do not improve fasting plasma glucose levels, hypertension or central obesity. Finally, lifestyle interventions have less negative side effects than medications (Sattar et al., 2010).

A number of lifestyle modification programs have shown to successfully improve the risk factors associated with MetS. These include the Lyon diet (de Lorgeril et al., 1999), the Ornish Heart Disease Reversing Program (Aldana et al., 2003) and the Pritikin program (Barnard, 2007; Sullivan & Samuel, 2006).

After reviewing the evidence from the three EUROASPIRE patient surveys conducted across 22 countries in Europe, Kotseva et al. (2009, p. 929) conclude that “to salvage the acute ischaemic myocardium without addressing the underlying cause of the disease is futile; we need to invest in prevention.” Alberti and Zimmet (2006, p. 261) concluded that for the treatment of MetS "at present there is only one magic bullet, and that is lifestyle management." However very few patients in usual primary care settings are offered the opportunity to participate in lifestyle interventions (Shurney, Hyde, & Hulsey, 2012), despite the fact that the current management paradigm for MetS, dominated by a heavy reliance on medication and surgery, is unable to cope with the increased burden of chronic disease that has occurred since the epidemiological transition (Campbell & Campbell, 2006, p. 327). It is in this climate that Lifestyle Medicine has emerged.

Bariatric surgery has been used for the treatment of extremely obese patients and has been shown to have a positive impact on T2D (Schauer et al.) and to decrease overall mortality in severely obese patients (Kwok et al., 2014; Sjöström et al., 2007). However there seems to be significant variation in outcomes between the various types of bariatric surgery with gastric banding being less effective than other procedures (Inabnet, Winegar, Sherif, & Sarr). Bariatric surgery, alone, without intensive lifestyle intervention does not treat the underlying cause of the obesity and Mets and as a major surgical

procedure carries the associated risks of surgery (Arterburn, Johnson, Butler, Fisher, & Bayliss, 2014).

2.5 Lifestyle Medicine: an overview

2.5.1 Introduction

The first of the two research questions which forms the basis of this dissertation is: How effective is the Coronary Health Improvement Program (CHIP) lifestyle intervention for reducing the selected risk factors of chronic disease that constitute the Metabolic Syndrome, when delivered by volunteers to free-living participants in their community? In order to answer this question it is necessary to present an overview of the development of Lifestyle Medicine as a background to the CHIP intervention. Epidemiological studies that are foundational to Lifestyle Medicine which demonstrate the relationship between lifestyle and disease will be presented. This is followed by an exploration of the work of several pioneering researchers who attempted to use lifestyle interventions to treat some chronic diseases. Finally the limitations of Lifestyle Medicine are discussed.

It has been long speculated that the way that we live has an impact on our health. Almost 2500 years ago Hippocrates advocated, "Let food be thy medicine and medicine thy food" (Smith, 2004). Yet by the 19th century, "food as medicine" had fallen into obscurity (Lucock, 2004).

In the early to mid-19th century a number of "health reformers" postulated that diet could have a significant effect on health and behaviour. Sylvester Graham had concluded, by 1837, that wheat as a food could sustain American bodies and prevent "immoral behaviour", particularly masturbation and sexual excitement (Tompkins, 2009). In 1863 Ellen G White, taking a more holistic approach, proposed eight laws of health by asserting, "Pure air, sunlight, abstemiousness, rest, exercise, proper diet, the use of

water, trust in divine power – these are the true remedies" (White, 1905, p. 127). These have been developed into the acronym NEW START, representing nutrition, exercise, water, sunshine, temperance, air, rest and trust (Weimar, 2012). Inspired by White, John Harvey Kellogg, who with his brother Will Keith Kellogg invented the cornflakes breakfast cereal, established the Battle Creek Sanitarium in the mid 1800's and promoted a vegetarian diet, exercise and absence from alcohol and tobacco. Kellogg's clients included former president William Howard Taft, Amelia Earhart, Henry Ford and Thomas Edison among others (Bull & Lockhart, 2007, p. 12; Wikipedia, 2012).

2.5.2 Epidemiological studies

By the mid-20th century researchers examining the interaction between diet and health began to suspect a correlation between diet and heart disease, referred to as the diet-heart concept. One of the key leaders in this research, Ancel Keys, postulated that a "rich" diet, high in saturated fats, may contribute to higher plasma cholesterol levels and a tendency towards coronary health disease (Macini & Stamler, 2004). Keys first investigated this hypothesis in a group of 300 businessmen from Minnesota, aged 45 to 55 years, who were recruited in 1947 for a prospective study that involved annual follow-ups. Key observed that the incidence of CVD was higher among individuals with higher levels of relative weight, body fatness, blood pressure and serum cholesterol concentrations. Of the measures studied, serum cholesterol concentration showed the most significant association to CVD (Keys et al., 1963). Further studies by Keys and associates, involving controlled feeding protocols in male residents of psychiatric institutions, showed a clear correlation between dietary intake and blood lipid levels (Keys, Anderson, & Grande, 1959; Keys & Parlin, 1966).

As is the case with most novel postulates, the diet-heart hypothesis was met with subtle and not so subtle "agnosticism"(Macini & Stamler, 2004). At a World Health Organisation meeting in 1955 where Keys proposed the link between diet, cholesterol and heart disease, expecting to be accepted without argument, he was publicly challenged by Sir Gregory Pickering. Pickering is reputed to have said with disdain, "Yes, and Prof Keys, would you be kind enough to cite for us the principal piece of evidence that you think supports this diet-heart theory of yours?"(Ordovas, 2005, p. 919).

Partly as an attempt to answer some of the criticism of the diet-heart theory Keys, along with prominent investigators from other countries, initiated the "Seven Countries Study" which was designed to be a 5-year prospective study of 12,770 men aged 49 to 59 from Finland, Greece, Italy, Japan, the Netherlands, the United States and Yugoslavia. The findings of the study were first published as a supplement in *Circulation* in 1970 and clearly showed that the incidence of deaths due to coronary heart disease was related to the prevalence of hypertension, serum cholesterol values and dietary saturated fat intake (Aravanis, Corcondilas, Dontas, Lekos, & Keys, 1970; Buzina et al., 1970; Fidanza, Puddu, Imbimbo, Menotti, & Keys, 1970; Keys, 1970; Kimura & Keys, 1970).

As a result of these findings, Keys became impressed with the benefits of the "Mediterranean diet". The Mediterranean diet is mainly vegetarian and incorporates a variety of pasta, leaves sprinkled with olive oil, a wide variety of vegetables in season, fruit as the standard dessert, and wine in moderation (Keys, 1995). Together with his wife, Margaret, Keys published a book *Eat Well and Stay Well* in 1959 promoting the benefits of the Mediterranean diet for the prevention of CVD and general longevity. Research into the Mediterranean diet and its benefits in relation to CVD have continued

in the Lyon Diet Heart Study which continues to show the protective effects of this eating pattern (de Lorgeril et al., 1994; de Lorgeril et al., 1999). While the Mediterranean diet has its detractors, the fact that Keys lived to the age of 100 and his wife to the age of 97 tended to lend credence to their hypothesis (VanItallie, 2005; Wright, 2011).

Several other studies were initiated in the middle of the 20th Century that complimented the work of Keys. The landmark Framingham Study begun in 1948 as an ongoing cardiovascular study on the residents of Framingham, Massachusetts. The study involved 5,209 subjects and showed a clear correlation between diet, cholesterol levels and heart disease (Caroline, 2006; Castelli et al., 1986). The Ireland–Boston Diet–Heart Study, a 20 year mortality study tracking 1,001 middle-aged men of Irish descent living in Boston, also showed a correlation between diet and the development of coronary heart disease (Kushi et al., 1985). The results from the Framingham study were used to generate the Framingham Risk Score which is still used to predict an individual's chance of having a heart attack in the next 10 years (National Cholesterol Education Program, 2002; Wilson et al., 1998).

The Iowa Women's Health Study which has followed 34,492 women since 1986 (Cutler et al., 2008; Jacobs, Meyer, Kushi, & Folsom, 1999; Jacobs, Meyer, Kushi, & Folsom, 1998; Jacobs, Pereira, Meyer, & Kushi, 2000) and the Nurses' Health Study which followed 121,700 women from 1976 (Hu, Manson, et al., 2001; Hu et al., 2000; Hu, Stampfer, et al., 2001; Oh, Hu, Manson, Stampfer, & Willett, 2005; Stampfer, Hu, Manson, Rimm, & Willett, 2000) have shown a clear correlation between lifestyle choices such as diet, exercise and smoking and chronic diseases such as CVD, T2D and some cancers.

The Adventist Health Studies began in 1960 with the Adventist Mortality Study and followed by Adventist Health Study One (AHS-1) 1974 – 1988, collected data on 34,192 participants and indicated that Seventh-day Adventist's had lower risk from cancers, CVD and diabetes than the general population. In 2002 the second Adventist Health Study (AHS-2) commenced, enrolling over 90,000 participants. These studies showed the advantages of lifestyle choices such as not smoking, a plant-based diet and exercise as being protective from certain types of cancer, T2D and CVD (Butler et al., 2008; Fraser, 2005; Fraser, Lindsted, & Beeson, 1995; Fraser & Swannell, 1981; Rizzo, Sabate, Jaceldo-Siegl, & Fraser, 2011; Tonstad, Butler, Yan, & Fraser, 2009; Tonstad et al., 2011).

The Cornell China Study which was a comparative dietary and mortality study from 65 counties and 130 villages in rural mainland China found that the mortality rates from coronary artery disease and some cancers for both genders in rural China were very strongly correlated to diet. There was a positive association with the intake of animal protein and the frequency of meat intake for CVD and certain cancers, and an inverse association with plant protein, legume and light-coloured vegetable intake (Campbell, Parpia, & Chen, 1998).

It is interesting that despite the findings of these large epidemiological studies clearly demonstrating a connection between diet and disease, some critics remain of the diet-disease connection, describing it as "quackery" (Le Fanu, 2000, p. 322). However there is a general consensus that a strong correlation exists between lifestyle practices and the prevalence of chronic disease. Nestle (1999, p. 216) concluded, "What does seem clear is that diets based largely on plant foods are most associated with health and

longevity, at least under conditions of food abundance. Substantial and compelling evidence supports the idea that people in industrialised and industrialising economies could reduce the risk factors for chronic disease if they increased intake of fruits, vegetables and grains in proportion to animal foods." Similarly, Fraser and Shavlik (2001) observed that lifestyle choices such as a adopting a plant-based diet, exercising, not smoking and maintaining a healthy body weight could influence the expected age of death by as much as a decade, and that these healthy choices also equate to a better quality of life at an older age.

2.5.3 Interventional studies

Following the evidence of the early epidemiological studies which showed that lifestyle choices, particularly relating to diet, smoking and exercise had an impact on health, several researchers began, in the 1970s, to investigate the possibility of using lifestyle change in an attempt to treat the risk factors associated with chronic diseases. Investigations were undertaken at both the societal level and individual level.

At the societal level, the North Karelia Project in Finland was commenced in 1972 with the aim of reducing smoking, salt intake and saturated dairy fat intake, and increasing the intake of vegetables in order to target high blood cholesterol and high blood pressure levels. Results published at 5 years, 10 years, 20 years and 30 years show reductions in population smoking rates, serum cholesterol and blood pressure levels in both male and female populations. Between 1969 to 2006, this resulted in a 62% reduction in all-cause mortality, and a 79% reduction in CVD mortality and a 65% reduction in all cancer mortality (Puska, 2008; Puska et al., 1985; Puska et al., 1983; Salonen, Puska, & Mustaniemi, 1979).

Researchers also began to look at the effects of implementing lifestyle interventions in small groups of individuals. Three of the pioneers of these group-based interventions were Nathan Pritikin, Dean Ornish and Caldwell Esselstyn. The contribution of these individuals is detailed below, followed by an examination of some large interventional studies including the Dietary Approach to Stop Hypertension (DASH), the Diabetes Prevention Program (DPP), the Look AHEAD Trial and the Portfolio diet.

*2.5.3.1 **Pritikin***

In 1958, curiosity led Nathan Pritikin to Lester Morrison who had begun an experiment in the late 1940s in which he took 100 patients with diagnosed atherosclerosis and randomised 50 of the patients to a low fat, low cholesterol diet while the remaining patients acted as controls and were given no specific treatment. After 12 years, 19 of the 50 patients on the low fat, low cholesterol diet were still alive while all of the 50 control patients had died (Morrison, 1960). While visiting Morrison, Pritikin discovered that his own cholesterol levels were high (280 mg/dL) and a stress electrocardiogram resulted in a diagnosis of asymptomatic coronary insufficiency. He was advised, by expert cardiologists, not to exert himself and informed that there was nothing he could do about his elevated cholesterol levels (Hubbard, Inkeles, & Barnard, 1985; Withnell, 2003). Following this diagnosis, Pritikin adopted a diet high in complex carbohydrates and low in fat and cholesterol. Combined with daily vigorous physical exercise, the “Pritikin Diet” resulted in significant reductions in Pritikin’s blood cholesterol levels (155mg/dL) within six months and he experienced substantial improvement in his health (Hubbard et al., 1985).

In the early 70s, Pritikin in association with John Kern (Trowell, 1977) studied the "Pritikin Diet" in a six-month trial of 38 individuals suffering from severe peripheral vascular disease. The control group were given usual care treatment and ate a Western diet consisting of approximately 30% low fibre starch, 45% fat, 12% protein, 13% sugar and salt as desired. This group made no significant improvements in treadmill performance, lipid profile or arteriograms. Conversely, the experimental group, who exercised vigorously and ate a diet which consisted of 80% rich unrefined starchy foods, 10% protein and 10% fat, and was low in cholesterol with no added sugar, salt and fat but fruits and vegetables as desired, made statistically significant improvements in their lipid profile, treadmill performance and arteriograms (Trowell, 1977).

In 1976, Pritikin established the Pritikin Longevity Research Institute, in Santa Barbara, California. The Institute offered a 30-day residential lifestyle program incorporating the Pritikin diet and daily exercise. The Pritikin diet constituted less than 10% fat, 10 to 15% protein and 75 to 80% complex carbohydrates, with less than 25 mg of cholesterol and 4 g of sodium chloride per day and 10 to 20 g per day of crude fibre. Protein was derived from plant sources with the exception of non-fat milk and a small amount of fish or poultry which was served once a week (Barnard, Lattimore, Holly, Cherny, & Pritikin, 1982; Pritikin, 1984).

Participants at the Pritikin Longevity Research Institute experienced significant reductions in FPG, with a number of patients being able to come off oral hypoglycaemic medication and, in some cases, cease using insulin. Follow-up studies showed that patients were able to maintain the improvements in fasting plasma glucose levels 2 and 3 years after participating in the program (Barnard, Massey, Cherny, O'Brien, & Pritikin,

1983). Participants also experienced significant improvements in serum triglyceride and cholesterol levels (Reddy et al., 1988; Weber, Barnard, & Roy, 1983).

Pritikin died in 1985. An autopsy of his heart showed no sign of atherosclerosis despite the diagnosis of heart disease 30 years earlier (Hubbard et al., 1985). The Institute Pritikin established continued following his death, as the Pritikin Longevity Centre, and publications continue to show significant improvements in fasting plasma glucose levels, lipid profiles, blood pressure and weight loss (Barnard, 1990, 1991; Barnard, Jung, & Inkeles, 1994; Rosenthal et al., 1985). The research from the Pritikin Longevity Centre indicates that a residential lifestyle program can have a statistically significant effect on the risk factors associated with chronic disease as defined by MetS (Barnard, 2007; Barnard & Wen, 1994).

More recent studies from the Pritikin Longevity Centre suggest that the Pritikin diet and lifestyle can also slow the progression of prostate cancer (Barnard, 2007; Barnard, Aronson, Tymchuk, & Ngo, 2002; Barnard, Kobayashi, & Aronson, 2008).

The studies arising from the Pritikin Longevity Centre indicate that a low-fat, low-protein, high-carbohydrate plant-based diet can be effective in reducing the risk factors associated with MetS and possibly even reversing heart disease (Withnell, 2003). The limitations of the studies from the Pritikin Longevity Centre were that the results were achieved in a controlled environment, namely a residential setting, using health professional presenters. Hence the program is relatively expensive making it inaccessible to many. Further, the sample size of the studies were small.

2.5.3.2 *Ornish*

Dean Ornish conducted his first clinical study on reversing heart disease between his second and third year at medical school, in 1977, after becoming convinced from the medical and scientific literature that diet and lifestyle choices had a significant impact on the underlying causes of coronary heart disease. In this first study Ornish took 10 patients who were considered inoperable because of cardiac disease, housed them in a hotel, and for 30 days worked with them using exercise, meditation, group activities and a low-fat vegetarian diet consisting predominantly of fruits, vegetables, grains and legumes in their natural forms. Fat comprised around 10% of calories in the diet and salt, alcohol and caffeine were not used. The study achieved a 91% reduction in the incidence of angina and using thallium scans showed improvement in myocardial perfusion (Ornish, 2002).

In 1980, Ornish conducted a randomised controlled trial with 48 patients randomly assigned to either the intervention group or control. The intervention consisted of stress management training, meditation, exercise and a vegan diet excluding salt, alcohol and caffeine. The control group continued their normal routine. After 30 days, the intervention group increased their exercise tolerance by 40% compared to the control group. The intervention group showed a 20.5% reduction in total cholesterol (TC) while there was no change in the control group. Similarly, significant reductions in triglycerides (TG) were observed in the intervention group but not in the control. The frequency of angina decreased from 10.1 episodes per week to 0.9 by the end of the 30 days. Medication usage reduced significantly in the intervention group only, with a number of participants having to discontinue antihypertensive medication (Ornish et al., 1983).

In 1986, Ornish initiated the Lifestyle Heart Trial which used qualitative coronary arteriography and cardiac PET scans to examine the progression of heart disease. Twenty-eight symptomatic patients were assigned to an experimental group and were asked to eat a low-fat vegetarian diet for at least 12 months. The diet included fruits, vegetables, grains, legumes and soya bean products without caloric restriction. No animal products were allowed except for egg white and 1 cup of non-fat milk or yoghurt per day. Cholesterol intake was limited to 5 mg per day or less. Approximate 10% of the calories in this diet were derived from fat, 15 to 20% protein and 70 to 75% complex carbohydrates. Caffeine was eliminated and alcohol consumption was discouraged and if consumed limited to no more than two units per day. Vitamin B12 supplementation was given. As a control, 20 patients were assigned to a usual-care group. The program also included exercise and stress management components. Compliance to the diet, exercise and stress management components was high in the experimental group.

After 12 months the experimental group showed a significant regression in arterial stenosis from 40% to 37.8%, while the control group showed a progression of arterial stenosis from 42.7% to 46.1%. The experimental group also showed significant reductions in chest pain frequency and severity. The experimental group showed a 24.3% reduction in TC and a 37.4% reduction in low density lipoproteins cholesterol (LDL). High density lipoprotein cholesterol (HDL) levels did not change significantly in either group. A strong dose relationship was demonstrated, with those participants in the experimental group who showed the strongest compliance with the program achieving the greatest benefits (Ornish et al., 1990a, 1990b).

A follow-up to this study showed that after 5 years, the experimental group which continued to be treated with a low-fat plant-based diet, exercise, stress management and group support, had decreased stenosis and improved myocardial perfusion compared to increased stenosis and reduced perfusion in the control group treated with standard antianginal therapy. Interestingly, compliance to treatment was significantly higher in the experimental group than in the control group (Gould et al., 1995; Ornish et al., 1998).

Ornish established the Preventive Medicine Research Institute in Northern California and conducted the Multicentre Lifestyle Demonstration Project. For this study, 333 patients who had angiographically documented coronary artery disease severe enough to warrant revascularisation were selected to participate in the study. Patients were offered the option of undertaking extensive lifestyle modification or the revascularisation procedure. A total of 194 patients agreed to undergo the lifestyle modification program, forming the experimental group, with the remaining 139 patients who underwent the revascularisation procedure forming the matched-control group. Staff were trained at various centres to offer the lifestyle intervention to the patients in the experimental group, who then met three times per week for 12 weeks and then once a week for the remaining nine months of the year-long study. Most sessions were four hours in duration and consisted of exercise, stress management techniques, group support and a group meal. The experimental group and control group were tracked for three years despite the fact that the intervention was only conducted for 12 months.

After three years, patients in the experimental group experienced a significant decrease in LDL cholesterol levels (122.9 mg/dL to 101.7 mg/dL) and TC (202 mg/dL to 183.4 mg/dL). HDL cholesterol levels initially decreased from 36.7 mg/dL at baseline to

32.8 mg/dL after three months, but increased to 42.2 mg/dL at three years. Triglyceride (TG) levels initially increased insignificantly from 229.8 mg/dL at baseline to 235.7 mg/dL after three months, but reduced to 200.8 mg/dL after three years which was not significant ($p = 0.339$). Mean body weight decreased from 187.3 lb at baseline to 170 lb after three months ($p < 0.001$) and was 179.9 lb after three years which was still significantly less than baseline ($p = 0.007$).

Participants in the experimental group also showed significant improvement in psychosocial quality-of-life factors (Dansinger, Gleason, Griffith, Selker, & Schaefer, 2005; Koertge et al., 2003), depression scores (Pischke et al., 2010) and endothelial function (Dod et al., 2010). This study suggested that individuals affected by heart disease could avoid revascularisation procedures for at least three years by making comprehensive lifestyle changes. Further, the lifestyle intervention showed a cost saving, estimated conservatively to have been $30,000 per participant in the experimental group in the first year (Ornish, 1998a, 2009a). Subsequently, other researchers have shown the benefits of the Ornish program in Rockford, Illinois (Aldana et al., 2004; Aldana et al., 2003), and across 2974 participants from 24 different hospital sites (Silberman et al., 2010). A limitation of the Ornish program is that it is very time intensive for the participant, at least 3 four hour sessions per week over a 12 week period, and requires intensive input from professional facilitators.

Recent studies from the Preventive Medicine Research Institute have shown positive changes in prostate gene expression in men undergoing intensive lifestyle intervention (Dewell et al., 2007; Ornish, Magbanua, et al., 2008; Ornish et al., 2005) and even increased telomerase activity (Ornish, Lin, et al., 2008; Skordalakes, 2008).

Furthermore, these positive effects may cause beneficial changes in gene expression in a relatively short period of time (Ornish, 2009b).

Ornish is adamant that in order to achieve reversal of coronary artery disease, aggressive fat reductions to around 10% of calories is appropriate and necessary (Ornish, 2004; Ornish & Brown, 1993; Ornish & Denke, 1994). Indeed, several of his studies have shown a "dose-response" relationship to dietary fat, cholesterol intake and changes in arterial stenosis (Ornish, 1998b). Importantly, this diet has been found to be nutritionally adequate (Dunn-Emke et al., 2005).

Ornish has met with considerable opposition from the medical establishment throughout the course of his career. He recounts the story of someone saying to him at the end of a lecture in Texas "Hey, your research is really pioneering. You know how we can tell pioneers here in Texas? By the arrows in their back"(Ornish, 2002, p. 273). Encouragingly, the Ornish Program for Reversing Heart Disease was awarded Medicare claimability in the United States in 2012.

2.5.3.3 Esselstyn

Caldwell Esselstyn, a surgeon from the Cleveland Clinic in Ohio, was frustrated with simply treating the symptoms of heart disease and not the underlying cause of the disease (Esselstyn, 2008). He became convinced from examining the literature that heart disease could be arrested and even reversed by dietary intervention if total serum cholesterol levels were reduced to 150 mg/dL or below and that such reductions could be achieved by consuming a very low-fat plant based diet. Accordingly, in 1985 he enrolled a group of 22 participants who had angiographically documented evidence of severe coronary heart disease in a five-year study to test his hypothesis that reversal was

possible. The participants were asked to adhere to a diet where less than 10% of calories were derived from fat and to avoid all animal products except for skimmed milk and non-fat yoghurt. Participants were also asked, but not required, to moderate their consumption of alcohol and caffeine. Low dose statins were used for some participants. Daily food diaries were kept, by the participants, and reviewed by Esselstyn every two weeks, during a one-on-one consult with each participant. Additionally, all participants were invited to a group-based support session four times per year.

Follow-up angiograms conducted five years later showed a mean 7% reduction in stenosis. The mean TC level of the participants which was 246 mg/dL at baseline was 132.4 mg/dL after five years (range = 109.9 to 149.9 mg/dL), showing that all participants could achieve a TC under 150 mg/dL (Esselstyn, Ellis, Medendorp, & Crowe, 1995).

Five participants dropped out of the study and resumed their pre-study dietary habits. These five participants all reported new cardiac events. Of the participants who remained in the study, no new infarctions or clinical evidence of progression of atherosclerotic lesions occurred (Esselstyn et al., 1995).

After 12 years, 17 patients remained compliant to the eating pattern prescribed in the study and none showed progression of the disease, new coronary events, or required any interventional procedures which is remarkable considering that they had collectively experienced 49 coronary events in the eight years before the study commenced. The mean TC of the 17 participants after 12 years in the study was 145 mg/dL (Esselstyn, 1999).

A 20-year follow-up of this cohort showed continued adherence by the participants to the lifestyle and successful arresting of the cardiac disease (Esselstyn,

2008). Rather than being unwilling to make drastic lifestyle changes Esselstyn has found that the majority of patients "rejoice" once they have been given a comprehensive understanding of the cause of the disease and how to halt its progression (Esselstyn, 2010).

Esselstyn states emphatically that the conventional medical treatment of coronary heart disease is not effective or efficient: "Our stop-gap, device-driven approach isn't working because it doesn't address our toxic food environment which is responsible for our epidemic"(Esselstyn, 2001, p. 41). In an editorial, in the *American Journal of Cardiology*, Esselstyn likened the current medical and surgical treatment of coronary artery disease to the radical mastectomy. While admitting that in a few acute cases stents and coronary artery bypasses may be lifesaving, Esselstyn asserts that none of the current medical therapies treat the cause: the Western diet (Esselstyn, 2010).

While the outcomes of Esselstyn's research have shown that dietary changes may be able to reverse coronary artery disease, it is limited by the small sample size of only 17 participants. A further limitation is the requirement for intensive and time-consuming input from the caring physician. Esselstyn advocates a five hour initial patient interview followed by hourly interviews every two weeks.

2.5.3.4 Dietary Approach to Stop Hypertension (DASH)

The Dietary Approach to Stop Hypertension (DASH) examined the effectiveness, in 459 adults, of eight weeks on the "DASH" diet compared to a control. The DASH diet was rich in fruits, vegetables, and low-fat dairy products and with reduced saturated and total fat. The control diet that was low in fruits, vegetables and dairy products with a fat concentration typical of the average diet in the United States. Body weight and sodium

intake were maintained at a constant levels. Significant reductions were found in both SBP and DBP for those on the intervention diet compared to the control. The authors concluded that a diet rich in fruits, vegetables and low-fat dairy foods can substantially lower blood pressure (Appel et al., 1997).

The DASH intervention was further explored in the PREMIER clinical trial which randomly assigned 810 adults with elevated BP to three groups: an "advice only" control group, a treatment group that implemented the "established" traditional recommendations for losing weight, reducing sodium intake, increasing physical activity and limiting alcohol intake, or the "established plus DASH" group which involved the recommendations for the "established" group as well as the DASH diet. The trial found both the "established" and "established plus DASH" group to show significantly greater reductions in BP than the control group after six months, but the "established plus DASH" group achieved the best outcomes (Appel et al., 2003; Elmer et al., 2006). The DASH intervention has also been shown to be effective for weight loss (Hollis et al., 2008).

On the basis of these and other trials, the American Heart Association recommends dietary modification to treat hypertension (Appel et al., 2006).

2.5.3.5 Diabetes Prevention Program (DPP)

The Diabetes Prevention Program Research Group (DPP) conducted a large, randomised clinical trial involving adults who were at high risk of developing T2D to determine whether lifestyle intervention could delay the onset of T2D. In the study, 3234 individuals with elevated FPG who were still non-diabetic (i.e. pre-diabetes) were randomly assigned either to a placebo, treatment with Metformin, or a lifestyle-modification program with the goal of at least 7% weight loss and a minimum of 150

minutes of physical activity per week. The primary outcome measure was the incidence of individuals developing T2D. Lifestyle intervention was almost twice as effective as Metformin for preventing the development of established T2D with a 58% reduction in the incidence of T2D in the lifestyle intervention group compared to the placebo group (Knowler et al., 2002). The Metformin group had a 31% reduction in the development of T2D compared to the placebo control. The lifestyle intervention also significantly reduced risk factors of CVD including BP and TC, and increased HDL, when compared to the placebo or Metformin groups (Ratner et al., 2005). In a cost analysis of the lifestyle and Metformin interventions, lifestyle was said to "dominate", costing $1,100 per QALY gained as opposed to $31,300 per QALY for the Metformin intervention (Herman et al., 2005).

Following the initial findings of this study, 88% of the original participants enrolled for a long-term follow-up study. Due to the findings of the initial study, all participants were offered group-implemented lifestyle intervention. After 10 years, the incidence of diabetes was reduced by 34% in those participants initially randomised to the lifestyle intervention group and 18% in the group initially randomised to the Metformin group, when compared to the placebo group. This study indicated that the benefits of lifestyle intervention can persist for at least 10 years (Knowler et al., 2009).

The DPP-based curriculum has been used in numerous interventions including a number of community-based, professionally-delivered programs, and has shown significant weight loss and reduction in symptoms of MetS (Jackson, 2009). It has also been found to be effective in a pilot study involving a church-based delivery of the DPP curriculum in rural African-American churches (Davis-Smith et al., 2007).

2.5.3.6 Look AHEAD Trail

Look AHEAD (Action for Health and Diabetes) included 5,145 overweight or obese men and women with T2D, from 16 sites throughout the US, who were randomly assigned to either an Intensive Lifestyle Intervention or the usual care of Diabetes Support and Education. The Intensive Lifestyle Intervention group met weekly for six months and then three times per month for the next six months and were prescribed a calorie goal of 1,200-1,800 kcal/day with 30% of the energy from fat and a physical activity goal of 175 minutes/week of moderate-intensity activity (Wing et al., 2011).

After four years, the Intensive Lifestyle Intervention group had better overall levels of glycaemic control, BP, HDL and TG than did the control group, resulting in lowered CVD risk (Wing & Look, 2010). The Intensive Lifestyle Intervention group was associated with a greater remission of T2D (Gregg et al., 2012) and was also shown to have more improvement in erectile function than the control group (Wing et al., 2010). In the Intensive Lifestyle Intervention group attendance at appointments was better than 80% and significant weight loss was achieved at one year across all categories of overweight and obese participants (Unick et al., 2011).

Funding for this study was stopped by the National Institute of Health in October 2012 as no difference in the number of cardiovascular events was noted between the two groups after 11 years (Despres & Poirier, 2013). It has been observed that several confounding factors could have contributed to the lack of difference in cardiac events between the two groups including: exclusion from the trial of participants unable to perform a maximal graded exercise test, the higher level of lipid-lowering and antihypertensive medication used in the control group, and the emphasis on calorie

restriction in the Intensive Lifestyle Intervention group (Despres & Poirier, 2013). The Intensive Lifestyle Intervention diet consisted of 30% of calories from fat and there was no emphasis on fruit and vegetables which are quite different from that advocated by studies discussed earlier which have been shown to alleviate and reverse CVD. Despite these limitations this study still showed significant advantages in the risk factors for MetS for the Intensive Lifestyle Intervention group.

2.5.3.7 Portfolio Diet

The Portfolio Diet is a low-fat vegetarian diet with the addition of "portfolio" foods and nutrients purported to reduce blood cholesterol levels, including plant sterols, viscous fibre, soy protein and almonds. In one study, 46 individuals with elevated cholesterol levels, but otherwise healthy, were randomised into either: (1) a control diet which was a very low saturated fat diet, (2)the control diet plus statin medication, or (3) the control diet plus portfolio foods. Significant and comparable reductions were observed in LDL and C-reactive protein levels in both the group treated with statins and the portfolio diet (Jenkins, Kendall, Marchie, Faulkner, Josse, et al., 2005; D. J. Jenkins et al., 2003; Jenkins, Kendall, Marchie, Faulkner, Wong, et al., 2005). A larger study, involving 345 participants, showed the Portfolio Diet to be effective in reducing LDL when compared to a control at six-months follow-up (Braun, 2012; Jenkins et al., 2011).The Portfolio diet has also shown benefits for the treatment of T2D (David JA Jenkins et al., 2003).

2.5.3.8 Other studies

A number of smaller studies have also shown benefits of dietary and lifestyle interventions for the management of chronic diseases and MetS. These include the Daniel

Diet (Bloomer et al., 2010), the NORDIET study (Adamsson et al., 2011) and a study conducted by the Physicians Committee for Responsible Medicine (Barnard et al., 2009). The studies showed positive outcomes for a plant-based diet but were limited by small sample sizes.

The Daniel Diet, involved a 21-day plant-based diet devoid of animal products and preservatives but including fruits, vegetables, whole grains, legumes, nuts and seeds. The 43 subjects enrolled in this study showed a 98.7% compliance with the diet and significant reductions in TC, LDL, HDL, SBP and DBP (Bloomer et al., 2010). Significant improvements in antioxidant status and oxidative stress markers were also observed (Bloomer, Kabir, Trepanowski, Canale, & Farney, 2011). This study was also limited by the short duration of only 21 days.

The NORDIET study analysed the benefits of the more traditional Nordic eating pattern which was primarily plant-based with a low intake of meat products, dairy products, sweets, deserts and alcoholic beverages, compared to the current Scandinavian diet (Adamsson et al., 2012). A randomised controlled trial of 88 mildly hypercholesterolaemic participants were assigned to either a typical Scandinavian diet or the NORDIET. Those participants on the NORDIET showed improved lipid profile, insulin sensitively and BP (Adamsson et al., 2011).

In a study that randomly assigned 99 participants to either an experimental group (n =49) assigned to total plant-based diet consisting of fruits, vegetables, grains and legumes with unrestricted portion size and energy intake (approximately 10% of energy from fat) and compared them with a control group (n = 50) assigned to the diet recommended by the 2003 American Diabetes Association, individuals consuming the

plant-based diet showed greater reductions in LDL, TC, and HbA1c through 74 weeks. When controlled for weight loss, the plant-based diet improved blood sugar control and plasma lipids more than the conventional diet (Barnard et al., 2009). The 74 weeks length of the study showed good long-term outcomes however it is limited by the relatively small sample size.

2.5.3.9 Exercise interventions

Most of the above studies have focused primarily on diet, however, the second pillar of Lifestyle Medicine is exercise and numerous studies have shown that exercise is an important component of lifestyle interventions for the management of chronic diseases and MetS. It is well established that improving cardiovascular fitness is an essential component of a lifestyle intervention targeting T2D and BP (Totsikas et al., 2011), reversing risk factors of MetS (Tjonna et al., 2008) and chronic inflammation (Mathur & Pedersen, 2008).

While trials focusing on exercise are not as common as those focused on overall lifestyle modification, there are several studies that show the benefits of exercise in isolation. A Canadian study randomly assigned a group of 216 participants with T2D but not on insulin therapy, to one of four groups: aerobic training, resistance training, combined aerobics and resistance training or a control group. Both aerobic and resistance training improved glycaemic control, however, the greatest improvements were achieved when both aerobic and resistance training were combined (Sigal et al., 2007). A Finnish study which randomised 56 men with type I diabetes into an exercise program or control found that exercise training improved lipid profiles, especially HDL/LDL ratios (Laaksonen et al., 2000). A meta-analysis of 12 studies looking at the effect of exercise found that

aerobic exercise when combined with dietary intervention had a beneficial effect on the lipid profile of overweight and obese adults for TC, LDL, TC/HDL ratio and TG but not HDL, but was unable to identify the independent effect of exercise or diet on the lipid profile (Kelley, Kelley, Roberts, & Haskell, 2012).

2.5.3.10 Definition of Lifestyle Medicine

The epidemiological studies reviewed earlier indicated that there was a link between lifestyle, MetS and chronic diseases such as T2D, CVD and some cancers. The interventional studies reviewed in this section show that for many individuals, it is possible to halt the progression of, and in some cases reverse the effects of, these diseases using aggressive lifestyle modification therapy. These studies, among others, have lead to the emergence of a discipline referred to as Lifestyle Medicine (Egger, Binns, & Rossner, 2009). It is a growing discipline that is now represented by the American College of Lifestyle Medicine (ACLM, 2013), the European Society of Lifestyle Medicine (ESLM, 2014) and the Australian Lifestyle Medicine Association (ALMA, 2013). Lifestyle Medicine has been defined as:

"The application of environmental, behaviour, medical, and motivational principles to the management of lifestyle-related health problem in a clinical setting … including self-care and self-management." (Egger et al., 2011) or simple as "the use of lifestyle interventions in the treatment and management of disease." (ACLM, 2013).

2.5.4 Limitations of Lifestyle Medicine

While we have seen that lifestyle can be effective in combating Mets and associated chronic diseases, it should be noted that there are some limitations with

Lifestyle Medicine. These limitations include environmental issues, study design issues, measurement issues and the wide variety of recommended interventions.

Lifestyle interventions tend to focus on the individual. The majority of lifestyle interventions attempt to get an individual to make significant changes in their lifestyle. While encouraging individuals to take responsibility for their own health can be seen as a positive move the downside of focusing on the individual is that for those individuals who for whatever reason struggle to comply with the intervention there can be a tendency to "shame and blame" (Larsen, 2012). Focusing on the individual rather than the environment and focusing on the individual level devoid of social context can in some cases lead to an ideology of individual responsibility and victim blaming (Korp, 2010).

While individual lifestyle adaption is important there also needs to be a societal approach incorporating multiple segments of society for intervention including the transport and infrastructure sector, the media and the food sector (Swinburn & Egger, 2002). Walls, Peeters, Proietto, and McNeil (2011) suggest that it is the obesogenic environment that is the primary cause of the exploding epidemic of obesity and this environment is best treated by enacting high-level policy and legislative changes to alter the environment rather than targeting individuals in lifestyle modification programs.

Lifestyle interventions do not lend themselves well to randomised controlled trials (RCT) because lifestyle interventions usually affects several diseases and the people who undergo lifestyle interventions also have an impact on their immediate environment as a result of undergoing the intervention which makes it difficult to measure exactly what has changed (Rosén, 1989). Also, given that there is no placebo for lifestyle, it is not possible to use blind or double-blind methodology in RCTs of Lifestyle Medicine (Tarasuk &

Brooker, 1997). When randomisation is attempted in studying lifestyle interventions it is usually done by randomly assigning participants to either a control group or to a lifestyle modification group however due to the prominence given to “healthy choices” promoted in the media it is not uncommon for members of the control group to make lifestyle changes independent of the research project which adds complications to the study (Merrill, Aldana, Greenlaw, Diehl, et al., 2008).

In evaluating lifestyle interventions obtaining accurate measures of compliance can be difficult. While some of the earlier researchers in lifestyle interventions used captive populations, such as psychiatric institutions (Keys et al., 1959), where lifestyle variables could be more easily controlled such studies are no longer considered ethical. In a free living populations it is “notoriously” difficult to accurately measure dietary intake (Tarasuk & Brooker, 1997). This makes it very difficult to accurately measure compliance and also to identify exactly what is responsible for the changes that may have taken place.

Another limitation in the analysis of Lifestyle Medicine interventions is the variability of the lifestyle interventions published in the literature (Yamaoka & Tango, 2012). There is still a lot of discussion as to what is considered to be appropriate lifestyle intervention and the information which the public receives is often incomplete and confusing (Weinberg, 2004; Yancy, Westman, French, & Califf, 2003).

2.6 The Coronary Health Improvement Program (CHIP)

While the epidemiological and interventional studies looking at Lifestyle Medicine showed promise, the evidence for the effectiveness of community-based lifestyle interventions, on the other hand, was initially disappointing. A meta-analysis of community-based lifestyle intervention programs delivered to free-living individuals concluded that these programs were only modestly effective, reducing cholesterol levels by about 3% (Tang et al., 1998). The editors of the *British Medical Journal* concluded that general population health education programs were of limited effectiveness in impacting lipid profile (Smith & Ebrahim, 1998). However, in the same year, Hans Diehl (1998) published a paper in *the American Journal of Cardiology* which showed significantly greater reductions in cholesterol levels and the risk factors of MetS through a community-based lifestyle intervention than those published in the *British Medical Journal* meta-analysis. This was the first peer-reviewed article relating to the Coronary Health Improvement Program (CHIP).

2.6.1 The Coronary Health Improvement Program (CHIP): a description

The CHIP program was developed in 1986 by Hans Diehl, after working for a period as the research director at the Pritikin Longevity Centre (Gidley, 2008). Diehl was impressed by the outcomes achieved by participants in the residential programs at the Pritikin Longevity Centre but recognised that the expense of the program was prohibitive to many individuals. Further, he observed that the lifestyle prescriptions the participants practiced in the residential program were more likely to be difficult to sustain when the participants returned to their home environment. In response, Diehl developed CHIP as a

community-based lifestyle intervention based on the lifestyle principles promoted at the Pritikin Longevity Centre.

CHIP was developed as a 30-day lifestyle intervention that encouraged participants to move towards what is referred to as the "Optimal Diet", engage in at least 30 minutes a day of aerobic exercise, and reduce stress. The Optimal Diet is defined as a whole-food plant-based diet, emphasising fruits, grains, legumes and vegetables *ad libitum,* with little or no animal products. This eating pattern recommended that no more than 15% of calories be derived from fat, and a daily intake of less than 10 teaspoons of added sugar, 5 g of salt and 15 mg of cholesterol. The consumption of 2 to 2.5 L of water each day was also recommended (Englert, Diehl, & Greenlaw, 2004).

The primary goals of CHIP were to substantially improve blood lipid levels, blood pressure and blood sugar levels. Secondary goals were to decrease weight, eliminate smoking, enhance daily exercise, improve stress coping strategies and decrease medication used for hypertension, diabetes and heart disease (Diehl, 1998).

The program delivery was in 16 two hour sessions over a four or five week period, and focused on developing intelligent self-care through a clearer understanding of the nature and aetiology of CVD and T2D, their epidemiology and risk factors (Englert et al., 2004).

CHIP incorporated accountability measures with a "health screen" conducted at the beginning and at the end of the intervention. The health screen included measurement of height, weight, BP, lipid profile and FPG. The results of the health screen were used to motivate participants to maintain lifestyle changes and to improve health-related self-efficacy through the intervention. Following the completion of the initial

intervention participants were encouraged to join a "CHIP Alumni" which meet on a monthly basis to provide ongoing support for the lifestyle changes initiated during the intervention.

The first program was conducted in British Columbia, Canada in 1988. This program consisted of a risk assessment of factors pertaining to MetS which included a brief medical history, BP, height, weight, food frequency and fasting blood drawn to measure TC, LDL, HDL, TG and FPG. The program involved participants meeting four nights a week for four weeks. Following the four-week program a further health appraisal was conducted. The final health appraisal showed a decrease in BP, resting heart rate, TC, LDL, TG, FPG and body weight (Gidley, 2008).

Following the success of the initial program numerous other programs were conducted in North America. In 1997 the program series was videotaped in front of a live audience at the Borgess Medical Center, Kalamazoo, Michigan, USA. A curriculum package was then built to support these recordings (Gidley, 2008). The results of CHIP programs in Kalamazoo, Michigan were published by Hans Diehl in 1998 in the *American Journal of Cardiology* (Diehl, 1998).

In 1999 CHIP was established in Rockford, Illinois at the invitation of Dr. Roger Greenlaw, Medical Director of the Centre for Contemporary Medicine attached to the Swedish-American Hospital, after he read Diehls' paper in the *American Journal of Cardiology*. Rockford has since become the business centre for CHIP (Diehl, 2003; Gidley, 2008) where the program is delivered by health professionals. There have been a number of articles published on the results obtained from the Rockford programs (Aldana, 2001; Aldana et al., 2002; Aldana et al., 2008; Aldana, Greenlaw, Diehl, Salberg, Merrill, &

Ohmine, 2005; Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Aldana, Greenlaw, et al., 2006; Englert, Dieh, Greenlaw, & Aldana, 2012; Englert et al., 2004; Englert et al., 2007; Gidley, 2008; Merrill & Aldana, 2008, 2009; Merrill, Aldana, Greenlaw, & Diehl, 2008; Merrill, Aldana, Greenlaw, Diehl, & Salberg, 2007; Merrill, Aldana, Greenlaw, Diehl, et al., 2008; Merrill, Massey, et al., 2008; Merrill, Taylor, & Aldana, 2008; Thieszen et al., 2011).

Subsequent to the success of the professional delivery of CHIP, Hans Diehl further developed CHIP so that it could be delivered by volunteers who were non-health professionals, to members of their local community, outside the confines of a recognised medical establishment. The program was made available to volunteer directors by supplying them with the recorded presentations on DVD, curriculum material for participants and with 2 days of training. To date approximately 50,000 people have completed the CHIP program in their communities under the guidance of volunteer directors. The researcher has been unable to find published research that has reported on the effectiveness of these volunteer directed programs.

CHIP has been delivered using paid professional facilitators and using community volunteers as facilitators. In a number of cases the volunteer facilitators delivering CHIP were health professionals however as volunteers they donated their time to facilitate the CHIP programs. The literature published to date examining CHIP is based on programs using paid health professionals as facilitators. The purpose of this dissertation is to look at the effectiveness of CHIP interventions delivered by volunteers. In this dissertation volunteer means that individual is not receiving payment or remuneration remuneration and donated their time when facilitating the CHIP programs.

2.6.2 Coronary Health Improvement Program publications

To date there have been 17 peer review articles published examining the effectiveness of the CHIP lifestyle intervention when delivered by professional facilitators. The first paper, mentioned above, arose from data collected by Hans Diehl in a CHIP intervention he personally presented (Diehl, 1998). Another 15 papers examined the effectiveness of CHIP using professional facilitators in clinical, workplace or community settings in Rockford, Illinois. In 2012, a paper was published documenting the effectiveness of CHIP in treating patients with T2D in a workplace setting (Shurney et al., 2012). An overview of these papers is presented below.

2.6.2.1 Diehl

In 1998 Hans Diehl published an article in the *American Journal of Cardiology* presenting the results from his CHIP program (Diehl, 1998). This article reports on a hospital-based educational program (n=288) aimed at substantially decreasing the risk of coronary artery disease and associated diseases such as T2D, essential hypertension and obesity through appropriate lifestyle change, delivered in Kalamazoo, Michigan, USA.

The pre-and post-health screen showed highly significant changes in lipid screen, blood pressure, weight and body mass index except for total-to-HDL cholesterol ratio and triglyceride levels in women as shown in Table 2-3.

