12-2014

The Impact of a Lifestyle Education Program (CHIP) on Cardiovascular Disease Risk Factors: An Australasian Study

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Avondale College of Higher Education
School of Education

THE IMPACT OF A LIFESTYLE EDUCATION PROGRAM (CHIP)
ON CARDIOVASCULAR DISEASE RISK FACTORS:
AN AUSTRALASIAN STUDY

A Masters Thesis

Presented in fulfilment
of the Requirements for the Award
of the Degree of

Master of Education (Research)

by

Cheryl Anne Carrasco, BSN, BEd (Secondary)

2014
Student Declaration

I, Cheryl Carrasco hereby declare that:

1. this thesis is my own work,

2. all persons consulted, and all assistance rendered are fully acknowledged,

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

4. the substance of this thesis has not been presented in whole, or part by me, to any University for a degree.

Date: 27th September 2013 Signature: Cheryl Carrasco
I would like to thank the following people who made this study possible.

Dr Darren Morton for his guidance and for his persistence to make this thesis the best it could be,

Dr Peter Morey for his countless hours teaching me statistical analysis.

Erik and my children, for their ongoing support and encouragement.

Mum for looking after the kids when I was at Avondale College Campus and attending the CHIP summit.

Andy and Teena for listening and giving advice.

And last but not least to God, who opened doors for me to complete this study.
Abstract

Cardiovascular disease (CVD) is a major cause of death and disability throughout Australasia, placing a burden on sufferers, carers and the population at large. The Coronary Health Improvement Project (CHIP) is a community-based lifestyle education program that has been shown to significantly reduce CVD risk factors when applied in the United States. The 30-day CHIP intervention involves 16 group sessions where participants are educated and encouraged to move towards a plant-based diet, engage in daily physical activity, cease substance usage and manage stress.

The primary aim of the study was to examine the impact of the CHIP educational program on participants’ CVD risk factors within Australasia. The second aim of the study was to assess the influence of the participants’ age, gender and marital status on their responsiveness to the program.

The pre-test post-test cohort study involved 836 individuals who participated in one of 18 CHIP interventions offered throughout Australia and New Zealand. Data collected included blood lipid profile, fasting plasma glucose levels, body weight, blood pressure and demographic details.

Significant reductions were observed in all measured CVD risk factors and participants at greatest risk experienced the greatest benefits. In general, males - those in the 50–59 years age group and married participants experienced the most successful outcomes.

The findings indicate that the CHIP lifestyle education program can effectively reduce CVD risk factors, suggesting that it may be an cost effective tool for combating the incidence of CVD in Australasia.
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Chapter 1: Introduction

In 2011, 45,600 deaths were attributed to CVD in Australia (ABS, 2013). CVD causes disability and death, as well as creating a financial burden to the individual and also to the health care systems. The current treatment of CVD is a predominately medical approach involving the use of pharmaceuticals and cardiac surgery.

It is widely recognized that CVD is largely a disease of comfort, caused by poor lifestyle choices (Choi, Hunter, Tsou, & Sainsbury, 2005). Given CVD’s lifestyle origins it is not surprising that an estimated 70 - 90% of coronary episodes can be avoided through positive lifestyle choices (Aldana et al., 2006).

Residential lifestyle education programs, such as those which are delivered at the Pritikin Longevity Centre, have demonstrated success in reducing CVD risk factors through lifestyle modification, however these programs are expensive and separate the participants from their “home” environment. The CHIP intervention is a lifestyle education program that can be delivered inexpensively by volunteers and operates in a community setting so that participants are educated on making positive lifestyle choices while living in their normal living environment.

CHIP involves 16 group sessions over 30 days in which participants are educated and encouraged to move towards a plant-based diet, engage in physical activity, adopt a positive mental attitude and cease substance usage, in particular tobacco and alcohol.

While the CHIP educational program has resulted in impressive results in a large cohort of over 5,000 participants in the United States, no studies have examined the effectiveness of the program in Australasia. The primary aim of this
study was therefore to examine the effect of the CHIP lifestyle education program in Australasia on the participants' risk factors for CVD. The second aim of the study was to explore the influence of participants’ age, gender and marital status on their responsiveness to the program.

Research Questions

The study examined the following research questions:

1. Can a 30-day lifestyle education program (CHIP) improve risk factors for CVD in Australasia?
2. How does the participants’ age, gender and marital status influence their responsiveness to the CHIP lifestyle education program?

Structure of the Thesis

The thesis consists of six chapters. Chapter 1 presents the rationale, purpose and aims of the study. Chapter 2 presents a review of the relevant literature. Chapter 3 provides information on the sample, methods and data analysis processes. Chapter 4 presents and discusses the results pertaining to Research Question 1, and Chapter 5 focuses on Research Question 2. The final chapter, Chapter 6 summarizes the results, identifies limitations of the study and offers suggestions for future research.
Chapter 2: Review of the Literature

Introduction

Cardiovascular disease (CVD) remains the biggest cause of death in Australia (ABS, 2013). It is predicted that by the year 2051 CVD could affect one in every four Australians (ABS 2013). The prevalence of CVD creates a considerable burden in terms of illness, disability and health care expenditure. Currently the dominant treatment for CVD is a medical paradigm, which includes surgical and pharmaceutical treatment (Esselstyn, 2010). While CVD is responsible for 18% of disease and injury, it has been suggested that 80% of cases are preventable (Osborne, 2009).

Another approach to managing CVD is through lifestyle modification, specifically through changes in diet and activity, cessation of substance usage, and more efficient stress management (Englert, Diehl, Greenlaw, Willich, & Aldana 2007; Esselstyn, 2010; Ornish et al., 1990b). The argued barriers to a lifestyle medicine approach include; the perception that patients will not make lifestyle changes, and the cost and the time intensiveness of administering lifestyle modification programs (Anderson, Boman, Jansson, Nilsson, & Lindahl, 2008; Wister et al., 2007). Despite the assumed difficulty of compliance, studies have documented maintenance of lifestyle changes from 18 months up to 20 years post intervention (Esselstyn, 2008; Merrill, Aldana, et al., 2008). Furthermore, studies have shown that compliance to a pharmaceutical regime can also be problematic (Lardizabal & Deedwania, 2010; Ward et al., 2007). The argument that the lifestyle medicine approach is costly is minimized when the projected cost for the next decade on statins to treat dyslipidemia alone exceeds one billion dollars per year in Australia (Clarke & Fitzgerald, 2010). The Coronary Health Improvement Project (CHIP) is a 30-day lifestyle education program targeting CVD, commonly facilitated by volunteers who are resourced with an educational package and curriculum that allows them to deliver a program in their local community. The program encourages a plant-based diet with emphasis on the consumption
of grains, legumes and fresh fruits and vegetables. The program also encourages daily exercise and offers stress management skills.

**CVD risk factors**

The Framingham study, which commenced in Massachusetts in 1948, was the first study to identify cigarette smoking, increased cholesterol levels, elevated blood pressure, obesity and inactivity as major CVD risk factors (O'Donnell & Elosua, 2008). More recently, the 52 country Interheart study identified a further six CVD risk factors: smoking, diabetes, abdominal obesity, psychological factors, lack of consumption of fruit and vegetables, and excessive alcohol consumption (Yusef et al., 2004). These lifestyle-related risk factors account for more than 80% of the risk of myocardial infarction (Yusuf et al., 2004).

The following CVD risk factors will be discussed in more detail below: cholesterol, blood pressure, obesity, smoking, diet, inactivity, diabetes and mental health. Whilst these risk factors are discussed separately it is important to note that they are clearly interrelated, for example obesity is related to an elevation in all other CVD risk factors (Gregg et al., 2005; Klein et al., 2004). Also the impact of coronary risk increases exponentially with an increase in the number of risk factors (Stamler, Stamler & Neaton, 1999). A cohort of men 55 years and older who had three coronary risk factors had 39 times more chance of a having a coronary event compared to those men with no risk factors and 10 times more compared to those with one or two risk factors (Yusuf, Giles, Croft, Anda, & Casper, 1998).

**Cholesterol**

Hypercholesterolemia is a primary cause of CVD (Cleeman, Grundy, Becker, & Clark, 2001; Krauss et al., 2000). Total cholesterol is made up of the cholesterol transporting fatty proteins: low-density lipoproteins (LDL), high-density lipoproteins (HDL) and triglycerides. LDL contributes to atherosclerosis and therefore increases the risk of CVD.
Conversely high levels of HDL are thought to have a protective effect against CVD (Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977).

Results of cholesterol lowering studies have found that a 1% reduction in the level of LDL results in a 1 – 1.5% decrease in the risk of a cardiac event (Assmann, Cullen, & Schulte, 1998; Wilson, Anderson, Castelli, & Kannel, 1991). In studies on high density lipoproteins (HDL) an increase of 2-3% led to a decrease in 2 – 4% of risk of a cardiac event independent of LDL levels (Brown et al., 2001).

**Blood Pressure**

High blood pressure (BP) is also a measurable and easily identifiable CVD risk factor (Rodgers, Lawes, & MacMahon, 2000; Vasan et al., 2001; Yusuf et al., 2004). The adverse effects of hypertension on CVD begin to occur when BP exceeds 120mmHg systolic and 80mmHg diastolic (Stamler, Stamler, & Neaton, 1993). As BP increases above these levels, the risk of CVD increases proportionally (Rodgers et al., 2000; Stamler, Stamler, et al., 1993; Vasan et al., 2001; Yusuf et al., 2004). The relationship between BP and CVD is so consistent that systolic blood pressure (SBP) has been accepted as a clinically useful predictor of CVD (Franklin et al., 2009).

**Obesity**

Being overweight or obese increases the risk of CVD, particularly when the weight is distributed around the abdomen. (Dagenais et al., 2005; Klein et al., 2004).
Smoking

It is widely accepted that cigarette smoking is a major cause of CVD (Ambrose & Barua, 2004; Benowitz, 2003; Burns, 2003; Yusuf et al., 2004). The cardiovascular risk caused by cigarette smoking increases with the number of cigarettes smoked per day as well as the length of time an individual has been a smoker (Burns, 2003; Yusuf et al., 2004). Smoking is also linked to other CVD risk factors including abnormal lipid profiles (Craig, Palomaki, & Haddow, 1989; Frati, Iniesta, & Ariza, 1996), hypertension and diabetes (Burns, 2003).

Benowitz (2003) has suggested that the extent to which cigarette smoking increases the risk of CVD is linked to genetic factors and insulin resistance; only smokers with insulin resistance appear at increased CVD risk (Freeman et al., 1993; Jeppesen, Hein, Suadicani, & Gyntelberg, 2001; Reaven, 2003).

Diet

Dietary intake is related to a number of CVD risk factors including hypertension, abnormal blood lipids and abdominal obesity (Yusuf et al., 2004). Cross-cultural studies focusing on dietary habits have revealed trends between high consumption of animal-derived foods, such as meat and dairy, and CVD. A study conducted in World War II revealed that when the Norwegian meat and dairy supply were cut off by the Germans, the incidence of death from heart attacks and stroke was reduced (Esselstyn, 2010). In another study the death rates amongst American men who consumed diets high in animal based foods were found to be 17 times higher than among rural Chinese men who consumed diets rich in plant based foods (Campbell, Parpia, & Chen, 1998). In other cultures that consume predominately plant-based foods, such as the Papua highlanders in New Guinea (Sinett & Whyte, 1973), Central Africans and the Tarahumara Indians of Northern Mexico (Connor et al., 1978), cholesterol levels are commonly below 3.88 mmol/ L, and CVD is practically nonexistent. When residents of these areas with low coronary artery
disease adopt a western diet (high in animal products and fat) the incidence of coronary
disease rises dramatically (Esselstyn 1999).

Inactivity

Physical inactivity is another major contributing factor to the risk of CVD. In a study
conducted over eight years on more than 40,000 men and women, it was observed that
those in the bottom 20% of fitness ranking were 65% more likely to die from heart attacks
and strokes than those in the top 20% of fitness ranking, as measured by treadmill test
performance (Blair et al., 1989).

A more recent study found that even sedentary women who had desirable blood
cholesterol levels, did not smoke, and had normal blood pressure, had six times the risk of
dying from heart disease compared to active women with the same cholesterol levels and
blood pressure (Mora & Lee, 2006).

There is evidence that the greatest CVD benefits are obtained by those who exercise
vigorously (Blair, Cheng, & Scott Holder, 2001).

Diabetes

Individuals with type II diabetes have two to six times greater risk for CVD than non-
diabetics (Gu, Cowie, & Harris, 1999; Kannel & McGee, 1979). Furthermore, complications
of diabetes led to multiple CVD risk factors including hyperglycemia, hypertension and
dyslipidemia (Stamler, Vaccaro, Neaton, & Wentworth, 1993).

Mental Health

The influence of psychological factors on CVD risk may have been underestimated in
the past, but it is becoming increasingly recognized as an important CVD risk factor. Yusuf
et al. (2004) found that depression increased the risk of myocardial infarction by four times.
Other studies have also found stress and anxiety to be strongly associated with CVD (Bunker et al., 2003; Katon, Lin, & Kroenke, 2007).

There appears to be ample evidence that suggests these lifestyle factors - cholesterol, blood pressure, obesity, smoking, diet, inactivity, diabetes and mental health - have a significant affect on CVD risk factors.

**Management of CVD**

The current approach to treatment is a medical approach, however there are complications associated with this strategy. Many of the pharmaceuticals have side effects, and in the treatment of atherosclerosis, when statins are unsuccessful as a treatment for dyslipidemia, a more aggressive surgical approach is taken including coronary artery bypass graft, atherectomy, angioplasty and stenting. These procedures can be life threatening with the American Heart Association reporting mortality rates of 1% for stents and 2.5 % for coronary bypass surgery (Esselstyn 2010). Where drug trials have been compared to the lifestyle intervention approach, the alteration of lifestyle factors has been found comparable to and in some cases superior for treating hypertension and dyslipidemia (Jenkins et al., 2003; Ratner et al., 2005). Considering the risk and side effects of the current treatment to CVD, it may be prudent to investigate the benefits of a lifestyle approach, which appears to have no side effects.

**The effect of lifestyle modifications on CVD risk factors**

It has been established that through the application of lifestyle factors, in particular a plant-based diet, increased physical activity, smoking cessation and management of mental attitude, CVD risk factors can be successfully reduced (Adlana et al., 2005; Ornish et al., 1990b). A further breakdown of the effect of these lifestyle factors on certain biometrics will be discussed below.
**Diet**

A plant based diet has been shown to have an influence on reducing weight, blood lipid levels, BP and Fasting plasma glucose, all of which are related to reducing CVD (Antonopoulos, 2002; Hu & Willett, 2002).

Dietary methods to reduce cholesterol levels mainly focus on decreasing the amount of fat in the diet. However, not all fats are equal in their effect on heart health. There are three main forms of dietary fatty acids: saturated fatty acid (SFAs), monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs). There are also trans unsaturated fats, produced by hydrogenation of vegetable oils.

Trans fatty acids have been found to increase LDL and reduce HDL (Sacks & Katan, 2002), therefore levels of LDL can be lowered by decreasing saturated fat (cholesterol, meats and dairy foods) and trans fatty acids in the diet. Dietary methods of reducing LDL are so effective that Jenkins et al (2006) point out that with a diet low in saturated fat, high in viscous fibers (oats, barley), plant sterols, vegetable protein, legumes and nuts, LDL reductions are as effective as those on statin treatment (Albert, Gaziano, Willett, & Manson, 2002; Fraser, Sabate, Beeson, & Strahan, 1992; Hu et al., 1998; Watts et al., 1992).

Whilst saturated fats are detrimental to heart health, MUFA’s and PUFAs are thought to have a protective effect, producing desirable changes in plasma lipids (Sacks & Katan, 2002). Omega 3 PUFA’s have been found to lower triglyceride levels (Albert, Campos, et al., 2002; Balk et al., 2006; Hu et al., 2002; Marchioli et al., 2002; Sacks & Katan, 2002) and decrease the incidence of sudden cardiac death (Lee & Lip, 2003). However an imbalance of the omega 6 to omega 3 fatty acids ratio increases the risk of CVD (Simopoulos, 2008). Simopoulos (2008) reported a ratio of 15:1 of omega 6 to omega 3 in the western diet, whereas a lower ratio is desirable. A ratio of 4: 1 was associated with a 70% reduction in death. Thus a reduction in omega 6 polyunsaturated fats may be necessary for optimal heart health.
The effect of the CHIP plant-based diet, low in saturated fat, trans fatty acids and cholesterol, and high in low glycemic index (GI) foods is also associated with a reduction in BP (Sacks et al., 2001), improved glycemic control, insulin sensitivity and a decreased incidence of diabetes (Chandalia et al., 2000; Costacou & Mayer-Davis, 2003; Fung et al., 2002; Hu et al., 2001; Järvi et al., 1999; Jenkins et al., 2002; Liu et al., 2000; Meyer, Kushi, Jacobs, & Folsom, 2001; Salmeron et al., 2001; Summers et al., 2002). In fact only one in ten type II diabetes (T2D) patients would require medication if they achieved ideal weight, consumed an optimal diet and exercised regularly (Pedersen & Nieman, 1998). Even without change of diet and weight loss, exercise has now emerged as one of the key treatment options for T2D, (Boulé, Haddad, Kenny, Wells, & Sigal, 2001; Knowler et al., 2002; LaMonte, Blair, & Church, 2005; Thomas, Zimmet, & Shaw, 2006; Tuomilehto et al., 2001).

**Exercise**

It is well known that exercise assists in weight reduction (Eyre et al., 2004). Interestingly, even when exercise does not result in weight loss it still can contribute to a reduction in abdominal fat, and fat in the abdominal region is a known factor contributing to CVD (Ross et al., 2000).

The effect of exercise on blood lipids varies with different exercise volumes. Greater changes have been seen in plasma lipids with increased exercise volume (Durstine et al., 2001; Kraus et al., 2002; Thompson et al., 2001), as demonstrated in endurance athletes who often have HDL levels 40 – 50% higher, TG levels 20% lower and LDL concentrations 5 -10% lower compared to the sedentary population (Franklin, Bonzheim, Gordon, & Timmis, 1998; Jones & Knapik, 1999; Maron et al., 1996; Sady, Cullinane, Saritelli, Bernier, & Thompson, 1988; Wood et al., 1976).