Table 2-3 Change, by gender, in coronary disease risk factors over four weeks in CHIP participants from Diehl 1998

	Means													
	Men (n = 123)							Women(n = 165)						
	Before	SD	After	SD	Change	SD	% Change	Before	SD	After	SD	Change	SD	% Change
Age	54.9	11						54.6						
Weight (lb)	202.5	38	195.7	35	-6.80	3	-3.4	172.1	43	166.9	41	-5.2	4	-3.0
BMI (kg/cm^2)	29.4	5	28.4	5	-1.00	1	-3.4	29.8	7	28.9	7	-0.9	0	-3.0
SBP (mmHg)	132.1	17	127.5	17	-4.60	16	-3.4	130.5	21	122.1	19	-8.3	14	-6.4
DBP (mmHv)	83.7	10	77.8	9	-6.00	8	-7.1	78.7	10	73.2	9	-5.5	9	-7.0
TC (mg/dL)	222.2	42	180.9	31	-41.30	33	-18.6	227.3	42	205.8	40	-21.6	28	-9.5
HDL (mg/dL)	35.9	9	32	8	-3.80	4	-10.6	48.9	12	42.3	11	-6.6	7	-13.5
TC /HDL	6.5	2	5.9	1	-0.60	1	-9.2	5	2	5.2	2	0.2	1	4.0
LDL (mg/dL)	149.8	34	116.1	28	-31.90	26	-21.3	147.2	38	131.1	34	-16.3	26	-11.1
TG (mg/dL)	190.5	116	163.2	84	-27.20	94	-14.3	159.4	87	164.1	101	4.8	77	3.0
FPG (mg/dL)	111.3	34	99.5	23	-11.80	2	-10.6	104.3	42	97.7	45	-6.7	26	-6.4

P <0.001 for all change except TG for women and total TC/HDL for women

(Diehl, 1998)

Diehl (1998) also stratified the participants according to their risk level at program entry. Table 2-4 shows the change in lipid profile and Table 2-5 show the change in FPG. In general, participants who were at the greatest risk experienced the greatest improvements. While triglycerides did not show a significant change in the aggregated data, participants with triglyceride levels greater than 200 mg/dL, particularly men, experienced significant reductions. Diehl (1998) identified that these reductions in triglyceride levels were not consistent with other studies that had shown increases in triglyceride levels with a very low-fat, high-carbohydrate diet. Some participants were able to decrease or discontinue antidiabetic, hypolipidaemic and antihypertensive medication as a result of the intervention.

Table 2-4 Changes in serum lipid levels degree of risk Diehl 1998

Change in Serum Lipid Levels by Degree of Risk and Gender in Chip Participants		
	Change (%)	
	Men	Women
Total cholesterol (mg/dL)		
<200	-11	-7
200 - 239	-17	-9
240 - 279	-21	-11
LDL cholesterol (mg/dL)%		
<100	-19	-6
100 - 129	-15	-6
130-159	-17	-9
160-189	-22	-14
Total/HDL cholesterol		
4.0-5.9	+2*	ND
6.0-7.9	-11	
>7.9	-15	
Triglycerides (mg/dL)		
<100	+21	+25
100-199	0*	+9
200-299	-18	-11
300-399	-25	-14*
400-599	-39	-20*
* p<0.05 change if not marked with asterisk		
ND = No Data		(Diehl, 1998)

Table 2-5 Changes in FPG levels degree of risk Diehl 1998

Change in Fasting Plasma Glucose Levels by Baseline Degree of Risk for Participants not Taking Antidiabetic Agents				
Category	n	Baseline	After	%Change
<101 (mg/dL)	64	91	88	-3
101-110(mg/dL)	59	105	97	-8
111-139(mg/dL)	25	116	98	-18
≥140*(mg/dL)	19	135	117	-18
* or by history of Diabetes				
p<0.001 for all changes				
				(Diehl, 1998)

The study demonstrated that measurable clinical improvements could occur within 30 days through a community-based lifestyle intervention at a relatively low cost compared to residential programs.

Diehl acknowledged several limitations of the study including its short duration and lack of a control group, and urged for a randomised control trial to further explore the short and longer term effectiveness of the CHIP intervention.

2.6.2.2 Rockford based studies

Following the publication of Diehl's 1998 article in the *American Journal of Cardiology*, Roger Greenlaw introduced CHIP to Rockford, Illinois. This project, working with the Swedish American Health System, aimed to enrol, within a seven-year period 7000 residents over the age of 40 years which represented 10% of the Rockford population, in a CHIP intervention (Englert et al., 2004). To date, 15 papers have been published in peer-reviewed journals examining different cohorts from the Rockford project. Some of the interventions were delivered in workplace settings and others in the community. In all cases the programs were delivered by professional facilitators.

Seven of the papers focused on the same cohort of 348 participants who were randomised to a treatment group who commenced a CHIP intervention immediately or a delay-control group who were stalled for six months before receiving the intervention. In all cases the intervention groups were shown to achieve significantly better outcomes than the control group (Aldana, Greenlaw, Diehl, Salberg, Merrill, & Ohmine, 2005; Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Aldana, Greenlaw, et al., 2006).

Of the Rockford papers, seven provided an analysis of changes in biometric data from baseline to post-intervention (Aldana et al., 2002; Aldana et al., 2008; Aldana, Greenlaw, Diehl, Salberg, Merrill, & Ohmine, 2005; Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Englert et al., 2004; Englert et al., 2007; Merrill & Aldana, 2008). A summary of these analyses is shown in Table 2-6. Several of these publications observed that the reductions in the biometrics were among the largest reported in the literature for a community-based lifestyle intervention (Aldana et al., 2002); (Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Aldana, Greenlaw, et al., 2006).

Significant improvements were observed in all biometrics, except for triglycerides in three of the papers (Aldana et al., 2008; Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Englert et al., 2007). These studies showed that participants in the highest risk category at baseline achieved the greatest results (Aldana et al., 2002; Aldana et al., 2008; Englert et al., 2004; Englert et al., 2007). Further, males were observed to achieve significantly better outcomes through the program than women in all biometric measures except for fasting plasma glucose (Aldana et al., 2002), a pattern also observed by Diehl (1998). Gender differences were particularly noteworthy for weight reduction, TC and TG. Several studies also reported significant reductions in medication usage (Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Diehl, 1998) by the end of the CHIP intervention. Merrill, Massey, et al. (2008) found that CHIP did not have a significant impact on C-reactive protein levels.

One initially concerning observation of these studies was a significant drop in HDL levels. While high levels of HDL are considered cardioprotective (Navab et al., 2006) , it was suggested that low HDL levels are only a problem in the context of high TC and LDL

levels, as typically found in Western society. In societies where TC and LDL levels are low, such as the Tarahumara Indians in Mexico, HDL levels are very low but there is virtually no CVD (Englert et al., 2004). Hence, it was asserted in these studies that the lowered HDL observed as a result of the CHIP intervention was not of concern due to the greater reduction in TC and LDL. The slight increase in TG observed in those participants with low baseline TG, was also not considered clinically significant (Diehl, 1998; Englert et al., 2004).

A number of the papers showed that the CHIP interventions had a positive impact on sleep, stress disorders, depression and mental health (Merrill, Aldana, Greenlaw, & Diehl, 2008; Merrill, Taylor, et al., 2008; Thieszen et al., 2011). It was also found that overall health and functional status improved for participants in the CHIP intervention (Merrill & Aldana, 2009).

Several studies followed the CHIP participants beyond the 30-day intervention. A 6 month follow up of 348 participants showed that continued improvement in weight, BMI, systolic and diastolic blood pressures and fasting plasma glucose levels were recorded after six months, but there was decay in the lipid measures (Aldana, Greenlaw, et al., 2006). An 18 month follow-up of this cohort showed that improvements in nutrition and physical activity health behaviours were still significant at 18 months although there was some decay following the end of the intervention (Merrill, Aldana, Greenlaw, Diehl, et al., 2008). A 12 month follow-up, of 1,712 participants, showed significant improvement for TC, HDL, LDL, TG, FPG, BMI and BP after 4 weeks and 12 month however 49% of the participants were lost to follow-up at 12 months (Merrill & Aldana, 2008).

Table 2-6 Summary of percentage mean change baseline vs post-intervention for various CHIP programs reported in literature

Percentage Change in Biometrics

Reference	Diehl 1998		Aldana 2002	Englert 2004		Adana 2005 A	Adana 2005 B	Englert 2007		Aldana 2008	Merrill 2008	
Gender	Male	Female	Mixed	Male	Female	Mixed	Mixed	Male	Female	Mixed	*	
n	123	165	442	88	164	64	167	400	649	714	862	841
Age	54.9	54.6	52.1	57	52	46.1	50.4	57.00	52.00	ND	55.2	55.2
Weight	-3.36	-3.02	-4.43	-3.79	-3.62	-3.25	-3.67	-4.42	-3.59	-4.01	ND	ND
BMI	3.40	-3.02	-4.38	-3.96	-3.72	-3.43	ND	-3.13	-3.03	-3.91	-3.85	-3.44
BP systolic	-3.48	-6.36	-5.41	-2.91	-2.14	-5.69	-5.56	-6.38	-5.67	-4.76	-4.64	-4.83
BP diastolic	-7.17	-6.99	-6.02	-3.63	-2.71	-6.31	-6.42	-5.81	-5.95	-4.02	-4.44	-5.08
Total cholesterol	-18.59	-9.50	-13.20	-14.27	-7.96	-8.02	-7.52	-16.00	-9.30	-10.89	-11.63	-10.19
HDL	-10.58	-13.50	-13.31	-10.20	-10.38	-6.77	-7.54	ND	ND	-14.19	-11.84	-11.81
LDL	-21.30	-11.07	-13.66	-15.66	-7.97	-9.58	-9.28	-18.55	-9.38	-12.22	-12.03	-10.9
Triglycerides	-14.28	3.01	-9.76	-22.37	-4.67	-2.53	2.63	-10.39	-1.96	-7.39	-9.27	-7.434
Glucose	-10.60	-6.42	-5.06	-8.53	-2.33	-4.00	-3.99	-7.02	-4.55	-6.52	-4.40	-5.301

* Merrill gives data for two groups: the first group is those who were followed up after 12 months the second group is those who were lost to follow-up 12 months
All figures are data after four weeks

Diehl 1998 $p < 0.001$ except for female triglycerides and female Cholesterol/HDL ratio
Merrill 2008 $p < 0.0001$ for all factors
Aldana 2005 A $p < 0.0001$ for weight and BMI, $p < 0.02$ for systolic and diastolic BP, $p < 0.005$ for total cholesterol HDL LDL and $p < 0.05$ for triglycerides and FPG
Aldan 2005 B $p < 0.0001$ for all factors except for FPG = 0.003 and triglycerides 0.9513
Aldana 2008 $p < 0.001$ except for triglycerides=0.0284
Englert 2004 $p<0.05$ for Female blood pressures and female triglycerides, $p<0.01$ for male and female glucose and female HDL, all other factors $p<0.001$
Englert 2007 $p<0.0001$ except for female triglycerides where $p=0.113$

2.6.2.3 *Other CHIP publications*

Recently, Shurney et al. (2012) enrolled 28 employees of Vanderbilt University with diagnosed T2D in a CHIP intervention delivered by health professionals. Participants' biometrics and health care expenditure were tracked for 26 weeks. Positive changes were observed in HBA1C, TC, LDL, HDL and TG. Approximately one quarter of the participants were able to eliminate one or more of their medications by the 26 week follow-up. Significant cost reductions were reported in the study with a total net saving of $67,582 within the initial six months for the 28 employees. Significant improvements were also recorded in the participants' Well-Being Indices.

The authors conclude that the CHIP intervention can improve well-being and health outcomes for individuals suffering with T2D and is capable of generating measurable savings with a meaningful return-on-investment within a relatively short time period of six months.

2.6.2.4 *Summary of CHIP publications*

These 17 published papers clearly showed that using paid health professionals as facilitators in workplace and community settings, the CHIP intervention can produce significant reductions in the risk factors associated with MetS. Further, the benefits of these reductions can still be seen through 18 months (Merrill, Aldana, Greenlaw, Diehl, et al., 2008). These papers also showed that CHIP can deliver significant improvements in quality of life, and psychological factors such as stress, sleep disorders and depression.

2.7 Volunteer delivery

The objective of this dissertation was to explore the effectiveness of the CHIP lifestyle intervention when delivered by volunteer facilitators in a community setting. In this dissertation the term volunteer is taken to mean a person who does not receive payment for the service they are delivering, the volunteer has donated his or her time. As demonstrated above, there is substantial evidence showing the effectiveness of lifestyle interventions, including CHIP, using professional facilitation. It has been suggested that more well-trained professionals achieve the best health care outcomes (Alter, 2007). Volunteers, in most cases, do not have the same training or experience as trained healthcare professional and because remuneration cannot be used as an inducement it can be more difficult to motivate and control volunteers. While these limitations affect volunteers, there is evidence in the literature that adequately resourced volunteers have been effectively utilised in the delivery of community programs.

Volunteers from Faith in Action groups were successfully used to deliver the *Strong for Life* program to elderly people and it was found that these trained volunteers could safely deliver programs to a wider community group of frail older persons (Etkin, Prohaska, Harris, Latham, & Jette, 2006). Further, Schneider et al (2007) concluded that volunteers can successfully deliver health promotion programs which allows for wider program dissemination than is possible using only professional leaders.

An obvious advantage of utilising volunteer facilitators is the cost saving when compared to utilising paid facilitators; however, there may be other advantages in the utilisation of volunteer community-based facilitators. In order for a community program to be successful, at least one person must champion the program. Indeed, a passionate

volunteer can incite his peers to action (Kong, 1997). Volunteers have relationships with individuals in the community and these relationships can be utilised to encourage members of that community to be proactive about their health and to make and sustain positive lifestyle changes (Kong, 1997).

While the ability to utilise volunteers depends on the availability and commitment of the volunteers, the utilisation of volunteers maximises the use of social and human capital. Yuasa et al (2007) observe that if a significant initial investment is made in social and human capital, other forms of capital, such as financial, physical and natural, are not always necessary. This may mean that the utilisation of voluntary facilitators can dramatically increase the scope and efficacy of lifestyle modification programs while keeping costs to a minimum.

In order to explore the effectiveness of volunteers in the delivery of a lifestyle modification program targeting the risk factors associated with the Metabolic Syndrome, this dissertation analyses the results achieved by 178 volunteer-facilitated, community-based CHIP interventions involving 5070 participants between 2006 and 2009 in multiple locations in the United States and Canada.

2.8 Conclusion

There is abundant evidence from the literature that lifestyle intervention can effectively reduce the risk factors associated with MetS and improve chronic disease status. The CHIP lifestyle intervention has been shown to be effective when delivered by professional facilitators, but its effectiveness when delivered by volunteers has not been examined. Further, there is a need for an understanding of how participant demographics, including age, gender and marital status, influence their responsiveness to lifestyle interventions. Presently, there is little work published in this area, yet this will be critical to the future designing of lifestyle interventions to be most efficacious.

The remainder of this dissertation examines the effectiveness of volunteer-delivered, community-based CHIP interventions in over 5000 CHIP participants and explores factors that influence the outcomes of these CHIP interventions.

Chapter 3 Methodology

3.1 Introduction

In this chapter the methodology used to address the research questions is presented. The study design is defined. An overview of the CHIP lifestyle intervention is presented, and the data collection and collation process is explained. The author's experience with, and contribution to, the CHIP intervention, as well as his role in the data collection process is also outlined. The measures analysed in this study are explained and an overview of the statistical analyses is presented.

3.2 Study Design

The study design that forms the basis of this dissertation was a pre-test post-test cohort design involving 5070 individuals who participated in one of 178 community-based CHIP interventions delivered by volunteers between 2006 to 2009 in the United States and Canada

3.3 Research Questions

The two research questions that form the basis of this dissertation are:

1. How effective is the Coronary Health Improvement Program (CHIP) lifestyle intervention for reducing the selected risk factors of chronic disease that constitute the Metabolic Syndrome, when delivered by volunteers to free-living participants in their community?

2. What is the impact of selected participant factors, including age, gender, marital status, religious affiliation, previous history, family history and body weight on the outcomes achieved by participants in the CHIP intervention?

3.4 The CHIP intervention

To answer these research questions question a large cohort (n = 5070) of individuals who participated in 178, volunteer-delivered, community-based, CHIP interventions between 2006 and 2009 in the United States of America and Canada has been analysed.

The CHIP interventions which provided the participants for this study were delivered by community based volunteer directors. In all cases the volunteer facilitators donated their time to conduct the CHIP interventions analysed in this dissertation. These volunteer directors were not required to be health professionals, although some were, and underwent a two-day training workshop at the cost of $250 per team of 3, during which they received instruction and detailed manuals regarding the programs philosophy, content and method. They were then provided with a comprehensive resource package that included all the materials required to deliver the program.

The majority of these CHIP interventions were conducted through local Seventh-day Adventist churches. Although detailed data is not available on how participants were recruited, in general, participants were recruited from the local community by word of mouth, advertising in local print and radio media and some cases physician referral.

Participants were charged to participate in the CHIP intervention and the normal fee was $250 per participant.

The CHIP intervention begins with an initial health appraisal which is then followed by an intensive education program consisting of 16 two and a half hour sessions delivered over a four or five week period. The focus is on developing a greater measure of intelligent self-care involving a clearer understanding of the nature and aetiology of CVD and T2D, their epidemiology and risk factors. The potential for prevention, arrest and reversal through better lifestyle choices in the areas of smoking, sedentary living, diet and stress management is outlined and participants are shown how to implement positive lifestyle change.

The CHIP curriculum includes the following topics:

- modern medicine its accomplishments and limitations
- atherosclerosis
- chronic disease risk factors
- smoking
- exercise
- dietary fibre
- cholesterol
- the optimal diet
- obesity
- diabetes
- hypertension
- hyperlipidaemia
- lifestyle and health
- behavioural change
- self-worth

(Englert et al., 2004).

Each session typically includes a 60 minute DVD presentation, a cooking demonstration, group discussion and encouragement to exercise. Also included is a shopping tour.

At the conclusion of the program a post-intervention health appraisal is conducted. Following a graduation ceremony participants are encouraged to join a CHIP Alumnus, which meets on a monthly basis to encourage the preservation of lifestyle changes.

The CHIP program encourages participants to move towards what is referred to as the Optimal Diet which is a whole-food, plant-based diet with little or no animal products. The recommendation is that on a daily basis: less than 15-20% of calories be derived from fat; less than 10 teaspoons of added sugar, 5 grams of salt and 15 mg of cholesterol be ingested; and 2 to 2.5 L of water be consumed (Diehl & Ludington, 2005, p. 197; Englert et al., 2004). CHIP recommends at least 30 minutes of moderate intensity aerobic exercise or 10,000 steps a day. The program also recommends the cessation of smoking and the implementation of strategies to reduce stress.

Diehl states that the primary goals of this comprehensive educational program are to substantially improve blood lipid levels, blood pressure and blood sugar levels. Secondary goals are to decrease weight, eliminate smoking, enhanced daily exercise, adopted better stress-coping strategies and decrease medication for hypertension, diabetes and heart disease (1998).

3.5 Theoretical framework of CHIP: A Theory of Planned Behaviour

CHIP builds off a number of behaviour change strategies but leans heavily on the Theory of Planned Behaviour (TPB). TPB was first proposed by Ajzen in 1985 as an

extension of the Theory of Reasoned Action published by Ajzen and Fishbein in 1975 (Ajzen, 1985).

Ajzen's theory was developed in an attempt to improve health education (Nutbeam, 2000) and has become one of the most frequently cited and influential models for the prediction of human behaviour (Ajzen, 2011). TPB has been successfully used to predict and explain a wide range of health behaviours and intentions including: smoking, drinking, health services utilization, breast-feeding and substance abuse (Glanz, Rimer, & Viswanath, 2008, p. 68).

The TPB is based on several assumptions including the assumption that the majority of an individual's everyday behaviours are under volitional control. The theory also assumes that most human behaviour is goal directed (Ajzen, 1985).

A schematic of the TPB is shown in Figure 2. As illustrated, the TPB suggests that behaviours, or actions, are premeditated by intentions for the behaviour. TPB suggests that between these beliefs and behaviours is the intentions that are formed by the individual to perform a given behaviour at a specific time and place (Ajzen, 2011). In essence TPB views an individuals' beliefs as predictors of their intentions or motivation which in turn predict their behaviour or actions. The theory acknowledges, however, that not all intentions are carried out.

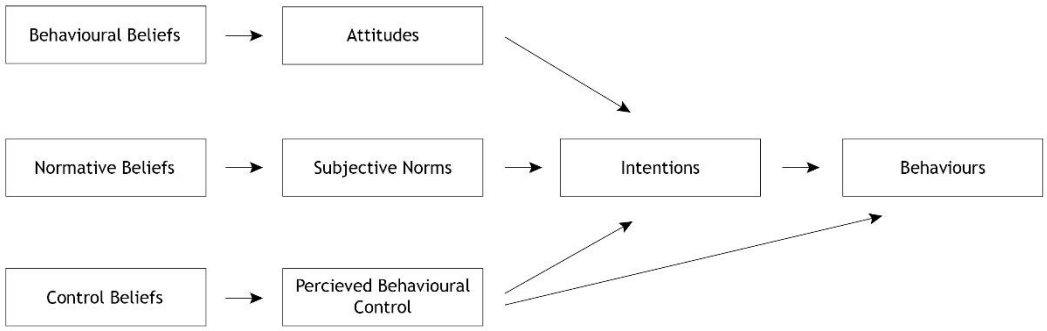


Figure 2 Theory of Planned Behaviour

The theory suggests that there are three determinants of an individual's intentions:

1. Attitude - the individual's positive or negative evaluation of the value of performing the behaviour. Typically a person would ask questions like: "What will be the result of performing this behaviour?" and "What is the consequence of performing this behaviour?" Driving a person's attitude are beliefs associated with that particular attitude which Ajzen refers to as behavioural beliefs. The stronger a person's attitude, the more likely that attitude is to drive intentions and ultimately drive behaviour (Ajzen, 2001).

2. Subjective Norms - the individuals' perception of the social pressure to perform or not perform a behaviour. It is in this category that an individual assesses the expectation of friends, family and society in performing a particular behaviour. Typically individuals would ask themselves: "Do the people in my sphere of influence approve, or disapprove of this behaviour?" Ajzen suggest that decisions are not always based just on an individual's judgement but can be significantly influenced by the judgement of significant others. The beliefs associated with subjective norms are called normative beliefs. In developing normative beliefs an individual makes a determination as to

whether social pressures from significant others or peers are valid. If the individual believes that the social pressures are valid, they are more likely to adopt the opinions of those around them.

3. Perceived Behavioural Control - the individuals' perception as to whether or not they will succeed or fail in the behaviour. Ajzen divides perceived behavioural control into two components: how much control a person has over the behaviour and how confident a person feels about being able to perform a particular behaviour. An individual's behaviour is strongly influenced by their confidence in their ability to perform a task, also referred to as self-efficacy. The person who is confident in their ability to master a skill is more like to succeed than one who doubts their ability to succeed, even if they have an equal capability of learning (Ajzen, 1991). The beliefs that influence perceived behavioural control are called control beliefs. These behavioural beliefs determine whether or not an individual believes that they are able to successfully perform a task.

In summary, according to TPB, an individual's beliefs drive their attitudes, subjective norms and perceived behavioural control which in turn predicts their intentions and subsequent behaviours.

Ajzen's theory has been criticised for not taking sufficient account of affective and emotional processes that are known to strongly influence intentions and behaviours (Armitage & Conner, 2001; Rapaport & Orbell, 2000). Ajzen (2011) has responded by suggesting that affect and emotions serve as background factors that influence beliefs which ultimately drive intentions and thus behaviour.

While CHIP is influenced by a number of well-established health behaviour theories including the Health-Belief Model (Rosenstock, 1974), the Social-Cognitive theory (Bandura, 2001), the Transtheoretical Model (also known as the Stages of Change) (Prochaska, Norcross, & DiClemente, 2013), the TPB is the most important theoretical framework for CHIP. CHIP deliberately impacts the three sets of beliefs, as proposed by TPB, that impact intentions and thus action in order to encourage and enable the participants to make positive changes to their lifestyle.

3.6 Data collection

The CHIP intervention incorporates a health assessment at baseline and the second health assessment postintervention. This health assessment includes measuring of height weight, blood pressure and blood samples taken to measure TC, HDL, LDL, TG and FPG. Where possible trained health professionals, normally nurses, were used for the measuring of blood pressure and blood samples were collected by trained phlebotomists from the local pathology laboratory services.

Once the results from the health assessment have been received by the volunteer director they are entered into a Microsoft Access™ based software package called the CHIP Assistant, which was provided to all directors of the CHIP interventions. The CHIP Assistant software package is used for the purpose of generating a basic report which is given to the CHIP participants to provide feedback on their health status, particularly in relation to lipid levels, blood pressure and BMI, compared to optimal levels.

The CHIP Assistant software package has provision for the data entered by the facilitators to be forwarded to a central collection point, managed by Dr John Gobble, as Microsoft Excel™ spreadsheets. The data thus collected records the name of the director,

the location, date and duration of the CHIP program but all participant data is anonymous.

3.7 The authors' involvement with CHIP

From January 2006 through December 2011, Paul Rankin (the author), was employed as the Health Director for the New Zealand Pacific Union Conference of the Seventh-day Adventist Church. As part of this role he oversaw the introduction and rollout of the CHIP program in New Zealand. During this period, the author facilitated the training of over 150 volunteer CHIP directors leading to over 100 CHIP interventions being offered in New Zealand, involving over 1000 participants. The author was responsible for the collection of the participant's biometric data from the pre-and post-intervention health appraisals.

The author supervised an extensive rewrite and reprint of the books and materials provided to the CHIP participants in New Zealand and Australia. This rewrite was necessary to contextualise the program into the Australasian context including utilising units of measurement that are familiar to Australian and New Zealand audiences, the adaptation of recipes for the Australasian market and localisation of spelling and grammar. He also supervised the production of an Australasian CHIP recipe book "Simple, Tasty, Good" providing CHIP recipes that taste good and use ingredients that are readily available in New Zealand supermarkets.

In 2009, the author was interested to determine the effectiveness of CHIP as delivered by volunteers and decided to undertake Ph.D. research to address this issue. His initial intention was to use the data that he had collected from New Zealand CHIP programs. He contacted Dr John Gobble to request any additional New Zealand data that

was available.

On requesting the data for New Zealand the author was informed that there was also a large amount of data from American participants, in the community-based, volunteer delivered CHIP interventions which has not been analysed. Following discussions with the custodians of this data, Dr Hans Diehl, Dr John Gobble, Dena Guthrie and Harold Burden an agreement was entered into to make this data available for use in this research project. The use of these data were provided to a large extent in recognition of the extensive contribution by the author to the development of the CHIP program in New Zealand and the substantial contribution he had made to the database.

While this dissertation focuses on the North American data set, the author has been involved in the publication of two papers examining the New Zealand data (Kent, Morton, Hurlow, Rankin, & Hanna, 2013; Morton et al., 2013).

3.8 Ethics consent

All participants in this study signed a form entitled "Request to Participate in the Coronary Health Improvement Program" at the time of enrolling in CHIP. This form included the following paragraph "I agree to my results being included as part of group analyses and publication of the program's results and understand that my particular results will not be disclosed to anyone without my express permission." Confidentiality of the participant data were protected by providing only anonymous data to the researchers. These data were only accessible to the research team and only aggregated data were presented in publications and public presentations.

Appropriate application forms were completed and submitted to the Avondale College of Higher Education Human Research Ethics Committee for permission to analyse

and publish this data in October 2009. Based on the consent form completed by the participants before the collection of this data, Avondale College Human Research Ethics Committee, on Monday, January 25, 2010, gave retrospective ethics approval for the use of this data (Approval number 20:10:07).

3.9 Collation of data

The extent of this data were some 7000 datasets collected since 2007 in North America, Australia and New Zealand from around 300 different CHIP programs. Because of the large size of the data set and to get a more homogenous sample a decision was made to concentrate on the North American data for this dissertation. The New Zealand and Australian data from this data set and the New Zealand data collected by Paul Rankin has been analysed independent of this research project and the results of the analysis were published in the *New Zealand Medical Journal* in February 2013 (Morton et al., 2013).

The data consisted of 183 separate Microsoft Excel™ spreadsheets containing 5581 datasets. As the data had been produced by different versions of the CHIP Assistant software the spreadsheets were in different formats so they had to be converted into a single format. The separate spreadsheets were combined into a single spreadsheet with all of the data appropriately aligned and formatted.

The data had been collected in both the United States and in Canada. The US data were in American units with TC, HDL, LDL, TG and FPG being in milligrams per decilitre (mg/dL) and height being in feet and inches with weight being in pounds while the Canadian data were in international units with TC, HDL, LDL, TG and FPG being in millimoles per litre (mmol/L), height in metres and weight in kilograms. Because the

majority of the data were from the US and the aim was to publish in US journals all of the Canadian data were converted to American units.

Once the data had been collated and converted into an appropriate format it was entered into the PASW Statistics 18 (for Microsoft Windows™) version of SPSS.

3.10 Measures analysed

The CHIP program commences with a health appraisal. Participants are asked to present themselves having fasted for at least eight hours, and a blood sample is taken and analysed for cholesterol levels (TC, HDL, LDL and TG) and FPG levels. This blood was collected by professional phlebotomists and analysed by professional laboratories. Participants' height, weight and blood pressure were measured by the trained volunteers. Participants were then requested to complete a lifestyle evaluation form which includes details of self-reported, exercise habits, past exposure to smoking, obesity, diabetes, high blood pressure and cardiac events as well as age, gender and religious affiliation. The lifestyle evaluation form also collects information pertaining to family histories of diabetes, cancer and coronary events. The health appraisal is performed again at the conclusion of the intervention.

The various measures, analysed in this dissertation, from this data are:

3.10.1 Body Mass Index (BMI)

BMI was used as a measure of obesity in this study as data were available on height and weight but no data were collected on waist circumference (WC) or on hip weight ratio (HWR). BMI is determined by the formula (weight in kilograms/height in metres^2) where (BMI units = kg/m^2). As imperial units were recorded for this dataset the BMI calculation was (weight in pounds/height in inches^2 X 703). Height and weight were

measured at the baseline and post-intervention health screens which were performed at the same time of day. Weight and height was measured with shoes removed and participants were asked to wear the same clothing for both health screens. The same instruments were used at baseline and post-intervention.

Participants were categorised by BMI at program entry according to the WHO BMI categories of underweight (< 18.5), normal (18.5 – 24.99), overweight (25 - 29.99), obese I (30 - 34.99), obese II (35 - 39.99) and obese III (> 40) (WHO, 2000).

BMI has been routinely used in clinical and public health practice to identify individuals and populations at risk of developing CVD and T2D for decades (Taylor et al., 2010). However in recent times BMI has been criticised as a measure of risk because it does not specifically identify abdominal adiposity (Mason, Craig, & Katzmarzyk, 2008). Waist circumference (WC) and waist-hip ratio (WHR) have been suggested as alternative measures to BMI with some studies finding these measures superior to BMI for predicting the onset of CVD andT2D (Dobbelsteyn, Joffres, MacLean, & Flowerdew, 2001; Yusuf et al., 2004). The Harmonized definition of MetS uses waist circumference to measure obesity (Alberti et al., 2009).

However Dalton et al. (2003) suggest that WC, BMI and WHR identify different portions of the population, as measures of both prevalence of obesity and CVD risk factors and Freiberg et al. (2008) found that BMI and WC were nearly identical in predicting the onset of the first cardiovascular event. Weight or BMI has been found to be correlated to waist circumference with a reduction of 1 cm in waist circumference being equivalent to approximately ¾ kilogram weight loss (Egger & Dobson, 2000). An analysis of 14,924 participants in the Third National Health and Nutrition Examination Survey

found that waist circumference cut-off points did not provide any greater prediction of health risk beyond that already predicted by BMI (Janssen, Katzmarzyk, & Ross, 2002). After examining the data from four large cohort studies to compare the association of BMI, WC and WHR for identifying persons at risk of future disease, Taylor et al. (2010) conclude that no strong evidence supports replacing BMI in clinical or public health practice with other adiposity measures.

The IDF definition of MetS stated that if the BMI was > 30 then central obesity could be assumed (K.G. Alberti et al., 2006).

3.10.2 Lipid Profile

Participants lipid profile was measured from fasting (> 8 hours) blood samples. Blood samples were collected by trained phlebotomists and analysed by local pathology laboratories to determine TC, HDL, LDL and TG. Units used for the lipid profile was milligrams per decilitre (mg/dL).

LDL levels are routinely calculated using the Friedewald formula which is LDL = TC - HDL - (TG/5) provided that TG levels are not greater than 400 mg/dL (Friedewald, Levy, & Fredrickson, 1972; National Cholesterol Education Program, 2002).

The National Cholesterol Education Program Adult Treatment Panel III classification system (National Cholesterol Education Program, 2002) was used to categorize LDL, HDL and TG (Table 3-1).

The Framingham Risk Classification (Wilson et al., 1998) was used for stratification of the TC data, because it includes 5 categories compared to only 3 in the National Cholesterol Education Program Adult Treatment Panel III classification system and thus allowing a more detailed analysis of the effect of the intervention on the highest

risk participants (Table 3-1).

Table 3-1 Lipid profile categories

TC categories	LDL categories	HDL categories	TG categories
<160	<100	<40	<100
160 – 199	100 – 129	42 – 60	100 – 199
200 – 239	130 – 159	>60	200 – 500
240 – 280	160 – 190		>500
>280	>190		

All units mg/dL

3.10.3 Fasting Plasma Glucose (FPG)

FPG levels were measured using the same blood samples collected for the lipid profile. FPG levels were categorised according to National Cholesterol Education Program Adult Treatment Panel III classification system. These categories were < 110, 110-125, > 125 and the units used were milligrams per decilitre (mg/dL) (National Cholesterol Education Program, 2002).

3.10.4 Blood Pressure

Systolic and diastolic blood pressures were measured using Sphygmomanometry and reported as millimetres of mercury (mmHg). BP was categorised according to the risk factor categories outlined by the National Cholesterol Education Program Adult Treatment Panel III classification system. For SBP the categories were ≤120, 121-139, 140-160 and >160. For DBP the categories were ≤80, 81-89, 90-100 and >100. (National Cholesterol Education Program, 2002).

There are several challenges with the measurement of blood pressure. Firstly blood pressure is notoriously labile and can change simply because it has been measured, a phenomenon referred to as white coat syndrome (Den Hond, Celis, Vandenhoven, O'Brien, & Staessen, 2003; Owens, Atkins, & O'Brien, 1999). Secondly, the indirect measurement of blood pressure using an occluding cuffs, stethoscope and manometer (Sphygmomanometry) has many possible causes of error and inaccuracy (Perloff et al., 1993). However it has been found that trained lay people can take accurate blood pressure readings (Kong, 1997). In this study the use of trained personnel, normally nurses, for the measurement of blood pressure and the large sample size, (4,560 for systolic blood pressure and 4,552 for diastolic blood pressure) should ameliorate some of the measurement errors.

3.10.5 Metabolic Syndrome status

MetS status was calculated using the Harmonized Definition (Alberti et al., 2009). In order to calculate MetS status participants BMI, BP, HDL, TG and FPG were categorised as either 0 if they did not meet the criteria for MetS or 1 if they did. If the total of the five MetS scores was three or more the participants was considered to be categorised as having MetS. The Harmonized Definition (Alberti et al., 2009) defined MetS as:

Any three (3) of

- Increased Waist Circumference
    - population-specific and countries-specific definitions as per IDF
- Raised Triglycerides
    - ≥ 150 mg/dL (1.7 mmol/L)
    - or specific drug treatment for this lipid abnormality
- Reduced HDL-cholesterol
    - < 40 mg/dL (1.03 mmol/L) in males
    - < 50 mg/dL (1.29 mmol/L) in females
    - or specific drug treatment for this lipid abnormality

- Raised Blood Pressure
    - Systolic BP≥ 130 or diastolic BP≥ 85mmHg
    - or specific drug treatment for previously diagnosed hypertension
- Raised Fasting Plasma Glucose
    - (FPG) ≥ 100 mg/dL (5.6 mmol/L)
    - or drug treatment for increased glucose.

(Eckel et al., 2010)

As waist circumference data were not available on this dataset a BMI of 30 or greater was used as the classification for BMI as per the IDF definition (K.G. Alberti et al., 2006).

3.10.6 Framingham Risk Score

The Framingham Risk Score is a widely used reference tool that estimates the 10-year risk of an individual developing coronary heart disease. This score was calculated using participants' age, TC, HDL, BP, FPG and self-reported smoking with points being assigned in each category as per the formula developed by Wilson et al. (1998) and published in the National Cholesterol Education Program Adult Treatment Panel III (National Cholesterol Education Program, 2002). The total points were then totalled to provide a raw Framingham Risk Score which could then converted to percentage risk of coronary heart disease within the next 10 years as also outlined by Wilson et al. (1998) (Table 3-2, Table 3-3).

Table 3-2 10-year risk estimates for men (Framingham risk score) (National Cholesterol Education Program, 2002)

Age	Points
20-34	-9
35-39	-4
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	11
70-74	12
75-79	13

Total Cholesterol (mg/dL)	Points at Ages 20-39	Points at Ages 40-49	Points at Ages 50-59	Points at Ages 60-69	Points at Ages 70-79
<160	0	0	0	0	0
160-199	4	3	2	1	0
200-239	7	5	3	1	0
240-279	9	6	4	2	1
≥280	11	8	5	3	1

	Points at Ages 20-39	Points at Ages 40-49	Points at Ages 50-59	Points at Ages 60-69	Points at Ages 70-79
Nonsmoker	0	0	0	0	0
Smoker	8	5	3	1	1

HDL (mg/dL)	Points
≥60	-1
50-59	0
40-49	1
<40	2

Systolic BP (mmHg)	If Untreated	If Treated
<120	0	0
120-129	0	1
130-139	1	2
140-159	1	2
≥ 160	2	3

Point Total	10-Year Risk	Point Total	10-Year Risk
<0	<1%	11	8%
0	1%	12	10%
1	1%	13	12%
2	1%	14	16%
3	1%	15	20%
4	1%	16	25%
5	2%	≥ 17	≥30%
6	2%		
7	3%		
8	4%		
9	5%		
10	6%		

Table 3-3 10-year risk estimates for women (Framingham risk score) (National Cholesterol Education Program, 2002)

Age	Points
20-34	-7
35-39	-3
40-44	0
45-49	3
50-54	6
55-59	8
60-64	10
65-69	12
70-74	14
75-79	16

Total Cholesterol (mg/dL)	Points at Ages 20-39	Points at Ages 40-49	Points at Ages 50-59	Points at Ages 60-69	Points at Ages 70-79
<160	0	0	0	0	0
160-199	4	3	2	1	1
200-239	8	6	4	2	1
240-279	11	8	5	3	2
≥280	13	10	7	4	2

	Points at Ages 20-39	Points at Ages 40-49	Points at Ages 50-59	Points at Ages 60-69	Points at Ages 70-79
Nonsmoker	0	0	0	0	0
Smoker	9	7	4	2	1

HDL (mg/dL)	Points
≥60	-1
50-59	0
40-49	1
<40	2

Systolic BP (mmHg	If Untreated	If Treated
<120	0	0
120-129	1	3
130-139	2	4
140-159	3	5
≥ 160	4	6

Point Total	10-Year Risk	Point Total	10-Year Risk
<9	<1%	20	11%
9	1%	21	14%
10	1%	22	17%
11	1%	23	22%
12	1%	24	27%
13	2%	≥25	≥30%
14	2%		
15	3%		
16	4%		
17	5%		
18	6%		
19	8%		

3.10.7 Gender

Gender was reported by participants as either male or female.

3.10.8 Age

Participant's age was calculated from the date of birth. Participants were grouped into decadal age groups.

3.10.9 Marital status

Marital status was reported by participants as either single, married, divorced or widowed. A limitation of the data collection on marital status was that data were not collected on de facto relationships as participants were only given the above four options to select for marital status.

3.10.10 Religious affiliation

As the majority of the programs analysed in this study were conducted under the auspices of Seventh-day Adventist churches, participants were asked to identify whether or not they were members of the Seventh-day Adventist Church. Using this information participants were categorised as either Seventh-day Adventists (SDA) or non-Seventh-day Adventists (non-SDA).

3.10.11 Family history and previous history

In the initial health appraisal participants were asked to complete a question relating to the participants' family medical history and another question relating to the participants' previous medical history. These two questions were analysed in order to assess the impact of family history and previous history on the outcomes of the CHIP intervention.

The first question related to family medical history and was:-

One or both of your parents died before age 60: of heart disease? □Yes □No of diabetes? □Yes □No

The second question related to participants' own medical history and was:-

Check (X) if you have ever been told by a physician that you have any of the following:

☐Angina (Yr)? _____	☐Abnormal EKG (last 3 yrs)	☐Gall bladder trouble	☐Ulcers
☐Heart attack (yr)? _____	☐Irregular heartbeats	☐Gout	☐Osteoarthritis
☐Angioplasty (Yr)? _____	☐Stroke (Yr)? _____	☐Kidney disease	☐Rheumatoid arthritis
☐Bypass (Yr)? _____	☐High blood pressure	☐Chronic bronchitis	☐Overweight
☐Heart failure (Yr)? ____	☐High cholesterol	☐Emphysema	☐Gout
☐Blood clotting problem	☐High triglycerides	☐Thyroid disorder	☐Cancer

3.10.12 Dietary intake and smoking

Unfortunately, while some data were collected on dietary intake at the health appraisal, the data were not entered into the CHIP Assistant Software and so we were unable to use it in this research project. Data were also collected with regard to participants' smoking history however the categories for smoking on the health screen questionnaire provided to participants were confusing and consequently the data collected was unreliable and was not suitable for use in this analysis.

3.11 Statistical analyses

Prior to analysis duplicates were removed and outliers were identified and eliminated from the data and the distribution of the change was tested for normality by means of graph construction and calculation of skewness and kurtosis. The distribution, for all biometrics was found to be near normal though due to the large sample size violations of the assumption of normality are of little concern (Coakes & Steed, 2010, p. 73; Lumley, Diehr, Emerson, & Chen, 2002). Once outliers and duplicates had been removed from the data set the clean dataset consisted of data on 5,070 participants from 178 CHIP interventions.

Detailed analysis of the data to address the research questions included, where appropriate, descriptive techniques, comparison of means, multivariate analysis and linear regression. Where necessary Bonferroni's post hoc analysis was used to perform pairwise comparisons following a significant overall test result. The effect size of any change was determined using Cohen's d. An effect size of 0.2 or less is considered a small change, an effect size > 0.2 but < 0.8 is considered a moderate change and an effect size of > 0.8 is considered large (Cohen, 1988, pp. 285-287).

Chapter 4 presents the results of the analyses that were performed to answer the first research question: "How effective is the Coronary Health Improvement Program (CHIP) lifestyle intervention for reducing selected risk factors of chronic disease associated with the metabolic syndrome, when delivered by volunteers to free-living participants in their community?"

In order to answer this research question the data from all the participants were grouped together independent of their respective demographic and initial health status.

Using this grouping, the change in each biometric, from baseline to post-intervention, was analysed, using t-tests to determine whether this change was significant and, if significant, to identify the extent of this change. The effect size was calculated using Cohen's d.

The effect of the intervention on the various risk factors was further analysed by stratifying the participants according to their risk factor status at baseline. Conventional risk factor categories were used to stratify the participants.

A paired t-test was used to determine if there was a significant change within each of the risk factor categories from baseline to post-intervention. Additionally, changes in the distribution of the number of participants within the risk categories from baseline to post-intervention was examined using appropriate the Chi square test.

Chapter 5 addresses the second research question: “What is the impact of selected participant factors, including age, gender, marital status, religious affiliation, previous history, family history and weight on the outcomes achieved by the CHIP intervention?” In order to answer this question the impact of selected participant factors on the participants’ outcome in relation to lipid profile, BMI, FPG and BP were examined. The participant factors that were examined are age, gender, gender differences across age range, marital status, religious affiliation, BMI, family history and previous history.

For each of the factors, differences in the biometrics at baseline between the categories were assessed using appropriate t-testing or ANOVA. Next the change in biometrics from baseline to post-intervention within the same category was examined using paired t-tests. If the change in biometrics was found to be significant within categories, the difference in change between categories was examined using appropriate

t-tests or ANOVA. When a significant difference between the categories was identified, Bonferroni's post hoc analyses were performed to identify where the differences occurred. Cohen's d was calculated to show the effect size where appropriate.

3.12 Summary

In brief, the following analysis steps were employed: each variable was examined for accuracy of data entry, variables were categorized as outlined above, and various statistical methods were used to identify differences in the data at baseline and then to analyse any changes in participants' biometrics from baseline to post-intervention. The results of these analyses are presented and discussed in the next two chapters.

Chapter 4 Results part 1: The effectiveness of the volunteer-delivered community-based CHIP interventions

4.1 Introduction

In this chapter the first research question, "How effective is the Coronary Health Improvement Program (CHIP) lifestyle intervention for reducing selected risk factors of chronic disease associated with the Metabolic Syndrome, when delivered by volunteers to free-living participants in their community?", is addressed.

To answer this question, this chapter presents the analysis of a large cohort (n = 5070) of individuals who participated in 178, volunteer-delivered, community-based, CHIP interventions between 2006 and 2009 in the United States of America and Canada. The chapter begins by examining the overall changes, from baseline to post-intervention (30-days), in the biometric scores for the participants in the CHIP interventions delivered by volunteers to free-living participants in their community. To assess the clinical significance of these results, the change in MetS status and Framingham Risk Score is shown. The results from the volunteer-delivered, community-based interventions included in this study are then compared with the published results of professionally delivered CHIP interventions. Thirdly, the participants are stratified according to their risk categories at baseline and the change in biometric scores from baseline to post-intervention are analysed. The key finding from these analyses are then discussed with reference to the literature before concluding.

The biometric measures analysed include: height and weight from which body mass index (BMI) was calculated, total cholesterol (TC), low density lipoprotein cholesterol (LDL), high density lipoprotein cholesterol (HDL), triglycerides (TG), fasting

plasma glucose (FPG), systolic blood pressure (SBP) and diastolic blood pressure (DBP). The ratios of TC to HDL and LDL to HDL have been calculated. Metabolic Syndrome (MetS) status of the participants and Framingham Risk Score was also calculated. The significance or otherwise of changes in these biometrics from baseline to post-intervention were assessed using paired t-tests and the impact factor is showing using Cohen's d.

4.2 Overall changes in the biometric scores from baseline to post-intervention

In this section the overall change in the participant's biometric scores (BMI, TC, HDL, LDL, DG, FPG, and BP) from baseline to postintervention is presented. This data set consisted of 5070 individuals who participated in one of 178 CHIP interventions. The mean age of the participants was 57.2 ± 12.9 years with 1690 (33.5%) males (57.86 ± 12.97 years) and 3353 (66.5%) females (56.9 ± 12.9 years). Using this grouping, the change in each biometric, from baseline to post-intervention, was analysed to determine whether this change was significant and, if significant, to identify the extent of this change.