Typically larger changes are seen in changes in HDL and TG than LDL; furthermore the effect of exercise on HDL and TG have been found to be greater in those with low HDL and elevated TG levels (Couillard et al., 2001; Durstine et al., 2001; Leon & Sanchez, 2001).
Exercise also benefits individuals with high BP. The effect from a single session of exercise may lower BP in the hypertensive individual into normal ranges for the major portion of the day (Hagberg, Park, & Brown, 2000). The chronic effects of exercise can benefit 75% of hypertensive individuals, decreasing systolic BP on average by 11mmHg and the diastolic BP by 8mmHG. These type of reductions have been shown to halve the risk of cardiovascular death (Hagberg et al., 2000; Wilmore et al., 2001). Greater benefits were obtained in women, in the middle aged, and in those exercising at an aerobic intensity (Hagberg et al., 2000).

**Mental health**

Mental health is a lifestyle factor that has gained more attention lately in its relationship to the effects on CVD risk factors. Poor mental health - specifically depression, anxiety and stress - contributes to an increase in cholesterol levels (Bajwa, Asnis, Sanderson, Irfan, & Van Praag, 1992; Helmut et al., 2002), hyperglycemia in type I and type II diabetics (Chandalia et al., 2000; De Groot, Anderson, Freedland, Clouse, & Lustman, 2001; Lustman et al., 2000; Pischke, Marlin, Weidner, Chi, & Ornish, 2006) and abdominal obesity (Tsatsoulis & Fountoulakis, 2006).

There have been inconclusive results in the relationship between BP, depression and anxiety. Some studies have found increased resting BP with depression (Rutledge & Hogan, 2002; Scherrer et al., 2003) and anxiety (Markovitz, Matthews, Kannel, Cobb, & D'Agostino, 1993; Paterniti et al., 1999), while others have found no relationship (Shinn, Poston, Kimball, St Jeor, & Foreyt, 2001; Yan et al., 2003). Some studies have even found a decrease in BP with anxiety and depression (Hildrum, Mykletun, Holmen, & Dahl, 2008; Hildrum et al., 2007; Paterniti et al., 1999).
Cessation of substance usage

In general the term “substance” refers to tobacco, alcohol and other drugs. However in the context of this study the focus will be on the effect of smoking and alcohol usage on blood lipids, FPG and BP. The effect of other drugs on the biometrics will not be discussed.

Smoking cessation has been found to increase HDL levels, but TC, LDL, and TG appear to be unaffected (Maeda, Noguchi, & Fukui, 2003). The authors found the increased HDL levels that occurred in response to smoking cessation reduced the risk of CHD by 7.4% in men and 12.5% in women. Most literature agree that smoking contributes to an elevation of blood sugar levels thus smoking cessation helps to combat diabetes (Filozof, Fernandez Pinilla, & Fernández Cruz, 2004; Frati et al., 1996).

Whilst cigarette smoking causes an acute rise in resting BP (Groppelli, Giorgi, Omoni, Parati, & Mancia, 1992), there was some disagreement regarding the long term effects of smoking. Some smokers were found to have no increases in BP, while others showed increases in BP (Gordon, Kannel, Dawber, & McGee, 1975; Green & Hrari, 1995; Groppelli et al., 1992; Lee, Ha, Kim, & Jacobs, 2001). The variation in the effect of smoking on blood pressure may be a result of smokers commonly having a lower body weight than non-smokers, which in turn tends to lower their BP (Higgins & Kjelsberg, 1967; Lee et al., 2001; Wilhelmsen, 1988). Whilst there is often a weight gain associated with smoking cessation, the health benefits of cessation have been found to exceed the health risks associated with the weight gain (Pistelli, Aquilini, & Carrozzi, 2009). Furthermore, most individuals tended to lose their post smoking weight gain over a period ranging from a few years (Gruber & Frakes, 2006) up to eight years (Perkins, 1993).

Light alcohol consumption, (1 drink per day for females; 1-2 drinks per day for males) is also thought to result in increases in HDL and improvements in insulin sensitivity (O’Keefe, Bybee, & Lavie, 2007). However, Yusuf et al (2004) found the protective effect was less than 1%. As it is difficult to predict which people are likely to develop a dependence on alcohol (Lucas, Brown, Wassef, & Giles, 2005), and the possibility of developing other alcohol
related diseases such as strokes, cancers, cirrhosis of the liver or experiencing an alcohol related accident, the consumption of alcohol for a mere 1% protective factor is not recommended (Collins & Lapsley, 2008; Yusuf et al., 2004).

**Lifestyle education programs**

As discussed above, the data indicates that lifestyle changes can make substantial differences to CVD risk factors. There are several lifestyle education programs that incorporate these lifestyle choices - diet, exercise, mental attitude and cessation of substance usage - and which have been shown to be beneficial.

The most notable lifestyle interventions targeting CVD are the Pritikin and the Ornish programs (Adlana et al., 2004; Barnard, 1990; Beard, Barnard, Robbins, Ordovas, & Schaefer, 1996; McClendon, Dunbar, Clark, & Coverson, 2010). These programs have demonstrated that heart disease is not only preventable but is also reversible through the implementation of lifestyle changes.

Nathan Pritikin, founder of the Pritikin Longevity Centre in California, designed a 3-week residential lifestyle program in the early 1980s. Dietary recommendations include less than 10% calories from fat, 15 – 20% from protein and the remainder from unrefined, complex carbohydrates. The program also involves daily exercise. In a study of 4587 participants in the Pritikin program, significant reductions in TC, LDL and TG levels were observed. Eighteen months later participants who complied with the dietary and exercise recommendations maintained cholesterol levels below 5.18 mmol/L (200mg/dl) (Barnard, 1991).

The intensive lifestyle modifications in the Ornish program involve a vegetarian diet with 7% of caloric intake from fat, use of stress management techniques, smoking cessation, moderate aerobic exercise and attendance at group support meetings. Changes after twelve months included a 20% average decrease in plasma TC and a 91% average reduction in frequency of angina (Adlana et al., 2004). In a landmark publication, 82% of participants in
the Ornish program with established CVD experienced a measurable regression in the narrowing of their coronary blood vessels (stenosis), whereas 53% of patients in the control group had documented progression of their stenosis at five years follow up (D. Ornish et al., 1990a; Parikh et al., 2005).

Caldwell Esselstyn is a further pioneer in the field of using lifestyle intervention for the treatment of CVD. Esselstyn (1999) in a study over 20 years demonstrated with a plant based diet cholesterol levels could be reduced below 3.88mmol/L.

The Pritikin, Ornish residential programs, and studies by Esselstyn have shown that the progression of CVD can be prevented, and even reversed, through lifestyle intervention (Esselstyn, 1999; Ornish et al., 1990a).

**Limitations of lifestyle education programs**

While residential programs such as the Pritikan and Ornish programs, have been found to be successful in reducing CVD and its risk factors, they are expensive and resource intensive (Diehl, 1998). The programs are also conducted out of the patients’ normal living environment. Consequently participants face issues at home that were not encountered in their program, such as a lack of psychological support (Ornish, Scherwitz, & Billings, 1998; Roberts & Barnard, 2005).

Recognizing these limitations, in 1987 Dr Hans Diehl designed a community-based lifestyle intervention called the Coronary Health Improvement Program (CHIP). The CHIP intervention has been presented by health professional since 1987 in hospital settings (Diehl, 1998), workplace environments and community venues (Adlana et al., 2008).
CHIP as a lifestyle education program.

The CHIP lifestyle education program is a 30-day intervention that aims to educate and support free-living participants to adhere to a whole-food, plant-based eating pattern with the emphasis on eating grains, legumes, fresh fruit and vegetables. Specifically, the program recommends less than 15% of calories be derived from fat, < 10 teaspoons of added sugar, < 5,000mg of salt and <50mg of cholesterol per day. The participants are also encouraged to drink 2 – 2.5 L of water per day. CHIP also encourages participants to eliminate tobacco usage, exercise daily, and utilize stress management techniques (Diehl, 1998). The intent of the program is to educate CHIP participants on the epidemiology, etiology and risk factors associated with CVD in order to empower individuals to make informed health choices.

The CHIP educational content includes modern medicine’s accomplishments and limitations, atherosclerosis, cardiovascular risk factors, smoking, exercise, dietary fiber, cholesterol, plant based nutrition, obesity, diabetes, hypertension, dyslipidemia, lifestyle and health, behavioral change and self worth (Englert, Diehl, & Greenlaw, 2004). The program occurs in a group context to foster social support. There is also an alumni, where at completion of the program participants are encouraged to attend monthly meetings for continued social support.

A recent study involving a large cohort of over 5,000 individuals from the United States who participated in a CHIP program between 2006 and 2009 showed significant reductions in basal metabolic index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), TC, LDL, TG and FPG in 30 days (Rankin et al., 2012). The most notable changes were in TC. The mean decrease of 21.3mg/dl in TC is thought to translate into a 20% reduction in relative risk for all cause mortality (Rankin et al., 2012). When the CVD risk factors of the CHIP participants were further classified into risk categories it was found that the greatest improvement was seen in those that were at highest risk at program entry (Rankin et al., 2012). Other CHIP studies have also demonstrated that those who made the
most improvement in their CVD risk factors were those who were at greatest risk at baseline (Adlana et al., 2006; Aldana et al., 2008; Englert et al., 2007; Englert et al., 2004).

Also after the stratification of the risk categories in the CHIP US cohort it was seen at the conclusion of the 30 day CHIP that 77% of participants moved from the highest TC risk category to a lower risk category. For FPG there was a 43% reduction in the number of participants in the highest risk category (Rankin et al., 2012). The changes in the blood lipid profile compared favorably to those achieved with statin medication (Gould, Davies, Alemao, Yin, & Cook, 2007). The change in these biometrics resulted in significant reductions in medication for participants, primarily for treatment of high cholesterol and BP (Rankin et al., 2012).

A unique aspect of the study was the CHIP interventions were delivered by volunteers, and that despite this potential disadvantage, the reduction in CVD risk factors was comparable to CHIP programs delivered by health professionals (Englert et al., 2007; Merrill, Taylor, & Adlana, 2008; Rankin et al., 2012).

The results of the United States based CHIP study have demonstrated that volunteers can be successful agents of change for health education within the community (Morton et al., 2013). The success of volunteers as facilitators of the US CHIP intervention was attributed to two main factors. Firstly the volunteers were resourced with prerecorded lectures by a cardiovascular epidemiologist. Secondly, it has been found that passionate volunteers are able to motivate participants into making changes to their health (Kong, 1997). Because many of the volunteers had a familiarity with the program, being past CHIP participants themselves, they may have been able to develop unexpected rapport and be highly inspirational to participants (Morton et al., 2013). Using volunteer facilitators to administer CHIP reduce costs, making the CHIP program available to the wider community. In turn, accessible and inexpensive CHIP programs will potentially yield substantial reductions in the fiscal burden associated with cardiovascular disease (Morton et al., 2013).
The success of the volunteer delivered CHIP has been established in the United States; however, there is a need to determine whether volunteers can reproduce the same successful outcomes in Australasia.

The effect of participant demographics on their responsiveness to CHIP

There is very little literature on the effects of participants’ age, gender and marital status on the outcomes achieved from a lifestyle education program targeting CVD. Specifically, the influence of participant demographics, with the exception of gender, has not been overly considered in the context of CHIP. These factors will be examined in this study.

Gender

Among the middle aged, CVD is 2 – 5 times more common in men than in women (Vartiainen & Puska, 1999). Interestingly men respond quicker to lifestyle modifications in the treatment of CVD than women, as shown by the following studies.

A health study conducted in 1995 on the relationship between weight loss and CVD risk factors found that there were certain differences between the outcomes for men and women. Initially men experienced greater decreases in BP, TG, waist to hip ratio (WHR) and greater increases in HDL with weight loss than women. However after 18 months, improvements in WHR were shown to be greater in women than men (Wing & Jeffery, 1995).

Few studies of CHIP have provided a gender breakdown of the outcomes, however, it appears that men tend to respond quicker to the lifestyle changes encouraged in the CHIP education program compared to women. (Diehl, 1998; Englert et al., 2004; Englert, Diehl, & Greenlaw, 2007; Pischke, Weidner, Elliott-Eller, & Ornish, 2007; Roberts & Barnard, 2005).
It is not clear why the CHIP intervention is greater on men than women, but the responsiveness of men could be explained by a combination of physiological or behavioral factors.

Behaviorally men differ from women in a number of ways relating to their attitude to health. Men are less concerned with being overweight (Gillon & McPherson, 2007), have poorer health yet use health services less, and have higher mortality rates (Courtney, 2000; Lee & G. Owens, 2002; Mansfield, Addis, & Mahalik, 2003; Tudiver & Talbot, 1999). Studies have suggested that men may feel the need, as a result of cultural pressure, to present a masculine identity (Lee & Owens, 2002), which involves an attitude of self reliance, independence, risk taking and superiority (Courtenay, 2000; Mahalik, Good, & Englar-Carlson, 2003) thus men delay seeking help when symptoms arise (Heidenreich, Trogdon, & Khavjou, 2011; Lee & Owens, 2002).

Women however are more concerned with their weight management and are more likely to attempt weight loss programs (McCreary & Sadava, 2001; Wardle & Johnson, 2002; Young et al., 2012).

However while women appear to be eager to change their behavior, this does not always translate into commitment (Vlerick Leuven Gent Working Paper Series 2012). It has been found that women who have joined weight loss programs have higher expectations and, due to disappointment, also have higher drop out rates (Vlerick Leuven Gent Working Paper Series 2012).

Men who join a weight loss program are more likely to complete the program and lose weight (Bautista-Castano, Molina-Cabrillana, Montoya-Alonso, & Serra-Majem, 2004). Men also appear to have more scope for changing their diet. The diets of men tend to be higher in red and processed meats and lower in fiber (Stibbe, 2004) compared to women who have been found to eat more fruit and vegetables (Blanck, Gillespie, Kimmons, Seymour, & Serdula, 2008; Friel, Kelleher, Nolan, & Harrington, 2003).

Having a supportive relationship may be another factor that favours males, they typically receive more support from their spouse than vice versa (Pischke et al., 2007).
Physiologically, men have the propensity to lay fat in the abdominal region, which has been found to be more metabolically active compared to fat on the hips and thighs (Chaston & Dixon, 2008; Lemon, Rosal, Zapka, Borg, & Anderson, 2009; Regitz - Zagrosek, Lehmkuhl, & Weickert, 2006). They also have more muscle mass, and therefore a higher metabolic rate (Janssen, Heymsfield, Wang, & Ross, 2000; Stiegler & Cunliffe, 2006). Indeed men are more able to lose weight more easily compared to women due to hormonal differences and generally they are heavier at the start of a weight loss program. Heavier people tend to lose more weight (Egger, 2007; Ramsden & Hunter, 2007).

In particular, gender differences in fat distribution and muscle mass, compliance and whether or not the participant was attending with a supportive spouse are important factors that may have contributed to the finding that men appear to have greater changes as a result of the CHIP intervention.

Age

CVD reaches epidemic proportions among older persons thus age is a major risk factor for CVD (Lakatta, 2002). One study found the incidence of CVD triples with each decade of life (Finegold, Asaria, & Francis, 2012).

Literature supports the importance of treating CVD with lifestyle intervention from an early age as it appears the absence of established risk factors at age 50 years has been associated with very low lifetime risk for CVD and markedly longer survival (Lloyd - Jones et al., 2006).

The Adventist Health Study has suggested that the maximum benefit from changes to lifestyle habits are obtained before the age of 30 (Fraser & Shavlik, 2001 ). Another study, has found that midlife fitness (mean age 49 years of age) was associated with a lower risk of common chronic health conditions in men and women older than 65 years enrolled in Medicare. It was concluded that higher midlife fitness may be associated with compression of morbidity in older age (Willis, Gao, Leonard, DeFina, & Berry, 2012).
No studies of CHIP have examined the influence of the participants’ age on the outcomes they achieve.

**Marital status**

Morbidity and mortality have been shown to be lower for married individuals (Kiecolt-Glaser & Newton, 2001). One meta-analysis found that single people have a 30% increased risk of mortality compared to married persons (Roelfs, Shor, Kalish, & Yogev, 2011). In regards to CVD, House, Landis, & Umberson, (1988) found that social integration had a positive impact on cardiovascular function.

Other studies have also found that social support and spouse support may be an important factor in achieving lifestyle change (Pischke et al., 2007; Verheijden & Kok, 2005). It appears that it is the female in a relationship who is typically more active in encouraging men to change their health habits (Kiecolt-Glaser & Newton, 2001; Umberson, 1992). Other factors such as age and also the quality of the relationship can also have an impact on health status (Troxel, Matthews, Gallo, & Kuller, 2005).

No studies of CHIP have considered the influence of marital status on the participants’ responsiveness to the program.

**Conclusion**

It is evident that CVD is a major health and financial burden in Australasia. The 30-day CHIP has been shown to successfully reduce the risk factors of CVD in the United States, even when the programs are facilitated by volunteers. To date, there are no studies of the effectiveness of CHIP in Australasia. The present study aims to examine the effect of the volunteer-delivered, 30-day CHIP lifestyle education program on CVD risk factors in Australasia. Further, the study aims to explore the influence of CHIP participants’ gender, age and marital status on their responsiveness to the lifestyle education program.
Chapter 3: Methodology

Introduction

Limited research has been conducted to establish the effectiveness of a volunteer delivered health education program. Such studies have been conducted in the United States, with successful results seen in reducing CVD risk factors. The aim of this study is to examine the changes in the CVD risk factors: total cholesterol, (TC), low density lipoprotein, (LDL), high density lipoprotein, (HDL), triglycerides, (TG), fasting plasma glucose, (FPG), body mass index,(BMI), and blood pressure (BP) as a result of the 30-day CHIP education program delivered by volunteers in Australasia. The biometric and demographic data taken from the CHIP survey instrument and blood screening results are recorded by the program coordinators and will be accessed by the researcher. Consent for the study was obtained from Avondale College of Higher Education Human Research Ethics Committee (Approval No. 20:10:07).

Study participants

The study involved 836 individuals located both in Australia and New Zealand across 18 different sites. The CHIP participants self-selected to participate in the program and were recruited via community advertising, newspapers and church-based advertising in local communities in Australia and New Zealand. In some instances local medical practitioners recommended their patients to the program.
Sample characteristics

Gender

The sample consisted of 265 males (35.19%) and 488 females (64.81%). Eighty-three of the participants did not indicate their gender.