The postintervention scores were lower than the baseline scores for all of the biometrics analysed at a significance of <0.001. The effect size ranged from small for TG and body weight to moderate for TC (Table 4-1).

Table 4-1 Overall change in biometrics post-intervention

Factor	N	Baseline		Post-intervention		Mean change	Percent change	t statistic	p value	Cohen's d
		Mean	(SD)	Mean	(SD)					
Weight (kg)	4,579	192.21	50.2	186.14	48.11	6.07	3.2%	80.36	<0.001	0.12
Body mass index (kg/m2)	4,508	31	7.28	30.02	7	0.98	3.2%	82.35	<0.001	0.14
Total cholesterol (mg/dL)	4,642	193.23	41.26	172.16	37.8	21.07	10.9%	55.90	<0.001	0.53
Low density lipoprotein (mg/dL)	4,539	130.67	61.18	113.7	54.35	16.97	13.0%	43.34	<0.001	0.29
High density lipoprotein (mg/dL)	4,638	54.71	25.49	49.97	23.06	4.74	8.7%	40.47	<0.001	0.20
Triglycerides (mg/dL)	4,628	141.24	83.48	131.49	72.59	9.75	6.9%	13.28	<0.001	0.12
Fasting plasma glucose (mg/dL L)	4,511	99.52	24.37	94.26	19.71	5.26	5.3%	26.69	<0.001	0.24
Systolic blood pressure (mmHg)	4,560	133.35	19.1	126.34	16.52	7.0	5.3%	31.54	<0.001	0.39
Diastolic blood pressure (mmHg)	4,552	79.86	11.1	75.69	9.89	4.2	5.2%	28.37	<0.001	0.40
Framingham score	3,670	12.18	9.29	10.38	7.84	1.8	14.8%	17.34	<0.001	0.21

SD – Standard deviation

4.2.1 Changes in Framingham Score and Metabolic Syndrome Status

To examine the clinical significance of these changes, the Framingham Risk Score (FRS) (National Cholesterol Education Program, 2002; Wilson et al., 1998) and Metabolic Syndrome Status was calculated at baseline and post-intervention and the differences compared. Data were available to calculate the FRS on 3,670 participants. The mean reduction of 1.8 in the Framingham Risk Score (Table 4-1) from baseline to post-intervention for the study cohort would be predicted to prevent approximately 70 coronary incidents over the next decade.

Data were available to determine the MetS status, at baseline and post-intervention, on 4,391 participants. A higher percentage of male participants (54.4%) could be categorised as having MetS at baseline than female participants (45.6%) [$\chi^2 = 19.417$, $p < 0.001$]. There was a significant reduction (Mantel-Haensizel chi squared score = 1474.619, df =1, p <0.001) between the number of participants who were categorised as having MetS at baseline (N= 2111, 48.1%) and post-intervention (N=1891, 43.1%). This represents a 10.4% reduction in the incident of MetS.

Table 4-2 Number of participants meeting the criteria for the MetS risk factors at program entry and post-intervention

Risk Factor	Baseline (N)	Post-intervention (N)	Change (N)
BMI	2170	1980	190
BP	2761	1994	767
FPG	1553	1113	440
TG	1523	1350	173
HDL	2050	2608	+558
MetS (≤3)	2111	1891	130

Table 4-2 shows that this overall reduction in MetS occurred because of the improved results for BMI, BP, FBG and TG, however during the intervention overall HDL levels dropped by 8.7%. This meant that an additional 558 participants met the MetS criteria for HDL at the end of the intervention (Table 4-1 and Table 4-2). By the end of the program 257 participants who were not

classified as having MetS at program entry acquired this status however, 157 of these individuals (61%) only did so because of reduced HDL levels. For these individuals, the TC: HDL and LDL: HDL ratios increased significantly from baseline to post-intervention (3.56 versus 4.12, $p<0.001$; 2.31 versus 2.44, $p=0.006$, respectively) as both TC (12%, $p<0.001$) and LDL (15%, $p<0.001$) did not decrease as much as HDL (21%, $p<0.001$).

4.2.2 Comparison with professionally delivered CHIP interventions

Table 4-3 shows a comparison of baseline to post-intervention change presented in seven papers published from the Rockford CHIP project andDiehl (1998) , outlined in chapter 2, with the results from this study. These eight papers, when aggregated, show the results for 4669 CHIP participants at the completion of the four week intervention where professional facilitators were used. This table shows that the outcomes achieved by the volunteer delivered programs, presented in this dissertation, are not dissimilar from those achieved by the professionally delivered.

Table 4-3 Comparison of volunteer facilitated CHIP interventions with professionally facilitated CHIP interventions

Percentage Change in Biometrics												
Reference		Professionally Facilitated										Volunteer Facilitated
		Diehl 1998 ++	Merrill 2008*		Aldana 2002	Adana 2005 A	Adana 2005 B	Aldana 2008	Englert 2004 ++	Englert 2007++	Professionally Facilitated Aggregated	
n		288	862	841	442	64	167	714	242	1049	4669	5042
Age		54.73	55.2	55.2	52.1	46.1	50.4		54.14	54.14	54.12#	57.29
Weight		3.17	ND	ND	4.43	3.25	3.68	4.01	3.68	3.91	3.89#	3.16
BMI		3.18	3.85	3.44	4.38	3.43	ND	3.91	3.82	3.07	3.47#	3.16
SBP		5.2	4.6	4.8	5.4	5.7	5.6	4.8	2.5	6.0	5.0#	5.3
DBP		7.0	4.4	5.4	6.0	6.3	6.4	4.0	3.1	5.9	5.2#	5.2
TC		13.36	11.63	10.19	13.20	8.02	7.52	10.89	10.66	12.16	11.29#	10.90
HDL		12.37	11.84	11.81	13.31	6.77	7.54	11.85	10.30	ND	9.05#	8.66
LDL		15.87	12.03	10.90	14.66	9.58	9.28	12.22	11.26	13.29	12.45#	12.99
TG		4.43	9.27	7.43	9.76	2.53	0.26	7.39	12.23	5.56	7.30#	6.90
FPG		8.15	4.40	5.30	5.06	4.00	4.00	6.52	4.98	5.60	5.46#	5.26

All figures are data after four weeks

* Merrill gives data for two groups the first group is those who were followed up after 12 months the second group is those who were lost to follow-up at 12 months but were followed up at 4 weeks

++ Data were presented for Male and Female for comparison purposes this has been combined using weighted means

Average of the Professionally Facilitated Programs calculated using weighted means

Volunteer Facilitated p< 0.0001 for all factors

Diehl 1998 p< 0.001 except for triglycerides Merrill 2008 p< 0.001 for all factors

Aldana 2002 Weight and BMI p<0.001, SBP p=0.0197, DBP p = 0.0175, TC p=0.0021, HDL p=0.0006, LDL p=0.0022, Tri p=0.0446 and FPG p=0.0263

Aldana 2005 A p< 0.0001 for weight and BMI, p< 0.02 for systolic and diastolic BP, p< 0.005 for TC HDL LDL and p< 0.05 for triglycerides and FPG

Aldan 2005 B p< 0.0001 for all factors except for FPG p= 0.003 and triglycerides p=0.9513

Aldana 2008 p< 0.001 except for triglycerides where p=0.0284

Englert 2004 p<0.05 for blood pressures and triglycerides, p<0.01 for glucose and HDL, all other factors p<0.001

Englert 2007 p<0.0001 except for triglycerides where p=0.113

These results clearly show that the volunteer delivered, community-based CHIP intervention can achieve significant improvements in selected chronic disease risk factors. Biometric changes from baseline to post-intervention are however more pronounced among the participants with higher risk profiles at program entry, as shown in the following section.

4.3 Results stratified for initial risk factor classification

The effect of the intervention on the various risk factors was further analysed by stratifying the participants according to their risk factor status at baseline. Conventional risk factor categories were used to stratify the participants. The National Cholesterol Education Program Adult Treatment Panel III classification system (National Cholesterol Education Program, 2002) was used to categorize the participants for all risk factors, except TC, for which the Framingham Risk Score Classification was used, and BMI, for which the WHO classification was used (WHO, 2000). The Framingham Risk Classification (Wilson et al., 1998) was used for stratification of the TC data, because it includes 5 categories compared to only 3 in the National Cholesterol Education Program Adult Treatment Panel III classification system, thus allowing a more detailed analysis of the effect of the intervention on the highest risk participants. The stratified analyses are shown in Table 4-4 and Table 4-5.

Table 4-4 Changes in risk factor levels within 30 days according to initial risk factor classification BMI and Lipid Profile

Risk Factor	N Baseline	N Post-intervention	χ^2* (p)	Baseline Mean	(SD)	Post-intervention Mean	(SD)	Mean Change	% Mean Change	t	p	Cohen's d
Body mass index (kg/m^2)												
< 18.5	27	33	(<0.001)	17.6	0.9	17.5	0.8	-0.1	0.8%	1.50	0.144	-0.12
18.5 - 24.9	884	1,086		22.7	1.6	22.3	1.7	-0.5	2.1%	17.78	<0.001	-0.24
25 – 30	1,470	1,539		27.5	1.4	26.6	1.5	-0.9	3.1%	51.08	<0.001	-0.62
> 30	2,242	1,965		36.6	6.1	35.4	6	-1.3	3.4%	59.56	<0.001	-0.20
Total cholesterol (mg/dL)												
< 160	631	1,862	1,950 (<0.001)	132.1	13.8	126.0	22.4	-6.1	4.6%	7.545	<0.001	-0.34
160 – 199	2,116	1,781		176.1	13.8	160.7	23.8	-17	8.8%	33.198	<0.001	-0.82
200 – 239	1,261	756		217.6	11.3	189.6	25.63	-27.1	12.7%	40.624	<0.001	-1.49
240 – 280	478	183		254.7	10.7	215.2	30.7	-39.5	15.5%	28.679	<0.001	-1.91
> 280	126	30		306.6	27.2	245.9	43.4	-60.7	19.8%	15.037	<0.001	-1.72
Low density lipoprotein (mg/dL)												
< 100	1,453	2,115	1,008 (<0.001)	80.6	15.1	75.3	209.1	-5.3	6.6%	10.928	<0.001	-0.29
100 - 129	1,345	1,326		114.6	8.3	102.1	20.2	-12.5	10.9%	23.794	<0.001	-0.88
130 - 159	905	588		142.4	8.5	120.1	21.8	-22.3	15.7%	31.843	<0.001	-1.47
160 - 190	377	197		172	8.2	141.6	27.1	-30.4	17.7%	22.486	<0.001	-1.72
> 190	488	342		273.9	67.9	229.8	73.1	-44.1	16.1%	20.218	<0.001	-0.63
High density lipoprotein (mg/dL)												
< 40	1,316	1,814	539 (<0.001)	34.2	4.8	33.2	7	-1	3.0%	6.15	<0.001	-0.17
40 - 60	2,097	1,912		48.9	5.3	45	7.8	-3.8	7.8%	27.29	<0.001	-0.60
> 60	1,261	948		86.3	29.8	76.2	28.5	-10.1	11.8%	30.15	<0.001	-0.35
Triglycerides (mg/dL)												
<100	3,053	3,232	109 (<0.001)	95.5	29.7	99.7	41.8	4.2	4.4%	-6.64	<0.001	0.12
100 - 199	753	765		171.9	13.9	158.1	53	-13.8	8.1%	7.17	<0.001	-0.41
200 - 500	820	663		270.5	62.4	220.1	81.8	-50.3	18.6%	18.995	<0.001	-0.70
> 500	45	11		634.7	114.2	354.8	158.5	-279.9	44.1%	10.431	<0.001	-2.05

* McNemar chi-square test. SD – Standard deviation.

Table 4-5 Changes in risk factor levels within 30 days according to initial risk factor classification FPG and BP.

Risk Factor	N Baseline	N Post-intervention	χ2* (p)	Baseline Mean (SD)		Post-intervention Mean (SD)		Mean Change	% Mean Change	t	p	Cohen's d
Fasting plasma glucose (mg/dL)												
< 110	3,716	4,026	265 (<0.001)	90.7	9.9	88.6	10.9	-2.1	2.3%	12.698	<0.001	-0.20
110 - 125	390	304		116.1	15.5	106	15.5	-10.1	8.7%	13.55	<0.001	-0.65
> 125	525	301		164	42.2	131.4	34.5	-32.6	19.9%	20.745	<0.001	-0.85
Systolic blood pressure (mmHg)												
≤ 120	1,279	1,866	662 (<0.001)	111.8	9	114.5	27	2.7	2.4%	-3.51	<0.001	0.15
121 - 139	1,719	1,788		129.9	5.1	125.2	27	-4.7	3.6%	7.28	<0.001	-0.29
140 - 160	1,127	743		147.2	5.8	134.3	13.1	-12.9	8.7%	31.82	<0.001	-1.37
> 160	454	182		170.7	11.9	147.3	17.6	-23.3	13.7%	27.49	<0.001	-1.59
Diastolic blood pressure (mmHg)												
≤ 80	2,619	3,364	560 (<0.001)	72.4	6.9	71.8	8.9	-0.7	0.9%	3.604	<0.001	-0.08
81 - 89	1,060	822		84.8	2.3	78.3	7.7	-6.4	7.6%	27.277	<0.001	-1.30
90 - 100	688	322		92.9	3.0	82.7	8.4	10.2	10.9%	31.608	<0.001	-1.79
> 100	210	69		106.2	13	87.7	10.3	-18.5	17.4%	16.963	<0.001	-1.59

* McNemar chi-square test. SD – Standard deviation.

While the overall group data presented in Section 4.2 shows significant changes in each of the biometrics, the stratified analyses present a more comprehensive picture. As shown in Tables 4-3 and 4-4, significant reductions were recorded in all risk categories for each of the biometrics at $p < 0.001$ with the exception of BMI < 18.5 where the change was not significant ($p = 0.144$) and the < 100 category for TG where there was a 4.4% increase ($p < 0.001$).

In general participants in the highest risk categories at baseline experienced the greatest change. For all biometrics the change was smallest in the lowest risk category and progressively increased as the level of risk increased, except for LDL where the second highest risk category (160 - 190 mg/dL) showed a slightly higher percentage mean reduction than the highest risk category (>190 mg/dL). The greatest variation of percentage mean change between categories was for TG where there was a 4.4% mean increase in the lowest risk category (< 100) and a 44.1% mean decrease in the highest risk category (> 500).

A similar trend was observed for effect size, as shown by Cohen's d, with the lowest risk categories experiencing the smallest effect size and the higher risk categories the largest effect size. In the higher risk categories, the effect sizes for the change from baseline to post-intervention were large. Interestingly, for all biometrics except for SPP and TG, where the highest category showed the largest effect size, the effect size was lower in the highest risk category than the second highest risk category. This may be explained by the fact that the standard deviations were large in the highest categories because there was no upper limit for these categories. Large effect sizes were observed in the higher risk categories for all biometrics except for HDL where the effect sizes were

moderate, and BMI where only the 25-30 risk category showed a moderate effect size with the other categories showing a small effect size.

There were significant reductions in the number of participants categorised as belonging to the highest risk categories from baseline to post-intervention. The change in the number of participants in the respective FPG categories is especially noteworthy. At baseline 525 participants recorded a FPG of >125 mg/dL which is indicative of diabetes, but this had reduced to 301 by the end of the program (Table 4-5), representing a 42.7% (N = 224) reduction.

The results of the stratified analyses indicate that the CHIP intervention is most effective for participants who exhibited the greatest risk level, at baseline, and therefore had the greatest need.

4.4 Discussion

In order to answer the first research question: "How effective is the Coronary Health Improvement Program (CHIP) lifestyle intervention for reducing the selected risk factors of chronic disease that constitute the Metabolic Syndrome, when delivered by volunteers to free-living participants in their community?" this chapter has examined the overall changes in the biometric scores from baseline to postintervention for the 5070 participants in this study and then the participants have been stratified according to baseline risk factor for further analysis. The key finding in this chapter is that the results of the overall and stratified analyses indicate that the CHIP intervention, when delivered by volunteer facilitators to free-living individuals in their community, effectively reduces risk factors associated with MetS. The stratified analyses showed that the CHIP intervention is most effective for those who need it most. Other findings are discussed below.

4.4.1 Metabolic Syndrome

In this study a 10.4% reduction in the number of participants being categorised as having MetS, from baseline to post-intervention was observed. Other studies, both human and animal studies, have shown that a low-fat plant-based diet and exercise can prevent and control MetS (Barnard & Wen, 1994). In a meta-analysis of lifestyle interventions Yamaoka and Tango (2012) concluded that long-term regular lifestyle modification programs reduce the prevalence of MetS and of the abnormalities associated with MetS.

The percentage of participants classified as having MetS in this study (52.6% of male participants and 45.6% of female participants) is higher than the prevalence reported within the general US population which has been estimated between 24 - 42%

for males and 23 - 38% for females (Alberti et al., 2009; Ford, Giles, & Dietz, 2002). The higher percentage of participants classified as having MetS in the present study than the general population may be explained by the CHIP intervention attracting participants who were more compromised in their health which is unremarkable given the intervention is marketed to individuals with elevated chronic disease risk factors. The prevalence of MetS has also been observed to rise with age, although attenuating in the over 70 years population (Hu et al., 2004). Given that the average age of participants in this study was 57.2 years which is substantially older than the average US population of 38.5 years (Howden & Meyer, 2010), age could also explain the high prevalence of MetS in this study.

In this study a 10.4% reduction in the number of participants being categorised as having MetS, from baseline to post-intervention was observed. Other studies, both human and animal studies, have shown that a low-fat plant-based diet and exercise can prevent and control MetS (Barnard & Wen, 1994). In a meta-analysis of lifestyle interventions Yamaoka and Tango (2012) concluded that long-term regular lifestyle modification programs reduce the prevalence of MetS and of the abnormalities associated with MetS.

4.4.2 Body Mass Index

The changes in BMI observed in this study were substantive given they were achieved in only 30 days. The mean 6.1 lb reduction in weight, equating to a 3% decrease in BMI, and a 3.4% reduction among those participants categorised as obese at program entry, might in itself be clinically significant.

Purposeful weight loss has been shown to improve exercise capacity, BP and lipid profile, as well as behavioural factors and quality of life (Aucott et al., 2005; Lavie, Milani,

Artham, Patel, & Ventura, 2009). While the benefits of weight loss are well recognised it is still frequently stated that lifestyle approaches to weight loss are ineffective (Goodpaster et al., 2010; Monkhouse, Morgan, Bates, & Norton, 2009). Surgical intervention is becoming increasingly popular. While weight loss can be achieved from surgical intervention, weight loss through lifestyle change has been found to be more effective in reducing blood pressure than equivalent weight loss from surgical intervention (Aucott et al., 2005) and successful maintenance of weight loss, even when achieved by surgical means, requires lifestyle change (Douketis, Macie, Thabane, & Williamson, 2005). Further weight loss by lifestyle seems to show greater reduction in risk factors associated with CVD and T2D than does weight loss from surgical means (Douketis et al., 2005).

The decrease in BMI observed in this study, of 3.2%, were substantive given they were achieved in only 30 days. Even modest weight loss can produce substantive health benefits (Goodpaster et al., 2010). It has been observed that in combating MetS and associated coronary vascular disease (CVD), a 10% reduction in body mass is the goal during the first year, and that 5% can be helpful (McClendon, Dunbar, Clark, & Coverson, 2010; Wing et al., 2011). The participants in the present study made good progress toward this goal within just 30 days.

Significant weight loss within the initial period of a weight loss program has been shown to be an important factor in successful weight maintenance (Elfhag & Rössner, 2005), as it may indicate greater compliance with the program (van Baak et al., 2003). The 3% mean weight loss in just 30 days, achieved by participants in this study may be beneficial long-term.

CHIP recommends a low-fat diet and increased exercise. Other studies have found that the lowering of fat intake in the diet can increase the effectiveness of a weight loss

program (Carmichael, Swinblirn, & Wilson, 1998) with the inclusion of physical exercise being particularly beneficial for weight loss (Goodpaster et al., 2010; Mekary, Feskanich, Hu, Willett, & Field, 2010).

4.4.3 Lipid Profile

The results of this study showed significant reductions in only 30 days in TC (10.9%), LDL (13.0%), HDL (8.7%) and TG (6.9%) (Table 4-1). These results are comparable to the published outcomes of the residential lifestyle interventions that promote a low-fat, plant-based eating pattern, such as the Ornish and Pritikin programs (Barnard, 1991; Beard, Barnard, Robbins, Ordovas, & Schaefer, 1996; Ornish et al., 1983), as well as the professionally delivered CHIP interventions (Aldana et al., 2002; Aldana et al., 2008; Diehl, 1998). Further, these changes in blood lipid profile compared favourably to those achieved with statin medication (Gould, Davies, Alemao, Yin, & Cook, 2007).

4.4.3.1 Low-Density Lipoprotein

A key target in coronary heart disease prevention is reduction of LDL levels, particularly very low density LDL (VLDL) (National Cholesterol Education Program, 2002). Participants in this study experience significant reductions in LDL, in only 30 days, with an overall mean reduction of 13% and greater reductions among those participants with elevated levels of LDL (Table 4-4). While changes in the size of LDL particles was not measured in this study, similar low-fat, plant-based interventions combined with exercise, have been shown to significantly increased the particle diameter of LDL (Beard et al., 1996).

It has been suggested that a 1 mmol/L (38.67 mg/dL) drop in LDL equates to a 40-50% reduction in coronary heart disease risk (Huxley, Lewington, & Clarke, 2002). The

overall mean decrease of 17 mg/dL observed in this study translates to an approximate 20% reduction in coronary heart disease risk. In those participants with the highest LDL classification (>190 mg/dL) at program entry, the mean reduction of 44.1 mg/dL represents an approximate 50% reduction in coronary heart disease risk. In conclusion, clinically significant reductions in LDL cholesterol levels were achieved by participants enrolled in the volunteer-delivered, community-based CHIP lifestyle interventions examined in this study.

4.4.3.2 Triglycerides

Two of the five criteria for MetS are related to lipid profile: elevated TG and lowered HDL. However, low-fat, plant-based diets, as advocated by CHIP, have been reported to elevate TG and lower HDL (Clevidence et al., 1992; Garg et al., 1994; McDougall, Litzau, Haver, Saunders, & Spiller, 1995). Consequentially, concern has been expressed by some authors as to the suitability of such diets for the prevention and management of MetS and associated T2D and CVD (Grundy et al., 2005; Lichtenstein et al., 2006; Lichtenstein & Van Horn, 1998; Weinberg, 2004).

The findings from this study show that TG levels increased marginally (4.4%) only for those participants who had the lowest level of TG (<100) at program entry which is well below the cut-off of 150 for a diagnosis of MetS. The effect size was small (0.12) and such minor changes in a healthy population are probably of minor clinical significance (Diehl, 1998). Conversely, overall the mean TG levels reduced by 6.9%, and participants in the highest risk category (>500) showed a 44.1% reduction with a very large effect size (-2.05) (Table 4-4). Of the 1523 participants who had a TG level above the MetS threshold at baseline, 173 participants fell below the threshold by the end of the intervention (Table 4-2). It would seem that a low-fat, plant-based diet, as recommended by the CHIP

intervention, can effectively reduce participants' TG levels for those with elevated TG levels. The findings of this study therefore support the recommendation of several authors who advocate a low-fat eating pattern for reducing TG levels (Barnard et al., 2009; Barnard et al., 2008; Beard et al., 1996; Daubenmier et al., 2007; Diehl, 1998; Englert et al., 2007; D. J. Jenkins et al., 2003; Ornish et al., 1983).

4.4.3.3 High-Density Lipoprotein

It is interesting that significant improvements in the risk factors that constitute MetS were observed in this study, except for HDL which significantly decreased (Table 4-2). Clearly, the change in the number of individuals meeting the HDL criteria for MetS demonstrated an opposing trend to the other 4 MetS risk factors, BMI, FBG, BP and TG.

Low levels of HDL, on a population level, are considered to be an important cardiovascular risk factor (Gordon & Rifkind, 1989). A strong inverse association between low HDL levels and the risk of cardiovascular events has been consistently observed in epidemiological studies (Brinton, Eisenberg, & Breslow, 1990; Rader, 2006). Low levels of HDL are included in the criteria for MetS. HDL has been shown to exhibit several potential anti-atherogenic properties, the most important of which is reverse cholesterol transport or cholesterol efflux which is the removal of cholesterol from peripheral tissues to the liver for catabolism (von Eckardstein, Nofer, & Assmann, 2001). More recently HDL has been shown to have many anti-atherogenic properties which include anti-inflammatory, anti-apoptotic, nitric oxide promoting, prostacyclin-stabilizing, and platelet-inhibiting functions.(Jensen, Rimm, Furtado, & Sacks, 2012; Leite & Fernandez, 2010) Indeed, it has been postulated that the anti-inflammatory properties of HDL and its ability to protect LDL from oxidation may be just as important as its role in reverse cholesterol transport.(Roberts, Ng, Hama, Eliseo, & Barnard, 2006)

However, HDL has been described as a “chameleon-like” lipoprotein as in its normal state it is anti-inflammatory but it can become pro-inflammatory during acute inflammatory responses. In its anti-inflammatory state the enzymes contained in HDL exhibit anti-atherogenic properties by destroying the oxidised lipids derived from LDL. In its pro-inflammatory state HDL, rather than inhibiting LDL oxidisation, enhances LDL oxidisation (Ansell et al., 2003; Navab et al., 2006; Navab, Van Lenten, Reddy, & Fogelman, 2001).

The result of this study which suggests that HDL levels may not be helpful for predicting cardiovascular risk in individuals consuming a low-fat, plant-based diet, is supported by other epidemiological and clinical studies. Over 30 years ago, Connor observed that the Tarahumara Indians of Mexico, who consumed a largely plant-based diet comprising approximately 12% fat, 13% protein (predominantly from corn and beans) and 85% carbohydrate, had very low rates of vascular disease and blood lipids, including HDL (Connor et al., 1978). However, blood lipids, including HDL, were observed to significantly increase after only five weeks when their traditional diet was changed to a Western diet (McMurry, Cerqueira, Connor, & Connor, 1991). It was argued that the increase in HDL was the “normal response to a high-fat diet” and that low-HDL in concert with low-LDL in a low-fat diet are associated with a low risk of coronary disease.

Other epidemiological findings also show that individuals who consume a plant-based diet are at lower risk of CVD and T2D, despite having lowered HDL levels (Ferdowsian & Barnard, 2009; Roberts et al., 2006). In addition, the Lifestyle Heart Trial, (Ornish et al., 1998) which incorporated a plant-based diet with less than 10% fat, showed a 7.9% improvement in measured coronary artery percent diameter stenosis after five years despite a 13% reduction in HDL. Similarly, individuals with diagnosed CVD and a

recommendation for bypass surgery who participated in the Pritikin residential program which recommends a plant-based diet (< 10% fat), experienced a 16% reduction in HDL but decreases in symptomatic angina (Barnard, Guzy, Rosenberg, & O'Brien, 1983). These patients averted surgery for more than five years after program entry despite sustained lowered HDL levels.

Lifestyle interventions that promote a plant-based diet have been shown to decrease HDL cholesterol levels despite concomitant reductions in atherosclerotic plaque and cardiac events (Esselstyn et al., 1995; Ornish, 1998b; Ornish et al., 1990a). This may be understood from the perspective that when all cholesterol subfractions are lowered there is not the demand for reverse cholesterol transport. In this study, there was a mean reduction in TC of 10.9% and LDL of 13.0% which means that the ratios of TC/HDL (3.53 at baseline to 3.45 post-intervention) and LDL/HDL (2.39 at baseline to 2.28 post-intervention) have actually improved.

Meta-analysis of the role of HDL in cardiovascular disease has indicated that increasing the levels of circulating HDL does not reduce the risk of coronary disease events, coronary heart disease deaths, or total deaths (Briel et al., 2009; Despres, 2013). In a review of published randomised clinical trials and observational studies Ferdowsian and Barnard (2009) found that decreased HDL associated with low-fat plant-based diets is not associated with poor cardiovascular health. Individuals who follow a plant-based eating pattern that is free from exogenous cholesterol typically have lower blood concentrations of all cholesterol subfractions, including HDL cholesterol. However, these individuals do not have compromised cardiovascular health and are not at increased risk of T2D (Ferdowsian & Barnard, 2009). Further in countries where a low-fat complex-carbohydrate diet is typically consumed population levels of HDL are low when compared

to countries consuming a typical Western diet however the rate of atherosclerosis is also very low (Barnard & Wen, 1994; Brinton et al., 1990)

There is also growing evidence that lifestyle interventions may be able to modulate the inflammatory or anti-inflammatory properties of HDL. In patients at risk of CVD, the anti-inflammatory properties of HDL improved following lifestyle modification, despite reductions in HDL (Roberts & Barnard, 2005). In another study, the HDL shifted from pro-inflammatory to anti-inflammatory in obese men, with MetS, who underwent a three-week intervention involving a low-fat, high- fibre diet and exercise (Roberts et al., 2006). More specifically, consumption of saturated fat reduces the anti-inflammatory potential of HDL, but consumption of polyunsaturated fat has been shown to increase it (Nicholls et al., 2006). The regulation and function of HDL appears more complex than originally thought, although high HDL levels are associated with reduced CVD at a population level, at an individual level HDL function may be more important than the actual HDL levels. (Khera et al., 2011).

Given the findings of this study, with regard to HDL and the literature mentioned above it would seem appropriate to challenge the inclusion of HDL as part of the MetS assemblage, especially when applied to vegetarian populations and when assessing the effectiveness of plant-based interventions targeting chronic disease. It may be more appropriate for the guidelines for diets targeting a reduction in atherosclerosis to emphasise the lowering of LDL rather than being concerned about HDL reduction (Brinton et al., 1990).

4.4.4 Fasting Plasma Glucose

In this study, significant reductions in mean FPG levels for all risk categories were observed. This was most clearly seen with those participants who had an FPG levels

indicative of T2D at program entry (>125 mg/dL) as this group showed a 19.9% reduction over the 30-day period of the intervention. Of the 525 participants who had an FPG indicative of T2D at baseline, 30 days later this number had reduced to 301 participants who had an FPG indicative of T2D , a 42.7% reduction.

The Finnish Diabetes Prevention Study Group showed that a lifestyle intervention emphasising a low-fat, high-fibre diet and exercise significantly reduced the incidence of T2D, particularly in participants in the highest risk categories (Tuomilehto et al., 2001). Similarly, the Diabetes Prevention Program Research Group found that for people with pre-diabetes, lifestyle intervention was twice as effective as Metformin for preventing progression to established diabetes. Compared to a placebo, lifestyle intervention resulted in a 58% reduction in the incidence of T2D as compared to only 31% for Metformin (Knowler et al., 2002). Another randomised controlled trial also showed a low-fat, plant-based diet to be more effective than a conventional diabetic treatment diet (2003 American Diabetes Association guidelines) for improving glycaemic control (Barnard et al., 2009; Barnard, Joshua, David, Gabrielle, & et al., 2006).

Significant reductions in FPG have been observed in professionally delivered CHIP interventions (Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Diehl, 1998; Englert et al., 2007), in the Multicentre Lifestyle Demonstration Project (Pischke et al., 2006) and in residential programs offered at the Pritikin Longevity Centre (Barnard et al., 1994; Barnard et al., 1982; Barnard, Massey, et al., 1983). The results of this study indicate that CHIP interventions delivered by volunteers can also effectively reduce FPG levels, particularly in those participants in the highest risk categories.

4.4.5 Systolic and Diastolic Blood Pressure

This study showed significant reductions in BP in just 30 days. These reductions were particularly noteworthy for those participants who had the highest blood pressures at baseline. Those participants who had a SBP of > 160, at baseline, experienced a 14% reduction in SBP, while those participants with a DBP of > 100, at baseline, experienced a 17% reduction in DBP with very large effect sizes. Similar results have been observed in reducing BP in the professional delivery of the CHIP intervention (Table 4-3).

Elevated BP is widely recognised as an important risk factor for strokes(Goldstein et al., 2011) and CVD (Lieb et al., 2013) and hence is included as a factor in the criteria for the MetS (Alberti et al., 2009) and as a component in calculating in the Framingham Risk Score (National Cholesterol Education Program, 2002; Wilson et al., 1998).

As discussed in the previous chapter there are a number of challenges in obtaining accurate BP measurement, however in this study the use of trained personnel, normally nurses, for the measurement of blood pressure and the large numbers of participants, (4,560 for SBP and 4,552 for DBP) should ameliorate some of the measurement errors.

The finding from this study that the lifestyle intervention can significantly reduce BP is reflected in a number of other lifestyle interventions including the PREMIER clinical trial (Appel et al., 2003), the McDougall program (McDougall et al., 1995), the Daniel diet (Bloomer et al., 2010; Trepanowski, Kabir, Alleman, & Bloomer, 2012), the Nordic diet (Adamsson et al., 2011) and the DASH diet (Appel et al., 2006). Significant reductions in blood pressure have also been observed in residential programs involving low-fat, plant-based diets such as the Pritikin Longevity Centre (Roberts, Vaziri, & Barnard, 2002; Weber et al., 1983) and the Ornish Program (Ornish et al., 1983).

4.4.6 Behaviour change

The results presented above show that participants in CHIP demonstrate improved biometrics post-intervention. The aim of the CHIP intervention is to improve health behaviour. Merrill, Aldana, Greenlaw, Diehl, et al. (2008) have shown that CHIP interventions can significantly improve nutrition and physical activity behaviours for up to 18 months. That use of the principles of the Theory of Planned Behaviour (TPB), in the development of CHIP may explain some of this improvement. TPB suggests that a person's intentions and subsequent behaviours are dictated by three sets of beliefs: behaviour beliefs leading to attitudes, normative beliefs leading to subjective norms and control beliefs leading to perceived behaviour control (Ajzen, 2011). CHIP attempts to influence all of these beliefs.

With regards to behavioural beliefs, the CHIP intervention endeavours to influence the participant's attitudes through education. Participants come away from CHIP well-informed about health and how to achieve it. Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al. (2005) showed that CHIP participants significantly improved their health knowledge during CHIP interventions. The answer the question "What is the consequence of performing this behaviour?" is presented with support from current research, using pre-recorded video presentations delivered by trustworthy presenters. Initially participants are presented with the negative consequences of an unhealthy lifestyle but they are then presented with the positive consequences of adopting a healthy lifestyle. The presentation of this information impacts the participants' behavioural belief and attitudes.

The CHIP intervention is presented in a group setting. The group dynamics of CHIP create a socially supportive environment as people journey together, problems solved

together overcome obstacles together, become accountable to each other and celebrate with each other. As observed earlier one of the reasons that male participants get better results than female participants could be that they are more likely to participate in the CHIP intervention with their partner. This group support influences participants Subjective Norms.

The health assessment performed at baseline and postintervention provide the CHIP participants with feedback on the changes in their biometric scores. The majority of the CHIP participants in these biometric scores improve during the intervention helping participants to realise that they can make a significant difference to their own health in as little as 30 days. This helps to increase the participant's belief that they can succeed in making lifestyle changes. CHIP encourages participants to believe that they can take some measure of control over their own health and the 30 days is pitched as a "self-experiment" in which the participant is challenged to give it a go and then observe the results. Participants in CHIP have been shown to experience improved biometrics but also experience improved sense of well-being, quality of life (Aldana, Whitmer, et al., 2006) and psychological health (Thieszen et al., 2011). These factors may have a positive impact on the participants Perceived Behavioural Control.

The fact that CHIP impacts the three sets of beliefs, as proposed by the Theory of Planned Behaviour, that impact intentions and thus action may contribute to the biometrics changes demonstrated in this study. More research is need to determine the factors that influence the behavior change that lead to the changes observed in the CHIP studies.

4.4.7 Volunteer vs. professional facilitators

A major focus of this dissertation is the efficacy of volunteer delivery CHIP interventions. This chapter has shown that volunteers, individuals who donate their time, can be effective in the delivery of CHIP interventions. Alter (2007) asserted the common perception that lifestyle modification programs that are intensely supported by trained, experienced health care professionals can be expected to have greater efficacy than those delivered by volunteers. However a comparison between the results from this study and the results obtained from published articles examining CHIP delivered by paid professional facilitator's shows similar outcomes (Table 4-3).

While no study of the effectiveness of the CHIP lifestyle intervention when delivered by volunteers had been published prior to this dissertation, there is evidence in the literature that the volunteers have been effectively utilised in the delivery of other community-based lifestyle modification programs. Volunteers, from Faith in Action groups, were successfully used to deliver the Strong for Life program to elderly people. It was found that these trained volunteers could safely deliver programs to a community group of frail older persons (Etkin et al., 2006). Further, Schneider et al (2007) concluded that volunteers can successfully deliver health promotion programs which allows for wider program dissemination than is possible using only professional leaders.

An obvious advantage of utilising volunteer facilitators is the cost saving when compared to utilising paid facilitators (Parkin & McKeganey, 2000). However, there may be other advantages in using volunteer, community-based facilitators to deliver health programs. It has been observed that for a community program to be successful, at least one person must champion the program (Steckler & Goodman, 1989). Volunteers, who are members of the local community, already have relationships with individuals in the

community and these relationships can be utilised to encourage members of that community to be proactive about their health and make necessary life changes (Kong, 1997). This means that they are often better placed than paid professionals to champion a program. As Kong (1997) suggests: a passionate volunteer can incite his peers to action. In addition, friendship and social networks which are tapped into by peer volunteers, have been shown to be protective when contributing towards positive health behaviour (Milburn, 1995).

Volunteering has also been shown to have significant benefits for those who volunteer (Grossman & Furano, 1999). A study of older volunteers reported that they felt they were "a great deal better off" for having volunteered and that the benefits of volunteering were also felt by their families (Morrow-Howell, Hong, & Tang, 2009). Volunteering in peer educational programs has been found to be a positive and rewarding experience (Karwalajtys et al., 2009). It has been found that volunteers in peer educational programs, targeting drug and sexual behaviour, make many positive lifestyle changes as a result of the information they learn while volunteering (Parkin & McKeganey, 2000).

Klein, Sondag, and Drolet (1994) found that the motivations for volunteering in university student peer health educators ranged from altruistic motives such as wanting to help others to self-efficacy beliefs, and the need to satisfy personal health goals. The motivation of volunteers in this study was probably similarly varied.

Yuasa et al (2007) observe that if a significant initial investment is made in social and human capital, other forms of capital, such as financial, physical and natural, are not always necessary. Using volunteers can maximise the use of social and human capital (Gratton & Ghoshal, 2003). The utilisation of voluntary facilitators can dramatically

increase the scope and efficacy of lifestyle modification programs while keeping costs to a minimum.

This study indicates that volunteers can be effective in the facilitation of lifestyle intervention programs.

4.5 Conclusion

The overall and stratified results outlined in this chapter provide evidence that the CHIP intervention, when delivered by volunteers to free-living participants in their community, can effect statistically significant reductions in chronic disease risk factors. The clinical significance of the results are indicated by the significant reductions observed in the Framingham Risk Score and MetS status from baseline to post-intervention. The results observed in this study showed similar outcomes to those reported in the literature for CHIP interventions delivered by paid, professional facilitators.

Chapter 5 Results part 2: The influence of selected participant characteristics on the effectiveness of the CHIP lifestyle interventions

5.1 Introduction

In the previous chapter, the first research question of this study which focused on the effectiveness of the volunteer-delivered, community-based CHIP lifestyle intervention, was addressed. In this chapter, the second research question: "What is the impact of selected participant factors, including age, gender, marital status, religious affiliation, previous history, family history and body weight on the outcomes achieved by participants in the CHIP intervention?" is examined.

To address the second research question, this chapter presents an analysis of the impact the selected participant factors including age, gender, marital status, religious affiliation, previous history, family history and body weight on changes in the outcome measures of lipid profile, BMI, FPG and BP. This chapter is presented in six sections which commences with this introduction. The second section presents an overview of the statistical methods used in this chapter. The third section examines the impact of the selected participant factors on the outcome measures. In the fourth section all the participant factors are included in regression models to determine their relative influences on the outcome measures and to control for the influence of selected factors on the outcomes of other factors. The fifth section presents a discussion of the key findings with reference to the literature and the chapter ends with a concluding statement.

5.2 Statistical analyses

In order to analyse participant factors, the participants in the study were grouped into appropriate categories:

Gender	Male, Female
Age	Decadal age groups
Marital Status	Single, Married, Divorced, Widowed
Religious Affiliation	Seventh-day Adventist, non-Seventh-day Adventist
Body Mass Index	WHO BMI Categories
Family History	family history, no family history
Previous History	previous history, no previous history

For each of the factors, differences in the biometrics at baseline between the categories were assessed using appropriate t-testing or ANOVA. Next the change in biometrics from baseline to post-intervention within the same category was examined using paired t-tests. If the change in biometrics was found to be significant within categories, the difference in change between categories was examined using appropriate t-tests or ANOVA. When a significant difference between the categories was identified, Bonferroni's post hoc analyses were performed to identify where the differences occurred. Cohen's d was calculated to show the effect size where appropriate.

5.3 Participant factors

5.3.1 Gender

Of the 5043 participants, who provided their gender, 1690 (33.5%) were male and 3353 (66.5%) were female. The age of male participants (57.86 ± 12.97) was slightly older than the female participants (56.98 ± 12.91) [t (5040) = 2.240, p = 0.025].

5.3.1.1 *Gender differences in biometrics at baseline*

At baseline, the male participants had significantly lower TC (10.8%), HDL (28.2%) and LDL (5.5%) but significantly higher TG (10.4%), FPG (12.5%), SBP (2.6%) and DBP (3.1%) than the female participants. There was no significant difference between the male and female participants' BMI at baseline (Table 5-1).

Table 5-1 Baseline biometric by gender

	Biometric by Gender										
	At Baseline										
	Male			Female			% Diff				
	N	Mean	SD	*N*	Mean	SD		t	df	Sig	Cohen's d
TC	1672	180.2	40.8	*3316*	199.7	41.00	-10.8	-15.86	4986	<0.001	-0.48
HDL	1671	46.0	21.4	3316	58.9	26.38	-28.2	-18.63	4024	<0.001	-0.54
LDL	1630	123.5	60.4	3270	133.7	61.51	-8.2	-5.45	4898	<0.001	-0.17
TG	1670	153.2	102.6	3312	137.3	82.16	10.4	5.52	2776	<0.001	0.17
FPG	1655	105.4	32.2	3275	92.2	26.82	12.5	6.75	2839	<0.001	0.45
BMI	1672	31.1	6.7	3247	31.0	7.62	0.1	0.17	3761	0.867	0.01
SBP	1667	135.4	19.0	3295	131.9	19.66	2.6	6.13	4960	<0.001	0.18
DBP	1667	81.5	11.0	3291	79.0	11.65	3.1	7.31	4956	<0.001	0.22

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.1.2 *Changes baseline to post-intervention for male and female participants*

There were significant (p<0.001) reductions from baseline to post-intervention in all eight biometric risk factors for both the male and female participants (Table 5-2 and Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-3).

Table 5-2 Changes in biometrics for male participants

	Baseline to Post-Intervention Changes for Male Participants											
	Baseline			Post-intervention								
	N	Mean	SD	N	Mean	SD	% Diff	Change	t	df	Sig	Cohen's d
TC	1563	180.80	40.80	1563	157.03	32.22	13.2	23.8	34.82	1562	<0.001	0.65
HDL	1561	46.13	1.54	1561	42.63	19.84	7.6	3.5	16.65	1560	<0.001	0.33
LDL	1512	124.61	61.32	1512	104.28	50.82	16.3	20.3	28.02	1511	<0.001	0.36
TG	1560	153.84	102.24	1560	136.31	80.40	11.4	17.5	9.90	1559	<0.001	0.19
FPG	1547	105.54	32.36	1547	96.87	22.27	8.2	8.7	16.20	1546	<0.001	0.32
BMI	1541	31.04	6.73	1541	29.96	6.36	3.5	1.1	44.88	1540	<0.001	0.17
SBP	1542	135.72	18.61	1542	128.23	16.07	5.5	7.5	19.55	1541	<0.001	0.43
DBP	1542	81.59	10.98	1542	76.79	9.98	5.9	4.8	19.53	1541	<0.001	0.46

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-3 Changes in biometrics for female participants

	Baseline to Post-Intervention Changes for Female Participants											
	Baseline			Post-intervention								
	N	Mean	SD	N	Mean	SD	% Diff	Change	t	df	Sig	Cohen's d
TC	3090	200.0	40.74	3090	179.7	36.81	10.1	20.3	42.44	3089	<0.001	0.52
HDL	3091	59.2	26.59	3091	53.8	23.80	9.2	5.4	32.98	3090	<0.001	0.22
LDL	3036	134.3	62.10	3036	118.9	56.18	11.5	15.5	31.62	3035	<0.001	0.26
TG	3088	138.1	82.70	3088	130.3	71.36	5.6	7.8	7.83	3087	<0.001	0.10
FPG	3038	99.1	26.79	3038	93.8	20.24	5.3	5.3	17.81	3037	<0.001	0.22
BMI	2974	31.0	7.58	2974	30.6	7.32	1.4	0.4	56.90	2973	<0.001	0.06
SBP	3016	131.9	19.56	30.16	126.0	29.18	4.5	6.0	11.41	3015	<0.001	0.24
DBP	3014	79.1	11.64	3014	75.2	9.87	5.0	3.9	20.10	3013	<0.001	0.36

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.1.3 *Difference in change of biometrics from baseline to post-intervention between male and female participants*

The males experienced significantly greater reductions in TC, LDL, TG, FPG, BMI and BP than the female participants, but a significantly lower reduction in HDL. However the effect size, as shown by Cohen's d, was small (Table 5-4).

Table 5-4 Difference in change pre- to post-intervention between male and female

	Change baseline to post-intervention between male and female											
	Male			Female								
	N	Mean	SD	N	Mean	SD	Difference	% Diff	t	df	Sig	Cohen's d
TC	1563	23.78	27.00	3090	20.29	26.57	3.49	14.7	4.21	4651	<0.001	0.13
HDL	1561	3.50	8.30	3091	5.41	9.13	-1.91	-54.6	-7.19	3406	<0.001	-0.22
LDL	1512	20.36	28.26	3036	15.46	26.93	4.90	24.1	5.70	4546	<0.001	0.18
TG	1560	17.53	69.94	3088	7.79	55.26	9.74	55.6	4.80	2568	<0.001	0.16
FPG	1547	8.68	21.08	3038	5.29	16.38	3.39	39.1	5.54	2519	<0.001	0.18
BMI	1540	1.08	0.93	2972	0.93	0.80	0.15	13.9	5.45	2723	<0.001	0.17
SBP	1542	7.49	10.79	3016	5.95	31.61	1.54	20.6	1.97	4556	0.049	0.07
DBP	1542	4.80	12.01	3014	3.90	13.49	0.90	18.8	2.70	4554	0.007	0.07

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.1.4 Summary

At baseline, the male participants had lower TC, HDL and LDL but higher TG, FPG, SBP and DBP than the female participants. There was no difference between male and female participants in baseline BMI. Both male and female participants achieved significant changes during intervention in all biometric scores but males achieved greater improvements in risk factor status with greater reductions in TC, LDL, TG, FPG, BMI, SBP and DBP but less reduction in HDL.