Age

The average age of the participants was 55.9±12.7 years. The participants were stratified into the following age groups: 39 years and younger (N=74, 10.9%), 40-49 years (N=121, 17.8%), 50–59 years (N=182, 26.8%), 60–69 years (N=212, 31.2%) and 70 and older (N=91, 13.4%). One hundred and fifty-six participants did not record their age.

Marital Status

From the 836 participants, 63 (9.5%) were single, 519 (77.9%) were married, 46 (6.9%) were divorced and 38 (5.7%) were widowed. One hundred and seventy participants did not record their marital status.

Family History of Cardiovascular Disease

Fifty-six (12.9%) CHIP participants recorded a family history of cardiovascular disease (CVD) while 379 (87.1%) had no history of CVD. There were no details recorded for family history of CVD for 401 of the CHIP participants.

Family History of Diabetes Mellitus

Twenty-four (5.7%) CHIP participants recorded a family history for diabetes mellitus, 400 (94.3%) reported no history of diabetes mellitus, and 412 of the participants did not indicate whether there was or was not any history of diabetes mellitus in their family.
The CHIP lifestyle education program

The intent of CHIP was to educate participants on the epidemiology, etiology and risk factors associated with CVD, and support them on a behavior change journey to adopt health-promoting lifestyle practices. The 30-day program involved 16 group sessions in which participants firstly viewed an educational pre-recorded lecture. Topics covered included atherosclerosis, coronary risk factors, smoking, exercise, dietary fiber, cholesterol, the optimal diet, obesity, diabetes, hypertension, hyperlipidemia, lifestyle and health behavior change, and self-worth. In addition to viewing the educational videos, the participants were involved in cooking demonstrations, group exercise and group discussions. Each session was between 1.5-2 hours in duration.

Nutrition was the focus of the CHIP program. Participants were encouraged to adopt a whole-food, plant-based diet with less than 15% of calories from fat, a daily intake of less than 10 teaspoons of sugar, less than 5000mg of salt, and less than 50mg of cholesterol. Also, consumption of 2 – 2.5 L of water daily was encouraged.

The CHIP participants were encouraged to engage in the program in consultation with their medical practitioner for monitoring of medication usage.

Of the 836 participants who enrolled in the program, 790 (94%) completed the 30-day CHIP. To qualify as completing the CHIP education program, a participant was required to attend 13 of the 16 sessions and undergo pre- and post-intervention blood testing. At the completion of CHIP they were encouraged to join a monthly support group, an exhortation the results of which were outside the scope of this study.
Program delivery and administration

There were 31 CHIP education programs presented in 18 locations throughout Australasia (731 participants from 14 sites in New Zealand and 105 participants from 4 Australian sites). The size of the CHIP groups ranged from 5 to 101 participants (mean group size = 26.3±23.0).

Volunteer directors, sourced primarily through the Seventh-day Adventist Church, facilitated these 31 CHIP education programs. The role of the volunteer director was to organise and facilitate the proceedings of the group sessions.

Training for these volunteer directors involved a two-day training program to teach group facilitator skills. In addition volunteers were provided with a curriculum guide for program delivery and pre-recorded educational lectures presented by cardiovascular epidemiologist, Dr Hans Diehl, which covered the topics as listed above.

The majority of the volunteers, twelve from the total of eighteen presenters, (age = 55.1±9.5, 5 males/13 females) were not health professionals but were individuals passionate about health.

Although the program was delivered by volunteers there was a fee to the participants of approximately $250 which was levied to cover the cost of venue hire, food samples distributed throughout the program, resources including reading materials, a pedometer and biomedical assessments.

Measurements and instruments

Informed written consent was obtained from the participants before commencing the CHIP program. Information about the participants’ personal demographics, including age, gender, marital status and personal medical history was collected using a questionnaire. Fasting blood samples were collected by trained phlebotomists and analyzed by local
pathology laboratories for TC, HDL, LDL, TG, BMI, SBP, DBP and FPG. Height, weight and blood pressure were also collected immediately before and after the program. Participants’ weight was measured with shoes removed. Height and weight were used to calculate the participants’ Body Mass Index (BMI), calculated as weight (kg) / height (m)².

**Data collection**

As the data for this study was collected at many sites across two countries, it was not possible for the candidate to personally collect the data and run all the CHIP programs. The candidate was, however, involved in the facilitation and data collection of two CHIP education programs at the Morrisset Health Centre and Cooranbong Sanitarium Health and Wellbeing Company, both of which are in Australia. The candidate was certified to facilitate the CHIP lifestyle education program through completion of a two-day facilitator training program at which a comprehensive CHIP resource package was provided.

Data from the other programs was collected in a uniform manner and entered into a specifically developed Microsoft Access program which was converted to Microsoft excel (Microsoft, Redmond, Washington) and then imported into PASW Statistics, version 20 (SAS Institute, Cary, North Carolina) for analysis.

**Data analysis**

For analysis the data was stratified using the conventional risk factor categories from the National Cholesterol Education Program Adult Treatment Panel 111 classification system (National Education Program, 2002), for all of the risk factors except for cholesterol. For TC categorization the Framingham classification (Wilson et al., 1998) was used dividing TC data into five categories instead of three. This allows for a more detailed analysis of the effect of the CHIP intervention on the highest risk participants.
The pre- and post-biometric measures and the pre- and post-lifestyle habits data were compared using SPSS 20.0 (SPSS 20.0 for windows, SPSS Inc).

Prior to statistical analyses the normality of the distribution of each of the biometrics was evaluated using graphical techniques and the calculation of the distributions skewness and kurtosis. The respective distributions were found to be near normal. Homogeneity of variances was tested using Levene’s test.

Significant differences in the biometrics and lifestyle habits from baseline to post intervention were assessed using independent t-tests, paired t-tests, and independent one way between group ANOVA with post hoc comparisons to locate any areas of significant differences between the respective risk factor categories. Descriptive statistics for each measure were determined. The changes in both the biometrics from baseline to post intervention were recorded and presented using descriptive statistics – mean+/−standard deviation. Regression analysis was used to assess the influence of the changes in the various lifestyle measures on the biometric changes. The effect size of each biometric change was determined by using Cohen’s d, where a value of 0.2 is considered a small change, 0.5 a moderate change and 0.8 or greater a large change.
Chapter 4: Effectiveness of the CHIP lifestyle education program for reducing CVD risk factors.

Introduction

In this chapter the changes in the participants' biometric risk factors from program entry to completion of the 30-day CHIP lifestyle education program are presented. This chapter specifically addresses research question 1: Can a 30-day lifestyle education program (CHIP) improve risk factors for CVD in Australasia?

The risk factors considered are: TC, LDL, HDL, TG, BMI, SBP, DBP and FPG. The chapter starts by presenting the overall changes for all participants combined. The participants are then stratified according to their initial risk level for each biometric.
Table 4.1: Mean changes in selected CVD risk factors from baseline to post-intervention.

<table>
<thead>
<tr>
<th>Factor</th>
<th>N</th>
<th>Baseline Mean (SD)</th>
<th>Post-intervention Mean (SD)</th>
<th>Mean Change</th>
<th>% Change</th>
<th>t statistic</th>
<th>p value</th>
<th>Cohen's d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>790</td>
<td>87.07 (22.47)</td>
<td>83.94 (21.52)</td>
<td>-3.2</td>
<td>-3.8%</td>
<td>37.531</td>
<td>&lt;0.001</td>
<td>0.143</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>718</td>
<td>31.18 (7.73)</td>
<td>30.04 (7.41)</td>
<td>-1.2</td>
<td>-3.8%</td>
<td>37.221</td>
<td>&lt;0.001</td>
<td>0.150</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>787</td>
<td>134.88 (18.99)</td>
<td>127.41 (16.75)</td>
<td>-7.46</td>
<td>-5.53%</td>
<td>13.390</td>
<td>&lt;0.001</td>
<td>0.417</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>787</td>
<td>79.96 (11.45)</td>
<td>76.26 (19.98)</td>
<td>-3.7</td>
<td>-4.6%</td>
<td>10.674</td>
<td>&lt;0.001</td>
<td>0.330</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>779</td>
<td>5.17 (1.08)</td>
<td>4.40 (0.96)</td>
<td>-0.76</td>
<td>-14.7%</td>
<td>30.046</td>
<td>&lt;0.001</td>
<td>0.75</td>
</tr>
<tr>
<td>Low density lipoprotein (mmol/L)</td>
<td>775</td>
<td>3.17 (0.95)</td>
<td>2.60 (0.83)</td>
<td>-0.57</td>
<td>-17.9%</td>
<td>26.145</td>
<td>&lt;0.001</td>
<td>0.638</td>
</tr>
<tr>
<td>High density lipoprotein (mmol/L)</td>
<td>779</td>
<td>1.32 (0.36)</td>
<td>1.21 (0.32)</td>
<td>-0.11</td>
<td>-8.3%</td>
<td>15.810</td>
<td>&lt;0.001</td>
<td>0.326</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>778</td>
<td>1.51 (0.98)</td>
<td>1.32 (0.71)</td>
<td>-0.19</td>
<td>-12.5%</td>
<td>6.749</td>
<td>&lt;0.001</td>
<td>0.219</td>
</tr>
<tr>
<td>Fasting plasma glucose (mmol/L)</td>
<td>772</td>
<td>5.55 (1.49)</td>
<td>5.24 (1.11)</td>
<td>-0.31</td>
<td>-5.6%</td>
<td>7.601</td>
<td>&lt;0.001</td>
<td>0.238</td>
</tr>
</tbody>
</table>

SD – Standard deviation.
Changes in risk factors from CHIP participation: group overview

As shown in Table 4.1, significant reductions were recorded in all the biometrics over the 30-days of the CHIP lifestyle education program with the largest changes occurring in LDL, TC and TC. The mean percentages for these changes ranged from a high of -17.9% for LDL to a low of -3.8% for BMI. While HDL also decreased following the intervention, the TC to HDL ratio improved. Cholesterol and LDL had a moderate effect size as shown by Cohen's d while all the other biometrics had a small effect size.