5.3.2 Age

Age data were available on 5042 participants. The mean age was 57.29 $\pm$ 12.91 years (range = 10 – 100 years).

The participants were grouped into decadal age categories. However, due to low numbers, all participants under the age of 30 were grouped into a single category, as were those over the age of 80 (Table 5-5).

Table 5-5 Age categories used for analysis

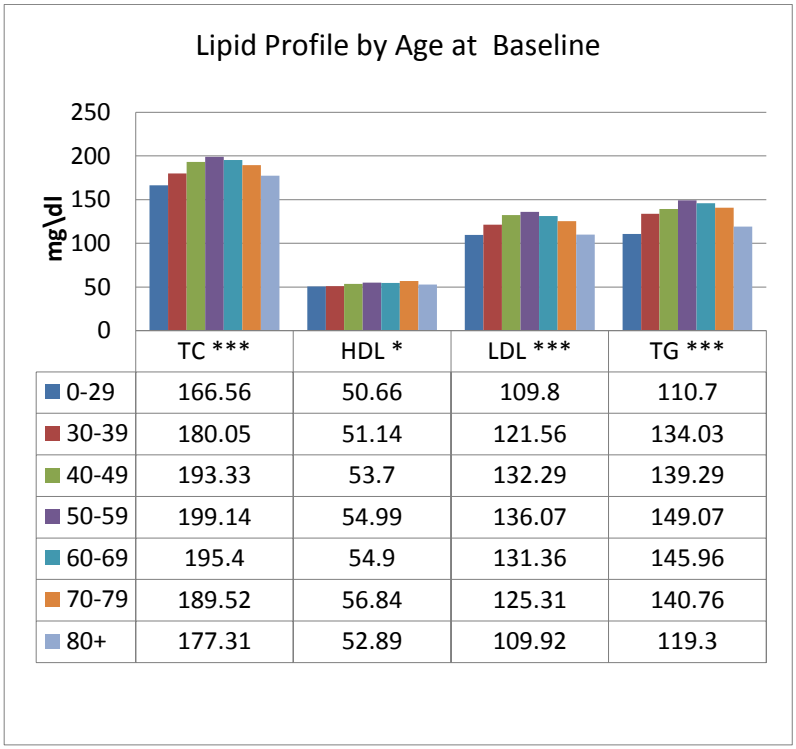
Age Range	Frequency	Percent
0 – 29	158	3.1
30-39	283	5.6
40-49	799	15.8
50-59	1549	30.7
60-69	1429	28.3
70-79	680	13.5
>80	144	2.9
Total	5042	99.9

5.3.2.1 Biometrics by age category at baseline

Using one-way ANOVA, significant differences between the participants in various age categories at baseline were identified: TC [$F(6, 4980) = 25.90$, $p < 0.001$], LDL [$F(6, 4892) = 9.66$, $p < 0.001$], TG [$F(6, 4974) = 7.22$, $p < 0.001$], for FPG [$F(6, 4922) = 16.04$, $p < 0.001$] BMI [$F(6, 4911) = 20.56$, $p < 0.001$] SBP [$F(6, 4954) = 92.67$, $p < 0.001$] and DBP [$F(6, 4950) = 1608$, $p<0.001$] and HDL [$F(6, 4979) = 2.70$, $p < 0.013$]

(Figure 5-3 and Figure 5-4).

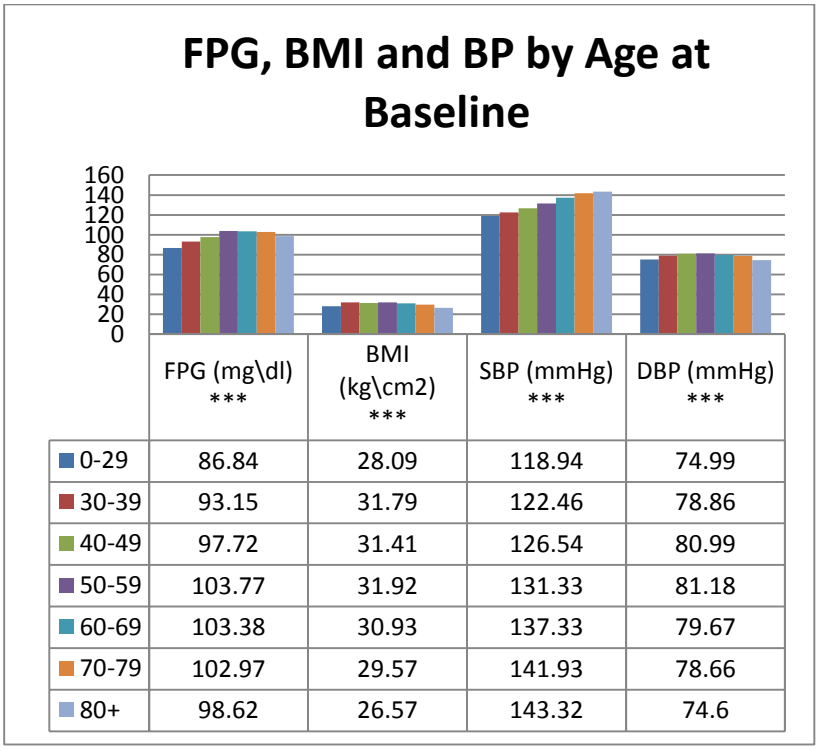
Post Hoc analysis identified that baseline TC, LDL, TG, FPG, BMI and DBP was lower in the younger and older age groups, peaking in the 50-59 age group. HDL levels rose with age peaking in the 70-79 age group. SBP continued to rise with age.



	TC ***	HDL *	LDL ***	TG ***
0-29	166.56	50.66	109.8	110.7
30-39	180.05	51.14	121.56	134.03
40-49	193.33	53.7	132.29	139.29
50-59	199.14	54.99	136.07	149.07
60-69	195.4	54.9	131.36	145.96
70-79	189.52	56.84	125.31	140.76
80+	177.31	52.89	109.92	119.3

*Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001

Figure 5-1 Lipid profile at baseline by age



	FPG (mg\dl) ***	BMI (kg\cm2) ***	SBP (mmHg) ***	DBP (mmHg) ***
0-29	86.84	28.09	118.94	74.99
30-39	93.15	31.79	122.46	78.86
40-49	97.72	31.41	126.54	80.99
50-59	103.77	31.92	131.33	81.18
60-69	103.38	30.93	137.33	79.67
70-79	102.97	29.57	141.93	78.66
80+	98.62	26.57	143.32	74.6

*Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001

Figure 5-2 Distribution of baseline FPG, BMI and BP by age

5.3.2.2 *Baseline to post-intervention changes within age groups*

With the exception of TG and FPG in the 0-29 age category significant change, was observed, baseline to post-intervention, for all age groups in all biometrics, at better than p = 0.05. Effect size ranged from small to moderate with the largest effect size for all age groups being for TC and the smallest effect sizes being for BMI and TG (Table 5-6 to Table 5-12).

Table 5-6 Biometric changes from baseline to post-intervention for the 0-29 age group

	Baseline to Post-Intervention Change 0-29 Age Category									
	Baseline		Post-intervention							
	Mean	SD	Post	SD	Change	%	t	df	p	Cohen's d
TC	166.8	30.64	151.5	30.72	15.3	9.2	7.51	143	<0.001	0.50
HDL	50.3	23.98	48.1	23.86	2.2	4.4	2.52	143	0.013	0.09
LDL	109.9	46.37	95.7	37.03	14.3	13.0	6.63	139	<0.001	0.34
TG	109.6	68.14	107.2	63.91	2.4	2.2	0.85	143	0.398	0.04
FPG	86.3	11.05	84.3	13.80	2.0	2.3	1.90	141	0.06	0.16
BMI	28.7	8.06	27.9	7.65	0.8	2.6	9.17	137	<0.001	0.10
SBP	118.7	15.15	114.0	12.55	4.7	4.0	4.58	137	<0.001	0.34
DBP	74.1	13.10	70.9	9.04	3.3	4.4	3.32	137	0.001	0.30

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-7 Biometric changes from baseline to post-intervention for the 30-39 age group

	Baseline to Post-Intervention Change 30-39 Age Category									
	Baseline		Post-intervention							
	Mean	SD	Post	SD	Change	%	t	df	p	Cohen's d
TC	182.3	40.33	163.4	37.69	19.0	10.4	12.21	254	<0.001	0.49
HDL	52.0	24.49	47.6	22.86	4.4	8.4	7.84	254	<0.001	0.18
LDL	124.5	57.56	110.1	52.57	14.4	11.6	9.23	251	<0.001	0.26
TG	135.9	97.30	121.8	70.63	14.1	10.4	3.11	254	0.002	0.17
FPG	92.9	26.85	87.8	15.60	5.1	5.5	4.55	252	<0.001	0.24
BMI	31.9	8.90	30.9	8.51	1.0	3.3	15.17	247	<0.001	0.12
SBP	122.4	17.19	117.9	13.25	4.5	3.6	5.78	251	<0.001	0.29
DBP	79.1	10.85	75.9	9.17	3.2	4.1	5.84	251	<0.001	0.32

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-8 Biometric changes from baseline to post-intervention for the 40-49 Age Group

	Baseline to Post-Intervention Change 40-49 Age Category									
	Baseline		Post-intervention							
	Mean	SD	Post	SD	Change	%	t	df	P	Cohen's d
TC	193.5	39.10	169.4	33.90	24.1	12.5	24.24	720	<0.001	0.66
HDL	53.6	24.45	48.4	20.78	5.3	9.8	15.25	719	<0.001	0.23
LDL	132.3	60.99	112.8	51.28	19.5	14.7	18.23	699	<0.001	0.35
TG	141.1	97.78	128.0	78.74	13.1	9.3	5.41	720	<0.001	0.15
FPG	97.6	26.37	92.4	19.65	5.2	5.3	8.13	709	<0.001	0.23
BMI	31.4	8.00	30.3	7.71	1.0	3.3	30.09	705	<0.001	0.13
SBP	126.5	16.51	119.9	14.91	6.6	5.2	13.42	715	<0.001	0.42
DBP	81.1	11.26	76.6	9.95	4.5	5.6	12.28	714	<0.001	0.43

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-9 Biometric changes from baseline to post-intervention for the 50-59 Age Group

	Baseline to Post-Intervention Change 50-59 Age Category									
	Baseline		Post-intervention							
	Mean	SD	Post	SD	Change	%	t	df	P	Cohen's d
TC	200.0	40.96	175.6	37.31	23.4	11.8	32.10	1442	<0.001	0.60
HDL	55.0	25.78	50.0	23.36	5.0	9.1	22.64	1441	<0.001	0.20
LDL	136.3	62.81	117.7	56.21	18.6	13.6	24.82	1402	<0.001	0.31
TG	149.0	95.18	136.1	75.93	12.9	8.7	7.32	1439	<0.001	0.15
FPG	103.7	32.44	96.3	22.38	7.5	7.2	13.82	1412	<0.001	0.27
BMI	31.9	7.58	30.9	7.29	1.1	3.3	46.59	1407	<0.001	0.14
SBP	131.5	18.24	126.3	33.18	5.3	4.0	5.13	1412	<0.001	0.20
DBP	81.2	11.13	77.0	9.69	4.2	5.2	15.25	1411	<0.001	0.40

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-10 Biometric changes from baseline to post-intervention for the 60-69 Age Group

	Baseline to Post-Intervention Change 60-69 Age Category									
	Baseline		Post-intervention							
	Mean	SD	Post	SD	Change	%	t	df	P	Cohen's d
TC	196.1	42.92	174.9	39.49	21.2	10.8	29.17	1315	<0.001	0.51
HDL	55.5	25.84	50.6	23.03	4.9	8.9	20.44	1316	<0.001	0.20
LDL	132.8	69.92	115.8	58.88	17.0	12.8	22.30	1296	<0.001	0.26
TG	146.7	86.33	136.7	73.87	10.0	6.8	6.14	1314	<0.001	0.12
FPG	103.3	28.48	96.2	20.49	7.1	6.9	15.02	1301	<0.001	0.29
BMI	31.0	6.64	30.0	6.36	1.0	3.2	44.33	1267	<0.001	0.15
SBP	137.3	18.93	129.8	16.13	7.5	5.5	16.94	1277	<0.001	0.43
DBP	79.84	10.84	75.5	9.85	4.3	5.4	15.00	1278	<0.001	0.42

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-11 Biometric changes from baseline to post-intervention for the 70-79 Age Group

	Baseline to Post-Intervention Change 70-79 Age Category									
	Baseline		Post-intervention							
	Mean	SD	Post	SD	Change	%	t	df	p	Cohen's d
TC	190.1	43.35	171.4	39.20	18.6	9.8	17.78	636	<0.001	0.45
HDL	57.0	28.84	52.6	26.19	4.4	7.7	11.82	636	<0.001	0.16
LDL	126.5	62.74	112.0	56.16	14.5	11.5	13.08	619	<0.001	0.24
TG	141.9	82.34	133.7	71.40	8.2	5.8	4.49	636	<0.001	0.11
FPG	103.0	27.29	96.8	22.58	6.3	6.1	8.74	628	<0.001	0.25
BMI	29.6	5.91	28.8	5.68	0.9	2.9	30.52	614	<0.001	0.15
SBP	141.8	19.43	133.6	16.46	8.1	5.7	12.33	626	<0.001	0.45
DBP	78.7	12.99	74.0	10.31	4.7	6.0	10.32	626	<0.001	0.40

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

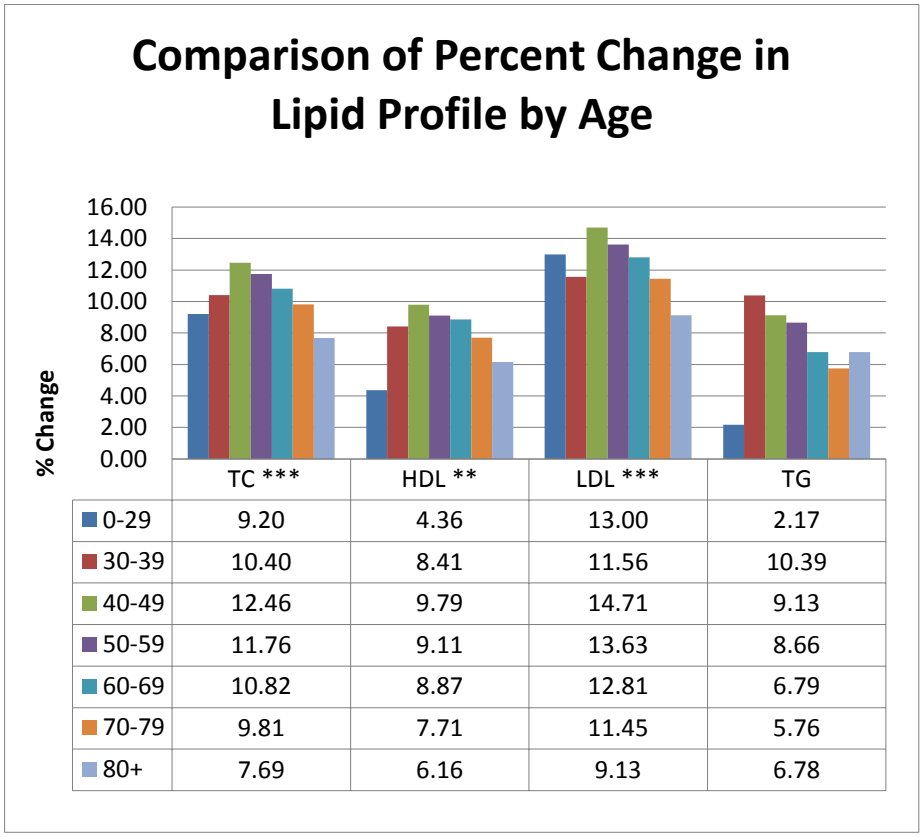
Table 5-12 Biometric changes from baseline to post-intervention for the >80 Age Group

	Baseline to Post-Intervention Change >80 Age Category									
	Baseline		Post-intervention							
	Mean	SD	Post	SD	Change	%	t	df	p	Cohen's d
TC	177.7	38.12	164.0	36.05	13.7	7.7	6.63	136	<0.001	0.37
HDL	53.1	18.54	49.8	16.96	3.3	6.2	4.34	136	<0.001	0.18
LDL	110.2	47.68	100.1	46.75	10.1	9.1	5.57	135	<0.001	0.21
TG	121.0	70.80	112.8	65.61	8.2	6.8	2.24	136	0.027	0.12
FPG	98.8	25.86	95.3	18.78	3.5	3.6	2.76	135	0.007	0.16
BMI	26.6	4.64	26.0	4.50	0.6	2.1	8.94	128	<0.001	0.12
SBP	143.7	21.09	137.4	20.19	6.2	4.3	3.79	133	<0.001	0.30
DBP	74.9	10.38	72.6	10.00	2.3	3.0	2.63	132	0.01	0.22

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.2.3 *Differences in change from baseline to post-intervention in biometric measures between age categories*

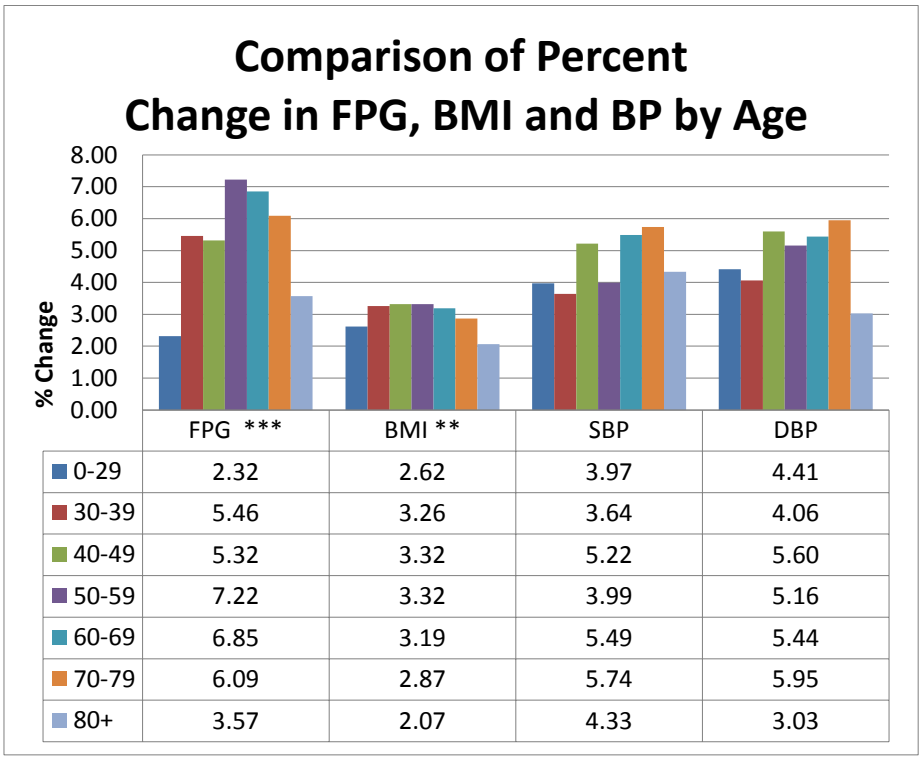
As shown in Figure 5-3 and Figure 5-4 there were significant differences between the various age categories in the change in TC [$F(6, 4646) = 7.24$, $p < 0.001$, LDL, $F(6, 4541) = 4.64$, $p < 0.001$], HDL [$F(6, 4645) = 3.50$, $p = 0.002$], FPG, [$F(6, 4578) = 3.89$, $p = 0.001$] and BMI [$F(6, 4505) = 12.80$ $p = 0.01$]. There was no difference between age categories for change in TG, SBP and DBP.



	TC ***	HDL **	LDL ***	TG
0-29	9.20	4.36	13.00	2.17
30-39	10.40	8.41	11.56	10.39
40-49	12.46	9.79	14.71	9.13
50-59	11.76	9.11	13.63	8.66
60-69	10.82	8.87	12.81	6.79
70-79	9.81	7.71	11.45	5.76
80+	7.69	6.16	9.13	6.78

*Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001

Figure 5-3 Comparison of percent change in lipid profile by age



	FPG ***	BMI **	SBP	DBP
0-29	2.32	2.62	3.97	4.41
30-39	5.46	3.26	3.64	4.06
40-49	5.32	3.32	5.22	5.60
50-59	7.22	3.32	3.99	5.16
60-69	6.85	3.19	5.49	5.44
70-79	6.09	2.87	5.74	5.95
80+	3.57	2.07	4.33	3.03

*Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001

Figure 5-4 Comparison of percent change in FPG, BMI and BP by age category

Post hoc analyses found that largest change in TC, LDL and HDL was achieved in the 40-49 age category. The greatest change in FPG and BMI occurred in the 50-59 age category, although these changes were not significantly higher than the 40-49 age category.

5.3.2.4 Summary

Baseline mean TC, LDL, TG, FPG levels, BMI and DBP tended to be lower in the younger and older age groups, peaking in the 50-59 age group. HDL levels rose with increasing age, peaking in the 70-79 age group. SBP continued to rise with age. Significant changes were observed from baseline to post-intervention in all biometrics in all age categories, except for TG and FPG in the 0-29 age categories. The largest changes in TC, LDL and HDL occurred in the 40-49 age category, and in the 50-59 age category for FPG and BMI, although these changes were not significantly higher than the 40-49 age group.

5.3.3 Gender differences across age ranges

Having observed that there were significant differences in the biometric baseline and change data in age and gender, this section compares the male and female participants across the different age categories.

The number of males and females in each age category is shown in Table 5-13.

Table 5-13 Age range by gender

	Male		Female	
Age Range (years)	Frequency	Percent	Frequency	Percent
0 – 29	56	3.3	102	3.0
30-39	83	4.9	200	6.0
40-49	249	14.7	550	16.4
50-59	504	29.8	1045	31.2
60-69	508	30.1	921	27.5
70-79	240	14.2	440	13.1
80+	50	3.0	94	2.8
Total	1690	100	3352	100

There were only slight differences in the number of male and female participants in each age category, however, these differences are significant [t (5040) = 2.19, p = 0.034].

5.3.3.1 *Differences in the change between baseline and post-intervention biometric measures by age categories and gender*

Two-way ANOVA analysis to examine the combined effect of age and gender, on change baseline to post-intervention, found that there were significant interaction effect between these variables for TC [F(6, 4639) = 2.91, p = 0.008], HDL [F(6, 4639) = 2.25, p = 0.036], LDL [F(6, 4639) = 2.25, p = 0.036], TG [F(6, 4634) = 5.10, p = <0.001] and FPG [F(6, 4634) = 5.10, p = <0.001].

Independent t-tests indicated that change in TC was significantly greater for males in 30-39, 40-49 and >80 age groups (Figure 5-5), change in HDL was significantly lower for

males in the 0-29, 40-49, 50-59 and 60-69 age groups (Figure 5-6). Males demonstrated significant greater change in LDL for all age groups except for the 70-79 category (Figure 5-7) and for TG the change was greater for males in the 30-39, 40-49 and 50-59 age groups (Figure 5-8). Males also showed greater change in FPG in the 0-29, 40-49, 50-59 and 60-69 age groups (Figure 5-9).

There was no significant interaction effect for BMI [$F(6, 4498) = 0.472$, $p = 0.830$] and the mean effect for both gender [$F(1, 4498) = 16.44$, $p = <0.001$] and age was significant, [$F(6, 4498) = 12.84$, $p = <0.001$]. Males exhibited greater change in BMI than did the females and at a younger age than did the females (Figure 5-10).

There was no significant interaction effect for either SBP [$F(6, 4544) = 0.39$, $p = 0.89$] or DBP [$F(6, 4542) = 1.33$, $p = 0.24$]. However neither of the mean effects for gender, SBP [$F(1, 4544) = 0.88$, $p = 0.35$], DBP [$F(1, 4542) = 3.28$, $p = 0.07$] or age SBP [$F(6, 4544) = 1.55$, $p = 0.16$] DBP [$F(6, 4542) = 1.79$, $p = 0.098$] were significant. (Figure 5-11 & Figure 5-12).

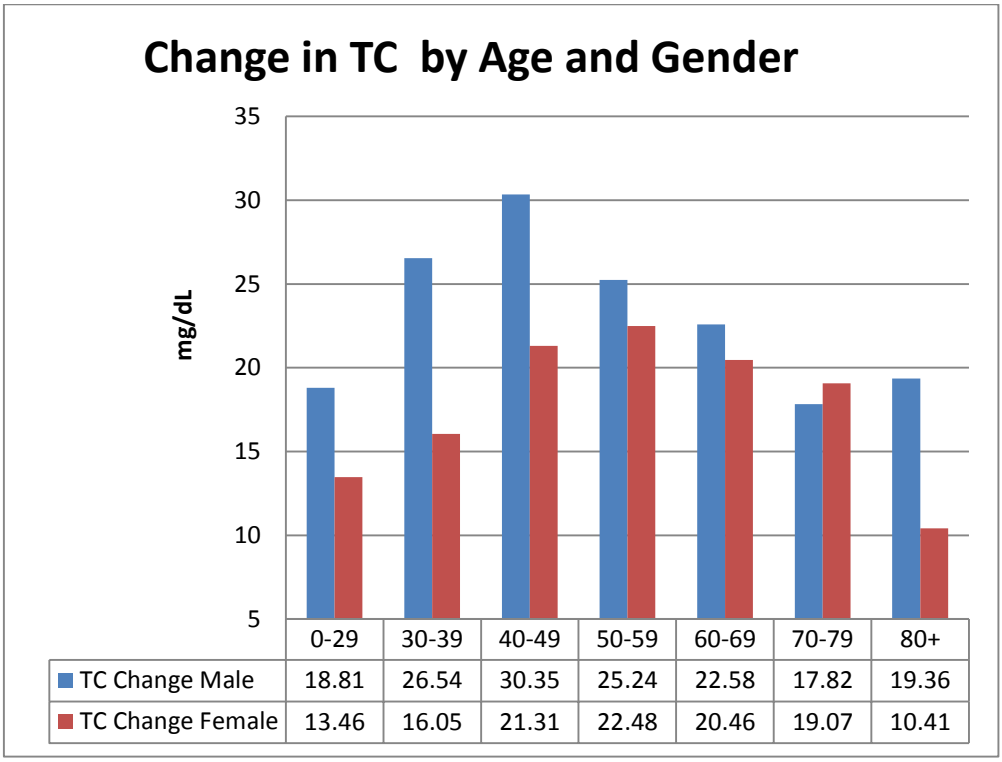


Figure 5-5 Change in TC by age and gender

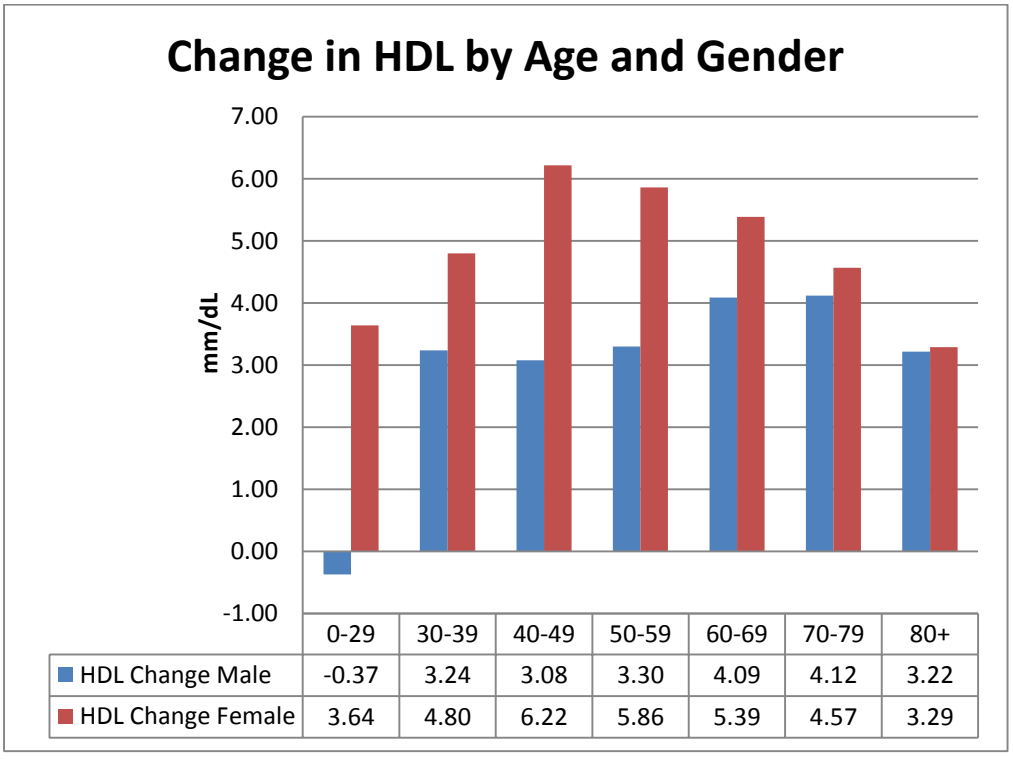


Figure 5-6 Change in HDL by age and gender

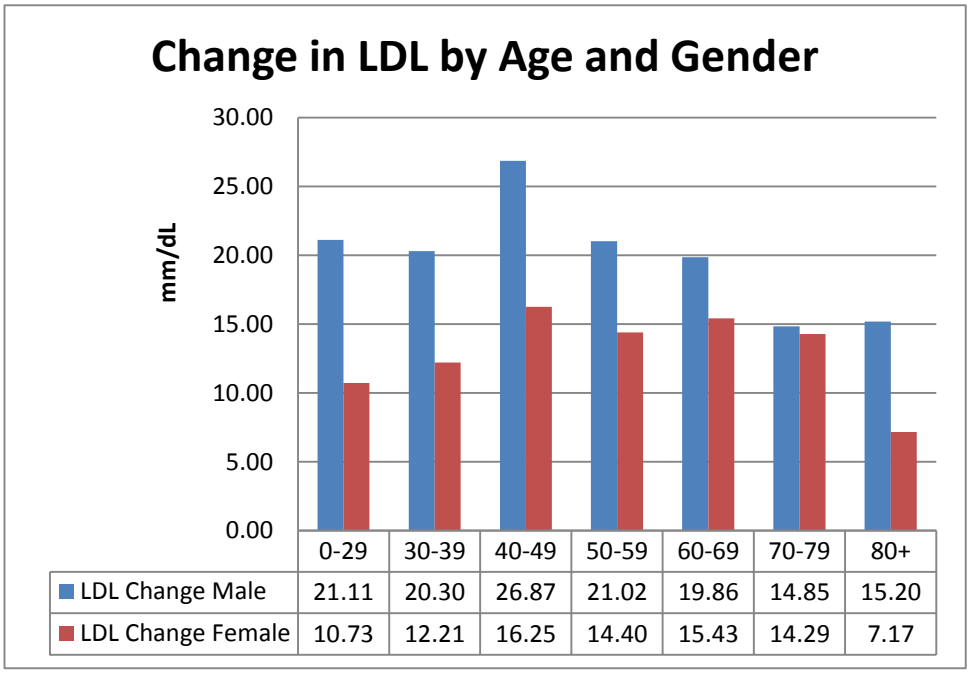


Figure 5-7 Change in LDL by age and gender

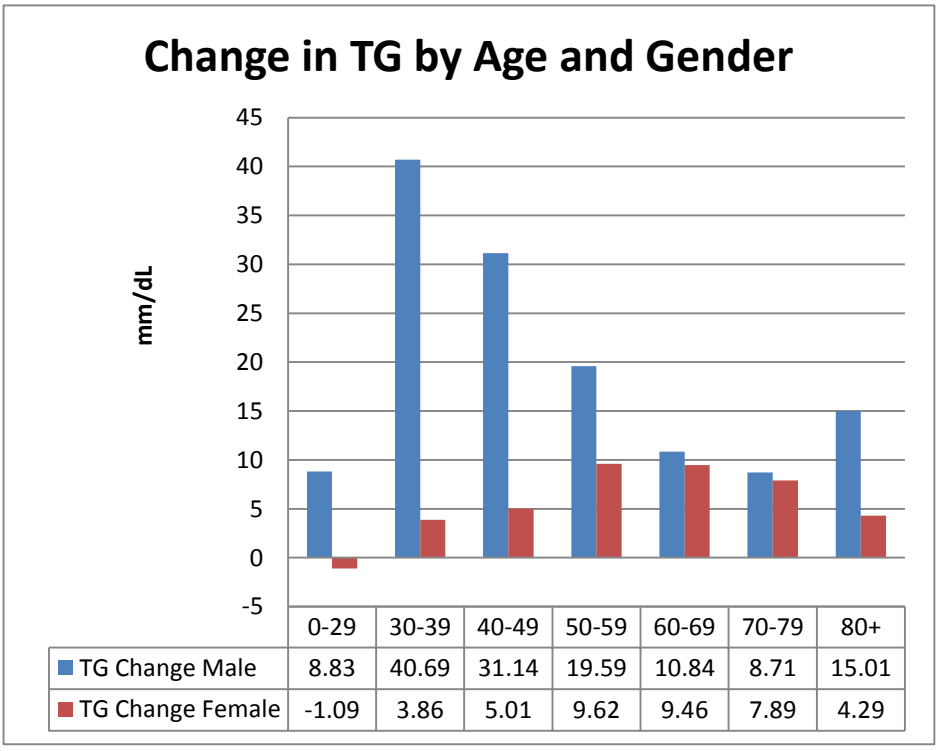


Figure 5-8 Change in TG by age and gender

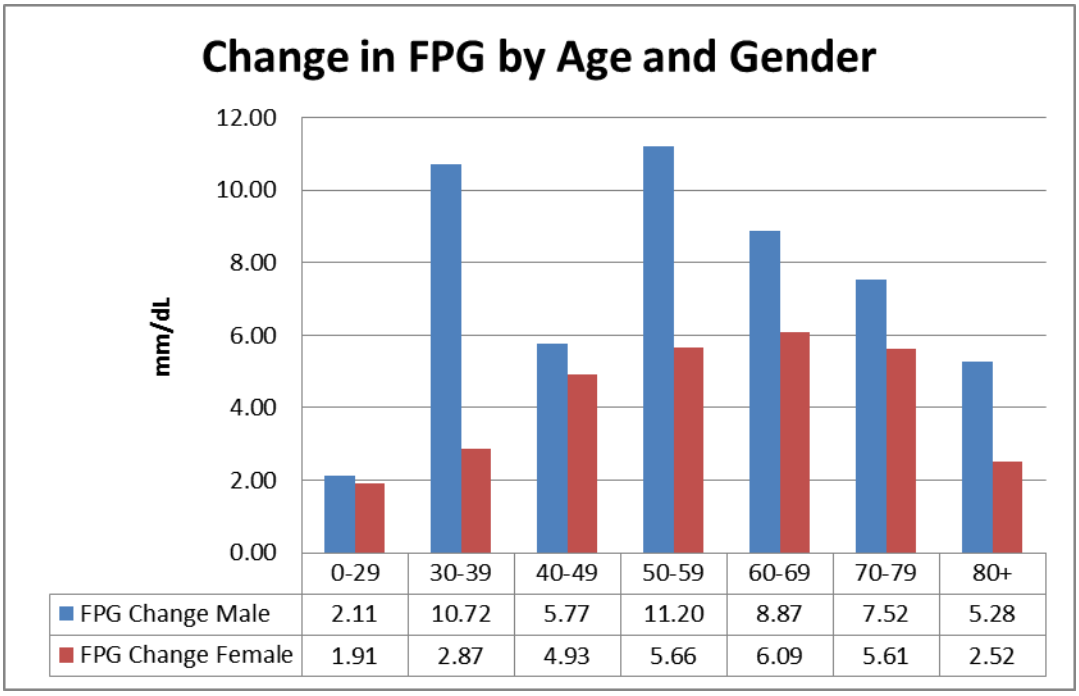


Figure 5-9 Change in FPG by age and gender

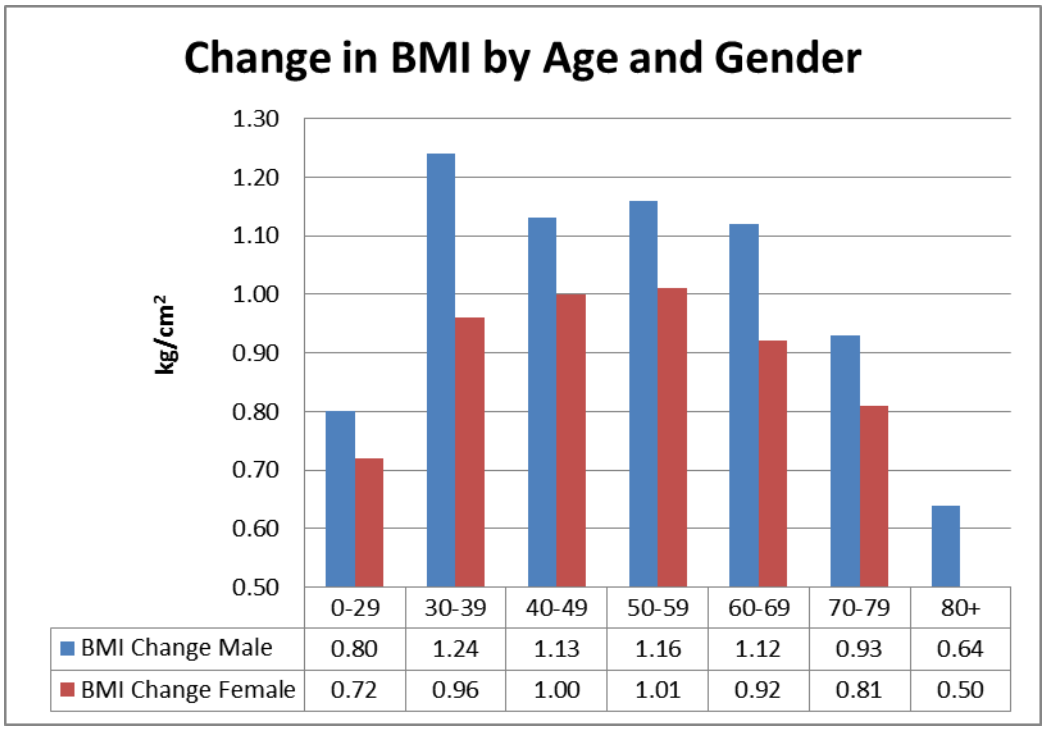


Figure 5-10 Change in BMI by age and gender

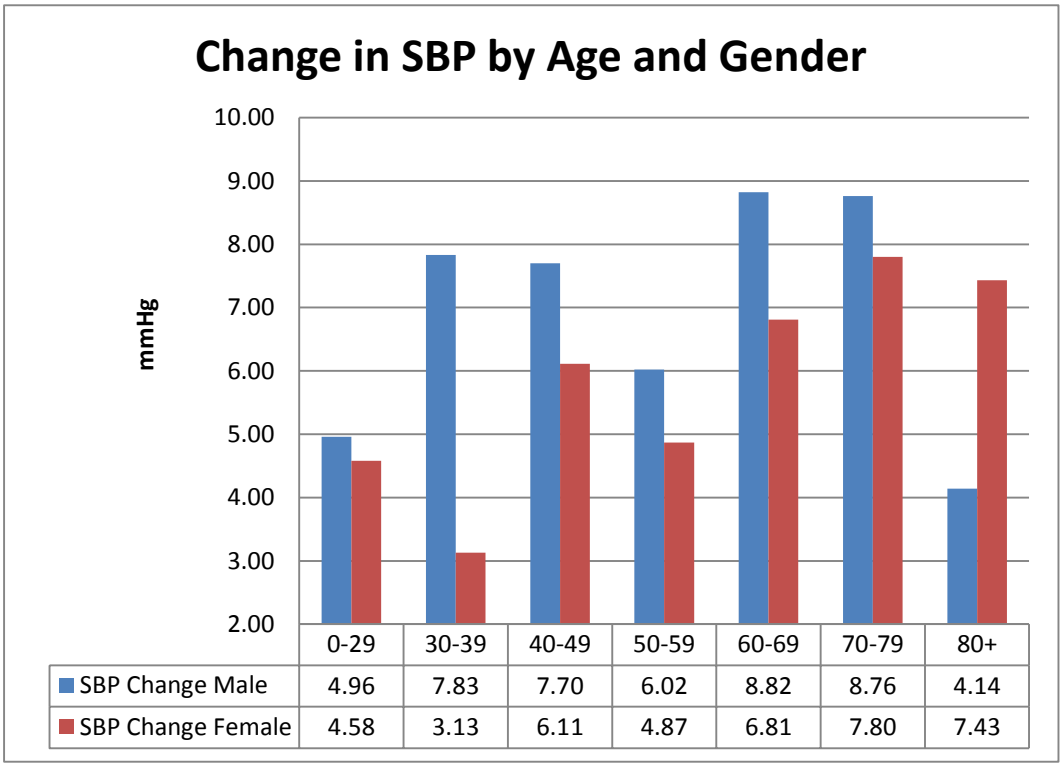


Figure 5-11 Change in SBP by age and gender

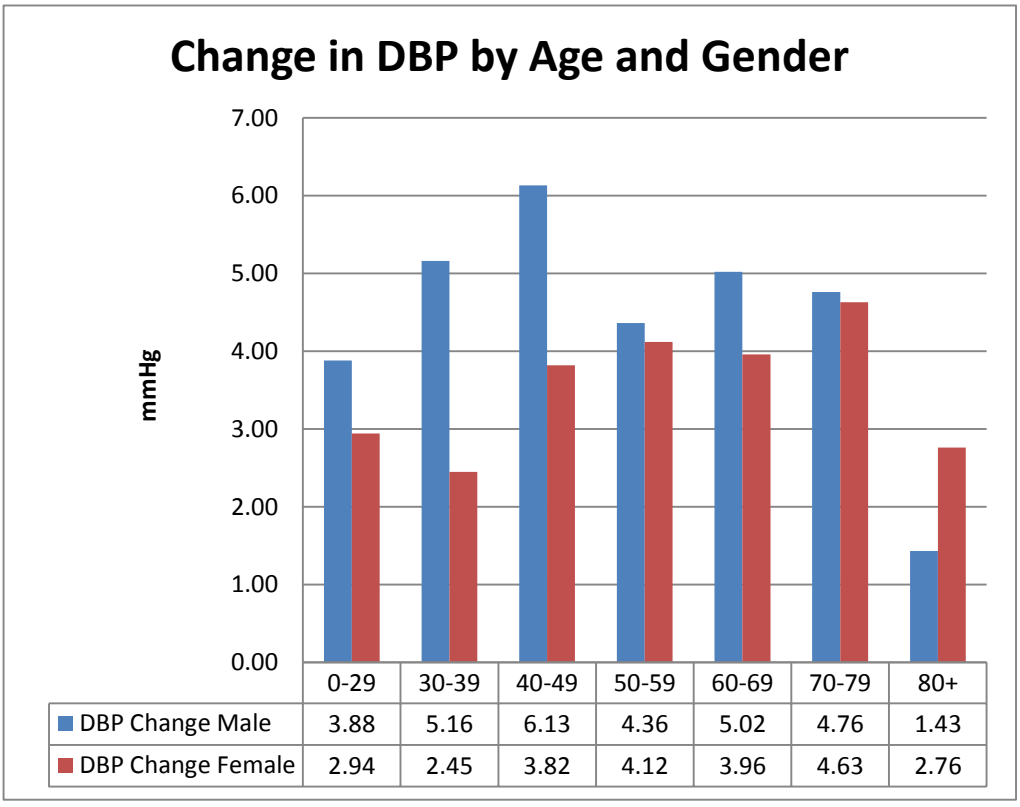


Figure 5-12 Change in Diastolic BP by age and gender

5.3.3.2 *Summary*

The preceding sections of this chapter have shown that significant reduction in risk factors for chronic disease can be achieved by both male and female participants at any age. In this section it has been demonstrated that the greatest reduction in risk factors occurs at a younger age for the male participants than the female participants with the greatest improvements occurring at least 1 decadal age category earlier in the male participants than in the female participants (Table 5-14).

Table 5-14 Age category at which greatest reduction in risk factor occurs by gender

	Age Category	
	Male	Female
TC (mg/dL)	40-49	50-59
HDL (mg/dL) (smallest reduction)	0-29	40-49
LDL (mg/dL)	40-49	60-69
TG (mg/dL)	30-39	60-69
FPG (mg/dL)	50-59	60-69
BMI (kg/cm^2)	30-39	50-59
SBP (mmHg)	60-69	70-79
DBP (mmHg)	40-49	70-79

5.3.4 Marital status

Marital status was captured on 4375 participants, as shown in (Table 5-15).

Table 5-15 Frequency table, mean age and gender for marital status

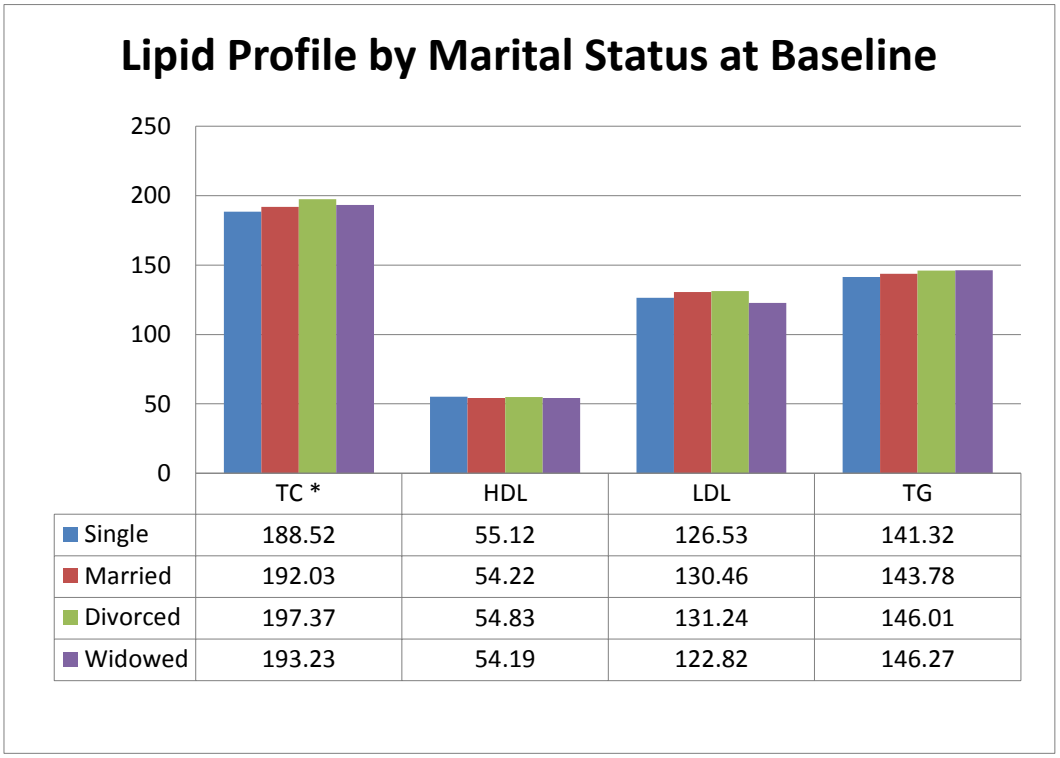
Marital status	Frequency	Percent	Mean Age	Male (%)	Female (%)
Single	422	9.6	47.10	114 (7.5)	308 (10.8)
Married	3237	74	57.61	1293 (85.2)	1944 (68.0)
Divorced	415	8.2	57.05	85 (5.6)	330 (11.6)
Widowed	301	5.9	70.17	26 (1.7)	275 (9.6)
Total	4375	100		1518 (100)	2857 (100)

Single participants were younger than the married and divorced participants, and the widowed participants were older than the other participants [$F(3, 4370) = 218.16$ ($p < 0.001$); Table 5-15]. There was no difference between the age of the married and divorced participants.