It is evident from these results that the volunteer delivered CHIP program was successful in reducing CVD risk factors for the CHIP participants.

Changes in risk factors for CHIP participants classified according to initial risk factors: group overview

Table 4.2 shows the changes in CVD risk factors categories after the 30 day CHIP intervention, when the participants were stratified according to their risk factor status at program entry. Conventional risk factor categories were used to stratify the data, as outlined in Chapter 3. The distributions of the changes in the respective biometrics were checked for normality and then a paired t - test was performed on each risk factor category.
Table 4.2 Mean changes in CVD risk factors in 30 days according to initial risk factor classification.

<table>
<thead>
<tr>
<th></th>
<th>N Baseline</th>
<th>N Post-intervention</th>
<th>Baseline Mean (SD)</th>
<th>Post-intervention Mean (SD)</th>
<th>Mean Change</th>
<th>% Mean Change</th>
<th>( t ) statistic</th>
<th>p</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Body mass index (kg/m(^2))</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18.5 - 24.9</td>
<td>137</td>
<td>168</td>
<td>22.7 1.6</td>
<td>22.2 1.6</td>
<td>-0.6</td>
<td>-2.5%</td>
<td>10.932</td>
<td>&lt;0.001</td>
<td>0.313</td>
</tr>
<tr>
<td>25 – 30</td>
<td>216</td>
<td>234</td>
<td>27.5 1.4</td>
<td>26.5 1.4</td>
<td>-1.0</td>
<td>-4.8%</td>
<td>24.045</td>
<td>&lt;0.001</td>
<td>0.714</td>
</tr>
<tr>
<td>&gt; 30</td>
<td>350</td>
<td>301</td>
<td>39.9 7.0</td>
<td>35.5 6.9</td>
<td>-1.4</td>
<td>-3.8%</td>
<td>30.917</td>
<td>&lt;0.001</td>
<td>0.201</td>
</tr>
<tr>
<td><strong>Systolic blood pressure (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;120</td>
<td>189</td>
<td>312</td>
<td>112.9 7.8</td>
<td>114.2 11.4</td>
<td>1.3</td>
<td>1.2%</td>
<td>-1.565</td>
<td>0.119</td>
<td>-0.134</td>
</tr>
<tr>
<td>120-139</td>
<td>314</td>
<td>297</td>
<td>130.2 4.9</td>
<td>125.1 12.6</td>
<td>-5.1</td>
<td>-3.9%</td>
<td>7.175</td>
<td>&lt;0.001</td>
<td>0.533</td>
</tr>
<tr>
<td>140-160</td>
<td>226</td>
<td>151</td>
<td>148.9 6.7</td>
<td>136.5 15.5</td>
<td>-12.4</td>
<td>-8.3%</td>
<td>12.359</td>
<td>&lt;0.001</td>
<td>1.047</td>
</tr>
<tr>
<td>&gt;160</td>
<td>58</td>
<td>27</td>
<td>177.1 12.8</td>
<td>147.5 16.5</td>
<td>-29.7</td>
<td>-16.8%</td>
<td>4.088</td>
<td>&lt;0.001</td>
<td>2.017</td>
</tr>
<tr>
<td><strong>Diastolic blood pressure (mmHg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;80</td>
<td>349</td>
<td>446</td>
<td>70.1 6.5</td>
<td>70.2 8.9</td>
<td>0.1</td>
<td>0.1%</td>
<td>-0.161</td>
<td>0.872</td>
<td>-0.013</td>
</tr>
<tr>
<td>80-89</td>
<td>277</td>
<td>258</td>
<td>82.9 2.8</td>
<td>78.2 8.5</td>
<td>-4.6</td>
<td>-5.5%</td>
<td>8.748</td>
<td>&lt;0.001</td>
<td>0.743</td>
</tr>
<tr>
<td>90-100</td>
<td>133</td>
<td>63</td>
<td>93.5 3.6</td>
<td>84.2 9.7</td>
<td>-9.4</td>
<td>-10.1%</td>
<td>10.941</td>
<td>&lt;0.001</td>
<td>1.271</td>
</tr>
<tr>
<td>&gt;100</td>
<td>28</td>
<td>20</td>
<td>108.8 5.3</td>
<td>93.7 10.9</td>
<td>-15.1</td>
<td>-13.9%</td>
<td>7.103</td>
<td>&lt;0.001</td>
<td>1.762</td>
</tr>
<tr>
<td><strong>Total cholesterol (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 4.00</td>
<td>93</td>
<td>268</td>
<td>3.49 0.4</td>
<td>3.23 0.55</td>
<td>-0.26</td>
<td>-7.4%</td>
<td>5.601</td>
<td>&lt;0.001</td>
<td>0.541</td>
</tr>
<tr>
<td>4.00–5.20</td>
<td>334</td>
<td>371</td>
<td>4.62 0.37</td>
<td>4.03 0.59</td>
<td>-0.59</td>
<td>-12.7%</td>
<td>19.803</td>
<td>&lt;0.001</td>
<td>1.198</td>
</tr>
<tr>
<td>5.21–5.99</td>
<td>172</td>
<td>94</td>
<td>5.59 0.21</td>
<td>4.74 0.63</td>
<td>-0.85</td>
<td>-15.2%</td>
<td>18.087</td>
<td>&lt;0.001</td>
<td>1.810</td>
</tr>
<tr>
<td>6.00–6.99</td>
<td>143</td>
<td>40</td>
<td>6.39 0.29</td>
<td>5.23 0.74</td>
<td>-1.16</td>
<td>-18.2%</td>
<td>18.026</td>
<td>&lt;0.001</td>
<td>2.064</td>
</tr>
<tr>
<td>&gt;7.00</td>
<td>37</td>
<td>6</td>
<td>7.62 0.49</td>
<td>6.01 0.99</td>
<td>-1.61</td>
<td>-21.1%</td>
<td>9.234</td>
<td>&lt;0.001</td>
<td>2.061</td>
</tr>
<tr>
<td></td>
<td>N Baseline</td>
<td>N Post-intervention</td>
<td>Baseline (SD)</td>
<td>Post-intervention (SD)</td>
<td>Mean Change</td>
<td>% Mean Change</td>
<td>t statistic</td>
<td>p</td>
<td>Cohen’s d</td>
</tr>
<tr>
<td>--------------------------</td>
<td>------------</td>
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<td>---------------</td>
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<td>-------------</td>
<td>---------------</td>
<td>-------------</td>
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</tr>
<tr>
<td><strong>Low density lipoprotein (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;2.50</td>
<td>215</td>
<td>381</td>
<td>2.06 0.41</td>
<td>1.80 0.50</td>
<td>-0.26</td>
<td>-12.6%</td>
<td>9.273</td>
<td>&lt;0.001</td>
<td>0.569</td>
</tr>
<tr>
<td>2.50-2.99</td>
<td>140</td>
<td>171</td>
<td>2.79 0.14</td>
<td>2.39 0.48</td>
<td>-0.40</td>
<td>-14.3%</td>
<td>9.818</td>
<td>&lt;0.001</td>
<td>1.131</td>
</tr>
<tr>
<td>3.00-4.00</td>
<td>271</td>
<td>181</td>
<td>3.49 0.29</td>
<td>2.84 0.58</td>
<td>-0.64</td>
<td>-18.3%</td>
<td>19.217</td>
<td>&lt;0.001</td>
<td>1.418</td>
</tr>
<tr>
<td>&gt;4.00</td>
<td>149</td>
<td>42</td>
<td>4.55 0.45</td>
<td>3.50 0.72</td>
<td>-1.05</td>
<td>-23.1%</td>
<td>17.967</td>
<td>&lt;0.001</td>
<td>1.789</td>
</tr>
<tr>
<td><strong>High density lipoprotein (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1.00</td>
<td>147</td>
<td>201</td>
<td>0.86 0.10</td>
<td>0.85 0.12</td>
<td>-0.01</td>
<td>-1.2%</td>
<td>0.605</td>
<td>0.546</td>
<td>0.091</td>
</tr>
<tr>
<td>1.00-1.55</td>
<td>439</td>
<td>470</td>
<td>1.26 0.16</td>
<td>1.17 0.19</td>
<td>-0.09</td>
<td>-7.1%</td>
<td>12.307</td>
<td>&lt;0.001</td>
<td>0.512</td>
</tr>
<tr>
<td>&gt;1.55</td>
<td>193</td>
<td>108</td>
<td>1.81 0.23</td>
<td>1.58 0.29</td>
<td>-0.24</td>
<td>-13.3%</td>
<td>12.869</td>
<td>&lt;0.001</td>
<td>0.879</td>
</tr>
<tr>
<td><strong>Triglycerides (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1.00</td>
<td>233</td>
<td>282</td>
<td>0.75 0.15</td>
<td>0.82 0.29</td>
<td>0.08</td>
<td>10.7%</td>
<td>-4.718</td>
<td>&lt;0.001</td>
<td>-0.303</td>
</tr>
<tr>
<td>1.00-2.25</td>
<td>433</td>
<td>428</td>
<td>1.47 0.33</td>
<td>1.33 0.48</td>
<td>-0.15</td>
<td>-10.2%</td>
<td>6.764</td>
<td>&lt;0.001</td>
<td>0.340</td>
</tr>
<tr>
<td>&gt;2.25</td>
<td>112</td>
<td>68</td>
<td>3.21 1.47</td>
<td>2.31 0.98</td>
<td>-0.91</td>
<td>-28.3%</td>
<td>5.914</td>
<td>&lt;0.001</td>
<td>0.720</td>
</tr>
<tr>
<td><strong>Fasting plasma glucose (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;5.60</td>
<td>530</td>
<td>605</td>
<td>4.91 0.42</td>
<td>4.87 0.56</td>
<td>-0.04</td>
<td>-0.8%</td>
<td>1.834</td>
<td>0.067</td>
<td>0.081</td>
</tr>
<tr>
<td>5.60-7.00</td>
<td>177</td>
<td>129</td>
<td>6.02 0.39</td>
<td>5.53 0.55</td>
<td>-0.48</td>
<td>-8.0%</td>
<td>12.208</td>
<td>&lt;0.001</td>
<td>1.028</td>
</tr>
<tr>
<td>&gt;7.00</td>
<td>65</td>
<td>38</td>
<td>9.47 2.28</td>
<td>7.42 2.27</td>
<td>-2.05</td>
<td>-21.6%</td>
<td>5.563</td>
<td>&lt;0.001</td>
<td>0.901</td>
</tr>
</tbody>
</table>
The 30-day CHIP intervention had very little effect on those participants who had normal levels of HDL, FPG, DBP and SBP at baseline. However, there were significant reductions in all other biometrics across all risk factor categories (p<0.001). The only increase observed over the 30 days was in TG among participants with normal levels at baseline. For all other participants, with the exception of BMI, those with the greatest risk at baseline experienced the greatest improvements during the intervention. Noteworthy, participants with the highest risk categories at baseline for TC, LDL, FPG and TG experienced over 20% decreases in these measures over the 30 days.