The influence of marital status on the biometrics (baseline and change) are presented below, however, it is acknowledged that there are potential confounders. Firstly, the difference in age between the marital status categories may have confounded the marital status effects. Secondly, a significantly higher proportion of the male participants (85.2%) were married than the female participants (68%) [$\chi^2(3,N = 4375) = 177.55$, $p < 0.001$; Table 5-15]. The linear regression analyses, shown later in this chapter, controls for these factors.

5.3.4.1 *Differences in Biometrics by Marital Status at Baseline*

One way ANOVA tests indicated that there were significant differences, at baseline, for marital status in TC levels [F(3, 4326) = 3.27, p = 0.02] but not for, HDL and LDL or TG (Figure 5-13). Post hoc analysis indicated that the single participants had significantly lower TC levels at baseline.

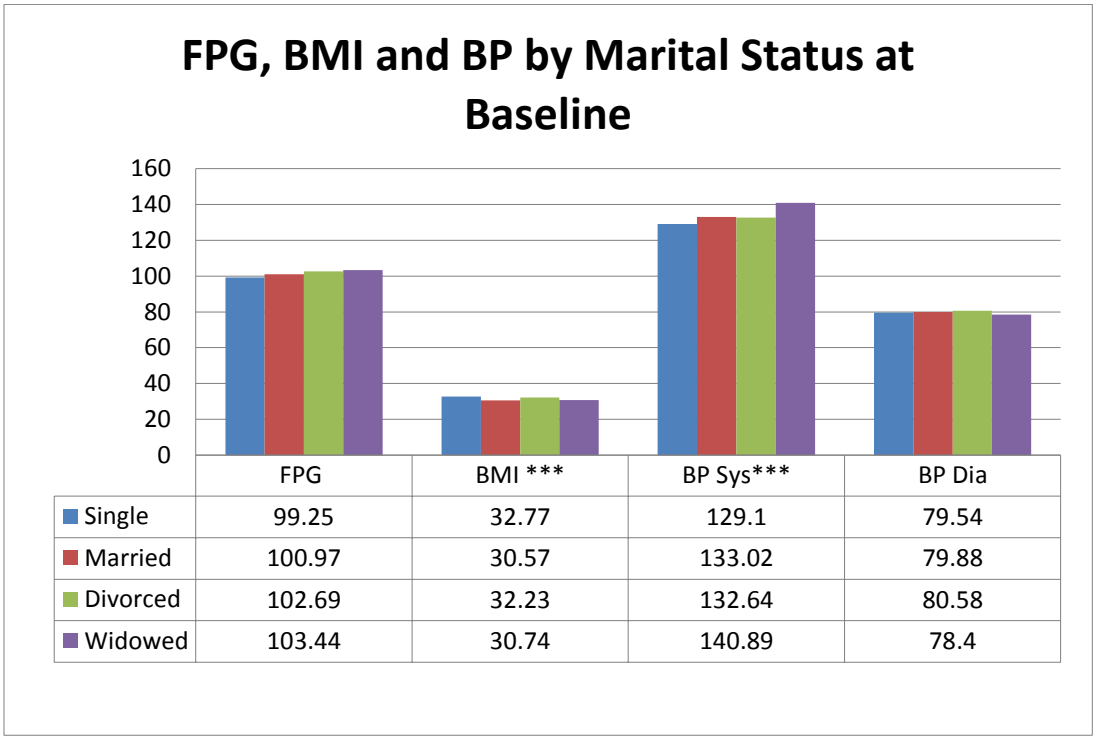


	TC *	HDL	LDL	TG
Single	188.52	55.12	126.53	141.32
Married	192.03	54.22	130.46	143.78
Divorced	197.37	54.83	131.24	146.01
Widowed	193.23	54.19	122.82	146.27

*Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001
Units: TC, HDL,LDL,TG,FPG = mg/dL:

Figure 5-13 Lipids Profile at baseline by marital status

There were significant differences, at baseline, between participants of various marital status for BMI [F(3, 4274) = 15.37, p <0.001] and SBP [F (3, 4311) = 22.33, p <0.001] but not for FPG or DBP(Figure 5-14). Post hoc analysis found that married and widowed participants had significantly lower BMIs than do the single or divorced participants and that single participants had significantly lower SBP than participants in other marital categories while the widowed participants had significantly higher SBP.



	FPG	BMI ***	BP Sys***	BP Dia
Single	99.25	32.77	129.1	79.54
Married	100.97	30.57	133.02	79.88
Divorced	102.69	32.23	132.64	80.58
Widowed	103.44	30.74	140.89	78.4

*Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001
Units: FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Figure 5-14 FPG, BMI, BP at baseline by marital status

5.3.4.2 *Baseline to Post-Intervention Changes within Marital Category*

Significant change from baseline to post-intervention was observed in all biometrics within all marital categories. The effect size was moderate for TC, LDL and BP for all marital categories and for FPG for all categories except the Single Marital Category. For all other biometrics the effect size was small (Table 5-16 through Table 5-19).

Table 5-16 Baseline to post-intervention change single marital category

	Single Marital Category									
	Baseline		Post-intervention		Change					
	Baseline	SD	Post	SD	Mean	%	t	df	p	Cohen's d
TC	189.5	44.44	170.0	36.67	-19.5	10.3	13.94	385	<0.001	0.48
HDL	54.9	27.73	50.6	25.99	-4.3	7.8	9.87	387	<0.001	0.16
LDL	126.9	62.05	111.8	56.52	-15.1	11.9	11.78	375	<0.001	0.25
TG	141.5	98.84	133.7	81.61	-7.9	5.6	2.37	386	0.018	0.09
FPG	98.7	29.74	94.7	24.13	-4.0	4.1	4.08	384	<0.001	0.15
BMI	32.7	9.64	31.8	9.22	-0.9	2.9	15.62	379	<0.001	0.10
SBP	129.5	20.18	123.9	17.07	-5.6	4.3	6.99	381	<0.001	0.30
DBP	79.8	12.19	76.5	10.88	-3.2	4.1	4.18	6.69	<0.001	0.28

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-17 Baseline to post-intervention change married marital category

	Married Marital Category									
	Baseline		Post-intervention		Change					
	Baseline	SD	Post	SD	Mean	%	t	df	p	Cohen's d
TC	192.5	41.13	170.7	37.36	-21.8	11.3	45.92	3014	<0.001	0.56
HDL	54.4	26.26	49.7	23.47	-4.8	8.7	30.14	3013	<0.001	0.19
LDL	131.2	63.58	113.5	57.75	-17.7	13.5	35.21	2944	<0.001	0.29
TG	144.7	88.90	132.2	73.03	-12.5	8.6	11.33	3010	<0.001	0.15
FPG	101.0	28.24	94.1	19.63	-6.9	6.8	20.64	2964	<0.001	0.29
BMI	30.6	6.90	29.6	6.59	-1.0	3.3	66.63	2935	<0.001	0.15
SBP	133.2	18.92	126.7	20.04	-6.6	4.9	12.42	2958	<0.001	0.34
DBP	80.0	11.19	75.7	9.61	-4.2	5.3	22.64	2957	<0.001	0.41

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-18 Baseline to post-intervention change divorced marital category

	Divorced Marital Category									
	Baseline		Post-intervention		Change					
	Baseline	SD	Post	SD	Mean	%	t	df	p	Cohen's d
TC	197.9	40.99	179.1	38.20	-18.8	9.5	13.16	371	<0.001	0.48
HDL	55.0	24.88	50.5	23.27	-4.4	8.1	8.73	370	<0.001	0.18
LDL	133.1	58.51	119.9	59.19	-13.2	9.9	9.64	364	<0.001	0.22
TG	146.7	88.34	138.6	79.58	-8.2	5.6	2.50	370	0.013	0.10
FPG	102.7	31.85	96.6	25.89	-6.1	5.9	7.20	366	<0.001	0.21
BMI	32.2	7.86	31.3	7.61	-0.9	2.8	22.47	354	<0.001	0.12
SBP	132.6	17.70	126.7	15.24	-5.9	4.4	8.17	363	<0.001	0.36
DBP	80.5	12.67	76.3	9.98	-4.2	5.3	6.78	363	<0.001	0.37

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

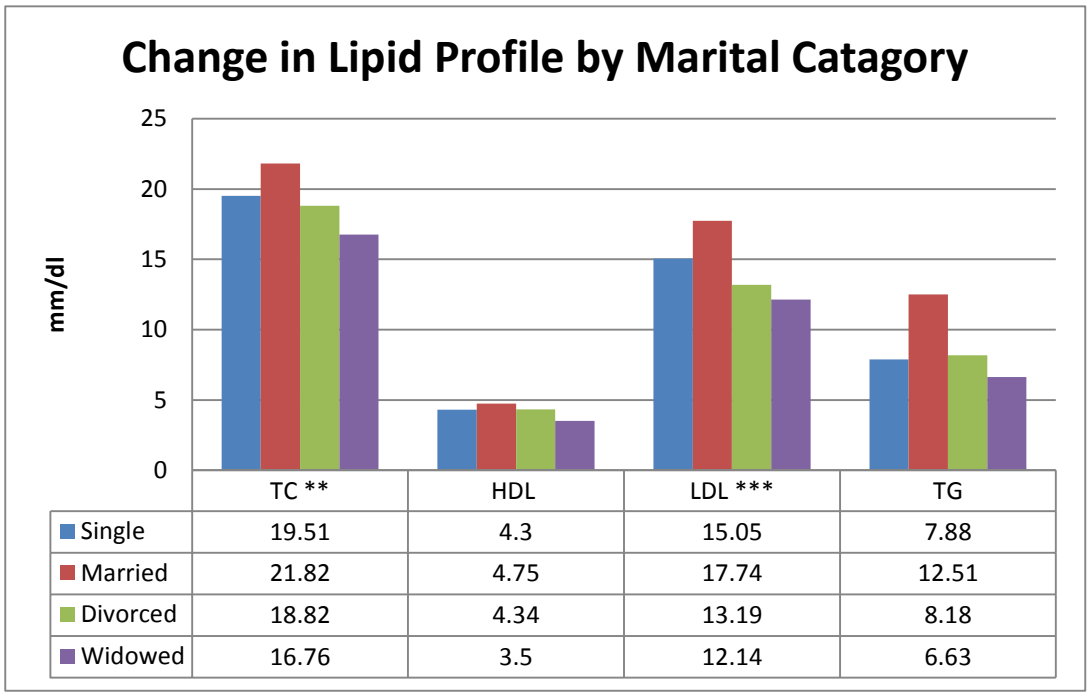
Table 5-19 Baseline to post-intervention change widowed marital category

	Widowed Marital Category									
	Baseline		Post-intervention		Change					
	Baseline	SD	Post	SD	Mean	%	t	df	p	Cohen's d
TC	194.0	41.30	177.2	36.14	-16.8	8.6	9.70	276	<0.001	0.43
HDL	54.8	21.67	51.3	19.82	-3.5	6.4	6.31	276	<0.001	0.17
LDL	123.9	52.97	111.8	48.94	-12.1	9.8	7.48	270	<0.001	0.24
TG	145.6	76.85	139.0	71.39	-6.6	4.6	2.19	276	0.03	0.09
FPG	103.3	26.32	97.8	20.30	-5.6	5.4	5.32	271	<0.001	0.24
BMI	30.7	7.30	29.9	6.95	-0.8	2.6	16.24	267	<0.001	0.11
SBP	141.3	19.95	133.2	17.72	-8.1	5.7	7.56	268	<0.001	0.43
DBP	78.7	11.40	73.9	9.81	-4.8	6.1	7.48	267	<0.001	0.45

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.4.3 *Differences in change from baseline to post-intervention in biometric measures between marital status categories*

There were significant differences between various marital status categories for changes in TC [F(3,4045)=4.54, p = 0.004] and LDL [F(3,3952) = 6.57, p < 0.001]. The differences in HDL and triglyceride change were not significant. Post hoc analysis showed that the married participants had significantly greater change in TC than the widowed participants and significantly greater change in LDL than the widowed and divorced participants (Figure 5-15).

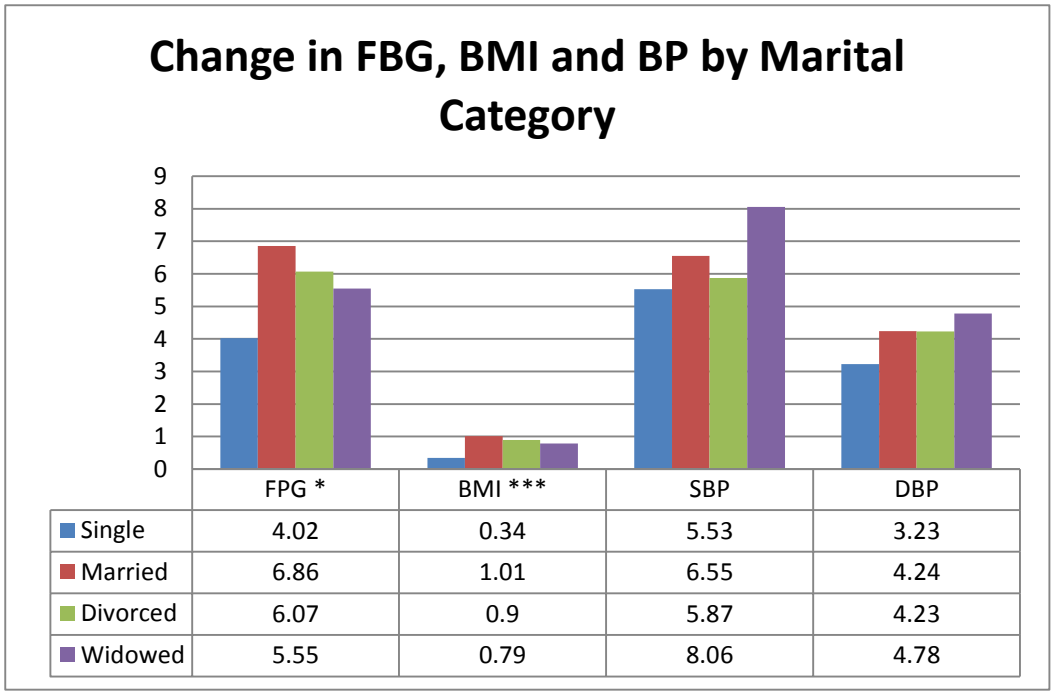


	TC **	HDL	LDL ***	TG
Single	19.51	4.3	15.05	7.88
Married	21.82	4.75	17.74	12.51
Divorced	18.82	4.34	13.19	8.18
Widowed	16.76	3.5	12.14	6.63

*Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001

Figure 5-15 Distribution of change in lipid profile by marital category

Significant differences were demonstrated between marital categories for changes in FPG [F(3, 3984) = 3.12, p = 0.025] and BMI [F(3, 3934) = 7.02, P < 0.001] but not for changes in SBP or DBP. Married participants showed significantly greater change in FPG then did single participants and significantly greater change in BMI than the widowed participants (Figure 5-16).



	FPG *	BMI ***	SBP	DBP
Single	4.02	0.34	5.53	3.23
Married	6.86	1.01	6.55	4.24
Divorced	6.07	0.9	5.87	4.23
Widowed	5.55	0.79	8.06	4.78

Sig at p= 0.05, ** Sig at p = 0.01, ***significant at p <0.001
Units: FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Figure 5-16 Distribution of change in FPG, BMI, SBP and DBP by marital category

5.3.4.4 Summary

Regardless of marital status category significant changes were achieved by the participants in volunteer-delivered, community-based CHIP interventions.

At baseline single participants had significantly lower SBP and TC while married and divorced participants had significantly lower BMI.

Marital status had a significant influence on the change in TC, LDL, FPG and BMI. Married participants had significantly greater reductions in TC, LDL, FPG and BMI. Widowed participants had smaller reductions in TC and LDL. Single participants had smaller reductions in BMI.

5.3.5 Religious affiliation

The majority of the programs analysed in this study were conducted under the auspices of the Seventh-day Adventist Church. Participants were therefore asked to identify whether or not they were members of the Seventh-day Adventist Church and this information was used to categorise them as either Seventh-day Adventists (SDA) or non-Seventh-day Adventists (non-SDA). Of the participants in this sample 1171 (23.2%) identified themselves as SDA and 3845 (76.2%) as being non-SDA, while 29 (0.6%) participants did not give their religious affiliation. There was no difference between the SDA and non-SDA participants in age [$t(1798) = -0.341$, $p = 0.733$] or gender [$t(5012) = -0.537$, $p = 0.591$].

5.3.5.1 Differences in biometrics at baseline by Religious Affiliation Category

SDA participants had lower biometric scores at baseline than the non-SDA participants for HDL, LDL, FPG and BMI at $p < 0.001$ and SBP at $p = 0.029$ however the effect size was small. There was no difference between the SDA and non-SDA participants for baseline TC, TG and DBP (Table 5-20)

Table 5-20 Baseline Biometrics by religious affiliation

	Biometrics at Baseline by Religious Affiliation											
	SDA			Non- SDA			Difference					
	N	Mean	SD	N	Mean	SD	Mean	%	t	df	Sig	Cohen's d
TC	1161	191.1	41.89	3800	193.7	41.96	-2.6	-1.4	-1.87	4959	0.061	-0.06
HDL	1162	49.8	16.49	3798	56.1	27.64	-6.4	-12.8	-9.68	3275	<0.001	-0.29
LDL	1147	118.9	46.34	3726	133.9	65.01	-15.0	-12.6	-8.65	2655	<0.001	-0.27
TG	1161	142.7	87.95	3794	142.8	90.59	-0.1	-0.1	-0.02	4953	0.984	0.00
FPG	1132	97.5	26.37	3771	102.5	29.56	-5.0	-5.2	-5.49	2057	<0.001	-0.18
BMI	1141	29.9	7.31	3752	31.3	7.32	-1.4	-4.7	-2.10	4935	<0.001	-0.19
SBP	1154	132.0	19.36	3783	133.4	19.56	-1.4	-1.1	-2.19	4935	0.029	-0.07
DBP	1154	79.8	11.65	3779	79.9	11.53	-0.1	-0.1	-0.21	4931	0.836	-0.01

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.5.2 Baseline to Post-Intervention Change within Religious Category

There were significant reductions from baseline to post-intervention in all biometrics ($p < 0.001$) for participants in both religious affiliation categories (Table 5-21, Table 5-22).

Table 5-21 Baseline to post-intervention change for SDA participants

	Baseline to Post-Intervention Changes for SDA Participants											
	Baseline			Post-intervention			Change					
	N	Mean	SD	N	Mean	SD	Mean	%	t	df	Sig	Cohen's d
TC	1075	191.6	41.64	1075	173.4	37.30	18.2	9.5	23.59	1074	<0.001	0.46
HDL	1076	49.7	16.60	1076	49.6	15.48	0.1	0.3	12.65	1075	<0.001	0.01
LDL	1060	119.6	47.07	1060	106.0	41.94	13.6	11.4	18.55	1059	<0.001	0.31
TG	1075	144.0	88.63	1075	131.8	74.96	12.3	8.5	7.12	1074	<0.001	0.15
FPG	1040	97.8	36.91	1040	92.9	20.74	4.9	5.1	9.63	1039	<0.001	0.17
BMI	1033	30.0	7.37	1033	29.1	6.99	0.9	2.9	32.25	1032	<0.001	0.12
SBP	1052	132.3	19.45	1052	125.6	31.33	6.7	5.1	7.03	1051	<0.001	0.26
DBP	1052	79.9	11.76	1052	75.6	10.24	4.3	5.4	13.85	1051	<0.001	0.39

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-22 Baseline to post-intervention change for non-SDA participants

	Baseline to Post-Intervention Changes for non-SDA Participants											
	Baseline			Post-intervention			Change					
	N	Mean	SD	N	Mean	SD	Mean	%	t	df	Sig	Cohen's d
TC	3551	194.1	41.80	3551	171.7	38.01	22.4	11.6	49.24	3550	<0.001	0.56
HDL	3549	56.5	27.83	3549	51.8	24.98	4.7	8.2	34.56	3548	<0.001	0.18
LDL	3461	135.7	65.69	3461	116.5	58.19	19.2	14.1	37.51	3460	<0.001	0.31
TG	3546	143.3	90.65	3546	132.6	74.59	10.7	7.5	10.30	3545	<0.001	0.13
FPG	3518	102.4	29.49	3518	95.5	20.07	6.9	6.8	31.95	3517	<0.001	0.28
BMI	3453	31.3	7.27	34.53	30.3	7.00	1.0	3.3	71.42	3452	<0.001	0.14
SBP	3481	133.5	19.30	3481	127.1	23.56	6.4	4.8	16.53	3480	<0.001	0.30
DBP	3479	79.9	11.39	3479	75.8	9.86	4.2	5.2	23.48	3478	<0.001	0.39

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.5.3 *Baseline to Post-Intervention Changes between Religious Categories*

Non-SDA participants showed significantly greater reductions, than SDA participants in TC (11.6% vs 9.5%), HDL (0.3% vs 8.2%), LDL (11.4% vs 14.1%), FPG (5.0% vs 6.9%) and BMI (2.9% vs 3.3%) at p <0.001. The differences in reductions in TG and BP were not significant (Table 5-23).

Table 5-23 Comparison of change post-intervention by religious category

	Change Post-Intervention By Religious Category											
	SDA			Non-SDA			Difference					
	N	Mean	SD	N	Mean	SD	Mean	%	t	df	Sig	Cohen's d
TC	1075	18.2	25.29	3551	22.4	27.15	-4.2	-23.3	-4.73	1885	<0.001	-0.16
HDL	1076	3.1	8.12	3549	5.3	9.09	-2.1	-68.4	-7.37	1962	<0.001	-0.25
LDL	1060	13.6	23.91	3461	18.2	28.47	-4.5	-33.3	-5.15	2060	<0.001	-0.17
TG	1075	12.3	56.52	3546	10.7	62.12	1.5	12.5	0.719	4619	0.472	0.03
FPG	1040	4.8	16.19	3518	6.9	18.7	-2.1	-43.3	-3.53	1932	<0.001	-0.12
BMI	1033	0.9	0.87	3453	1.0	0.84	-0.2	-17.2	-4.89	4484	<0.001	-0.18
SBP	1052	6.7	30.75	3481	6.4	22.92	0.3	3.8	0.28	4531	0.777	0.01
DBP	1052	4.3	10.11	3476	4.6	10.45	-0.2	-5.6	-5.68	4593	0.570	-0.02

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.5.4 *Comparison of Biometrics at Baseline and Post-Intervention by Religious Category*

Table 5-24 shows a comparison of the baseline and post-intervention biometric values for the SDA and non-SDA participants. At baseline the SDA participants had

significantly better biometric than did the non-SDA participants however the non-SDA participants had greater improvements in biometrics during the intervention. The difference between SDA and non-SDA has reduced in just 30 days.

Table 5-24 Comparison of biometrics at baseline and post-intervention by religious category

Comparison of Biometrics between SDA and non-SDA at Baseline and Post-Intervention						
	Baseline			Post-Intervention		
	SDA	non-SDA	% Difference	SDA	non-SDA	% Difference
TC	191.1	193.7	-1.4	173.3	171.7	0.9
HDL	49.8	56.1	-12.8	46.5	51.1	-9.9
LDL	118.9	133.9	-12.6	106.0	116.3	-9.8
TG	142.7	142.8	NS	132.0	132.8	NS
FPG	97.5	102.5	-5.2	93.1	95.4	-2.5
BMI	29.9	31.3	-4.7	29.1	30.3	-4.2
SBP	132.0	133.4	NS	125.5	127.1	NS
DBP	79.8	79.9	NS	75.6	75.8	NS

NS = not significant
Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.5.5 Summary

While the SDA participants showed significantly better risk factor status at baseline than the non-SDA participants, the non-SDA participants showed significantly greater improvement in these risk factors, from baseline to post-intervention, than did the SDA participants. The health status of the SDA participants was still better post-intervention than the non-SDA participants but the gap between the SDA and the non-SDA had reduced over the period of intervention.

5.3.6 Body Mass Index

In this section the impact of the participants' level of obesity at program entry, as measured by a BMI, on their response to the intervention was examined.

Data needed to calculate BMI was available on 4912 participants. Participants were grouped by weight at program entry according to the WHO BMI categories of Underweight, Normal, Overweight, Obese I, Obese II and Obese III (WHO, 2000). As only 37 participants were classified as underweight, they were included in the normal weight category. Table 5-25 shows the distribution of the participants across the various BMI categories. Also shown is the mean age participants and percentage of males in each category.

Table 5-25 Frequency table and mean age for BMI categories

BMI Range (kg/cm2)	Frequency	Percent	Mean age	% Male
Underweight + Normal <25	994	20.0	57.4±15.3	26.7
Overweight 25-29.99	1504	30.6	58.5±12.8	35.4
Obesity Class I 30 – 34.99	1206	24.6	57.8±11.8	34.7
Obesity Class II 35 – 39.99	659	13.4	55.7±11.9	33.5
Obesity Class III >40	549	11.2	54.5±11.4	29.1
Total	4912	100.0		

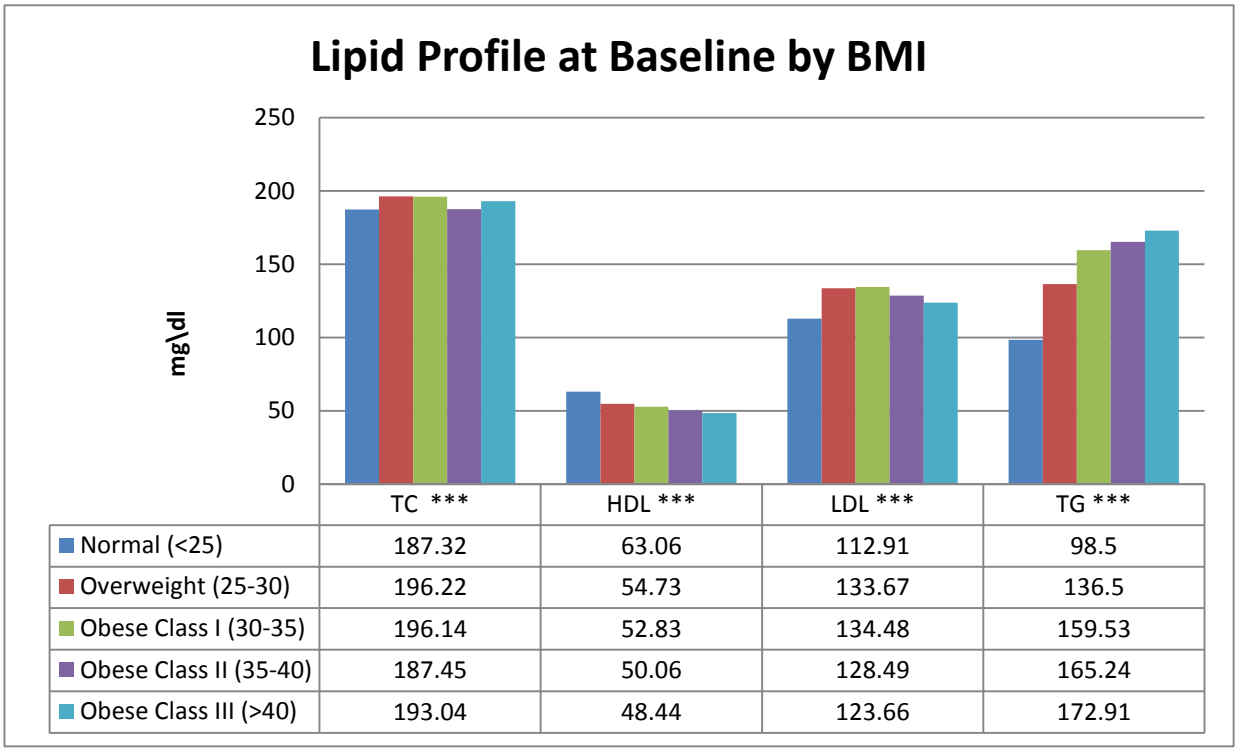
There were significant differences between the mean ages of the different BMI categories [F(4, 4903) = 12.949 (p = < 0.001)]. Post hoc analysis indicated that the Obese Class II and Obese Class III participants were significantly younger than the lower BMI

categories. There was no significant gender difference between the various BMI categories (t(4911) = 0.145, p = 0.885)

5.3.6.1 *Comparison of basline biometrics by Body Mass Index at program entry*

There were significant differences at baseline between the participants in the various BMI categories for TC, [F(4, 4868) = 10.89], HDL cholesterol, [F(4, 4867) = 42.91], LDL cholesterol [F(4, 4785) = 6.09] and, TG [F(4, 4863) = 106.39] at p < 0.001 (Figure 5-17).

Post hoc analysis indicated that TC levels at baseline were significantly lower for participants in the Normal BMI categories than for participants in the Overweight, Obese Class I and the Obese Class III categories, but not significantly different from participants in the Obese Class II. Baseline LDL was lowest for participants in the normal category, peaking in the Overweight and Obese Class I category, but dropping off again in the two extreme obese categories. HDL was highest in those participants in the Normal BMI category and decreased as the BMI category increased. TG levels were lowest in the Normal category and increased as the BMI increased (Figure 5-17).



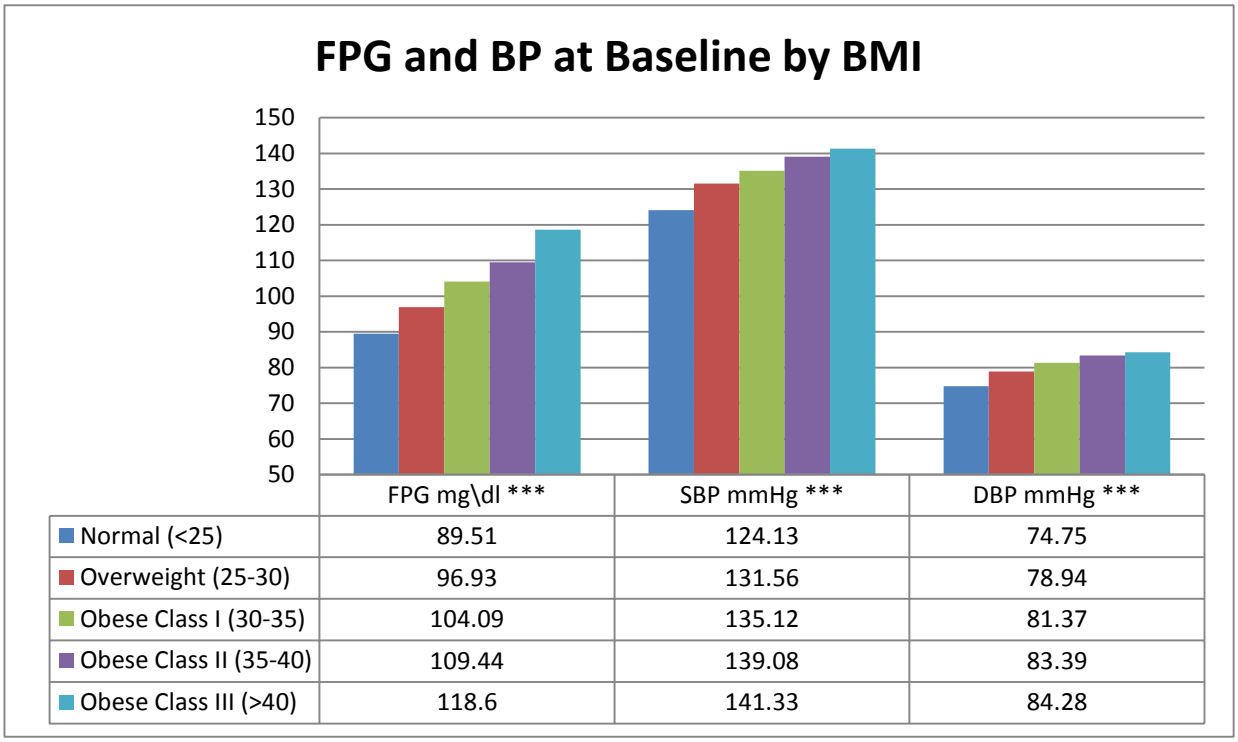
	TC ***	HDL ***	LDL ***	TG ***
Normal (<25)	187.32	63.06	112.91	98.5
Overweight (25-30)	196.22	54.73	133.67	136.5
Obese Class I (30-35)	196.14	52.83	134.48	159.53
Obese Class II (35-40)	187.45	50.06	128.49	165.24
Obese Class III (>40)	193.04	48.44	123.66	172.91

**sig at p= 0.05, ** sig at p = 0.01, ***significant at p <0.001*

Figure 5-17 Comparison of baseline lipid profile by BMI category

Significant differences were also observed, between participants in various BMI categories at program entry, for FPG [$F(4, 481) = 123.58$], SBP [$F(4, 4864) = 105.34$] and DBP [$F(4, 4860) = 98.39$] at $p < 0.001$.

Post hoc analysis confirmed that for baseline FPG and BP, the lowest scores were in the Normal baseline BMI category and that the scores increased as the BMI increased peaking in the Obese Class III category (Figure 5-18).



	FPG mg\dl ***	SBP mmHg ***	DBP mmHg ***
Normal (<25)	89.51	124.13	74.75
Overweight (25-30)	96.93	131.56	78.94
Obese Class I (30-35)	104.09	135.12	81.37
Obese Class II (35-40)	109.44	139.08	83.39
Obese Class III (>40)	118.6	141.33	84.28

*sig at p= 0.05, ** sig at p = 0.01, ***significant at p <0.001

Figure 5-18 FPG and BP at baseline by BMI

5.3.6.2 *Comparison of changes in biometric measures from baseline to post-intervention within Body Mass Index categories*

Significant changes occurred from baseline to post-intervention in the biometric scores within all BMI categories at $p < 0.001$ except for TG in the Obese Class III category where $p = 0.011$ and for SBP in the normal category where there was no significant change (Table 5-26 to Table 5-30).

Table 5-26 Baseline to post-intervention change in participants with Normal BMI category

	Normal BMI < 25						
	Baseline	Post	Change	%	t	df	p
TC	188.0	169.8	18.2	9.7	23.51	912	<0.001
HDL	63.7	59.0	4.7	7.4	13.10	912	<0.001
LDL	126.5	111.6	14.9	11.8	17.19	904	<0.001
TG	98.8	94.2	4.6	4.6	3.69	912	<0.001
FPG	89.8	86.7	3.1	3.4	6.35	893	<0.001
BMI	22.6	22.1	0.5	2.2	27.49	897	<0.001
SBP	122.0	124.3	-2.23	-1.9	1.47	893	0.143
DBP	74.7	72.0	2.7	3.7	8.45	893	<0.001

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-27 Baseline to post-intervention change in participants with Overweight BMI category

	Overweight BMI 25 – 29.99						
	Baseline	Post	Change	%	t	df	p
TC	196.3	172.8	23.5	12.0	32.25	1408	<0.001
HDL	54.1	49.9	4.2	7.7	21.38	1408	<0.001
LDL	133.9	114.8	19.1	14.3	25.90	1381	<0.001
TG	136.9	124.2	12.7	9.3	6.98	1408	<0.001
FPG	96.9	91.7	5.2	5.4	12.72	1387	<0.001
BMI	27.5	26.6	0.9	3.2	51.23	1387	<0.001
SBP	131.8	125.1	6.7	5.1	16.47	1377	<0.001
DBP	79.1	74.7	4.4	5.5	16.36	1377	<0.001

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-28 Baseline to post-intervention change in participants with Obese Class I BMI category

	Obese Class I BMI 30 - 34.99						
	Baseline	Post	Change	%	t	df	p
TC	196.2	173.0	23.2	11.9	27.60	1130	<0.001
HDL	52.8	48.1	4.7	8.90	20.36	1129	<0.001
LDL	134.9	116.4	18.5	13.7	21.45	1098	<0.001
TG	160.9	145.4	15.6	9.7	8.38	1129	<0.001
FPG	103.9	96.8	7.1	6.9	13.53	1121	<0.001
BMI	32.3	31.2	1.1	3.4	46.86	1125	<0.001
SBP	135.4	128.0	7.4	5.4	17.01	1120	<0.001
DBP	81.6	77.0	4.5	5.6	13.74	1117	<0.001

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-29 Baseline to post-intervention change in participants with Obese Class II BMI category

	Obese Class II BMI 35 – 39.99						
	Baseline	Post	Change	%	t	df	p
TC	193.4	171.0	22.4	11.6	20.70	607	<0.001
HDL	50.2	45.6	4.6	9.2	14.09	607	<0.001
LDL	128.7	111.3	17.3	13.5	16.09	588	<0.001
TG	166.4	155.8	10.6	6.4	4.06	605	<0.001
FPG	109.4	101.3	8.1	7.4	9.60	601	<0.001
BMI	37.2	35.9	1.3	3.3	33.78	603	<0.001
SBP	139.0	130.2	8.8	6.3	13.19	601	<0.001
DBP	83.1	78.4	4.7	5.7	11.17	601	<0.001

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-30 Baseline to post-intervention change in participants with Obese Class III BMI category

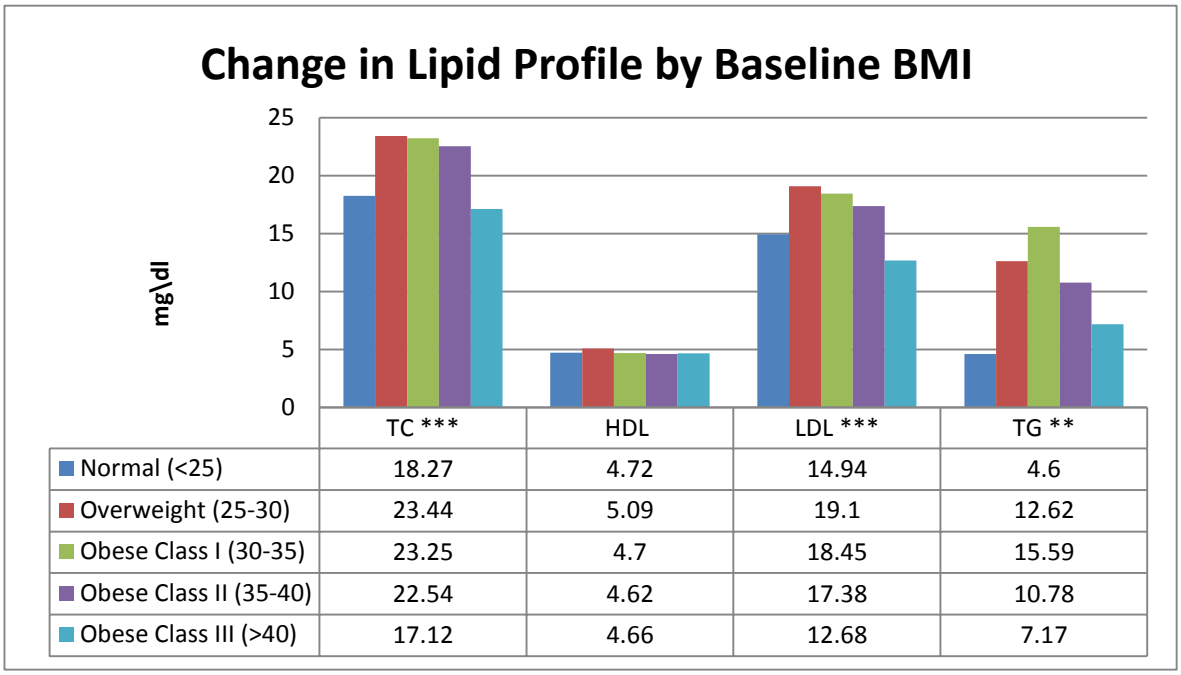
	Obese Class III						
	Baseline	Post	Change	%	t	df	p
TC	188.0	170.9	17.1	9.1	14.83	498	<0.001
HDL	48.7	44.0	4.7	9.6	12.83	498	<0.001
LDL	124.6	112.0	12.6	10.2	9.93	485	<0.001
TG	172.8	165.6	7.2	4.2	2.55	497	0.011
FPG	118.9	106.4	12.5	10.5	10.38	490	<0.001
BMI	45.8	44.2	1.6	3.5	29.49	497	<0.001
SBP	141.5	132.9	8.6	6.1	10.47	486	<0.001
DBP	84.4	79.3	5.1	6.0	10.36	487	<0.001

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.6.3 Difference in change between baseline Body Mass Index categories

There were significant differences between the participants in various BMI categories, in the changes, from baseline to post-intervention, in TC, [F(4, 4546) = 10.04] and in LDL cholesterol, [F(4, 4447) = 7.08] at P < 0.001 and in TG [F(4, 4542) = 4.87] at p = 0.001. However there was no significant difference in changes in HDL cholesterol.

Post hoc analysis showed that change in TC and LDL cholesterol was greatest in the overweight, Obese Class I and Obese Class II categories but was significantly lower in the normal category and Obese Class III categories. For TG the change was significantly less for the normal BMI category than for the other BMI categories (Figure 5-19).



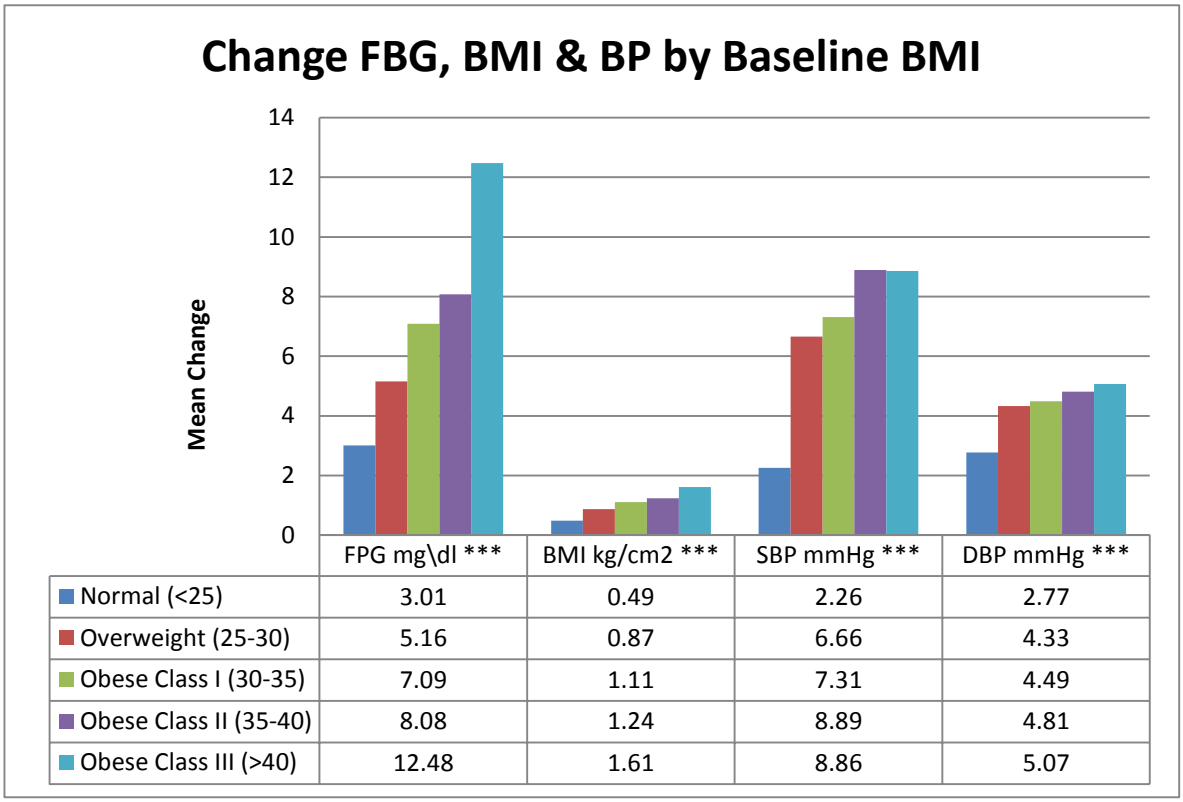
	TC ***	HDL	LDL ***	TG **
Normal (<25)	18.27	4.72	14.94	4.6
Overweight (25-30)	23.44	5.09	19.1	12.62
Obese Class I (30-35)	23.25	4.7	18.45	15.59
Obese Class II (35-40)	22.54	4.62	17.38	10.78
Obese Class III (>40)	17.12	4.66	12.68	7.17

** sig at p= 0.05, ** sig at p = 0.01, ***significant at p <0.001*

Figure 5-19 Change in Lipid Profile by baseline BMI Category

There were significant differences between the changes achieved in FPG, [F(4, 4483) = 25.30], BMI, [F(4, 4500) = 200.49], SBP, [F(4, 4468) = 8.99] and DBP, [F(44, 4466) = 5.91] at $p < 0.001$ between participants in various baseline BMI categories.

Changes in FPG and BMI were significantly lower in the normal baseline BMI category and increase significantly as the baseline BMI level increased. For changes in SBP and DBP the participants in the normal BMI category had significantly less change when compared with participants in the other BMI categories.



	FPG mg\dl ***	BMI kg/cm2 ***	SBP mmHg ***	DBP mmHg ***
Normal (<25)	3.01	0.49	2.26	2.77
Overweight (25-30)	5.16	0.87	6.66	4.33
Obese Class I (30-35)	7.09	1.11	7.31	4.49
Obese Class II (35-40)	8.08	1.24	8.89	4.81
Obese Class III (>40)	12.48	1.61	8.86	5.07

** sig at p= 0.05, ** sig at p = 0.01, ***significant at p <0.001*

Figure 5-20 Change in FPG, BMI and BP by baseline BMI

5.3.6.4 Summary

At baseline those participants in the normal BMI category demonstrated the lowest levels for the biometrics tested. For TG, FPG, SBP and DBP the level increased with each rise in BMI category. HDL levels decreased with increase in BMI. TC and LDL levels peaked in the overweight and obese I categories. The greatest degree of change from baseline to post-intervention occurred in participants in the overweight and obese I categories but significantly less change occurred in participants in the normal category and in the extremely obese categories for TC, LDL and TG. For changes in both FPG, BMI, SBP and DBP the amount of change increases as the BMI category increases. Differences in change in HDL across BMI categories were not significant.