**Discussion**

The Australasian CHIP facilitated by volunteers produced substantial reductions in the CVD risk factors examined. As can be seen in Table 4.3, outcomes achieved by this Australasian study were similar to the recent study on over 5,000 CHIP participants in the United States. The results of this study indicate that the CHIP lifestyle education program, when delivered by volunteers, can improve participants’ CVD risk factors. Importantly, those participants at greatest risk experience the greatest improvements.
Table 4.3 Comparison of the mean changes in selected chronic disease risk factors from baseline to post intervention between Australia and US participants.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Australasian CHIP (Percentage change)</th>
<th>US CHIP (Percentage change)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index</td>
<td>-3.8%</td>
<td>-3.2%</td>
</tr>
<tr>
<td>Systolic Blood Pressure</td>
<td>-5.6%</td>
<td>-4.9%</td>
</tr>
<tr>
<td>Diastolic Blood Pressure</td>
<td>-4.6%</td>
<td>-5.3%</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>-14.7%</td>
<td>-11.0%</td>
</tr>
<tr>
<td>Low Density Lipoprotein</td>
<td>-17.9%</td>
<td>-13.0%</td>
</tr>
<tr>
<td>High Density Lipoprotein</td>
<td>-8.3%</td>
<td>-8.6%</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>-12.5%</td>
<td>-7.7%</td>
</tr>
<tr>
<td>Fasting Plasma Glucose</td>
<td>-5.6%</td>
<td>-6.1%</td>
</tr>
</tbody>
</table>

(Rankin et al., 2012).

In 30 days there was over 20% improvement in the participants from the highest risk categories, (TC 21%, LDL 23%, TG 28% and FPG 22%). These results have exceeded the expectations for lowering blood lipids in health studies (Tang, Armitage, & Lancaster, 1998) and the reductions in TC and LDL in this study have been found to be comparable to the treatment of these blood lipids with statins (Gould et al., 2007).

While the reductions in TC and LDL are beneficial, the reductions in HDL may appear to be detrimental for CVD. HDL has been viewed as cardio protective; however, this has recently come under scrutiny. A recent meta analysis revealed that increasing levels of circulating HDL does not reduce the risk of coronary disease events, coronary heart disease deaths, or total deaths (Briel et al., 2009). Other studies have also found that plant-based diets typically lower HDL (Ferdowsian & Barnard, 2009), yet despite decreased HDL levels a reduction in atherosclerotic plaque was still observed (Ornish et al 1990). Further research is needed to clarify the role of HDL as well as to understand more fully the implications of
decreased HDL levels commonly observed when individuals move towards a plant-based eating pattern.

The other possible counter productive result was the slight rise in TG levels in those with normal levels (less than 1mmol/L) at baseline. Diehl (1988) found similar results with participants on a low fat diet, but described these changes as of minor clinical significance.

Improvements in the Australasian CHIP participants’ biometrics resulted in lower risk factor classifications for the participants. As shown in Table 4.2, only 6 of the 37 individuals who initially had cholesterol levels above 7mmol/L maintained these levels at the end of the 30-day program. Overall a total of 73 CHIP participants were able to reduce their levels below 4mmol/L in three weeks. This is clinically significant, as CVD has been found to be practically non-existent when cholesterol levels fall below 3.88mmol/L (Connor et al., 1978; Esselstyn, 1999; Sinett & Whyte, 1973).

Several studies have demonstrated the potential for reduction of CVD factors through lifestyle intervention, mainly through the adoption of a whole food, plant based diet (Esselstyn, 1999; Ornish et al.,1990b). Similarly the role of a plant-based diet on the reversal of type II diabetes has also been demonstrated (Anderson & Ward, 1979; O’Dhea, 1984).

Barnard et al (1994) also found that after a 26 day residential program involving a low fat near vegetarian diet and exercise regime, 40% of individuals with type II diabetes could discontinue insulin usage. From 65 participants, 27 (over 40%) with FPG levels indicative of diabetes at pre intervention reduced their FPG levels below 7mmol/L. This observation is also comparable to the CHIP study with over 5,000 participants’ in the United States (Rankin et al., 2012).
The reduction in the biometrics as a result of this 30-day CHIP intervention achieved clinically meaningful outcomes. Manson, Tosteson & Ridker, (1992) found that coronary risk declines by 3% for every 1% reduction in serum cholesterol. In the present study this translates to a 63% reduced coronary risk for those participants who entered the program in the highest TC risk category.

According to cholesterol lowering studies, a 1% reduction in LDL results in a 1 – 1.5% decrease in the risk of a cardiac event (Assmann et al., 1998; Wilson et al., 1991), thus a possible decrease in the chance of a cardiac event by 23 – 34.5% in those participants’ in the highest risk category for LDL (Ferdowsian & Barnard, 2009; Ornish et al., 1990b).

There was a DBP reduction of 15.1 mmHg for those in the highest risk category. Manson et al (1992) states that for every 1 mmHg reduction in elevated DBP the risk is reduced by 2-3%, thus a coronary risk reduction of 30 – 45%.

The results of this study support other studies in its deduction that lifestyle factors, such as diet and exercise, can be associated with significant improvements in CVD risk factors (Ornish et al., 1998; Roberts & Barnard, 2005; Unal, Crithley, & Capewell, 2005).

A unique element of this health education program is the use of trained volunteers instead of professionals to facilitate the program. There is very little literature that discusses the success of volunteers as health education program facilitators. It has been established that volunteers can be successful in motivating their peers to action programs (Kong, 1997). The results of this study indicate that the use of volunteer facilitators is a cost effective and successful way of delivering the CHIP program to the wider community.

**Conclusion**

The results of this Australasian-based study of the CHIP intervention indicate that clinically significant decreases in CVD risk factors can be achieved in 30 days. Participants at highest risk tended to achieve the best outcomes. The results of this study were similar to data from the cohort of 5,000 CHIP participants from the United States. It seems therefore,
that the CHIP lifestyle education program may be a valuable asset in reducing the burden of CVD in Australasia. These results are related to short term outcomes, 30 days, and there is a need to assess the effectiveness of CHIP long term.
Chapter 5: The influence of gender, age and marital status on the effectiveness of the CHIP lifestyle education program

Introduction

This chapter explores the influence of gender, age and marital status on the participants’ responsiveness to the CHIP lifestyle education program which addresses research question 2: How do age, gender and marital status influence the responsiveness of participants to the CHIP lifestyle education program? For each of these demographics, ANOVAs were used to examine differences in the participants’ baseline biometric risk factors as well as changes in these measures over the 30-day intervention. Regression analyses were also performed using the biometric risk factors as dependent variables and gender and age as independent variables, along with baseline BMI, BMI change, initial biometric at baseline entry, family history of cardiovascular disease mortality and family history of diabetes mellitus.