5.3.7 Family history and previous history

At baseline participants were asked to complete questions relating to their family medical history and own previous medical history. These questions were:

1. One or both of your parents died before age 60: of heart disease? □Yes □No
of diabetes? □Yes □No

2. Check (X) if you have ever been told by a physician that you have any of the following:

☐Angina (Yr)? _____	☐Abnormal EKG (last 3 yrs)	☐Gall bladder trouble	☐Ulcers
☐Heart attack (yr)? _____	☐Irregular heartbeats	☐Gout	☐Osteoarthritis
☐Angioplasty (Yr)? _____	☐Stroke (Yr)? _____	☐Kidney disease	☐Rheumatoid arthritis
☐Bypass (Yr)? _____	☐High blood pressure	☐Chronic bronchitis	☐Overweight
☐Heart failure (Yr)? ____	☐High cholesterol	☐Emphysema	☐Gout
☐Blood clotting problem	☐High triglycerides	☐Thyroid disorder	☐Cancer

The participant responses to these questions were compared using t-test analysis to identify differences in baseline biometrics and change from baseline to post-intervention, in the biometrics. Next all of the participants who had indicated a positive response to any of the above questions were grouped together and they were compared with those participants who had indicated that there was no previous history or family history were compared.

5.3.7.1 Family History

Of the 3398 participants who responded to the question relating to a family history of heart disease 379 (11.02%) participants indicated that they had had a parent die from heart disease before the age of 60. In response to the question about a family history of diabetes 144 (4.4%) of the 3266 participants who responded indicated that they had a parent die from diabetes before the age of 60.

At baseline, participants who reported that one or both parents had died from heart disease before the age of 60 had significantly higher TG, FPG and SBP (Table 5-31) than those participants who indicated that they had not had a parent die from heart disease before the age of 60.

Table 5-31 Comparison of family history vs no family history of heart disease mortality at baseline

	Family History of Heart Disease Mortality At baseline									
	Family History			No Family History						
	N	Mean	SD	N	Mean	SD	t	df	Sig	Cohen's d
TC	375	191.0	42.84	2989	193.2	41.65	-0.97	3362	0.330	-0.05
HDL	375	53.3	28.67	2988	55.0	26.70	-1.15	3361	0.252	-0.06
LDL	366	129.1	62.88	2938	131.2	63.14	-0.61	3302	0.540	-0.03
TG	374	156.7	97.19	2985	143.1	87.99	2.57	453	0.001	0.15
FPG	371	106.1	30.92	2953	100.9	27.59	3.09	447	0.002	0.18
BMI	366	31.5	7.52	2970	31.0	7.45	1.14	3334	0.250	0.06
SBP	376	136.1	19.33	2990	132.8	19.22	3.10	3364	0.002	0.17
DBP	375	79.8	11.92	2988	79.8	11.21	0.03	3361	0.980	0.00

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Those participants who indicated that one or both of their parents had died from heart disease before the age of 60 had significantly greater reductions in FPG levels than did those participants who had indicated that they had not had a parent die from heart disease before the age of 60 (Table 5-32). Effect size however was small.

Table 5-32 Comparison of family history vs no family history of heart disease mortality post-intervention

	Family History of Heart Disease Mortality Percentage Change Post-intervention									
	Family History			No Family History						
	N	Mean	SD	N	Mean	SD	t	df	Sig	Cohen's d
TC	353	10.2	13.93	2817	10.1	12.21	0.06	423	0.960	0.00
HDL	353	6.6	20.08	2816	7.5	13.96	-1.05	3167	0.293	-0.05
LDL	343	10.5	22.18	2754	11.4	19.19	-0.51	408	0.610	-0.04
TG	352	-0.0	0.49	2814	0.0	0.37	-1.16	3164	0.250	-0.07
FPG	349	6.2	13.12	2782	4.5	12.84	2.29	436	0.020	0.13
BMI	344	3.0	2.40	2811	3.2	4.12	-0.98	3153	0.327	-0.06
SBP	374	2.9	40.74	2793	4.3	16.69	-1.15	3138	0.250	-0.05
DBP	374	4.5	12.98	2791	4.2	12.46	0.41	3136	0.680	0.02

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Those participants who reported that one or both parents had died from diabetes before the age of 60 had significantly lower TC, but higher FPG, BMI and SBP levels at baseline than did those participants who indicated that they had not had a parent die from diabetes before the age of 60. (Table 5-33).

Table 5-33 Comparison of family history vs no family history of diabetes mortality at baseline

	Family History of Diabetes Mortality At baseline									
	Family History			No Family History						
	N	Mean	SD	N	Mean	SD	t	df	Sig	Cohen's d
TC	141	184.8	41.55	3091	193.1	41.90	-2.326	3230	0.020	-0.20
HDL	142	53.4	26.08	3089	54.8	26.71	-0.572	3229	0.567	-0.05
LDL	140	122.2	56.25	3035	131.0	63.38	-1.603	3173	0.109	-0.15
TG	142	157.6	90.50	3085	143.8	88.43	1.817	3225	0.069	0.15
FPG	140	120.4	41.13	3053	100.6	36.94	5.531	144	<0.001	0.51
BMI	140	33.1	8.37	3069	31.0	7.46	3.194	3207	0.001	0.26
SBP	142	136.5	17.47	3092	132.9	19.32	2.203	3232	0.028	0.20
DBP	140	39.7	10.37	3091	78.8	11.16	-0.052	3229	0.958	-3.63

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Those participants who indicated that one or both of their parents had died from diabetes before the age of 60 had greater reductions in TC, LDL, FPG levels but

significantly less reductions in BMI than did those participants who had indicated that they had not had a parent die from diabetes before the age of 60 (Table 5-34). The effect size was small.

Table 5-34 Comparison of family history vs no family history of diabetes mortality percentage change post-intervention

	Family History of Diabetes Mortality Percentage Change Post-intervention									
	Family History			No Family History						
	N	Mean	SD	N	Mean	SD	t	df	Sig	Cohen's d
TC	133	5.8	13.48	2917	10.3	12.35	-4.135	3048	<0.001	-0.35
HDL	134	5.0	14.11	2915	7.5	14.93	-1.923	3047	0.055	-0.17
LDL	132	5.9	21.46	2847	11.3	19.61	-3.078	2977	0.002	-0.26
TG	134	-0.6	0.66	2912	0.0	0.37	-1.795	3044	0.073	-1.10
FPG	133	8.8	18.29	2879	4.5	12.60	2.669	138	0.009	0.28
BMI	132	2.1	5.59	2902	3.1	2.41	-2.181	133	0.031	-0.27
SBP	132	-2.6	64.67	2889	4.4	16.47	-1.247	132	0.215	-0.17
DBP	132	3.7	14.11	2887	4.7	12.33	-0.530	3017	0.596	-0.07

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.3.7.2 Previous History

The question regarding previous history was completed by 5046 participants. The number of respondents who indicated that they had been told that they had various medical conditions is shown in Table 5-35.

Table 5-35 Distribution of participants self-reported previous history

Previous history	n	%	Previous history	n	%
Angina	175	3.5	Gallbladder	425	8.4
Heart Attach	210	4.2	Gout	121	2.4
Angioplasty	160	3.2	Kidney Disease	113	2.2
Bypass	110	2.2	Diabetes Myelitis	707	14.0
Heart Failure	71	1.4	Peptic Ulcer	178	3.5
Blood Clots	135	2.7	Thyroid	700	13.9
Abnormal EKG	194	3.8	Osteoporosis	360	9.4
Arrhythmia	566	11.2	Osteoarthritis	476	9.4
Stroke	101	2.0	Rheumatoid Arthritis	162	3.2
High Blood Pressure	1695	33.6	Overweight	2190	43.4
High Cholesterol	1807	35.8	Bronchitis Emphysema	355	7.0
High TG	1009	20.0	Cancer	437	8.7

At baseline it was found that generally, those participants who indicated a previous medical history had significantly lower scores, for TC, LDL, HDL and DBP however significantly higher baseline scores for TG, FPG, BMI and SBP (Table 5-36) than those participants who did not indicate a previous medical history. A previous history of various components of vascular disease or diabetes impacted the greatest number of biometrics.

Post-intervention it was found that there were much fewer differences between the two groups and that a previous history of various components of vascular disease did not seem to have an impact. However participants with a previous history of diabetes show a significantly less reductions in lipid profile, and BMI with a greater reduction in FPG (Table 5-37).

*5.3.7.3 **Summary***

Previous medical history had a significant impact on the participants' baseline biometrics. Those participants with a previous medical history of diabetes had greater change from baseline to post-intervention in lipid profile, FPG and BMI.

A factor that needs to be considered when the impact of previous history is examined is that it is more likely that participants who indicated a previous history are taking medication for these conditions and that this medication could be a confounding factor. Unfortunately data were not collected on medication usage.

Table 5-36 Comparison baseline biometrics for previous history

	Comparison of Baseline Biometrics by Previous History																											
Previous history	True		False		T Cholest			HDL			LDL			Triglycerides			FPG			BMI			Bp Sys			Bp Dia		
	N	%	N	%	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p
Angina	175	3.5	4872	96.5	181.43	193.57	0.001	56.60	54.53	0.413	131.04	130.28	0.896	164.06	141.85	0.001	111.53	100.94	<0.001	32.58	30.93	0.004	136.61	132.92	0.014	78.33	79.92	0.074
Heart Attach	210	4.2	4837	95.8	173.25	194.02	<0.001	49.68	54.82	0.004	109.91	131.19	<0.001	170.68	141.39	<0.001	115.20	100.71	<0.001	32.98	30.90	<0.001	136.84	132.89	0.017	77.61	79.97	0.004
Angioplasty	160	3.2	4887	96.8	172.40	193.84	<0.001	46.96	54.85	<0.001	108.17	193.03	<0.001	169.61	141.73	0.006	111.64	100.97	0.001	32.13	30.95	0.047	135.26	132.98	0.148	76.73	79.97	0.001
Bypass	110	2.2	4937	97.8	168.92	193.70	<0.001	48.26	54.74	0.008	106.46	130.85	<0.001	154.35	142.35	0.168	111.12	101.10	<0.001	31.54	30.98	0.346	138.24	132.94	0.005	76.63	79.94	0.003
Heart Failure	71	1.4	4976	98.6	176.94	193.38	0.001	47.95	54.70	0.028	117.26	130.49	0.080	173.31	142.18	0.015	117.13	101.08	<0.001	34.57	30.94	0.006	140.04	132.95	0.012	75.87	79.92	0.003
Blood Clots	135	2.7	4912	97.3	193.04	193.15	0.974	51.64	54.68	0.176	131.54	130.28	0.817	163.97	142.03	0.005	111.07	101.04	0.001	34.60	30.89	<0.001	135.67	132.98	0.117	79.43	79.88	0.660
Abnormal EKG	194	3.8	4853	96.2	184.88	193.49	0.010	52.46	54.69	0.233	125.97	130.49	0.320	151.27	142.29	0.172	104.04	101.20	0.183	31.75	30.96	0.160	135.39	132.96	0.093	78.88	79.91	0.231
Arrhythmia	566	11.2	4481	88.8	190.36	193.51	0.094	53.67	54.72	0.359	127.49	130.67	0.253	155.92	140.92	0.001	103.24	101.06	0.094	31.64	30.91	0.027	134.70	132.84	0.034	78.85	80.00	0.026
Stroke	101	2.0	4946	98.0	180.00	193.42	0.002	55.83	54.58	0.627	123.40	130.45	0.260	139.11	142.69	0.644	114.34	101.04	0.001	31.60	30.98	0.408	137.44	132.96	0.023	77.99	79.90	0.099
High Blood Pressure	1695	33.6	3352	66.4	191.23	194.12	0.024	53.16	55.33	0.005	128.08	131.43	0.078	162.66	132.45	<0.001	109.03	97.41	<0.001	33.47	29.74	<0.001	141.37	128.83	<0.001	82.40	78.58	<0.001
High Cholesterol	1807	35.8	3240	64.2	200.76	188.88	<0.001	52.90	55.55	<0.001	135.05	127.68	<0.001	164.81	130.19	<0.001	105.99	98.68	<0.001	31.82	30.53	<0.001	134.58	132.20	<0.001	79.61	80.01	0.231
High Triglycerides	1009	20.0	4038	80.0	197.58	192.05	0.001	47.28	56.23	<0.001	124.89	131.63	0.002	196.74	129.17	<0.001	111.08	98.86	<0.001	33.09	30.47	<0.001	136.90	132.08	<0.001	80.13	79.80	0.390
Gallbladder	425	8.4	4622	91.6	192.94	193.17	0.913	52.83	54.77	0.099	126.68	130.65	0.207	166.91	140.36	<0.001	107.29	100.75	<0.001	34.25	30.69	<0.001	136.77	132.71	<0.001	79.69	79.88	0.747
Gout	121	2.4	4926	97.6	179.88	193.48	<0.001	46.88	54.79	0.001	117.90	130.61	0.027	183.45	141.61	<0.001	118.45	100.89	<0.001	37.48	30.83	<0.001	143.52	132.79	<0.001	80.60	79.82	0.093
Kidney Disease	113	2.2	4934	97.8	192.36	193.17	0.864	55.50	54.58	0.785	128.21	130.36	0.717	161.03	142.18	0.025	108.94	101.13	0.005	32.16	30.96	0.142	138.12	132.94	0.006	79.72	79.87	0.893
Diabetes Myelitis	707	14.0	4340	86.0	178.60	195.53	<0.001	48.29	55.47	<0.001	115.28	132.70	<0.001	180.22	136.49	<0.001	138.06	95.28	<0.001	35.27	30. 3	<0.001	138.61	132.14	<0.001	78.94	80.02	0.021
Peptic Ulcer	178	3.5	4869	96.5	190.15	193.26	0.332	55.68	54.56	0.648	129.63	130.33	0.882	156.29	142.15	0.071	107.40	101.09	0.027	32.01	30.95	0.062	136.14	132.94	0.033	78.72	79.91	0.177
Thyroid	700	13.9	4347	86.1	194.81	192.88	0.261	57.40	54.15	0.004	134.27	129.67	0.105	148.06	141.73	0.084	102.04	101.19	0.457	31.97	30.84	0.001	133.66	132.95	0.379	78.83	80.03	0.011
Osteoporosis	360	7.1	4687	92.9	196.97	192.85	0.073	63.08	53.94	<0.001	132.79	130.12	0.428	131.80	143.56	0.018	94.48	101.53	0.021	28.63	31.17	<0.001	134.82	132.92	0.076	78.15	80.00	0.004
Osteoarthritis	476	9.4	4571	90.6	196.59	192.79	0.610	55.77	54.67	0.011	136.81	129.63	0.040	159.20	140.88	<0.001	104.58	100.97	0.010	33.17	30.76	<0.001	137.26	132.61	<0.001	79.35	79.92	0.255
Rheumatoid Arthritis	162	3.2	4885	96.8	195.52	193.07	0.467	52.74	54.66	0.350	128.93	130.35	0.778	172.38	141.64	<0.001	109.25	101.05	0.003	33.05	30.92	<0.001	139.40	132.84	<0.001	81.26	79.82	0.118
Overweight	2190	43.4	2857	56.6	193.63	192.78	0.479	52.07	56.55	<0.001	129.48	130.49	0.413	164.57	125.76	<0.001	107.81	92.29	<0.001	34.92	27.98	<0.001	136.87	130.10	<0.001	81.11	78.91	<0.001
Bronchitis Emphysema	355	7.0	4692	93.0	195.32	192.99	0.315	54.93	54.58	0.800	130.35	130.31	0.990	154.09	141.75	0.013	106.76	100.90	<0.001	33.79	30.78	<0.001	135.75	132. 85	0.007	80.17	39.84	0.607
Cancer	437	8.7	4610	91.3	192.12	193.25	0.593	56.19	54.45	0.176	127.75	130.56	0.366	148.44	142.06	0.158	103.08	101.14	0.186	30.87	31.00	0.724	137.99	132.58	<0.001	79.69	79.88	0.743

Participants with previous history had significantly **higher** results

Participants with previous history had significantly **lower** results

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

Table 5-37 Comparison of baseline to post-intervention percentage change by previous history

					Comparison PostIntervention % Change by Previous History																						
Previous history	True		False		T Cholest			HDL			LDL			Triglycerides			FPG			BMI			Bp Sys			Bp Dia		
	N	%	N	%	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	F Mean	p	T Mean	Mean	p	T Mean	F Mean	p	T Mean	F Mean	p
Angina	175	3.5	4872	96.5	9.700	10.300	0.560	6.65	7.41	0.524	10.94	11.24	0.860	0. 015	-0.002	0.582	4.83	4.47	0.788	3.11	3.05	0.816	4.840	3.62	0.567	4.46	4.30	0.877
Heart Attach	210	4.2	4837	95.8	10.24	10.280	0.970	5.32	7.48	0.241	10.73	11.25	0.776	-0.013	-0.001	0.668	8.06	4.33	0.002	3.05	3.06	0.968	2.850	3.69	0.668	4.53	4.29	0.804
Angioplasty	160	3.2	4887	96.8	9.68	10.290	0.630	6.72	7.41	0.585	11.14	11.23	0.964	0.016	-0.002	0.591	5.19	4.46	0.644	3.23	3.05	0.423	4.320	3.64	0.763	2.68	4.35	0.129
Bypass	110	2.2	4937	97.8	11.19	10.260	0.469	3.77	7.48	0.246	11.32	11.23	0.975	0.014	-0.002	0.686	6.87	4.44	0.198	2.96	3.06	0.718	4.840	3.63	0.652	2.87	4.33	0.265
Heart Failure	71	1.4	4976	98.6	0.341	10.280	0.760	7.39	7.39	0.999	9.01	11.26	0.384	0.031	-0.002	0.485	7.21	4.45	0.190	3.38	3.05	0.435	4.580	3.65	0.937	2.81	4.32	0.341
Blood Clots	135	2.7	4912	97.3	11.28	10.250	0.374	8.04	7.37	0.650	14.00	11.15	0.134	-0.006	-0.001	0.895	6.40	4.43	0.101	2.81	3.06	0.304	5.180	3.62	0.521	4.75	4.29	0.703
Abnormal EKG	194	3.8	4853	96.2	8.75	10.340	0.103	7.11	7.40	0.797	9.98	11.28	0.413	-0.366	0.000	0.216	5.60	4.44	0.302	3.06	3.06	0.969	5.080	3.60	0.470	6.03	4.23	0.073
Arrhythmia	566	11.2	4481	88.8	9.83	10.330	0.390	6.78	7.47	0.320	10.03	11.38	0.183	0.272	-0.005	0.071	4.98	4.42	0.361	3.04	3.06	0.895	2.960	3.75	0.521	4.13	4.32	0.746
Stroke	101	2.0	4946	98.0	9.75	10.290	0.691	6.71	7.40	0.658	13.28	11.19	0.313	-0.024	-0.001	0.570	8.87	4.40	0.012	2.06	3.07	0.126	4.490	3.64	0.760	2.43	4.34	0.162
High Blood Pressure	1695	33.6	3352	66.4	10.71	10.060	0.107	7.37	7.40	0.941	12.26	10.71	0.018	0.007	-0.006	0.272	6.03	3.70	<0.001	3.30	2.93	<0.001	5.210	2.85	0.004	4.71	4.09	0.123
High Cholesterol	1807	35.8	3240	64.2	10.63	10.070	0.176	7.17	7.52	0.443	11.74	10.94	0.224	0.002	-0.004	0.617	5.51	3.90	<0.001	3.19	2.98	0.008	3.840	3.56	0.727	4.50	4.18	0.425
High Triglycerides	1009	20.0	4038	80.0	9.91	10.370	0.380	6.20	7.69	0.006	10.17	11.50	0.135	0.020	-0.007	0.062	5.93	4.12	0.001	3.27	3.00	0.008	4.470	3.45	0.291	4.68	4.20	0.316
Gallbladder	425	8.4	4622	91.6	9.95	10.310	0.596	7.07	7.42	0.662	9.56	11.39	0.096	0.028	-0.004	0.070	5.48	4.39	0.117	3.27	3.04	0.103	5.090	3.53	0.264	2.22	2.31	0.897
Gout	121	2.4	4926	97.6	9.28	10.300	0.406	5.61	7.43	0.209	9.33	11.28	0.406	0.013	-0.002	0.686	7.93	4.04	0.043	3.61	3.04	0.033	2.430	3.69	0.650	8.00	4.21	0.003
Kidney Disease	113	2.2	4934	97.8	9.94	10.280	0.790	6.86	7.40	0.592	11.76	11.22	0.785	0.044	-0.002	0.235	3.77	4.50	0.702	2.72	3.06	0.200	5.850	3.61	0.398	4.94	4.29	0.617
Diabetes Myelitis	707	14.0	4340	86.0	8.48	10.570	<0.001	5.90	7.63	0.006	8.73	11.62	0.001	-0.017	0.001	0.264	10.37	3.52	<0.001	2.68	3.12	<0.001	4.220	3.57	0.559	4.42	4.28	0.804
Peptic Ulcer	178	3.5	4869	96.5	8.26	10.350	0.041	5.91	7.44	0.202	8.92	11.31	0.156	-0.953	0.002	0.073	5.59	4.45	0. 397	2.84	3.06	0.310	-0.650	3.82	0.339	2.49	4.37	0.234
Thyroid	700	13.9	4347	86.1	9.33	10.430	0.040	7.62	7.35	0.665	10.10	11.42	0.134	-0.006	-0.001	0.763	4.34	4.49	0.923	2.97	3.07	0.371	4.500	3.52	0.388	4.43	4.28	0.788
Osteoporosis	360	7.1	4687	92.9	8.07	10.440	0.001	5.79	7.51	0.044	7.22	11.54	<0.001	-0.033	0.001	0.128	3.85	4.53	0.369	2.63	3.09	0.003	4.010	3.63	0.805	4.83	4.26	0.451
Osteoarthritis	476	9.4	4571	90.6	8.67	10.440	0.006	6.17	7.51	0.077	9.47	11.41	0.068	-0.021	-0.001	0.273	3.98	4.54	0.399	3.02	3.06	0.766	5.240	3.50	0.196	4.90	4.24	0.317
Rheumatoid Arthritis	162	3.2	4885	96.8	8.66	10.330	0.123	6.91	7.43	0.331	9.25	11.29	0.258	0.005	-0.002	0.849	7.08	4.40	0.017	2.81	3. 06	0.268	1.700	3.72	0.697	5.81	4.25	0.157
Overweight	2190	43.4	2857	56.6	10.16	10.360	0.590	7.48	7.32	0.723	10.47	11.81	0.033	0.002	-0.004	0.049	5.55	3.66	<0.001	3.30	2.86	<0.001	4.780	2.76	0.010	4.71	3.89	0. 061
Bronchitis Emphysema	355	7.0	4692	93.0	9.89	10.300	0.570	8.09	7.34	0.381	9.96	11.33	0.254	-0.020	0.000	0.365	4.03	4.52	0.525	3. 1	3.05	0.775	4.200	3.62	0.704	4.01	4.32	0.678
Cancer	437	8.7	4610	91.3	10.63	10.24	0.561	7.34	7.39	0.947	11.410	11.210	0.853	0.018	-0.003	0.299	5.35	4.20	0.167	3.21	3.04	0.237	6.090	3.42	0.053	4.60	4.27	0.635
	Participants with previous history had significantly **higher** results																											
	Participants with previous history had significantly **lower** results																											

Units: TC, HDL,LDL,TG,FPG = mg/dL: SBP, DBP = mmHg: BMI = kg/m2

5.4 Predictors of change in biometric measures: regression analyses

In the previous section the influence of the selected participant characteristics (age, gender, marital status, religious affiliation, BMI, family history and previous history) on the biometrics have been examined in isolation of the other characteristics. In this section the results of regression analyses which were used to explore the influence of these participant characteristics as predictors of the change in the biometric measures are presented. Multiple regression was carried out using backwards stepping criteria.

The change from baseline to post-intervention in TC, HDL, LDL, TG, BMI, FPG, SBP and DBP was used as dependent variables. The impact of the following 11 independent variables, on each dependent variable was considered.

1. Age — age at baseline
2. Gender — gender (1 male, 2 female)
3. Religiosity — self-reported affiliation to the Seventh-day Adventist Church (1 SDA, 2 not SDA)
4. Marital Status — self-reported marital status (1 married, 2 single, divorced or widowed)
5. BMI — weight in kilograms divided by height in centimetres squared at baseline
6. Participants baseline level of the dependent variable
7. Family History of CVD Mortality — self-reported (1 family history, 2 no family history)
8. Family History of Diabetes Myelitis — self-reported (1 family history, 2 no family history)
9. Previous History Diabetes Myelitis — self-reported (1 previous history, 2 no previous history)
10. Previous History High Blood Pressure, — self-reported (1 previous history, 2 no previous history)
11. Previous History High Cholesterol — self-reported (1 previous history, 2 no previous history)

Assumptions of normality, linearity, homoscedasticity and independence of residuals were examined using residual scatterplots for each set of regression analyses. Further, the statistic variance inflation factor (VIF) was used to test for high levels of

collinearity and multicollinearity. Analysis of each set of data indicated these assumptions were appropriate.

5.4.1 Summary of predictors of change

Table 5-38 presents a summary of the impact of the 11 independent variables analysed in the regression analyses to predict the changes in the biometrics examined.

Table 5-38, Summary the beta values associated with the respective predictors of change for TC, HDL, LDL, TG, FPG, BMI, SBP and DBP

Summary Predictors of Change Showing R Squared and Beta Values									
Predictors	TC	HDL	LDL	TG	FPG	BMI	SBP	DBP	No biometrics influenced
R Square	0.256	0.237	0.235	0.316	0.480	0.213	0.142	0.344	
Age	-0.087	0.012	-0.068				-0.092		4
Gender (male/female)	-0.190		-0.129	-0.032		-0.090			4
SDA/non-SDA	0.086	0.084				0.059			3
Marital Status			-0.039		-0.029	-0.079			3
Baseline BMI		0.021		-0.118	-0.049	0.454	-0.048	-0.096	6
Baseline Level of Factor	0.503	0.476	0.464	0.591	0.771	0.454	0.400	0.615	8
Family History of CVD									0
Family History of Diabetes	0.044		0.042	0.067		0.048	0.095		5
Prev Hist Diabetes					0.125	0.094		-0.059	3
Prev Hist High BP	-0.040		-0.046					0.078	3
Prev Hist High Cholesterol				0.059				-0.032	2
Independent Variables Count	6	4	6	5	4	6	4	5	

Values shown were significant at the 0.05 level

Table 5-38 is replicated in Table 5-39 with beta values replaced by arrows. An up arrow (↑) indicates that the greater change is toward the value shown in brackets and a down arrow (↓) indicates greater change is away from the value shown in brackets. For example the ↑ for Age (Younger) under TC indicates that younger participants had

greater change in TC while the ↓ for SDA (Yes) indicates that participants indicating an affiliation with the Seventh-day Adventist Church had less change in TC.

Table 5-39 Graphical summary of predictors of change for TC, HDL, LDL, TG, FPG, BMI, SBP and DBP

Summary Predictors of Change Showing R Square and Beta Values										
Predictors		TC	HDL	LDL	TG	FPG	BMI	SBP	DBP	Count
	R Square	0.256	0.237	0.235	0.316	0.480	0.213	0.142	0.344	
Age	(Younger)	↑	↓	↑				↑		4
Gender	(Male)	↑		↑	↑		↑			4
SDA	(Yes)	↓	↓				↓			3
Marital Status	(Married)			↓		↓	↓			3
Baseline BMI	(Higher)		↑		↓	↓	↑	↓	↓	6
Baseline Level of Factor	(Higher)	↑	↑	↑	↑	↑	↑	↑	↑	8
Family History of CVD	(Yes)									0
Family Hist of Diabetes	(Yes)	↓		↓	↓		↓	↓		5
Prev Hist Diabetes	(Yes)					↓	↓		↑	3
Prev Hist High BP	(Yes)	↑		↑					↓	3
Prev Hist High Chol	(Yes)				↓				↑	2
	Count	6	4	6	5	4	6	4	5	

The R Square shows that this group of predictors accounted for around 48.0% of the variance in FPG but only 14.2% of the variance in SBP. This indicates that there are other factors that contributed the variance. Unfortunately a key factor of the CHIP program and a potential predictor of changes in participant's biometric measures, that was not able to be analysed due to a lack of data, is the change in participant's diet. The CHIP program encourages participants to move towards a plant-based diet but data relating to changes in this area were not collected. Also there was no data collected on compliance with the intervention.

The one consistently strong predictor of change was the baseline level of that biometric and this was the strongest predictor of change for all eight biometrics

examined. A family history of CVD, however, did not register as a predictor for any of the biometric measures analysed.

Of interest is the fact that the while, Baseline BMI showed a positive relationship to change in HDL an inverse relationship to change was found for TG, FPG, SBP and DBP. In this population those with a higher BMI at baseline had significantly less change in TG, FPG, SBP and DBP than participants with a lower BMI at baseline. This seems to be different from what would be expected as it appears that a higher BMI is normally correlated with a higher level of these biometrics and thus if there is a positive correlation between the baseline level of these biometrics and change, one would normally assume that a higher BMI at baseline would also be positively correlated with change in the biometric. This is not the case in this analysis.

The regression analyses showed that younger participants had greater change in TC, LDL, FPG and SBP however age was not a predictor for the other dependent variables analysed. With regard to gender, male participants had greater change in TC, LDL cholesterol and BMI than did the female participants, however gender was not significant for the other biometrics analysed. With respect to religiosity, participants indicating that they had an affiliation with the Seventh-day Adventist Church were shown to have less change in TC, HDL and BMI, however religiosity was not shown as a predictor for change in the other biometrics analysed. Marital status was only shown to be a predictor for change in BMI with those participants who were married experiencing less change in BMI than the non-married participants.

With regards to family history as an independent variable, a family history of CVD was not a significant predictor for any of the biometrics. A family history of diabetes was a

significant predictor for changes in LDL, BMI and SBP with those participants who indicated that they had a family history of diabetes mellitus achieving less change in LDL, BMI and SBP than those participants who indicated they did not have a family history of diabetes myelitis.

In relation to participant previous medical history, those participants indicating they had a personal history of diabetes myelitis experienced less change in TG, FPG and BMI than those participants who did not indicate having a previous history of diabetes myelitis however those participants who indicated they had a previous history of diabetes myelitis achieved greater change in TC and DBP. A previous history of higher blood pressure was significant for changes in DBP and LDL. Less change in DBP was realised by those for those participants who indicated they had a previous history of high blood pressure and greater change in LDL was shown for those participants with a previous history high blood pressure. A previous history of high cholesterol was only a significant predictor for changes in TG with those participants who indicated they had a previous history of high cholesterol showing less change in TG.

5.4.2 Summary

Previous sections have shown that all participants can achieve significant improvements in risk factors of chronic disease from volunteer-delivered, community based CHIP interventions. This regression analyses demonstrates that those participants with the greatest risk, at baseline achieve the greatest benefits during the intervention. It also appears that greater change in the biometric indices is achieved by those participants who are younger, male and non-SDA. However it would appear that those participants with the highest BMI at baseline achieve less change than those participants with lower

BMI at baseline, for some biometrics. It would also appear that family history and previous medical history of the participants has some impact as a predictor of change in the biometrics analysed.

5.5 Discussion

The aim of this chapter was to address the second research question: "What is the impact of selected participant factors, including age, gender, marital status, religious affiliation, previous history, family history and body weight on the outcomes achieved by participants in the CHIP intervention?" The key finding of this chapter was that regardless of age, gender, marital status, religious affiliation, body weight, family history or previous history, significant improvements in the risk factors associated with MetS can be achieved through the CHIP intervention when delivered by volunteers to free-living individuals in their community. The analysis presented in this chapter also indicated that participant factors do factors influence the responsiveness of participants to the intervention. The effect of these factors is discussed in this section.

5.5.1 Biometric level at baseline

The greatest predictor of the change achieved in a particular biometric by participants in the CHIP intervention was the baseline level of that biometric. Participants in the highest risk category consistently achieved the greatest amount of change. This was demonstrated in the overall results (presented in Chapter 4), and the regression analysis reported in this chapter. The trend for participants with the greatest need achieving the greatest improvement has also been demonstrated in professionally delivered CHIP interventions (Aldana et al., 2008; Diehl, 1998; Englert et al., 2004; Englert et al., 2007) and in other professionally delivered lifestyle interventions (Ellsworth et al., 2004).

5.5.2 Gender

A significant finding of this study was that gender had an impact on the outcome achieved by participants. This study found that at baseline, male participants had significantly lower mean TC (10.8%), HDL (28.2%) and LDL (5.5%) but significantly higher TG (10.4%), FPG (12.5%), SBP (2.6%) and DBP (3.1%) than did the female participants, however there was no difference between male and female participants for BMI at baseline. From baseline to postintervention, male participants showed significantly greater reductions in TC (14.7 %), LDL (24.1%), TG (55.6%), FPG (39.1%), BMI (13.9%), SBP (20.6%) and DBP (18.7%) and significantly lower reductions in HDL (55.5%) this indicates that male participants achieved better outcomes, during the intervention, than did the female participants. It was also found that male participants demonstrated the greatest change at a younger age than did the female participants.

It is commonly perceived that there is a difference in health status between males and females. Men die younger, underreport their symptoms and their pride stops them from asking for help when they do get sick (Moynihan, 1998). But while men die younger it is often perceived that women have poorer health (Macintyre, Hunt, & Sweeting, 1996). Women generally have higher mortality and morbidity than men after a heart attack, angioplasty or bypass surgery (Koertge et al., 2003) but are less likely to be referred to cardiac rehabilitation programs following a coronary incident (Allen, Scott, Stewart, & Young, 2004).

It has also been reported that men have a higher incidence of MetS. In a review article Regitz-Zagrosek, Lehmkuhl, and Mahmoodzadeh (2007) suggest that MetS is more common among men, however the incidents of MetS is rising in the female population

particularly among younger females. Ford et al. (2002) found that the age adjusted prevalence of MetS in the overall US population, was similar for males (24.0%) and females (23.4%) and Alberti et al. (2009) found that the unadjusted prevalence of MetS in the US population was 41.9% in males and 38.3% in females. As observed previously, the prevalence of MetS, in the participants in this study, of 52.6% for males and 45.6% for females is higher than in the general US population with males having a significantly higher prevalence of MetS than females.

However, as with most questions relating to health, issues of gender are not simply issues of gender but are complicated by other factors such as ethnicity, socio-economic status and education (Read & Gorman, 2006). Both physiological and psychological issues need to be considered.

This study found that at baseline, male participants had significantly lower mean TC (10.8%), HDL (28.2%) and LDL (5.5%) but significantly higher TG (10.4%), FPG (12.5%), SBP (2.6%) and DBP (3.1%) than did the female participants, however there was no difference between male and female participants for BMI at baseline. These findings are comparable to other studies showing gender differences in health status on entry into lifestyle interventions (Cannistra, Balady, O'Malley, Weiner, & Ryan, 1992; Koertge et al., 2003).

While significant reductions were observed, from baseline to post-intervention, on all biometric measures for both male and female participants, male participants showed significantly greater reductions in TC (14.7 %), LDL (24.1%), TG (55.6%), FPG (39.1%), BMI (13.9%), SBP (20.6%) and DBP (18.7%) and significantly lower reductions in HDL (55.5%). This indicates that male participants actually achieved better health outcomes from CHIP,

in relation to MetS, than did the female participants. Other studies (Daubenmier et al., 2007; Pischke, Weidner, Elliott-Eller, & Ornish, 2007; Roberts & Barnard, 2005) have also demonstrated greater change in male participants. While this study found that when adjustments were made for age and other baseline risk factors, gender was significant for TC and LDL and BMI but not for other risk factors Englert et al. (2007) found that gender was significant across all risk factors. In comparison the Multicentre Lifestyle Demonstration Project found that the improvement of female participants was similar to that experienced by the male participants (Koertge et al., 2003).

There are physiological differences between males and females that need to be considered. Males have significantly greater muscle mass than do females, even among the elite athletes (Alway, Grumbt, Gonyea, & Stray-Gundersen, 1989). It has been suggested that there are gender specific pathophysiological differences in the in relation to MetS in that woman tend to develop peripheral adiposity with subcutaneous fat accumulation whereas men are more prone to central or android obesity. (Regitz-Zagrosek, Lehmkuhl, & Weickert, 2006). The central and android fat is more metabolically active and therefore easier to remove than fat on the hips and thighs.

In studies looking at barriers to change it has been found that the most important barrier for women is self-esteem while for men it is time (Mosca, McGillen, & Rubenfire, 1998). It also seems that different approaches need to be taken to encourage men to lose weight than that taken for women. In the Australian experience, it has been found that the hardest part is getting men to admit that they have a problem. It has also been found that humour is very effective when working with a male audience, but obesity is not a laughing matter for females (Egger, 2007, p. 212; Plater & Egger, 2011). There are,

however, some who do not think that humour should be used in such a weighty topic as obesity (Egger, 2010; Frank & Kahan, 2010).

There are a number of factors that could have contributed to the greater benefits achieved by male participants. Marital status may be a factor with a significantly greater percentage of male participants (85.2%), than female participants (68%) indicating that they were married. Also, as two thirds of the participants in these CHIP programs were female (66.5%) as opposed to only one third being male (33.5%) it would seem that there was a greater chance that the male participants were attending the CHIP program with a spouse and while we do not have the data to analyse this detail others have shown that social support and spouse support may be an important factor in achieving the lifestyle changes (Verheijden & Kok, 2005). This is a question that needs to be researched in more detail in further studies.

As pointed out in an earlier section of this chapter males achieve greater change in a younger age bracket then did the females. This would indicate that benefit in regards to Quality Adjusted Life Years may actually be higher for male participants then for female participants.

While it is not fully understood why the effect of the CHIP intervention is greater for men than women the propensity for men to lay fat in the abdominal region which is more metabolically active and therefore easier to remove than fat on the hips and thighs may offer a physiological explanation. In addition, men tend to have more muscle mass and therefore a higher metabolic rate than women, so increases in physical activity and higher intakes of health promoting plant foods may be expected to more quickly ameliorate the previous adverse effect of lifestyle. A further explanation may be that

married men often attended with their spouses and therefore benefited from the household changes made by the women.

5.5.3 Age

For the CHIP participants in this study, it can be seen that at baseline mean TC, LDL, TG, FPG levels, BMI and DBP are lower in the younger and the older age groups peaking in the 50-59 age group while HDL levels rise with age peaking in the 70-79 age group and SBP continues to rise with age.

The increasing prevalence of symptoms associated with MetS with age has been observed in the US populations (Ford et al., 2002; Ford et al., 2004) and worldwide (Regitz-Zagrosek et al., 2006). The attenuation of MetS in individuals 70+ has also been noted in general population studies of MetS (Hu et al., 2004).

In this study, significant change was observed in all biometrics in all age categories except for TG and FPG in the 0-29 age categories. When the change was observed across age categories it was found that the largest changes were achieved for TC, LDL and HDL in the 40-49 age category and in the 50-59 age category for FPG and BMI. Although the changes are maximum for the 50-59 age group these are not significantly higher than the 40-49 age group. This is consistent with the fact that these age groups showed the highest risk, at baseline.

The indicators of risk for chronic disease can change with age. For instance, in relation to BP as a predictor of Chronic Heart Disease (CHD) risk Franklin et al. (2001) have shown that for patients <50 years of age DBP is the strongest predictor of CHD risk while for the 50 to 59 year old both BP indexes were compatible predictors. For those in the

60+ age group DBP was negatively related to CHD risk and pulse pressure became superior to SBP as a predictor of CHD risk (Franklin et al., 2001).

The results of the current study, in relationship to age, would seem to indicate that if we can enrolled individuals in a lifestyle intervention program, such as CHIP, before they turn 40 we can have a significant impact on the risk factors for MetS and associated mortality and morbidity such as CVD and T2D, thus significantly impacting the lifespan and quality of life for these individuals. Fraser and colleagues suggest, from reviewing the Adventist Health Study data, that maximum benefit from changes to lifestyle habits is obtained if the changes are made before the age of 30, though significant benefits can be gained at any age (Fraser & Shavlik, 2001).

5.5.4 Marital status

A limitation of the data collection on marital status was that participants were only given before options of single, married, divorced or widowed and this did not make provision for participants who were in de facto or other relationships.

A further finding of the study was that married participants generally achieve better outcomes than did the widowed, divorced or single participants. There has long been debate about the effect of marital status on the health of individuals. In 1980 it was suggested that marital status had no effect on health status (Hunt et al., 1980), however a number of epidemiological studies have since shown a clear interaction between social integration and mortality and morbidity, and evidence began to accrue that social integration had an impact on health including cardiovascular function (House, Landis, & Umberson, 1988). Marital status is a significant component of social interaction and marriage is the central relationship for a majority of adults. Being married is now seen as

having major health benefits. Morbidity and mortality has been shown to be lower for married individuals than unmarried individuals (Kiecolt-Glaser & Newton, 2001) and a meta-analysis found that singles have a 30% increased risk of mortality relative to married persons (Roelfs, Shor, Kalish, & Yogev, 2011). As with most issues relating to health the correlation between marital status and health status is not a simple relationship, factors such as age and also the quality of that relationship can have an impact on health status (Troxel, Matthews, Gallo, & Kuller, 2005).

Gallo et al. (2003) suggest that the health benefits of marriage are greater for men than for women. A possible explanation for the greater health benefits for men in marriage could be that women are more likely than men to attempt to control others' health behaviour and that the female partner is more active in encouraging the male partner to improve his health habits than vice versa (Kiecolt-Glaser & Newton, 2001; Umberson, 1992).

Significant reductions in the risk factors relating to MetS were observed in this study by participants in all marital status categories. Further, analysis of the reduction in risk factors for MetS achieved during the intervention, by marital status, showed that participants in the married category had significantly greater reductions in TC, LDL, FPG and BMI. When other factors such as age and gender were corrected for by regression analysis, it was found that marital status was a significant predictor of change in LDL, FPG and BMI.

5.5.5 Religious affiliation

An analysis of the participants in the CHIP lifestyle intervention, in this study, showed that the SDA participants had significantly better biometric data, in regard to risk

factors associated with MetS, at baseline than the non-SDA participants. However the gap between the Adventist and the non-Adventist, in regard to risk factors associated with MetS, was reduced over the period of intervention.

An ongoing research project from Loma Linda University has compared the health status of Seventh-day Adventist's with that of the general population. These studies began with the Adventist Mortality Study from 1960-66 and then the First Adventist Health Study, from 1974-88 focusing on Adventists in California. Then in 2002 Adventist Health Study-2 began to analyse the health status of over 90,000 Seventh-day Adventists across North America. These studies have found that Adventists, as a population, have lower risk for most cancers, CVD and T2D which equates to decreased mortality and morbidity (Beeson, Mills, Phillips, Andress, & Fraser, 1989; Butler et al., 2008; Fraser, 1988, 1994, 2005; Fraser & Swannell, 1981; Heuch, Jacobsen, & Fraser, 2005; Key et al., 1999; Slavicek et al., 2008; Tonstad et al., 2011). A more detailed analysis of the Seventh-day Adventist population has shown that Adventists who adopt a plant-based diet have significant advantages over those Adventists consuming a meat-based diet in such diverse areas as the onset of dementia (Giem, Beeson, & Fraser, 1993), prostate cancer (Mills, Beeson, Phillips, & Fraser, 1989), CVD (Slavicek et al., 2008), T2D (Tonstad et al., 2009) and MetS (Rizzo et al., 2011). It has been suggested that the advantages observed in the Adventist population, of up to 10 years additional lifespan, are the effect of healthier choices made by this population (Fraser & Shavlik, 2001).

An analysis of the participants in the CHIP lifestyle intervention, in this study, showed that the SDA participants had significantly better biometric data, in regard to risk factors associated with MetS, at baseline than the non-SDA participants. This is consistent

with the findings of the Adventist Health Studies outlined above. It was also observed in the study that the gap between the Adventist and the non-Adventist, in regard to risk factors associated with MetS, was reduced over the period of intervention. This would tend to support the suggestion made by Fraser and Shavlik (2001) that it would seem that the health advantages experienced by Seventh-day Adventists are a factor of lifestyle choices and that these advantages can be achieved by others who choose to make these same lifestyle choices.

5.5.6 Body Mass Index

A further key finding was that participants in all BMI categories, at baseline, were able to achieve significant weight loss during the CHIP intervention. CHIP recommends a dietary fat intake of less than 20% of calories per day which on an 1800 calorie diet equates to around 40g of fat per day. While some studies have found that there is no correlation between fat intake and weight change(Forouhi et al., 2009) other researchers have found that a significant reduction in fat intake is associated with greater weight loss (Zulet, Berkenpas, & Martinez, 2005). Carmichael et al. (1998) found that greater initial weight loss was associated with greater decrease in fat intake and weight loss occurred in all participants who reported a fat intake of 40 g per day or less. In a review article of epidemiological and intervention studies Gaesser (2007) concluded that a low-fat diet with emphasis on fibre rich carbohydrates, particularly cereal fibre could be beneficial for weight loss.

There has also been shown to be a correlation between fat intake and weight gain. Researchers from the Guttenberg study found a strong correlation between percentage of calories from fat and weight gain in women (Heitmann, Lissner, Sorensen, & Bengtsson,

1995; Lissner & Heitmann, 1995). An analysis of the Nurses' Health Study found only a weak positive association between overall percentage of calories from fat consumption and weight gain, however it found a strong association with percentage of calories from animal, saturated and trans fat and weight gain (Field, Willett, Lissner, & Colditz, 2007). Weight loss with a low-fat diet in obese men with MetS has been found to decrease LDL and have positive effects on HDL (Ng, Watts, Barrett, Rye, & Chan, 2007) and a low-fat diet was shown to have a positive impact on the risk factors for cardiovascular disease within one week (Slavicek et al., 2008). The results from this analysis of CHIP participants would seem to support the benefits of a low-fat, high unrefined carbohydrate diet.

Overall it was found that significant changes in the risk factors for MetS were achieved by participants regardless of baseline BMI, with those participants with the highest BMI, at baseline, achieving the greatest reductions in FPG, BMI, systolic and diastolic blood pressures. However the greatest changes in TC, LDL and TG were experienced by those participants in the Overweight or Obese I category, while those participants in the Obese II and Obese III categories, at baseline, experience significantly less change in these components of the lipid profile. While it has been suggested that lifestyle intervention may not be effective in the severely obese (National Institute of Health, 1998) it has been clearly shown that the extremely obese do respond very well to appropriate lifestyle intervention (Goodpaster et al., 2010; Unick et al., 2011).

In this study regression analysis showed a negative correlation between baseline BMI and changes in LDL, FPG, SBP and DBP. This result was unexpected. A closer examination of the data shows that those participants in the Obese Class II and Obese Class III (BMI > 35) do not experience as much change in these risk factors as do those

participants who are overweight or categorised as Obese Class I (BMI < 35). It would seem that the extremely obese participants do not respond as well to this lifestyle intervention in the short term as do the less severely obese individuals in reducing their lipid profile, FPG and BP. However over the long term, moderate weight loss of 5% even in the extremely obese has been shown to be beneficial (Pasanisi, Contaldo, de Simone, & Mancini, 2001). Those in the Obese Class III showed an average drop in BMI of 1.61 BMI units which equates to a 4% drop in BMI for those with a BMI of 40 in just 30 days indicating that the CHIP intervention provided substantial benefits even for the extremely obese.

5.6 Conclusion

In this chapter an analysis of how selected participant factors contribute to the outcomes achieved in community-based, volunteer-delivered CHIP interventions has been presented.

Male participants were shown to achieve greater improvement, in the risk factors associated with MetS, than the female participants, and to achieve the greatest change at a younger age than the female participants. Across all participants, greatest improvement occurred in the 40-49 age group and married participants did better than the single, divorced or widowed participants. With regard to religious affiliation it was found that SDA participants had better biometric scores at baseline however the non-SDA participants achieved greater improvements in the biometrics during CHIP intervention and that the gap between SDA and non-SDA participants had narrowed significantly by the end of the intervention. Participants with the highest BMI at baseline achieve the greatest improvements in BMI, FPG and BP however those participants in the highest

obesity categories, (obese II and obese III) showed significantly less improvement in their lipid profile than did participants whose weight place them in the overweight and obese I categories at baseline.

The regression analysis showed that the single biggest predictor of change in the participant's biometric profile was the participant's baseline biometric level with those participants having the worst biometrics at baseline achieving the best results. However even this generalisation is not clear-cut, as discussed above, participants baseline BMI level did not follow this trend. Clearly the biochemical interactions that take place during a lifestyle intervention such as CHIP are very complex and only poorly understood.

Unfortunate, no measures of compliance were taken for this data set. It can probably be assume that those who are most compliant achieved the greatest results. Ornish has shown a very clear dose response between compliance and outcome (Ornish, 2009b; Ornish et al., 1990a). There is a need for more research looking at compliance level particularly with regard to dietary factors.

This analysis has shown that regardless of age, gender, marital status, religious affiliation, baseline weight, previous history or family history, significant improvements in the risk factors associated with MetS can be achieved by participants in the CHIP lifestyle intervention when delivered by volunteers in their local community.

Chapter 6 Conclusion: Key findings, limitations and recommendations

This dissertation makes a valuable contribution to the understanding of utilising lifestyle interventions to reduce the risk factors associated with MetS and chronic disease. Unique elements of the study include the large sample size and the use of volunteer facilitators in the context of a community-based lifestyle intervention.

The study was guided by two research questions which are shown below with the key findings of the study:

1. How effective is the Coronary Health Improvement Program (CHIP) lifestyle intervention for reducing selected risk factors of chronic disease associated with the Metabolic Syndrome, when delivered by volunteers to free-living participants in their community?

This large cohort study has shown that significant and meaningful reductions in the risk factors associated with the Metabolic Syndrome can be achieved by the CHIP lifestyle intervention when delivered by volunteers to free-living participants in their community.

2. What is the impact of selected participant factors including age, gender, marital status, religious affiliation, previous history, family history and body weight on the outcomes achieved by the CHIP intervention?

This study has shown that regardless of age, gender, marital status, religious affiliation, previous history, family history and body weight, participants in the CHIP intervention can achieve significant improvements in the risk factors associated with the

Metabolic Syndrome, however, participant factors do influence the responsiveness of the participants to the program. In general, male participants achieved better results than females, and males tended to achieve their best outcomes at a younger age than the female participants. Married participants achieved better outcomes than single, divorced or widowed participants. SDA participants had better risk profiles at baseline, however, the non-SDA participants achieved better outcomes during the intervention. Participants who had the highest BMI at baseline achieved the greatest changes in BMI, FPG and BP, however, those participants with a BMI greater than 35 kg/m^2 showed significantly less improvement in their lipid profile than those participants with a BMI between 25 and 35 kg/m^2.

In this final chapter, the key conclusions from the study are presented. A discussion of the limitations of this research project is then presented followed by recommendations for further study.

6.1 Key findings

The key findings of this study include:

1. The CHIP life-style intervention achieves, within 30 days, significant reductions in the various risk factors associated with MetS.
2. The CHIP intervention achieves the best outcomes among individuals with the highest level of risk at program entry. It was found that CHIP works best for those who need it the most.
3. Significant improvements in the risk factors of MetS and chronic disease can be achieved by participants, in CHIP interventions, regardless of age, sex, gender, marital status, religious affiliation, previous history, family history or health status. However, some groups of participants achieve better results than others.
4. Volunteers, who have donated their time, can deliver CHIP lifestyle interventions to free-living participants in their community that achieve significant and meaningful reductions in the risk factors for chronic disease. Further, that the outcomes achieved by volunteers are comparable to those achieved by paid health professionals.
5. The utilisation of volunteer facilitators in the delivery of CHIP interventions maximizes the use of human and social capital. Volunteer facilitators provide the opportunity for a cost-effective, accessible and easily scalable approach for the delivery of lifestyle interventions

6.2 Limitations

This study has shown that the CHIP intervention when delivered by volunteer facilitators achieve significant results over 30 days. However a significant limitation of this study is that the effects have only been demonstrated in the short term, data has only been collected for a 30 day period. Further research is needed to establish the long-term effectiveness of the CHIP intervention.

Several factors may have influenced the results observed in this study. Firstly, data were not collected on the participants' compliance to the program and therefore the extent to which the participants adhered to the lifestyle changes promoted in the CHIP intervention cannot be factored into the analysis of the biometric changes. It can be assumed that not all participants completely embraced the lifestyle behaviour changes advocated in the program, however, this would have only diluted the overall significance of the results. The second limiting factor was that accurate data were not collected on changes in medication use throughout the 30-day intervention. Anecdotal reports from facilitators suggest that many participants, in consultation with their personal physicians, decreased or even ceased their medication use during the intervention. While this is a desirable outcome, it would have once again had the effect of diluting the overall significance of the results and diminishing the apparent effectiveness of the program as observed in the mean changes from baseline to post-intervention.

A further limitation of the study is that participants were self-selected. As the participants were self-selected, they likely entered the program with an elevated readiness for change and hence willingness to engage in the intervention. In accordance with the transtheoretical model of behaviour change (Prochaska et al., 2013), a key

objective of the first few sessions of the CHIP intervention is to move individuals from pre-contemplation to action. Yet the participants were probably beyond the pre-contemplation stage at program entry. It would be interesting to compare the outcomes observed in this study with participants who had not shown an initial interest in the program.

In the absence of a control group, the extent to which regression to the mean explains the observed improvements cannot be determined. Consistent with regression to the mean is that the individuals with the most extreme baseline measures tended to experience the greatest improvements and hence inclination towards the norm. However, given the large size of the sample and that in some of the outcomes measured the high risk classifications moved 1.5 to 2 standard deviations, regression to the mean likely only explains a small component of the observed results. Noteworthy, several studies of CHIP in the United States have demonstrated the effectiveness of the intervention using a randomised control design and the magnitude of change observed in the present study is similar to the treatment groups of these studies (Aldana, Greenlaw, Diehl, Salberg, Merrill, Ohmine, et al., 2005; Englert et al., 2007; Merrill, Massey, et al., 2008).

Another potential confounder of the outcomes observed in this study is the Hawthorne effect. While the research team were not responsible for conducting the interventions, the participants' behaviours and level of engagement with the program was undoubtedly influenced by the blood measures taken at baseline and post-intervention. Given that the pre and post blood work is a standard component of the CHIP intervention, improvements achieved as a result of these accountability measures could

be considered part of the intervention itself. However, further research is needed to elucidate the influence of the unique lifestyle recommendations of the CHIP intervention—namely its emphasis on a whole-food, plant-based eating pattern—from the motivational properties of the pre and post-intervention measurements made on the participants. Certainly, the inclusion of accountability measures is likely to be an important component of lifestyle interventions targeting chronic disease.

6.3 Recommendations for further study

This study has made an important contribution to the understanding of how lifestyle interventions can be utilised to combat chronic disease. While this study makes a valuable contribution, it also demonstrates the enormous potential for further research in this area.

To address some of the limitations of the study outlined above there is a need for further studies of a randomised control design. Further studies also need to collect validated data on compliance to the intervention relating to changes in dietary habits and exercise levels. As the data analysed in this study was short-term (30 days), there is a need for long-term studies of the interventions effectiveness.

While this study has explored the impact of some participant factors such as age, gender, religiosity, marital status, previous history and the baseline biometric levels, there is a need for more research in this area.

There is also a need to study the impact the facilitator has on the outcomes achieved by the CHIP interventions. For instance, does the facilitator's personal compliance to the recommendations of the intervention influence the outcomes achieved by the participants? Also, do voluntary facilitators who have some medical training, such as nurses, doctors or allied health professionals achieve better outcomes than those facilitators with no medical background?

One of the primary questions facing Lifestyle Medicine is "How can lifestyle interventions be most effectively implemented?" The current study further expanded the body of knowledge surrounding this question by showing, in a large study, that volunteers can effectively facilitate lifestyle interventions. However, there is much to be learned.

7 References

ACLM. (2013). *American College of Lifestyle Medicine Webpage.* Retrieved October 10, 2013, from http://www.lifestylemedicine.org/

Adamsson, V., Reumark, A., Cederholm, T., Vessby, B., Riserus, U., & Johansson, G. (2012). What is a healthy Nordic diet? Foods and nutrients in the NORDIET study. *Food Nutr Res, 56*.

Adamsson, V., Reumark, A., Fredriksson, I. B., Hammarstrom, E., Vessby, B., Johansson, G., et al. (2011). Effects of a healthy Nordic diet on cardiovascular risk factors in hypercholesterolaemic subjects: a randomized controlled trial (NORDIET). *J Intern Med, 269*(2), 150-159.

Agatston, A. (2012). Why America Is Fatter and Sicker Than Ever. *Circulation, 126*(1), e3-e5.

Ajzen, I. (1985). From Intentions to Actions: A Theory of Planned Behavior. In J. Kuhl & J. Beckmann (Eds.), *Action Control* (pp. 11-39): Springer Berlin Heidelberg.

Ajzen, I. (1991). The theory of planned behavior. *Organ Behav Hum Decis Process, 50*(2), 179-211.

Ajzen, I. (2001). Nature and operation of attitudes. *Annu Rev Psychol, 52*(1), 27-58.

Ajzen, I. (2011). The theory of planned behaviour: Reactions and reflections. *Psychology & Health, 26*(9), 1113-1127.

Alberti, K. G., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., et al. (2009). Harmonizing the Metabolic Syndrome: A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation, 120*(16), 1640-1645.

Alberti, K. G., & Zimmet, P. (2006). The metabolic syndrome: time to reflect. *Curr Diab Rep, 6*(4), 259-261.

Alberti, K. G., Zimmet, P., & Shaw, J. (2005). The metabolic syndrome-a new worldwide definition. *The Lancet, 366*(9491), 1059.

Alberti, K. G., Zimmet, P., & Shaw, J. (2006). Metabolic syndrome--a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabet Med, 23*(5), 469-480.

Alberti, K. G., Zimmet, P., Shaw, J., & Grundy, S. (2006). The IDF consensus worldwide definition of the Metabolic Syndrome. Brussels: Internation Diabetes Federation.

Alberti, K. G., & Zimmet, P. Z. (1998). Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabet Med, 15*(7), 539-553.

Aldana, S. G. (2001). Financial impact of health promotion programs: a comprehensive review of the literature. *Am J Health Promot, 15*(5), 296-320.

Aldana, S. G., Greenlaw, R., Diehl, H. A., Englert, H., & Jackson, R. (2002). Impact of the coronary health improvement project (CHIP) on several employee populations. *J Occup Environ Med, 44*(9), 831-839.

Aldana, S. G., Greenlaw, R., Thomas, D., Salberg, A., DeMordaunt, T., Fellingham, G. W., et al. (2004). The influence of an intense cardiovascular disease risk factor modification program. *Prev Cardiol, 7*(1), 19-25.

Aldana, S. G., Greenlaw, R. L., Diehl, H. A., Merrill, R. M., Salberg, A., & Englert, H. (2008). A video-based lifestyle intervention and changes in coronary risk. *Health Educ Res, 23*(1), 115-124.

Aldana, S. G., Greenlaw, R. L., Diehl, H. A., Salberg, A., Merrill, R. M., & Ohmine, S. (2005). The effects of a worksite chronic disease prevention program. *J Occup Environ Med, 47*(6), 558-564.

Aldana, S. G., Greenlaw, R. L., Diehl, H. A., Salberg, A., Merrill, R. M., Ohmine, S., et al. (2005). Effects of an intensive diet and physical activity modification program on the health risks of adults. *J Am Diet Assoc, 105*(3), 371-381.

Aldana, S. G., Greenlaw, R. L., Diehl, H. A., Salberg, A., Merrill, R. M., Ohmine, S., et al. (2006). The behavioral and clinical effects of therapeutic lifestyle change on middle-aged adults. *Prev Chronic Dis, 3*(1), A05.

Aldana, S. G., Whitmer, W. R., Greenlaw, R., Avins, A. L., Salberg, A., Barnhurst, M., et al. (2003). Cardiovascular risk reductions associated with aggressive lifestyle modification and cardiac rehabilitation. *Heart Lung, 32*(6), 374-382.

Aldana, S. G., Whitmer, W. R., Greenlaw, R., Avins, A. L., Thomas, D., Salberg, A., et al. (2006). Effect of intense lifestyle modification and cardiac rehabilitation on psychosocial cardiovascular disease risk factors and quality of life. *Behav Modif, 30*(4), 507-525.

Allen, J. A. (2009). *Heart disease cost US $503 billion in 2010.* Retrieved November 4, 2010, from http://www.reuters.com/article/idUSTRE5BG52I20091217

Allen, J. K., Scott, L. B., Stewart, K. J., & Young, D. R. (2004). Disparities in Women's Referral to and Enrollment in Outpatient Cardiac Rehabilitation. *J Gen Intern Med, 19*(7), 747-753.

ALMA. (2013). *Australian Lifestyle Medicine Association Website.* Retrieved 10 October, 2013, from https://lifestylemedicine.com.au/

Alter, D. M. D. P. (2007). Therapeutic lifestyle and disease-management interventions: pushing the scientific envelope. *Can Med Assoc J, 177*(8), 887.

Alwan, A. (2011). *Global status report om noncommunicable diseases 2010.* Retrieved August 31, 2012, from http://www.who.int/nmh/publications/ncd_report2010/en/

Alway, S. E., Grumbt, W. H., Gonyea, W. J., & Stray-Gundersen, J. (1989). Contrasts in muscle and myofibers of elite male and female bodybuilders. *J Appl Physiol, 67*(1), 24-31.

Ansell, B. J., Navab, M., Hama, S., Kamranpour, N., Fonarow, G., Hough, G., et al. (2003). Inflammatory/Antiinflammatory Properties of High-Density Lipoprotein Distinguish Patients From Control Subjects Better Than High-Density Lipoprotein Cholesterol Levels and Are Favorably Affected by Simvastatin Treatment. *Circulation, 108*(22), 2751-2756.

Appel, L. J., Brands, M. W., Daniels, S. R., Karanja, N., Elmer, P. J., & Sacks, F. M. (2006). Dietary Approaches to Prevent and Treat Hypertension. *Hypertension, 47*(2), 296-308.

Appel, L. J., Champagne, C. M., Harsha, D. W., Cooper, L. S., Obarzanek, E., Elmer, P. J., et al. (2003). Effects of comprehensive lifestyle modification on blood pressure control: main results of the PREMIER clinical trial. *JAMA, 289*(16), 2083-2093.

Appel, L. J., Moore, T. J., Obarzanek, E., Vollmer, W. M., Svetkey, L. P., Sacks, F. M., et al. (1997). A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. *N Engl J Med, 336*(16), 1117-1124.

Aravanis, C., Corcondilas, A., Dontas, A. S., Lekos, D., & Keys, A. (1970). Coronary heart disease in seven countries. IX. The Greek islands of Crete and Corfu. *Circulation, 41*(4 Suppl), I88-100.

Armitage, C. J., & Conner, M. (2001). Efficacy of the Theory of Planned Behaviour: A meta-analytic review. *Br J Soc Psychol, 40*(4), 471-499.

Arterburn, D., Johnson, E. S., Butler, M. G., Fisher, D., & Bayliss, E. A. (2014). Predicting 90-day mortality after bariatric surgery: an independent, external validation of the OS-MRS prognostic risk score. *Surg Obes Relat Dis*.

Ascunce, R., Berger, J., Weintraub, H., & Schwartzbard, A. (2012). The Role of Statin Therapy for Primary Prevention: What is the Evidence? *Curr Atheroscler Rep, 14*(2), 167-174.

Aucott, L., Poobalan, A., Smith, W. C. S., Avenell, A., Jung, R., & Broom, J. (2005). Effects of Weight Loss in Overweight/Obese Individuals and Long-Term Hypertension Outcomes: A Systematic Review. *Hypertension, 45*(6), 1035-1041.

Balkau, B., & Charles, M. A. (1999). Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). *Diabet Med, 16*(5), 442-443.

Bandura, A. (2001). Social Cognitive Theory: An Agentic Perspective. *Annu Rev Psychol, 52*(1), 1-26.

Barnard, N. D., Cohen, J., Jenkins, D. J., Turner-McGrievy, G., Gloede, L., Green, A., et al. (2009). A low-fat vegan diet and a conventional diabetes diet in the treatment of type 2 diabetes: a randomized, controlled, 74-wk clinical trial. *Am J Clin Nutr, 89*(5), 1588S-1596S.

Barnard, N. D., Joshua, C., David, J. A. J., Gabrielle, T.-M., & et al. (2006). A Low-Fat Vegan Diet Improves Glycemic Control and Cardiovascular Risk Factors in a Randomized Clinical Trial in Individuals With Type 2 Diabetes. *Diabetes Care, 29*(8), 1777.

Barnard, R. J. (1990). Short-term reductions in serum lipids through diet and exercise. *N Engl J Med, 323*(16), 1142-1143.

Barnard, R. J. (1991). Effects of life-style modification on serum lipids. *Arch Intern Med, 151*(7), 1389-1394.

Barnard, R. J. (2007). Prostate cancer prevention by nutritional means to alleviate metabolic syndrome. *Am J Clin Nutr, 86*(3), 889S-893.

Barnard, R. J., Aronson, W. J., Tymchuk, C. N., & Ngo, T. H. (2002). Prostate cancer: another aspect of the insulin-resistance syndrome? *Obes Rev, 3*(4), 303-308.

Barnard, R. J., Guzy, P. M., Rosenberg, J. M., & O'Brien, L. T. (1983). Effects of an intensive exercise and nutrition program on patients with coronary artery disease: five-year follow-up. *J Cardiac Rehab, 3*, 183-190.

Barnard, R. J., Jung, T., & Inkeles, S. B. (1994). Diet and exercise in the treatment of NIDDM. The need for early emphasis. *Diabetes Care, 17*(12), 1469-1472.

Barnard, R. J., Kobayashi, N., & Aronson, W. J. (2008). Effect of diet and exercise intervention on the growth of prostate epithelial cells. *Prostate Cancer And Prostatic Diseases, 11*(4), 362-366.

Barnard, R. J., Lattimore, L., Holly, R. G., Cherny, S., & Pritikin, N. (1982). Response of non-insulin-dependent diabetic patients to an intensive program of diet and exercise. *Diabetes Care, 5*(4), 370-374.

Barnard, R. J., Massey, M. R., Cherny, S., O'Brien, L. T., & Pritikin, N. (1983). Long-term use of a high-complex-carbohydrate, high-fiber, low-fat diet and exercise in the treatment of NIDDM patients. *Diabetes Care, 6*(3), 268-273.

Barnard, R. J., & Wen, S. J. (1994). Exercise and diet in the prevention and control of the metabolic syndrome. *Sports Med, 18*(4), 218-228.
Bays, H., & Dujovne, C. A. (2006). Adiposopathy is a more rational treatment target for metabolic disease than obesity alone. *Curr Atheroscler Rep, 8*(2), 144-156.
Bays, H. E. (2009). Lorcaserin and adiposopathy: 5-HT2c agonism as a treatment for 'sick fat' and metabolic disease. *Expert Rev Cardiovasc Ther, 7*(11), 1429-1445.
Bays, H. E., González-Campoy, J. M., Bray, G. A., Kitabchi, A. E., Bergman, D. A., Schorr, A. B., et al. (2008). Pathogenic potential of adipose tissue and metabolic consequences of adipocyte hypertrophy and increased visceral adiposity. *Expert Rev Cardiovasc Ther, 6*(3), 343-368.
Beard, C. M., Barnard, R. J., Robbins, D. C., Ordovas, J. M., & Schaefer, E. J. (1996). Effects of diet and exercise on qualitative and quantitative measures of LDL and its susceptibility to oxidation. *Arterioscler Thromb Vasc Biol, 16*(2), 201-207.
Beck-Nielsen, H. (1999). General characteristics of the insulin resistance syndrome: prevalence and heritability. European Group for the study of Insulin Resistance (EGIR). *Drugs, 58 Suppl 1*, 7-10; discussion 75-82.
Beeson, W. L., Mills, P. K., Phillips, R. L., Andress, M., & Fraser, G. E. (1989). Chronic disease among Seventh-day Adventists, a low-risk group. Rationale, methodology, and description of the population. *Cancer, 64*(3), 570-581.
Bloomer, R. J., Kabir, M. M., Canale, R. E., Trepanowski, J. F., Marshall, K. E., Farney, T. M., et al. (2010). Effect of a 21 day Daniel Fast on metabolic and cardiovascular disease risk factors in men and women. *Lipids Health Dis, 9*, 94.
Bloomer, R. J., Kabir, M. M., Trepanowski, J. F., Canale, R. E., & Farney, T. M. (2011). A 21 day Daniel Fast improves selected biomarkers of antioxidant status and oxidative stress in men and women. *Nutr Metab (Lond), 8*, 17.
Bloomgarden, Z. T. (2009). The 6th Annual World Congress on the Insulin Resistance Syndrome. *Diabetes Care, 32*(11), e127-e133.
Boudreau, D. M., Malone, D. C., Raebel, M. A., Fishman, P. A., Nichols, G. A., Feldstein, A. C., et al. (2009). Health care utilization and costs by metabolic syndrome risk factors. *Metab Syndr Relat Disord, 7*(4), 305-314.
Braun, L. T. (2012). People with hyperlipidaemia who followed a dietary portfolio of cholesterol-lowering foods achieved a greater reduction in LDL cholesterol over 6 months than those who received advice to follow a low-saturated fat diet. *Evid Based Nurs, 15*(2), 57-58.
Briel, M., Ferreira-Gonzalez, I., You, J. J., Karanicolas, P. J., Akl, E. A., Wu, P., et al. (2009). Association between change in high density lipoprotein cholesterol and cardiovascular disease morbidity and mortality: systematic review and meta-regression analysis. *BMJ, 338*, b92.
Brinton, E. A., Eisenberg, S., & Breslow, J. L. (1990). A low-fat diet decreases high density lipoprotein (HDL) cholesterol levels by decreasing HDL apolipoprotein transport rates. *J Clin Invest, 85*(1), 144-151.
Bruce, K. D., & Hanson, M. A. (2010). The Developmental Origins, Mechanisms, and Implications of Metabolic Syndrome. *J Nutr, 140*(3), 648-652.
Bull, M., & Lockhart, K. (2007). *Seeking Sanctuary Seventh-day Adventistism and the American Dream*. Bloomington, IN: Indiana University Press.
Butler, T. L., Fraser, G. E., Beeson, W. L., Knutsen, S. F., Herring, R. P., Chan, J., et al. (2008). Cohort profile: The Adventist Health Study-2 (AHS-2). *Int J Epidemiol, 37*(2), 260-265.

Buzina, R., Keys, A., Mohacek, I., Marinkovic, M., Hahn, A., & Blackburn, H. (1970). Coronary heart disease in seven countries. V. Five-year follow-up in Dalmatia and Slavonia. *Circulation, 41*(4 Suppl), I40-51.

Caballero, B. (2007). The Global Epidemic of Obesity: An Overview. *Epidemiol Rev, 29*(1), 1-5.

Caldwell, J. C. (2001). Population health in transition. *Bull World Health Organ, 79*, 159-160.

Campbell, T. C., & Campbell, T. M. (2006). *The China Study: Startling Implications for Diet, Weight Loss and Long-Term Health*. Dallas: Benbella Books.

Campbell, T. C., Parpia, B., & Chen, J. (1998). Diet, lifestyle, and the etiology of coronary artery disease: the Cornell China study. *Am J Cardiol, 82*(10B), 18T-21T.

Cannistra, L. B., Balady, G. J., O'Malley, C. J., Weiner, D. A., & Ryan, T. J. (1992). Comparison of the clinical profile and outcome of women and men in cardiac rehabilitation. *Am J Cardiol, 69*(16), 1274-1279.

Carmichael, H. E., Swinblirn, B. A., & Wilson, M. R. (1998). Lower Fat Intake as a Predictor of Initial and Sustained Weight Loss in Obese Subjects Consuming an Otherwise ad Libitum Diet. *J Am Diet Assoc, 98*(1), 35-39.

Caroline, R. (2006). Obituaries: Thomas Royle Dawber. *BMJ, 332*(7533), 122-122.

Castelli, W. P., Garrison, R. J., Wilson, P. W., Abbott, R. D., Kalousdian, S., & Kannel, W. B. (1986). Incidence of coronary heart disease and lipoprotein cholesterol levels. The Framingham Study. *JAMA, 256*(20), 2835-2838.

Chew, G. T., Gan, S. K., & Watts, G. F. (2006). Revisiting the metabolic syndrome. *Med J Aust, 185*(8), 445-449.

Chiuve, S. E., McCullough, M. L., Sacks, F. M., & Rimm, E. B. (2006). Healthy lifestyle factors in the primary prevention of coronary heart disease among men: benefits among users and nonusers of lipid-lowering and antihypertensive medications. *Circulation, 114*(2), 160-167.

Clarke, P. M., & Fitzgerald, E. M. (2010). Expiry of patent protection on statins: effects on pharmaceutical expenditure in Australia. *Med J Aust, 192*(11), 633-636.

Clevidence, B. A., Judd, J. T., Schatzkin, A., Muesing, R. A., Campbell, W. S., Brown, C. C., et al. (1992). Plasma lipid and lipoprotein concentrations of men consuming a low-fat, high-fiber diet. *Am J Clin Nutr, 55*(3), 689-694.

Coakes, S. J., & Steed, M. G. (2010). *SPSS Analysis Without Anguish: version 17 for Windows*. Sydney: Australia:: John Wiley & Sons.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*: Lawrence Erlbaum.

Connor, W. E., Cerqueira, M. T., Connor, R. W., Wallace, R. B., Malinow, M. R., & Casdorph, H. R. (1978). The plasma lipids, lipoproteins, and diet of the Tarahumara Indians of Mexico. *Am J Clin Nutr, 31*(7), 1131-1142.

Cornier, M.-A., Dabelea, D., Hernandez, T. L., Lindstrom, R. C., Steig, A. J., Stob, N. R., et al. (2008). The Metabolic Syndrome. *Endocr Rev, 29*(7), 777-822.

Cutler, G. J., Nettleton, J. A., Ross, J. A., Harnack, L. J., Jacobs, D. R., Jr., Scrafford, C. G., et al. (2008). Dietary flavonoid intake and risk of cancer in postmenopausal women: the Iowa Women's Health Study. *Int J Cancer, 123*(3), 664-671.

Dalton, M., Cameron, A. J., Zimmet, P. Z., Shaw, J. E., Jolley, D., Dunstan, D. W., et al. (2003). Waist circumference, waist–hip ratio and body mass index and their correlation with cardiovascular disease risk factors in Australian adults. *J Intern Med, 254*(6), 555-563.

Dansinger, M. L., Gleason, J. A., Griffith, J. L., Selker, H. P., & Schaefer, E. J. (2005). Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA, 293*(1), 43-53.

Daskalopoulou, S. S., Athyros, V. G., Kolovou, G. D., Anagnostopoulou, K. K., & Mikhailidis, D. P. (2006). Definitions of metabolic syndrome: Where are we now? *Curr Vasc Pharmacol, 4*(3), 185-197.

Daubenmier, J., Weidner, G., Sumner, M., Mendell, N., Merritt-Worden, T., Studley, J., et al. (2007). The contribution of changes in diet, exercise, and stress management to changes in coronary risk in women and men in the multisite cardiac lifestyle intervention program. *Ann Behav Med, 33*(1), 57-68.

Davis-Smith, Y. M., Boltri, J. M., Seale, J. P., Shellenberger, S., Blalock, T., & Tobin, B. (2007). Implementing a diabetes prevention program in a rural African-American church. *J Natl Med Assoc, 99*(4), 440-446.

de la Monte, S. M., & Wands, J. R. (2008). Alzheimer's disease is type 3 diabetes-evidence reviewed. *J Diabetes Sci Technol, 2*(6), 1101-1113.

de Lorgeril, M., Renaud, S., Mamelle, N., Salen, P., Martin, J. L., Monjaud, I., et al. (1994). Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease. *Lancet, 343*(8911), 1454-1459.

de Lorgeril, M., Salen, P., Martin, J. L., Monjaud, I., Delaye, J., & Mamelle, N. (1999). Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation, 99*(6), 779-785.

Den Hond, E., Celis, H., Vandenhoven, G., O'Brien, E., & Staessen, J. A. (2003). Determinants of white-coat syndrome assessed by ambulatory blood pressure or self-measured home blood pressure. *Blood Press Monit, 8*(1), 37-40.

Despres, J. P. (2013). HDL cholesterol studies--more of the same? *Nat Rev Cardiol, 10*(2), 70-72.

Despres, J. P., & Poirier, P. (2013). Diabetes: Looking back at Look AHEAD[mdash]giving lifestyle a chance. *Nat Rev Cardiol, 10*(4), 184-186.

Dewell, A., Weidner, G., Sumner, M. D., Barnard, R. J., Marlin, R. O., Daubenmier, J. J., et al. (2007). Relationship of dietary protein and soy isoflavones to serum IGF-1 and IGF binding proteins in the Prostate Cancer Lifestyle Trial. *Nutr Cancer, 58*(1), 35-42.

Diehl, H. A. (1998). Coronary Risk Reduction Through Intensive Community-based Lifestyle Intervention: The Chip Experience. *Am J Cardiol, 82*, 83T-87T.

Diehl, H. A. (2003). *Reversing Disease with Fork and Knife*. Loma Linda: Lifestyle Medicine Institute.

Diehl, H. A., & Ludington, A. (2005). *Dynamic Living: How to Take Charge of Your Health*. Hagerstown: Review and Herald Publishing Association.

Dobbelsteyn, C. J., Joffres, M. R., MacLean, D. R., & Flowerdew, G. (2001). A comparative evaluation of waist circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk factors. The Canadian Heart Health Surveys. *Int J Obes Relat Metab Disord, 25*(5), 652-661.

Dod, H., Bhardwaj, R., Sajja, V., Weidner, G., Hobbs, G., Konat, G., et al. (2010). Effect of intensive lifestyle changes on endothelial function and on inflammatory markers of atherosclerosis. *Am J Cardiol, 105*(3), 362-367.

Douketis, J. D., Macie, C., Thabane, L., & Williamson, D. F. (2005). Systematic review of long-term weight loss studies in obese adults: clinical significance and applicability to clinical practice. *Int J Obes, 29*(10), 1153-1167.

Dunn-Emke, S., Weidner, G., Pettengill, E., Marlin, R., Chi, C., & Ornish, D. (2005). Nutrient adequacy of a very low-fat vegan diet. *J Am Diet Assoc, 105*(9), 1442-1446.

Eckel, R., Alberti, K. G., Grundy, S., & Zimmet, P. (2010). The metabolic syndrome. *Lancet, 375*(9710), 181.

Eckel, R. H., Grundy, S. M., & Zimmet, P. Z. (2005). The metabolic syndrome. *Lancet, 365*(9468), 1415-1428.

Egger, G. (2007). The Australian Experience. In A. White & M. Pettifer (Eds.), *Hazardous Waist: Tackling Male Weight Problems* (pp. 205). Oxford: Radcliffe Publishing.

Egger, G. (2010). Reply to Frank et al.,'The Light Look at a Heavy Problem'cartoon series letter. *Obes Rev, 11*(5), 400-400.

Egger, G., Binns, A., & Rossner, S. (2011). *Lifestyle Medicine: Managing Diseases of Lifestyle on the 21st Century* (2nd ed.). North Ryde: MacGraw-Hill Australia.

Egger, G., & Dobson, A. (2000). Clinical measures of obesity and weight loss in men. *Int J Obes Relat Metab Disord, 24*(3), 354-357.

Egger, G., & Swinburn, B. (1997). An "ecological" approach to the obesity pandemic. *BMJ, 315*(7106), 477-480.

Egger, G. J., Binns, A. F., & Rossner, S. R. (2009). The emergence of "lifestyle medicine" as a structured approach for management of chronic disease. *Med J Aust, 190*(3), 143-145.

Elfhag, K., & Rössner, S. (2005). Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *Obes Rev, 6*(1), 67-85.

Ellsworth, D. L., O'Dowd, S. C., Salami, B., Hochberg, A., Vernalis, M. N., Marshall, D., et al. (2004). Intensive lifestyle modification: impact on cardiovascular disease risk factors in subjects with and without clinical cardiovascular disease. *Prev Cardiol, 7*(4), 168-175.

Elmer, P. J., Obarzanek, E., Vollmer, W. M., Simons-Morton, D., Stevens, V. J., Young, D. R., et al. (2006). Effects of comprehensive lifestyle modification on diet, weight, physical fitness, and blood pressure control: 18-month results of a randomized trial. *Ann Intern Med, 144*(7), 485-495.

Englert, H. S., Dieh, H. A., Greenlaw, R. L., & Aldana, S. (2012). The Effects of Lifestyle Modification on Glycemic Levels and Medication Intake: The Rockford CHIP. In O. Capelli (Ed.), *Primary Care at a Glance - Hot Topics and New Insights*. Online: InTech.

Englert, H. S., Diehl, H. A., & Greenlaw, R. L. (2004). Rationale and design of the Rockford CHIP, a community-based coronary risk reduction program: results of pilot phase. *Preventative Medicine, 38*, 432-441.

Englert, H. S., Diehl, H. A., Greenlaw, R. L., Willich, S. N., & Aldana, S. (2007). The effect of a community-based coronary risk reduction: the Rockford CHIP. *Prev Med, 44*(6), 513-519.

ESLM. (2014). *European Society of Lifestyle Medicine Webpage.* Retrieved 29 April, 2014, from http://eu-lifestylemedicine.org/

Esselstyn, C. B. (1999). Updating a 12-year experience with arrest and reversal therapy for coronary heart disease (an overdue requiem for palliative cardiology). *Am J Cardiol, 84*(3), 339-341, A338.

Esselstyn, C. B. (2001). Resolving the Coronary Artery Disease Epidemic Through Plant-based Nutrition. *Prev Cardiol*(Fall).

Esselstyn, C. B. (2008). *Prevent and Reverse Heart Disease*. New York: Avery.

Esselstyn, C. B. (2010). Is the present therapy for coronary artery disease the radical mastectomy of the twenty-first century? *Am J Cardiol, 106*(6), 902-904.
Esselstyn, C. B., Ellis, S. G., Medendorp, S. V., & Crowe, T. D. (1995). A strategy to arrest and reverse coronary artery disease: a 5-year longitudinal study of a single physician's practice. *J Fam Pract, 41*(6), 560-568.
Etkin, C. D., Prohaska, T. R., Harris, B. A., Latham, N., & Jette, A. (2006). Feasibility of implementing the Strong for Life program in community settings. *Gerontologist, 46*(2), 284-292.
Ferdowsian, H. R., & Barnard, N. D. (2009). Effects of plant-based diets on plasma lipids. *Am J Cardiol, 104*(7), 947-956.
Ferenczi, E. A., Asaria, P., Hughes, A. D., Chaturvedi, N., & Francis, D. P. (2010). Can a statin neutralize the cardiovascular risk of unhealthy dietary choices? *Am J Cardiol, 106*(4), 587-592.
Fidanza, F., Puddu, V., Imbimbo, A. B., Menotti, A., & Keys, A. (1970). Coronary heart disease in seven countries. VII. Five-year experience in rural Italy. *Circulation, 41*(4 Suppl), I63-75.
Field, A. E., Willett, W. C., Lissner, L., & Colditz, G. A. (2007). Dietary Fat and Weight Gain Among Women in the Nurses' Health Study. *Obesity, 15*(4), 967-976.
Ford, E. M. M., Li, C. M. P., & Sattar, N. M. P. (2008). Metabolic Syndrome and Incident Diabetes: Current state of the evidence. *Diabetes Care, 31*(9), 1898.
Ford, E. S., Giles, W. H., & Dietz, W. H. (2002). Prevalence of the metabolic syndrome among us adults: Findings from the third national health and nutrition examination survey. *JAMA, 287*(3), 356-359.
Ford, E. S., Giles, W. H., & Mokdad, A. H. (2004). Increasing prevalence of the metabolic syndrome among U.S. Adults. *Diabetes Care, 27*(10), 2444-2449.
Forouhi, N. G., Sharp, S. J., Du, H., van der A, D. L., Halkjær, J., Schulze, M. B., et al. (2009). Dietary fat intake and subsequent weight change in adults: results from the European Prospective Investigation into Cancer and Nutrition cohorts. *Am J Clin Nutr, 90*(6), 1632-1641.
Frank, A., & Kahan, S. (2010). Re: 'The Light Look at a Heavy Problem' cartoon series. *Obes Rev, 11*(5), 399-399.
Franklin, S. S., Larson, M. G., Khan, S. A., Wong, N. D., Leip, E. P., Kannel, W. B., et al. (2001). Does the Relation of Blood Pressure to Coronary Heart Disease Risk Change With Aging? : The Framingham Heart Study. *Circulation, 103*(9), 1245-1249.
Fraser, G. E. (1988). Determinants of ischemic heart disease in Seventh-day Adventists: a review. *Am J Clin Nutr, 48*(3 Suppl), 833-836.
Fraser, G. E. (1994). Diet and coronary heart disease: beyond dietary fats and low-density-lipoprotein cholesterol. *Am J Clin Nutr, 59*(5 Suppl), 1117S-1123S.
Fraser, G. E. (2005). A comparison of first event coronary heart disease rates in two contrasting California populations. *J Nutr Health Aging, 9*(1), 53-58.
Fraser, G. E., Lindsted, K. D., & Beeson, W. L. (1995). Effect of risk factor values on lifetime risk of and age at first coronary event. The Adventist Health Study. *Am J Epidemiol, 142*(7), 746-758.
Fraser, G. E., & Shavlik, D. J. (2001). Ten years of life: Is it a matter of choice? *Arch Intern Med, 161*(13), 1645-1652.
Fraser, G. E., & Swannell, R. J. (1981). Diet and serum cholesterol in Seventh-day Adventists: a cross-sectional study showing significant relationships. *J Chronic Dis, 34*(9-10), 487-501.

Freemantle, N., Holmes, J., Hockey, A., & Kumar, S. (2008). How strong is the association between abdominal obesity and the incidence of type 2 diabetes? *Int J Clin Pract, 62*(9), 1391-1396.

Freiberg, M. S., Pencina, M. J., D'Agostino, R. B., Lanier, K., Wilson, P. W., & Vasan, R. S. (2008). BMI vs. waist circumference for identifying vascular risk. *Obesity, 16*(2), 463-469.

Friedewald, V. E., Grundy, S., Gotto, A. M., Jr., Haffner, S., Denke, M. A., Hollander, P., et al. (2007). The Editor's Roundtable: the metabolic syndrome. *Am J Cardiol, 99*(3), 382-389.

Friedewald, W. T., Levy, R. I., & Fredrickson, D. S. (1972). Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma, Without Use of the Preparative Ultracentrifuge. *Clin Chem, 18*(6), 499-502.

Gaesser, G. A. (2007). Carbohydrate quantity and quality in relation to body mass index. *J Am Diet Assoc, 107*(10), 1768-1780.

Gale, A. E. (1998). *Chronic fatigue syndrome associated with insulin resistance.* Retrieved 28/7/2013, 2013, from http://www.agale.com.au/CFS.htm

Gallo, L. C., Troxel, W. M., Kuller, L. H., Sutton-Tyrrell, K., Edmundowicz, D., & Matthews, K. A. (2003). Marital status, marital quality, and atherosclerotic burden in postmenopausal women. *Psychosom Med, 65*(6), 952-962.

Gardner, G., Halweil, B., & Peterson, J. A. (2000). *Underfed and overfed : the global epidemic of malnutrition*. Washington: Worldwatch Institute.

Garg, A., Bantle, J. P., Henry, R. R., Coulston, A. M., Griver, K. A., Raatz, S. K., et al. (1994). Effects of varying carbohydrate content of diet in patients with non-insulin-dependent diabetes mellitus. *JAMA, 271*(18), 1421-1428.

Gidley, V. (2008). *Chip Leaders Training Manual*. Mapleton, Queensland: Lifestyle Medicine Institute (Australia).

Giem, P., Beeson, W. L., & Fraser, G. E. (1993). The incidence of dementia and intake of animal products: preliminary findings from the Adventist Health Study. *Neuroepidemiology, 12*(1), 28-36.

Glanz, K., Rimer, B. K., & Viswanath, K. (2008). *Health behavior and health education: theory, research, and practice*. San Francisco, CA: Jossey-Bass.

Goldstein, L. B., Bushnell, C. D., Adams, R. J., Appel, L. J., Braun, L. T., Chaturvedi, S., et al. (2011). Guidelines for the Primary Prevention of Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke, 42*(2), 517-584.

Goodpaster, B. H., DeLany, J. P., Otto, A. D., Kuller, L., Vockley, J., South-Paul, J., et al. (2010). Effects of diet and physical activity interventions on weight loss and cardiometabolic risk factors in severely obese adults: A randomized trial. *JAMA, 304*(16), 1795-1802.

Gordon, D. J., & Rifkind, B. M. (1989). High-density lipoprotein--the clinical implications of recent studies. *N Engl J Med, 321*(19), 1311-1316.

Gould, A. L., Davies, G. M., Alemao, E., Yin, D. D., & Cook, J. R. (2007). Cholesterol reduction yields clinical benefits: meta-analysis including recent trials. *Clin Ther, 29*(5), 778-794.

Gould, K. L., Ornish, D., Scherwitz, L., Brown, S., Edens, R. P., Hess, M. J., et al. (1995). Changes in myocardial perfusion abnormalities by positron emission tomography after long-term, intense risk factor modification. *JAMA, 274*(11), 894-901.

Gratton, L., & Ghoshal, S. (2003). Managing Personal Human Capital:: New Ethos for the 'Volunteer' Employee. *European Management Journal, 21*(1), 1-10.

Gregg, E. W., Chen, H., Wagenknecht, L. E., Clark, J. M., Delahanty, L. M., Bantle, J., et al. (2012). Association of an intensive lifestyle intervention with remission of type 2 diabetes. *JAMA, 308*(23), 2489-2496.

Gregoire, F. M. (2001). Adipocyte differentiation: from fibroblast to endocrine cell. *Exp Biol Med, 226*(11), 997-1002.

Gregoire, F. M., Smas, C. M., & Sul, H. S. (1998). Understanding adipocyte differentiation. *Physiol Rev, 78*(3), 783-809.

Grossman, J. B., & Furano, K. (1999). Making the most of volunteers. *Law and Contemp. Probs., 62*, 199.

Grundy, S. M. (2006a). Drug therapy of the metabolic syndrome: minimizing the emerging crisis in polypharmacy. *Nat Rev Drug Discov, 5*(4), 295-309.

Grundy, S. M. (2006b). Metabolic syndrome: connecting and reconciling cardiovascular and diabetes worlds. *J Am Coll Cardiol, 47*(6), 1093-1100.

Grundy, S. M., Brewer, H. B., Jr., Cleeman, J. I., Smith, S. C., Jr., & Lenfant, C. (2004). Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation, 109*(3), 433-438.

Grundy, S. M., Cleeman, J. I., Daniels, S. R., Donato, K. A., Eckel, R. H., Franklin, B. A., et al. (2005). Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation, 112*(17), 2735-2752.

Heitmann, B. L., Lissner, L., Sorensen, T. I., & Bengtsson, C. (1995). Dietary fat intake and weight gain in women genetically predisposed for obesity. *Am J Clin Nutr, 61*(6), 1213-1217.

Herman, W. H., Hoerger, T. J., Brandle, M., Hicks, K., Sorensen, S., Zhang, P., et al. (2005). The cost-effectiveness of lifestyle modification or metformin in preventing type 2 diabetes in adults with impaired glucose tolerance. *Ann Intern Med, 142*(5), 323-332.

Heuch, I., Jacobsen, B. K., & Fraser, G. E. (2005). A cohort study found that earlier and longer Seventh-day Adventist church membership was associated with reduced male mortality. *J Clin Epidemiol, 58*(1), 83-91.

Hill, J. O., & Peters, J. C. (1998). Environmental Contributions to the Obesity Epidemic. *Science, 280*(5368), 1371-1374.

Hoerger, T. J., & Ahmann, A. J. (2008). The impact of diabetes and associated cardiometabolic risk factors on members: strategies for optimizing outcomes. *J Manag Care Pharm, 14*(1 Suppl C), S2-14; quiz 15-16.

Hollis, J. F., Gullion, C. M., Stevens, V. J., Brantley, P. J., Appel, L. J., Ard, J. D., et al. (2008). Weight loss during the intensive intervention phase of the weight-loss maintenance trial. *Am J Prev Med, 35*(2), 118-126.

Horton, E. S. (2009). Effects of Lifestyle Changes to Reduce Risks of Diabetes and Associated Cardiovascular Risks: Results from Large Scale Efficacy Trials. *Obesity, 17*(n3s), S43-S48.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science, 241*(4865), 540-545.

Howden, L. M., & Meyer, J. A. (2010). Age and sex composition: 2010. *2010 Census Briefs, US Department of Commerce, Economics and Statistics Administration. US CENSUS BUREAU*.

Hu, F. B., Manson, J. E., Stampfer, M. J., Colditz, G., Liu, S., Solomon, C. G., et al. (2001). Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. *N Engl J Med, 345*(11), 790-797.

Hu, F. B., Stampfer, M. J., Manson, J. E., Grodstein, F., Colditz, G. A., Speizer, F. E., et al. (2000). Trends in the incidence of coronary heart disease and changes in diet and lifestyle in women. *N Engl J Med, 343*(8), 530-537.

Hu, F. B., Stampfer, M. J., Solomon, C., Liu, S., Colditz, G. A., Speizer, F. E., et al. (2001). Physical activity and risk for cardiovascular events in diabetic women. *Ann Intern Med, 134*(2), 96-105.

Hu, G., Qiao, Q., & Tuomilehto, J. (2004). Prevalence of the metabolic syndrome and its relation to all-cause and cardiovascular mortality in nondiabetic european men and women. *Arch Intern Med, 164*(10), 1066-1076.

Hubbard, J. D., Inkeles, S., & Barnard, R. J. (1985). Nathan Pritikin's heart. *N Engl J Med, 313*(1), 52-52.

Hunt, S. M., McKenna, S. P., McEwen, J., Backett, E. M., Williams, J., & Papp, E. (1980). A quantitative approach to perceived health status: a validation study. *J Epidemiol Community Health, 34*(4), 281-286.

Huxley, R., Lewington, S., & Clarke, R. (2002). Cholesterol, coronary heart disease and stroke: a review of published evidence from observational studies and randomized controlled trials. *Semin Vasc Med, 2*(3), 315-323.

Inabnet, W. B., III, Winegar, D. A., Sherif, B., & Sarr, M. G. Early Outcomes of Bariatric Surgery in Patients with Metabolic Syndrome: An Analysis of the Bariatric Outcomes Longitudinal Database. *J Am Coll Surg, 214*(4), 550-556.

Jackson, L. (2009). Translating the Diabetes Prevention Program into practice: a review of community interventions. *Diabetes Educ, 35*(2), 309-320.

Jacobs, D. R., Jr., Meyer, K. A., Kushi, L. H., & Folsom, A. R. (1999). Is whole grain intake associated with reduced total and cause-specific death rates in older women? The Iowa Women's Health Study. *Am J Public Health, 89*(3), 322-329.

Jacobs, D. R., Meyer, K. A., Kushi, L. H., & Folsom, A. R. (1998). Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study. *Am J Clin Nutr, 68*(2), 248-257.

Jacobs, D. R., Pereira, M. A., Meyer, K. A., & Kushi, L. H. (2000). Fiber from whole grains, but not refined grains, is inversely associated with all-cause mortality in older women: the Iowa women's health study. *J Am Coll Nutr, 19*(3 Suppl), 326S-330S.

Janssen, I., Katzmarzyk, P. T., & Ross, R. (2002). Body mass index, waist circumference, and health risk: Evidence in support of current national institutes of health guidelines. *Arch Intern Med, 162*(18), 2074-2079.

Jenkins, D. J., Jones, P. J., Lamarche, B., Kendall, C. W., Faulkner, D., Cermakova, L., et al. (2011). Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. *JAMA, 306*(8), 831-839.

Jenkins, D. J., Kendall, C. W., Marchie, A., Faulkner, D. A., Josse, A. R., Wong, J. M., et al. (2005). Direct comparison of dietary portfolio vs statin on C-reactive protein. *Eur J Clin Nutr, 59*(7), 851-860.

Jenkins, D. J., Kendall, C. W., Marchie, A., Faulkner, D. A., Wong, J. M., de Souza, R., et al. (2003). Effects of a dietary portfolio of cholesterol-lowering foods vs lovastatin on serum lipids and C-reactive protein. *JAMA, 290*(4), 502-510.

Jenkins, D. J., Kendall, C. W., Marchie, A., Faulkner, D. A., Wong, J. M., de Souza, R., et al. (2005). Direct comparison of a dietary portfolio of cholesterol-lowering foods with a statin in hypercholesterolemic participants. *Am J Clin Nutr, 81*(2), 380-387.

Jenkins, D. J., Kendall, C. W., Marchie, A., Jenkins, A. L., Augustin, L. S., Ludwig, D. S., et al. (2003). Type 2 diabetes and the vegetarian diet. *Am J Clin Nutr, 78*(3), 610S-616S.

Jensen, M. K., Rimm, E. B., Furtado, J. D., & Sacks, F. M. (2012). Apolipoprotein C-III as a Potential Modulator of the Association Between HDL-Cholesterol and Incident Coronary Heart Disease. *J Am Heart Assoc, 1*(2).

Jia, H., & Lubetkin, E. I. (2010). Trends in quality-adjusted life-years lost contributed by smoking and obesity. *Am J Prev Med, 38*(2), 138-144.

Kahn, R., Buse, J., Ferrannini, E., & Stern, M. (2005). The metabolic syndrome: time for a critical appraisal: joint statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetes Care, 28*(9), 2289-2304.

Karwalajtys, T., McDonough, B., Hall, H., Guirguis-Younger, M., Chambers, L., Kaczorowski, J., et al. (2009). Development of the Volunteer Peer Educator Role in a Community Cardiovascular Health Awareness Program (CHAP): A Process Evaluation in Two Communities. *J Community Health, 34*(4), 336-345.

Kelley, G. A., Kelley, K. S., Roberts, S., & Haskell, W. (2012). Combined effects of aerobic exercise and diet on lipids and lipoproteins in overweight and obese adults: a meta-analysis. *J Obes, 2012*, 985902.

Kent, L., Morton, D., Rankin, P., Ward, E., Grant, R., Gobble, J., et al. (2013). The effect of a low-fat, plant-based lifestyle intervention (CHIP) on serum HDL levels and the implications for metabolic syndrome status - a cohort study. *Nutr Metab, 10*(1), 58.

Kent, L., Morton, D. P., Hurlow, T., Rankin, P., & Hanna, A. (2013). Long-term effectiveness of the community-based Complete Health Improvement Program (CHIP) lifestyle intervention: a cohort study. *BMJ Open, in press*.

Kent, L. M., & Worsley, A. (2009). Trends in BMI, diet and lifestyle between 1976 and 2005 in North Sydney. *Asia Pac J Clin Nutr, 18*(3), 453-461.

Key, T. J., Fraser, G. E., Thorogood, M., Appleby, P. N., Beral, V., Reeves, G., et al. (1999). Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. *Am J Clin Nutr, 70*(3 Suppl), 516S-524S.

Keys, A. (1970). Coronary heart disease in seven countries. *Circulation, 41*(1), 186-195.

Keys, A. (1995). Mediterranean diet and public health: personal reflections. *Am J Clin Nutr, 61*(6 Suppl), 1321S-1323S.

Keys, A., Anderson, J. T., & Grande, F. (1959). Serum cholesterol in man: diet fat and intrinsic responsiveness. *Circulation, 19*(2), 201-214.

Keys, A., & Parlin, R. W. (1966). Serum cholesterol response to changes in dietary lipids. *Am J Clin Nutr, 19*(3), 175-181.

Keys, A., Taylor, H. L., Blackburn, H., Brozek, J., Anderson, J. T., & Simonson, E. (1963). Coronary Heart Disease among Minnesota Business and Professional Men Followed Fifteen Years. *Circulation, 28*(3), 381-395.

Khera, A. V., Cuchel, M., de la Llera-Moya, M., Rodrigues, A., Burke, M. F., Jafri, K., et al. (2011). Cholesterol efflux capacity, high-density lipoprotein function, and atherosclerosis. *N Engl J Med, 364*(2), 127-135.

Kiecolt-Glaser, J. K., & Newton, T. L. (2001). Marriage and health: his and hers. *Psychol Bull, 127*(4), 472-503.

Kimura, N., & Keys, A. (1970). Coronary heart disease in seven countries. X. Rural southern Japan. *Circulation, 41*(4 Suppl), I101-112.
Klein, N. A., Sondag, K. A., & Drolet, J. C. (1994). Understanding volunteer peer health educators' motivations: Applying social learning theory. *J Am Coll Health, 43*(3), 126-130.
Knowler, W. C., Barrett-Connor, E., Fowler, S. E., Hamman, R. F., Lachin, J. M., Walker, E. A., et al. (2002). Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med, 346*(6), 393-403.
Knowler, W. C., Fowler, S. E., Hamman, R. F., Christophi, C. A., Hoffman, H. J., Brenneman, A. T., et al. (2009). 10-year follow-up of diabetes incidence and weight loss in the Diabetes Prevention Program Outcomes Study. *Lancet, 374*(9702), 1677-1686.
Koertge, J., Weidner, G., Elliott-Eller, M., Scherwitz, L., Merritt-Worden, T., Marlin, R., et al. (2003). Improvement in medical risk factors and quality of life in women and men with coronary artery disease in the Multicenter Lifestyle Demonstration Project. *Am J Cardiol, 91*(11), 1316-1322.
Kong, B. W. (1997). Community-based hypertension control programs that work. *J Health Care Poor Underserved, 8*(4), 409-415.
Korp, P. (2010). Problems of the Healthy Lifestyle Discourse. *Sociology Compass, 4*(9), 800-810.
Kotseva, K., Wood, D., De Backer, G., De Bacquer, D., Pyorala, K., Keil, U., et al. (2009). Cardiovascular prevention guidelines in daily practice: a comparison of EUROASPIRE I, II, and III surveys in eight European countries. *Lancet, 373*(9667), 929-940.
Kushi, L. H., Lew, R. A., Stare, F. J., Ellison, C. R., el Lozy, M., Bourke, G., et al. (1985). Diet and 20-Year Mortality from Coronary Heart Disease. *N Engl J Med, 312*(13), 811-818.
Kwok, C. S., Pradhan, A., Khan, M. A., Anderson, S. G., Keavney, B. D., Myint, P. K., et al. (2014). Bariatric surgery and its impact on cardiovascular disease and mortality: A systematic review and meta-analysis. *Int J Cardiol, 173*(1), 20-28.
Laaksonen, D. E., Atalay, M., Niskanen, L. K., Mustonen, J., Sen, C. K., Lakka, T. A., et al. (2000). Aerobic exercise and the lipid profile in type 1 diabetic men: a randomized controlled trial. *Med Sci Sports Exerc, 32*(9), 1541-1548.
Larsen, L. T. (2012). The Leap of Faith from Disease Treatment to Lifestyle Prevention: The Genealogy of a Policy Idea. *J Health Polit Policy Law, 37*(2), 227-252.
Larsson, S. C., & Wolk, A. (2012). Red and processed meat consumption and risk of pancreatic cancer: meta-analysis of prospective studies. *Br J Cancer, 106*(3), 603-607.
Lavie, C. J., Milani, R. V., Artham, S. M., Patel, D. A., & Ventura, H. O. (2009). The obesity paradox, weight loss, and coronary disease. *Am J Med, 122*(12), 1106-1114.
Le Fanu, J. (2000). *The Rise and Fall of Modern Medicine*. New York: Carroll & Graf Publishers, Inc.
Leite, J. O., & Fernandez, M. L. (2010). Should we take high-density lipoprotein cholesterol levels at face value? *Am J Cardiovasc Drugs, 10*(1), 1-3.
Levesque, J., & Lamarche, B. (2008). The metabolic syndrome: definitions, prevalence and management. *J Nutrigenet Nutrigenomics, 1*(3), 100-108.
Liberopoulos, E. N., Tsouli, S., Mikhailidis, D. P., & Elisaf, M. S. (2006). Preventing type 2 diabetes in high risk patients: an overview of lifestyle and pharmacological measures. *Curr Drug Targets, 7*(2), 211-228.

Lichtenstein, A. H., Appel, L. J., Brands, M., Carnethon, M., Daniels, S., Franch, H. A., et al. (2006). Diet and Lifestyle Recommendations Revision 2006. *Circulation, 114*(1), 82-96.
Lichtenstein, A. H., & Van Horn, L. (1998). Very Low Fat Diets. *Circulation, 98*(9), 935-939.
Lieb, W., Jansen, H., Loley, C., Pencina, M. J., Nelson, C. P., Newton-Cheh, C., et al. (2013). Genetic Predisposition to Higher Blood Pressure Increases Coronary Artery Disease Risk. *Hypertension, 61*(5), 995-1001.
Lissner, L., & Heitmann, B. L. (1995). The dietary fat: carbohydrate ratio in relation to body weight. *Curr Opin Lipidol, 6*(1), 8-13.
Lopez, A. D., & Mathers, C. D. (2006). Measuring the global burden of disease and epidemiological transitions: 2002-2030. *Ann Trop Med Parasitol, 100*(5-6), 481-499.
Lucock, M. (2004). Is folic acid the ultimate functional food component for disease prevention? *Br Med J, 328*(7433), 211+.
Luengo-Fernández, R., Leal, J., Gray, A., Petersen, S., & Rayner, M. (2006). Cost of cardiovascular diseases in the United Kingdom. *Heart, 92*(10), 1384-1389.
Lumley, T., Diehr, P., Emerson, S., & Chen, L. (2002). The importance of the normality assumption in large public health data sets. *Annu Rev Public Health, 23*, 151-169.
Macini, M., & Stamler, J. (2004). Diet for preventing cardiovascular diseases: Light from Ancel Keys, Distinguished Centenarian Scientist. *Nutr Metab Cardiovasc Dis, 14*(1), 52-57.
Macintyre, S., Hunt, K., & Sweeting, H. (1996). Gender differences in health: Are things really as simple as they seem? *Soc Sci Med, 42*(4), 617-624.
Mackenbach, J. P. (1994). The epidemiologic transition theory. *J Epidemiol Community Health, 48*(4), 329-331.
Magee, M. J., Henry, M. B., & Narayan, K. M. V. (2011). Commentary: Co-occurrence of tuberculosis and diabetes: new paradigm of epidemiological transition. *Int J Epidemiol, 40*(2), 428-431.
Martin, S. S., Metkus, T. S., Horne, A., Blaha, M. J., Hasan, R., Campbell, C. Y., et al. (2012). Waiting for the National Cholesterol Education Program Adult Treatment Panel IV Guidelines, and in the meantime, some challenges and recommendations. *Am J Cardiol, 110*(2), 307-313.
Mason, C., Craig, C. L., & Katzmarzyk, P. T. (2008). Influence of central and extremity circumferences on all-cause mortality in men and women. *Obesity, 16*(12), 2690-2695.
Mathur, N., & Pedersen, B. K. (2008). Exercise as a mean to control low-grade systemic inflammation. *Mediators Inflamm, 2008*, 109502.
McClendon, D. A., Dunbar, S. B., Clark, P. C., & Coverson, D. L. (2010). An analysis of popular weight loss diet types in relation to metabolic syndrome therapeutic guidelines. *Medsurg Nurs, 19*(1), 17-24.
McDougall, J., Litzau, K., Haver, E., Saunders, V., & Spiller, G. A. (1995). Rapid reduction of serum cholesterol and blood pressure by a twelve-day, very low fat, strictly vegetarian diet. *J Am Coll Nutr, 14*(5), 491-496.
McMurry, M. P., Cerqueira, M. T., Connor, S. L., & Connor, W. E. (1991). Changes in lipid and lipoprotein levels and body weight in Tarahumara Indians after consumption of an affluent diet. *N Engl J Med, 325*(24), 1704-1708.

McNaughton, S. A., Marks, G. C., & Green, A. C. (2005). Role of Dietary Factors in the Development of Basal Cell Cancer and Squamous Cell Cancer of the Skin. *Cancer Epidemiol Biomarkers Prev, 14*(7), 1596-1607.
Mekary, R. A., Feskanich, D., Hu, F. B., Willett, W. C., & Field, A. E. (2010). Physical Activity in Relation to Long-term Weight Maintenance After Intentional Weight Loss in Premenopausal Women. *Obesity, 18*(1), 167-174.
Merrill, R. M., & Aldana, S. G. (2008). Cardiovascular risk reduction and factors influencing loss to follow-up in the coronary health improvement project. *Med Sci Monit, 14*(4), PH17-25.
Merrill, R. M., & Aldana, S. G. (2009). Improving overall health status through the CHIP intervention. *Am J Health Behav, 33*(2), 135-146.
Merrill, R. M., Aldana, S. G., Greenlaw, R. L., & Diehl, H. A. (2008). The Coronary Health Improvemint Projects Impact on Lowering Eating, Sleep, and Depressive Disorders. *American Journal of Health Education, 39*(6), 337-344.
Merrill, R. M., Aldana, S. G., Greenlaw, R. L., Diehl, H. A., & Salberg, A. (2007). The effects of an intensive lifestyle modification program on sleep and stress disorders. *J Nutr Health Aging, 11*(3), 242-248.
Merrill, R. M., Aldana, S. G., Greenlaw, R. L., Diehl, H. A., Salberg, A., & Englert, H. (2008). Can newly acquired healthy behaviors persist? An analysis of health behavior decay. *Prev Chronic Dis, 5*(1), A13.
Merrill, R. M., Massey, M. T., Aldana, S. G., Greenlaw, R. L., Diehl, H. A., & Salberg, A. (2008). C-reactive protein levels according to physical activity and body weight for participants in the coronary health improvement project. *Prev Med, 46*(5), 425-430.
Merrill, R. M., Taylor, P., & Aldana, S. G. (2008). Coronary Health Improvement Project (CHIP) is associated with improved nutrient intake and decreased depression. *Nutrition, 24*(4), 314-321.
Milburn, K. (1995). A critical review of peer education with young people with special reference to sexual health. *Health Educ Res, 10*(4), 407-420.
Mills, P. K., Beeson, W. L., Phillips, R. L., & Fraser, G. E. (1989). Cohort study of diet, lifestyle, and prostate cancer in Adventist men. *Cancer, 64*(3), 598-604.
Mokdad, A. H., Marks, J. S., Stroup, D. F., & Gerberding, J. L. (2004). Actual causes of death in the United States, 2000. *JAMA, 291*(10), 1238-1245.
Monkhouse, S. J., Morgan, J. D., Bates, S. E., & Norton, S. A. (2009). An overview of the management of morbid obesity. *Postgrad Med J, 85*(1010), 678-681.
Morrison, L. M. (1960). DIet in coronary atherosclerosis. *JAMA, 173*(8), 884-888.
Morrow-Howell, N., Hong, S.-I., & Tang, F. (2009). Who Benefits From Volunteering? Variations in Perceived Benefits. *The Gerontologist, 49*(1), 91-102.
Morton, D. P., Rankin, P., Morey, P., Kent, L., Hurlow, T., Chang, E., et al. (2013). The effectiveness of the Complete Health Improvement Program (CHIP) in Australasia for reducing selected chronic disease risk factors: a feasibility study. *N Z Med J, 126*(1370), 43-54.
Mosca, L., McGillen, C., & Rubenfire, M. (1998). Gender differences in barriers to lifestyle change for cardiovascular disease prevention. *J Womens Health, 7*(6), 711-715.
Moynihan, C. (1998). Theories in health care and research: theories of masculinity. *BMJ, 317*(7165), 1072-1075.
Nabel, E. G., & Braunwald, E. (2012). A tale of coronary artery disease and myocardial infarction. *N Engl J Med, 366*(1), 54-63.

National Cholesterol Education Program. (2002). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation, 106*(25), 3143-3421.

National Institute of Health. (1998). Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. *Obes Res, 6 Suppl 2*, 51S-209S.

Navab, M., Anantharamaiah, G. M., Reddy, S. T., Van Lenten, B. J., Ansell, B. J., & Fogelman, A. M. (2006). Mechanisms of Disease: proatherogenic HDL-an evolving field. *Nat Clin Pract End Met, 2*(9), 504-511.

Navab, M., Van Lenten, B. J., Reddy, S. T., & Fogelman, A. M. (2001). High-Density Lipoprotein and the Dynamics of Atherosclerotic Lesions. *Circulation, 104*(20), 2386-2387.

Nestle, M. (1999). Animal v. plant foods in human diets and health: is the historical record unequivocal? *Proc Nutr Soc, 58*(2), 211-218.

Ng, T. W. K., Watts, G. F., Barrett, P. H. R., Rye, K.-A., & Chan, D. C. (2007). Effect of Weight Loss on LDL and HDL Kinetics in the Metabolic Syndrome. *Diabetes Care, 30*(11), 2945-2950.

Nicholls, S. J., Lundman, P., Harmer, J. A., Cutri, B., Griffiths, K. A., Rye, K. A., et al. (2006). Consumption of saturated fat impairs the anti-inflammatory properties of high-density lipoproteins and endothelial function. *J Am Coll Cardiol, 48*(4), 715-720.

Nutbeam, D. (2000). Health literacy as a public health goal: a challenge for contemporary health education and communication strategies into the 21st century. *Health Promotion International, 15*(3), 259-267.

Ogden, C. l., Carroll, M. D., Kit, B. K., & Flegal, K. M. (2012). Prevalence of obesity and trends in body mass index among us children and adolescents, 1999-2010. *JAMA, 307*(5), 483-490.

Oh, K., Hu, F. B., Manson, J. E., Stampfer, M. J., & Willett, W. C. (2005). Dietary Fat Intake and Risk of Coronary Heart Disease in Women: 20 Years of Follow-up of the Nurses' Health Study. *Am J Epidemiol, 161*(7), 672-679.

Olshansky, S. J., & Ault, A. B. (1986). The fourth stage of the epidemiologic transition: the age of delayed degenerative diseases. *Milbank Mem Fund Q*, 355-391.

Omran, A. R. (1971). The epidemiologic transition. A theory of the epidemiology of population change. *Milbank Mem Fund Q, 49*(4), 509-538.

Orchard, T. J., Temprosa, M., Goldberg, R., Haffner, S., Ratner, R., Marcovina, S., et al. (2005). The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: the Diabetes Prevention Program randomized trial. *Ann Intern Med, 142*(8), 611-619.

Ordovas, J. M. (2005). Diet-heart hypothesis: will diversity bring reconciliation? *Am J Clin Nutr, 82*(5), 919-920.

Ornish, D. (1998a). Avoiding revascularization with lifestyle changes: The Multicenter Lifestyle Demonstration Project. *Am J Cardiol, 82*(10B), 72T-76T.

Ornish, D. (1998b). Serum lipids after a low-fat diet. *JAMA, 279*(17), 1345-1346.

Ornish, D. (2002). Dean Ornish, MD: a conversation with the editor. Interview by William Clifford Roberts, MD. *Am J Cardiol, 90*(3), 271-298.

Ornish, D. (2004). Low-carbohydrate diets. *Ann Intern Med, 141*(9), 738; author reply 738-739.

Ornish, D. (2009a). Intensive lifestyle changes and health reform. *Lancet Oncol, 10*(7), 638-639.
Ornish, D. (2009b). Mostly plants. *Am J Cardiol, 104*(7), 957-958.
Ornish, D., & Brown, S. (1993). Treatment of and screening for hyperlipidemia. *N Engl J Med, 329*(15), 1124-1125; author reply 1127-1128.
Ornish, D., Brown, S. E., Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., et al. (1990a). Can lifestyle changes reverse coronary heart disease? The Lifestyle Heart Trial. *Lancet, 336*(8708), 129-133.
Ornish, D., Brown, S. E., Scherwitz, L. W., Billings, J. H., Armstrong, W. T., Ports, T. A., et al. (1990b). Lifestyle changes and heart disease. *Lancet, 336*(8717), 741-742.
Ornish, D., & Denke, M. (1994). Dietary treatment of hyperlipidemia. *J Cardiovasc Risk, 1*(4), 283-286.
Ornish, D., Lin, J., Daubenmier, J., Weidner, G., Epel, E., Kemp, C., et al. (2008). Increased telomerase activity and comprehensive lifestyle changes: a pilot study. *Lancet Oncol, 9*(11), 1048-1057.
Ornish, D., Magbanua, M. J., Weidner, G., Weinberg, V., Kemp, C., Green, C., et al. (2008). Changes in prostate gene expression in men undergoing an intensive nutrition and lifestyle intervention. *Proc Natl Acad Sci U S A, 105*(24), 8369-8374.
Ornish, D., Scherwitz, L., Doody, R., Kesten, D., McLanahan, S., Brown, S., et al. (1983). Effects of stress management training and dietary changes in treating ischemic heart disease. *JAMA, 249*(1), 54-59.
Ornish, D., Scherwitz, L. W., Billings, J. H., Brown, S. E., Gould, K. L., Merritt, T. A., et al. (1998). Intensive lifestyle changes for reversal of coronary heart disease. *JAMA, 280*(23), 2001-2007.
Ornish, D., Weidner, G., Fair, W., Marlin, R., Pettengill, E., Raisin, C., et al. (2005). Intensive lifestyle changes may affect the progression of prostate cancer. *J Urol, 174*(3), 1065-1069; discussion 1069-1070.
Owens, P., Atkins, N., & O'Brien, E. (1999). Diagnosis of White Coat Hypertension by Ambulatory Blood Pressure Monitoring. *Hypertension, 34*(2), 267-272.
Pan, A., Keum, N., Okereke, O. I., Sun, Q., Kivimaki, M., Rubin, R. R., et al. (2012). Bidirectional Association Between Depression and Metabolic Syndrome. *Diabetes Care, 35*(5), 1171-1180.
Pan, A., Sun, Q., & Bernstein, A. M. (2012). Red meat consumption and mortality: Results from 2 prospective cohort studies. *Arch Intern Med, 172*(7), 555-563.
Parkin, S., & McKeganey, N. (2000). The rise and rise of peer education approaches. *Drugs: Education, Prevention, and Policy, 7*(3), 293-310.
Pasanisi, F., Contaldo, F., de Simone, G., & Mancini, M. (2001). Benefits of sustained moderate weight loss in obesity. *Nutr Metab Cardiovasc Dis, 11*(6), 401-406.
Pereira, M., Kottke, T., Jordan, C., O'Connor, P., Pronk, N., & Carreón, R. (2009). Preventing and Managing Cardiometabolic Risk: The Logic for Intervention. *Int J Environ Res Public Health, 6*(10), 2568.
Perk, J., De Backer, G., Gohlke, H., Graham, I., Reiner, Z., Verschuren, M., et al. (2012). European Guidelines on cardiovascular disease prevention in clinical practice (version 2012): The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts) * Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). *Eur Heart J, 33*(13), 1635-1701.

Perloff, D., Grim, C., Flack, J., Frohlich, E. D., Hill, M., McDonald, M., et al. (1993). Human blood pressure determination by sphygmomanometry. *Circulation, 88*(5), 2460-2470.
Pischke, C. R., Frenda, S., Ornish, D., & Weidner, G. (2010). Lifestyle changes are related to reductions in depression in persons with elevated coronary risk factors. *Psychol Health, 25*(9), 1077-1100.
Pischke, C. R., Weidner, G., Elliott-Eller, M., & Ornish, D. (2007). Lifestyle changes and clinical profile in coronary heart disease patients with an ejection fraction of <or=40% or >40% in the Multicenter Lifestyle Demonstration Project. *Eur J Heart Fail, 9*(9), 928-934.
Pischke, C. R., Weidner, G., Elliott-Eller, M., Scherwitz, L., Merritt-Worden, T. A., Marlin, R., et al. (2006). Comparison of coronary risk factors and quality of life in coronary artery disease patients with versus without diabetes mellitus. *Am J Cardiol, 97*(9), 1267-1273.
Pladevall, M., Singal, B., Williams, L. K., Brotons, C., Guyer, H., Sadurni, J., et al. (2006). A Single Factor Underlies the Metabolic Syndrome. *Diabetes Care, 29*(1), 113-122.
Plater, S., & Egger, G. (2011). How hard? *Obes Rev, 12*(4), 303-303.
Pollan, M. (2007). *The omnivore's dilemma: the search for a perfect meal in a fast-food world*: Bloomsbury Publishing.
Potenza, M. V., & Mechanick, J. I. (2009). The metabolic syndrome: definition, global impact, and pathophysiology. *Nutr Clin Pract, 24*(5), 560-577.
Potter, J. D. (2005). Cancer Prevention: The Gap between What We Know and What We Do. *Health Research and Policy*, 11.
Pritikin, N. (1984). The pritikin diet. *JAMA, 251*(9), 1160-1161.
Prochaska, J. O., Norcross, J. C., & DiClemente, C. C. (2013). Applying the stages of change. *Psychotherapy in Australia, 19*(2), 11.
Puska, P. (2008). The North Karelia Project. *Diabetes Voice*, 26.
Puska, P., Nissinen, A., Tuomilehto, J., Salonen, J. T., Koskela, K., McAlister, A., et al. (1985). The Community-Based Strategy to Prevent Coronary Heart Disease: Conclusions from the Ten Years of the North Karelia Project. *Annu Rev Public Health, 6*(1), 147-193.
Puska, P., Salonen, J. T., Nissinen, A., Tuomilehto, J., Vartiainen, E., Korhonen, H., et al. (1983). Change in risk factors for coronary heart disease during 10 years of a community intervention programme (North Karelia project). *Br Med J (Clin Res Ed), 287*(6408), 1840-1844.
Rader, D. J. (2006). Molecular regulation of HDL metabolism and function: implications for novel therapies. *J Clin Invest, 116*(12), 3090-3100.
Rankin, P., Morton, D. P., Diehl, H., Gobble, J., Morey, P., & Chang, E. (2012). Effectiveness of a volunteer-delivered lifestyle modification program for reducing cardiovascular disease risk factors. *Am J Cardiol, 109*(1), 82-86.
Rapaport, P., & Orbell, S. (2000). Augmenting the theory of planned behaviour: Motivation to provide practical assistance and emotional support to parents. *Psychology & Health, 15*(3), 309-324.
Ratner, R., Goldberg, R., Haffner, S., Marcovina, S., Orchard, T., Fowler, S., et al. (2005). Impact of intensive lifestyle and metformin therapy on cardiovascular disease risk factors in the diabetes prevention program. *Diabetes Care, 28*(4), 888-894.
Read, J. G., & Gorman, B. K. (2006). Gender inequalities in US adult health: the interplay of race and ethnicity. *Soc Sci Med, 62*(5), 1045-1065.

Reaven, G. M. (1988). Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes, 37*(12), 1595-1607.
Reddy, B. S., Engle, A., Simi, B., O'Brien, L. T., Barnard, R. J., Pritikin, N., et al. (1988). Effect of low-fat, high-carbohydrate, high-fiber diet on fecal bile acids and neutral sterols. *Prev Med, 17*(4), 432-439.
Regitz-Zagrosek, V., Lehmkuhl, E., & Mahmoodzadeh, S. (2007). Gender aspects of the role of the metabolic syndrome as a risk factor for cardiovascular disease. *Gend Med, 4 Suppl B*, S162-177.
Regitz-Zagrosek, V., Lehmkuhl, E., & Weickert, M. (2006). Gender differences in the metabolic syndrome and their role for cardiovascular disease. *Clin Res Cardiol, 95*(3), 136-147.
Rizzo, N. S., Sabate, J., Jaceldo-Siegl, K., & Fraser, G. E. (2011). Vegetarian dietary patterns are associated with a lower risk of metabolic syndrome: the adventist health study 2. *Diabetes Care, 34*(5), 1225-1227.
Roberts, C. K., & Barnard, R. J. (2005). Effects of exercise and diet on chronic disease. *J Appl Physiol, 98*(1), 3-30.
Roberts, C. K., Ng, C., Hama, S., Eliseo, A. J., & Barnard, R. J. (2006). Effect of a short-term diet and exercise intervention on inflammatory/anti-inflammatory properties of HDL in overweight/obese men with cardiovascular risk factors. *J Appl Physiol, 101*(6), 1727-1732.
Roberts, C. K., Vaziri, N. D., & Barnard, R. J. (2002). Effect of Diet and Exercise Intervention on Blood Pressure, Insulin, Oxidative Stress, and Nitric Oxide Availability. *Circulation, 106*(20), 2530-2532.
Roche, A. F. (1981). The adipocyte-number hypothesis. *Child Dev, 52*(1), 31-43.
Roelfs, D. J., Shor, E., Kalish, R., & Yogev, T. (2011). The Rising Relative Risk of Mortality for Singles: Meta-Analysis and Meta-Regression. *Am J Epidemiol, 174*(4), 379-389.
Roger, V. L., Go, A. S., Lloyd-Jones, D. M., Benjamin, E. J., Berry, J. D., Borden, W. B., et al. (2012). Heart disease and stroke statistics--2012 update: a report from the American Heart Association. *Circulation, 125*(1), e2-e220.
Rosén, M. (1989). On randomized controlled trials and lifestyle interventions. *Int J Epidemiol, 18*(4), 993-994.
Rosenstock, I. M. (1974). The Health Belief Model and Preventive Health Behavior. *Health Educ Behav, 2*(4), 354-386.
Rosenthal, M. B., Barnard, R. J., Rose, D. P., Inkeles, S., Hall, J., & Pritikin, N. (1985). Effects of a high-complex-carbohydrate, low-fat, low-cholesterol diet on levels of serum lipids and estradiol. *Am J Med, 78*(1), 23-27.
Sacks, F. M., & Katan, M. (2002). Randomized clinical trials on the effects of dietary fat and carbohydrate on plasma lipoproteins and cardiovascular disease. *Am J Med, 113 Suppl 9B*, 13S-24S.
Salehi, N., Hanifi, Z., Khaleghparast, S., Ghadrdoost, B., & Zamari Nobari Shabnam, V. Z. A. (2011). Prevalence of obesity and diabetes in coronary artery disease. *Iranian Heart Journal*.
Salonen, J. T., Puska, P., & Mustaniemi, H. (1979). Changes in morbidity and mortality during comprehensive community programme to control cardiovascular diseases during 1972-7 in North Karelia. *BMJ, 2*(6199), 1178-1183.
Sattar, N., Preiss, D., Murray, H. M., Welsh, P., Buckley, B. M., de Craen, A. J., et al. (2010). Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials. *Lancet, 375*(9716), 735-742.

Schauer, P. R., Bhatt, D. L., Kirwan, J. P., Wolski, K., Brethauer, S. A., Navaneethan, S. D., et al. (2014). Bariatric Surgery versus Intensive Medical Therapy for Diabetes — 3-Year Outcomes. *N Engl J Med, In Press*.

Schneider, E. C., Altpeter, M., & Whitelaw, N. (2007). An innovative approach for building health promotion program capacity: a generic volunteer training curriculum. *Gerontologist, 47*(3), 398-403.

Shurney, D., Hyde, S., & Hulsey, K. (2012). CHIP Lifestyle Program at Vanderbilt University Demonstrates an Early ROI for Diabetic Cohort in Workplace Setting: A Case Study. *Journal of Managed Care Medicine, 15*(4), 5-15.

Sigal, R. J., Kenny, G. P., Boule, N. G., Wells, G. A., Prud'homme, D., Fortier, M., et al. (2007). Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes: a randomized trial. *Ann Intern Med, 147*(6), 357-369.

Silberman, A., Banthia, R., Estay, I., Kemp, C., Studley, J., Hareras, D., et al. (2010). The effectiveness and efficacy of an intensive cardiac rehabilitation program in 24 sites. *Am J Health Promot, 24*(4), 260-266.

Sjöström, L., Narbro, K., Sjöström, C. D., Karason, K., Larsson, B., Wedel, H., et al. (2007). Effects of Bariatric Surgery on Mortality in Swedish Obese Subjects. *N Engl J Med, 357*(8), 741-752.

Skordalakes, E. (2008). Telomerase and the benefits of healthy living. *Lancet Oncol, 9*(11), 1023-1024.

Slavicek, J., Kittnar, O., Fraser, G. E., Medova, E., Konecna, J., Zizka, R., et al. (2008). Lifestyle decreases risk factors for cardiovascular diseases. *Cent Eur J Public Health, 16*(4), 161-164.

Smith, G. D., & Ebrahim, S. (1998). Community based heart health promotion project in England. Authors conclusions are unjustified and misleading. *BMJ, 316*(7132), 705.

Smith, R. (2004). "Let food be thy medicine…". *BMJ, 328*(7433).

St-Onge, M. P., Janssen, I., & Heymsfield, S. B. (2004). Metabolic syndrome in normal-weight Americans: new definition of the metabolically obese, normal-weight individual. *Diabetes Care, 27*(9), 2222-2228.

Stampfer, M. J., Hu, F. B., Manson, J. E., Rimm, E. B., & Willett, W. C. (2000). Primary prevention of coronary heart disease in women through diet and lifestyle. *N Engl J Med, 343*(1), 16.

Steckler, A., & Goodman, R. M. (1989). How to Institutionalize Health Promotion Programs. *Am J Health Promot, 3*(4), 34-43.

Sturm, R. (2002). The Effects Of Obesity, Smoking, And Drinking On Medical Problems And Costs. *Health Aff (Millwood), 21*(2), 245-253.

Sullivan, S., & Samuel, S. (2006). Effect of Short-Term Pritikin Diet Therapy on the Metabolic Syndrome. *The Journal of Cardiometabolic Syndrome, 1*(5), 308-312.

Swinburn, B., & Egger, G. (2002). Preventive strategies against weight gain and obesity. *Obes Rev, 3*(4), 289-301.

Tang, J., Yan, H., & Zhuaig, S. (2012). Inflammation and Oxidative Stress in Obesity-Related Glomerulopathy. *In J Nephrology, 2012*, 11.

Tang, J. L., Smith, G. D., Armitage, J. M., Lancaster, T., Silagy, C. A., Fowler, G. H., et al. (1998). Systematic review of dietary intervention trials to lower blood total cholesterol in free-living subjectsCommentary: Dietary change, cholesterol reduction, and the public health—what does meta-analysis add? *BMJ, 316*(7139), 1213-1220.

Tarasuk, V. S., & Brooker, A.-S. (1997). Interpreting Epidemiologic Studies of Diet-Disease Relationships. *The Journal of Nutrition, 127*(9), 1847-1852.
Taubes, G. (1998). As Obesity Rates Rise, Experts Struggle to Explain Why. *Science, 280*(5368), 1367-1368.
Taylor, A. E., Ebrahim, S., Ben-Shlomo, Y., Martin, R. M., Whincup, P. H., Yarnell, J. W., et al. (2010). Comparison of the associations of body mass index and measures of central adiposity and fat mass with coronary heart disease, diabetes, and all-cause mortality: a study using data from 4 UK cohorts. *Am J Clin Nutr, 91*(3), 547-556.
Thieszen, C. L., Merrill, R. M., Aldana, S. G., Diehl, H. A., Mahoney, M. L., Greenlaw, R. L., et al. (2011). The Coronary Health Improvement Project (CHIP) for lowering weight and improving psychosocial health. *Psychol Rep, 109*(1), 338-352.
Tjonna, A. E., Lee, S. J., Rognmo, O., Stolen, T. O., Bye, A., Haram, P. M., et al. (2008). Aerobic Interval Training Versus Continuous Moderate Exercise as a Treatment for the Metabolic Syndrome: A Pilot Study. *Circulation, 118*(4), 346-354.
Tompkins, K. W. (2009). Sylvester Graham's Imperial Dietetics. *Gastronomica, 9*(1), 50-60.
Tonstad, S., Butler, T., Yan, R., & Fraser, G. E. (2009). Type of vegetarian diet, body weight, and prevalence of type 2 diabetes. *Diabetes Care, 32*(5), 791-796.
Tonstad, S., Stewart, K., Oda, K., Batech, M., Herring, R. P., & Fraser, G. E. (2011). Vegetarian diets and incidence of diabetes in the Adventist Health Study-2. *Nutr Metab Cardiovasc Dis*.
Totsikas, C., Rohm, J., Kantartzis, K., Thamer, C., Rittig, K., Machann, J., et al. (2011). Cardiorespiratory fitness determines the reduction in blood pressure and insulin resistance during lifestyle intervention. *J Hypertens, 29*(6), 1220-1227.
Trepanowski, J. F., Kabir, M. M., Alleman, R. J., Jr., & Bloomer, R. J. (2012). A 21-day Daniel fast with or without krill oil supplementation improves anthropometric parameters and the cardiometabolic profile in men and women. *Nutr Metab (Lond), 9*(1), 82.
Trowell, H. (1977). Food and Dietary Fibre. *Nutr Rev, 35*(3), 6-11.
Troxel, W., Matthews, K. A., Gallo, L. C., & Kuller, L. H. (2005). Marital quality and occurrence of the metabolic syndrome in women. *Arch Intern Med, 165*(9), 1022-1027.
Tsai, A. G., Williamson, D. F., & Glick, H. A. (2011). Direct medical cost of overweight and obesity in the USA: a quantitative systematic review. *Obes Rev, 12*(1), 50-61.
Tuomilehto, J., Lindstrom, J., Eriksson, J. G., Valle, T. T., Hamalainen, H., Ilanne-Parikka, P., et al. (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med, 344*(18), 1343-1350.
Tuttle, K. R., Shuler, L. A., Packard, D. P., Milton, J. E., Daratha, K. B., Bibus, D. M., et al. (2008). Comparison of Low-Fat Versus Mediterranean-Style Dietary Intervention After First Myocardial Infarction (from The Heart Institute of Spokane Diet Intervention and Evaluation Trial). *Am J Cardiol, 101*(11), 1523-1530.
Umberson, D. (1992). Gender, marital status and the social control of health behavior. *Soc Sci Med, 34*(8), 907-917.
Unick, J. L., Beavers, D., Jakicic, J. M., Kitabchi, A. E., Knowler, W. C., Wadden, T. A., et al. (2011). Effectiveness of lifestyle interventions for individuals with severe obesity and type 2 diabetes: results from the Look AHEAD trial. *Diabetes Care, 34*(10), 2152-2157.

van Baak, M. A., van Mil, E., Astrup, A. V., Finer, N., Van Gaal, L. F., Hilsted, J., et al. (2003). Leisure-time activity is an important determinant of long-term weight maintenance after weight loss in the Sibutramine Trial on Obesity Reduction and Maintenance (STORM trial). *The American Journal of Clinical Nutrition, 78*(2), 209-214.

VanItallie, T. B. (2005). Ancel Keys: a tribute. *Nutr Metab, 2*(1), 4.

Verheijden, M. W., & Kok, F. J. (2005). Public health impact of community-based nutrition and lifestyle interventions. *Eur J Clin Nutr, 59*(S1), S66.

Vogels, N., Egger, G., Plasqui, G., & Westerterp, K. R. (2004). Estimating changes in daily physical activity levels over time: implication for health interventions from a novel approach. *Int J Sports Med, 25*(8), 607-610.

von Eckardstein, A., Nofer, J.-R., & Assmann, G. (2001). High Density Lipoproteins and Arteriosclerosis: Role of Cholesterol Efflux and Reverse Cholesterol Transport. *Arterioscler Thromb Vasc Biol, 21*(1), 13-27.

Walls, H., Peeters, A., Proietto, J., & McNeil, J. (2011). Public health campaigns and obesity - a critique. *BMC Public Health, 11*(1), 136.

Weber, F., Barnard, R. J., & Roy, D. (1983). Effects of a high-complex-carbohydrate, low-fat diet and daily exercise on individuals 70 years of age and older. *J Gerontol, 38*(2), 155-161.

Weimar. (2012). *The History of NEWSTART.* Retrieved July 23, 2012, from http://www.newstart.com/about/the-history-of-weimar/

Weinberg, S. L. (2004). The diet–heart hypothesis: a critique. *J Am Coll Cardiol, 43*(5), 731-733.

White, E. G. (1905). *The Ministry of Healing*. Mountain View, CA: Pacific Press Publishing Association.

WHO. (2000). Obesity: preventing and managing the global epidemic. Report of a WHO Consultation. (Vol. Part 1). Geneva.

Wikipedia. (2012). *John Harvey Kellog --- Wikipedia, The Free Encyclopedia.* Retrieved July 23, 2012, from http://en.wikipedia.org/w/index.php?title=John_Harvey_Kellogg&oldid=498656279

Wild, S., Roglic, G., Green, A., Sicree, R., & King, H. (2004). Global Prevalence of Diabetes. *Diabetes Care, 27*(5), 1047-1053.

Wilson, P. W. F., D'Agostino, R. B., Levy, D., Belanger, A. M., Silbershatz, H., & Kannel, W. B. (1998). Prediction of Coronary Heart Disease Using Risk Factor Categories. *Circulation, 97*(18), 1837-1847.

Wing, R. R., Lang, W., Wadden, T. A., Safford, M., Knowler, W. C., Bertoni, A. G., et al. (2011). Benefits of Modest Weight Loss in Improving Cardiovascular Risk Factors in Overweight and Obese Individuals With Type 2 Diabetes. *Diabetes Care, 34*(7), 1481-1486.

Wing, R. R., & Look, A. R. G. (2010). Long-term effects of a lifestyle intervention on weight and cardiovascular risk factors in individuals with type 2 diabetes mellitus: four-year results of the Look AHEAD trial. *Arch Intern Med, 170*(17), 1566-1575.

Wing, R. R., Rosen, R. C., Fava, J. L., Bahnson, J., Brancati, F., Gendrano Iii, I. N., et al. (2010). Effects of weight loss intervention on erectile function in older men with type 2 diabetes in the Look AHEAD trial. *J Sex Med, 7*(1 Pt 1), 156-165.

Withnell, A. (2003). The natural cure of coronary heart disease. *Nutr Health, 17*(1), 55-60.

World Health Organisation. (1999). Definition, diagnosis and classification of diabetes mellitus and its complications: report of WHO Consultation. Part 1: diagnosis and

classification of diabetes mellitus. Geneva, Switzerland: World Health Organisation.
Wright, C. M. (2011). Biographical notes on Ancel Keys and Salim Yusuf: Origins and significance of the Seven Countries Study and the INTERHEART Study. *J Clin Lipidol, 5*(6), 434-440.
Yamaoka, K., & Tango, T. (2012). Effects of lifestyle modification on metabolic syndrome: a systematic review and meta-analysis. *BMC Med, 10*(1), 138.
Yancy, W. S., Westman, E. C., French, P. A., & Califf, R. M. (2003). Diets and Clinical Coronary Events: The Truth Is Out There. *Circulation, 107*(1), 10-16.
Yanovski, S. Z., & Yanovski, J. A. (2011). Obesity Prevalence in the United States — Up, Down, or Sideways? *N Engl J Med, 364*(11), 987-989.
Yuasa, M., de Sa, R. F., Pincovsky, S., & Shimanouchi, N. (2007). Emergence Model of social and human capital and its application to the Healthy Municipalities project in Northeast Brazil. *Health Promot Int, 22*(4), 292-298.
Yusuf, S., Hawken, S., Ounpuu, S., Dans, T., Avezum, A., Lanas, F., et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet, 364*(9438), 937-952.
Zhang, P., Zhang, X., Brown, J., Vistisen, D., Sicree, R., Shaw, J., et al. (2010). Global healthcare expenditure on diabetes for 2010 and 2030. *Diabetes Res Clin Pract, 87*(3), 293-301.
Zulet, M. A., Berkenpas, M. E., & Martinez, J. A. (2005). Comparison of Dietary Approaches to Treat Obesity Based on the Different Carbohydrate/Fat Content: Impact on Weight Loss and Lipid Profile. *Cur Nut Food Sci, 1*(1), 13-21.