Gender

Of the 836 participants, 265 were males (35.2%) and 488 were female (64.8%). There were no significant differences between the males and females at program entry in BMI and FPG. However, the males had slightly higher TG, SBP and DBP at baseline. These differences were relatively small (Cohen’s d values were lower than 0.4) and the mean values for the males and females were still in the same risk category at baseline. The females had significantly higher TC, LDL and HDL than the males at program entry, but once again the mean values for the males and females were within the same risk category.

As shown in Table 5.1, the males had greater changes during the 30-days of the CHIP intervention compared to the females in the majority of the biometrics. The differences between the changes achieved by the males and females are illustrated graphically in
Figures 5.1 and 5.2. The males had larger changes in BMI, TG and despite the males entering the program with lower LDL and TC levels, they had significantly greater changes in these biometrics compared to the females. The greatest percentage changes for both the males and females were seen in LDL. Other noteworthy changes within the males were the 20% decrease in TG and 18% reduction in TC. The effect sizes for the change in LDL, TG and TC for the males and the females were small. The females had a larger change in HDL, but the effect size for this difference was also small (Cohen’s d = 0.21). There were no significant differences in SBP, DBP, and FPG between male and the female participants.
Table 5.1 Changes between male and female biometrics from program entry to post-intervention.

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Males change Mean (SD)</th>
<th>N</th>
<th>Females change Mean (SD)</th>
<th>% change male</th>
<th>% change female</th>
<th>t statistic</th>
<th>p value</th>
<th>Cohen's d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index (kg/m²)</td>
<td>248</td>
<td>1.30 0.9</td>
<td>470</td>
<td>1.05 0.76</td>
<td>4.2%</td>
<td>3.3%</td>
<td>3.91</td>
<td>&lt;0.001</td>
<td>0.300</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>245</td>
<td>8.43 16.57</td>
<td>465</td>
<td>7.10 15.34</td>
<td>6.1%</td>
<td>5.3%</td>
<td>1.31</td>
<td>0.192</td>
<td>0.08</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>245</td>
<td>3.88 9.74</td>
<td>465</td>
<td>3.75 9.82</td>
<td>4.7%</td>
<td>4.8%</td>
<td>1.67</td>
<td>0.867</td>
<td>0.014</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>244</td>
<td>0.89 0.71</td>
<td>459</td>
<td>0.72 0.71</td>
<td>18.2%</td>
<td>13.6%</td>
<td>3.08</td>
<td>0.002</td>
<td>0.24</td>
</tr>
<tr>
<td>Low density lipoprotein (mmol/L)</td>
<td>243</td>
<td>0.68 0.58</td>
<td>457</td>
<td>0.53 0.62</td>
<td>22.3%</td>
<td>16.4%</td>
<td>3.03</td>
<td>0.003</td>
<td>0.25</td>
</tr>
<tr>
<td>High density lipoprotein (mmol/L)</td>
<td>244</td>
<td>0.09 0.17</td>
<td>459</td>
<td>0.13 0.21</td>
<td>8.0%</td>
<td>9.2%</td>
<td>-2.60</td>
<td>0.010</td>
<td>0.21</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>244</td>
<td>0.32 0.92</td>
<td>458</td>
<td>0.13 0.71</td>
<td>20.0%</td>
<td>9.0%</td>
<td>3.13</td>
<td>0.004</td>
<td>0.23</td>
</tr>
<tr>
<td>Fasting plasma glucose (mmol/L)</td>
<td>240</td>
<td>0.36 1.07</td>
<td>454</td>
<td>0.28 0.96</td>
<td>6.4%</td>
<td>5.1%</td>
<td>0.92</td>
<td>0.357</td>
<td>0.079</td>
</tr>
</tbody>
</table>
Figure 5.1 Percentage change in blood lipid profile for the males and females.

Figure 5.2 Percentage change in FPG, BMI and BP for the males and females.
Age

To examine the influence of age on the participants' responsiveness to the 30-day intervention, the participants were categorised into the following age groupings: less than 40 years, 40 – 49 years, 50 – 59 years, 60 – 69 years and 70 years and older.

At baseline there were no significant differences between the respective age categories in BMI, HDL, TG, and DBP. There were significant differences in TC, LDL and FPG between the <39 years age group and the 50 – 59 years age group. There were also significant differences (p< 0.001) in SBP between all of the age categories except for the <39 years and the 40 – 49 years.

Table 5.2 shows the differences in the changes in the biometric risk factors for the different age categories. The 50 -59 year age group had the greatest changes in systolic BP, TC, FPG, BMI, LDL and HDL. According to Cohen's d the change in TC and LDL experienced by the 50 – 59 years age group was a large change. Although the 50 -59 year age group had larger changes, they were only significantly different to the other age categories in SBP, TC, LDL, and HDL. The 50 -59 years age group had a significantly greater change in TC, LDL and HDL than the <39 years group and in SBP than the 40 – 49 years age category. The 40 – 49 years age group had the largest changes in TG, and the 70 or older age group had the largest changes in DBP. The differences in the percentage biometric changes across age categories can be seen in Figures 5.3 and 5.4.
Table 5.2 Differences in biometric change across age

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Age categories</th>
<th>Number in each category</th>
<th>Pre - intervention Mean (SD)</th>
<th>Post intervention Mean (SD)</th>
<th>Mean change (SD)</th>
<th>Percentage mean change (%)</th>
<th>t</th>
<th>p</th>
<th>Cohen’s d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index (kg/m²)</td>
<td>1(&lt;39yrs)</td>
<td>69</td>
<td>31.83 8.77</td>
<td>30.75 8.62</td>
<td>1.09 0.98</td>
<td>-3.4%</td>
<td>9.203</td>
<td>&lt;0.001</td>
<td>0.11</td>
</tr>
<tr>
<td></td>
<td>2(40-49)</td>
<td>112</td>
<td>31.62 8.39</td>
<td>30.52 8.07</td>
<td>1.09 0.73</td>
<td>-3.4%</td>
<td>15.780</td>
<td>&lt;0.001</td>
<td>0.12</td>
</tr>
<tr>
<td></td>
<td>3(50-59)</td>
<td>175</td>
<td>32.03 8.82</td>
<td>30.60 8.37</td>
<td>1.28 0.94</td>
<td>-4.0%</td>
<td>18.030</td>
<td>&lt;0.001</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>4(60-69)</td>
<td>210</td>
<td>30.42 5.63</td>
<td>29.29 5.33</td>
<td>1.14 0.75</td>
<td>-3.7%</td>
<td>21.943</td>
<td>&lt;0.001</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td>5(70+)</td>
<td>89</td>
<td>29.65 6.22</td>
<td>28.65 6.08</td>
<td>0.99 0.63</td>
<td>-3.3%</td>
<td>14.857</td>
<td>&lt;0.001</td>
<td>0.16</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>1(&lt;39yrs)</td>
<td>67</td>
<td>123.31 15.89</td>
<td>119.49 17.48</td>
<td>3.82 12.42</td>
<td>-3.1%</td>
<td>2.518</td>
<td>0.014</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>2(40-49)</td>
<td>110</td>
<td>127.56 18.26</td>
<td>123.32 15.78</td>
<td>4.25 15.08</td>
<td>-3.3%</td>
<td>2.953</td>
<td>0.004</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>3(50-59)</td>
<td>174</td>
<td>135.27 18.69</td>
<td>125.69 15.16</td>
<td>9.56 13.82</td>
<td>-7.1%</td>
<td>9.120</td>
<td>&lt;0.001</td>
<td>0.56</td>
</tr>
<tr>
<td></td>
<td>4(60-69)</td>
<td>208</td>
<td>138.19 18.150</td>
<td>129.13 16.59</td>
<td>9.13 16.44</td>
<td>-6.6%</td>
<td>8.012</td>
<td>&lt;0.001</td>
<td>0.52</td>
</tr>
<tr>
<td></td>
<td>5(70+)</td>
<td>88</td>
<td>144.58 20.128</td>
<td>135.27 15.30</td>
<td>8.76 18.71</td>
<td>-6.1%</td>
<td>4.93</td>
<td>&lt;0.001</td>
<td>0.52</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>1(&lt;39yrs)</td>
<td>67</td>
<td>78.00 11.56</td>
<td>74.94 11.47</td>
<td>3.06 8.64</td>
<td>-4.0%</td>
<td>2.900</td>
<td>0.005</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>2(40-49)</td>
<td>110</td>
<td>80.42 12.18</td>
<td>77.69 11.70</td>
<td>2.73 9.13</td>
<td>-3.9%</td>
<td>3.133</td>
<td>0.002</td>
<td>0.26</td>
</tr>
<tr>
<td></td>
<td>3(50-59)</td>
<td>174</td>
<td>81.84 11.61</td>
<td>77.73 9.97</td>
<td>3.93 9.50</td>
<td>-4.8%</td>
<td>5.457</td>
<td>&lt;0.001</td>
<td>0.34</td>
</tr>
<tr>
<td></td>
<td>4(60-69)</td>
<td>208</td>
<td>80.52 11.45</td>
<td>75.49 11.04</td>
<td>5.02 10.23</td>
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<td>7.086</td>
<td>&lt;0.001</td>
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</tr>
<tr>
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<td>5(70+)</td>
<td>88</td>
<td>79.23 12.46</td>
<td>73.86 11.28</td>
<td>5.36 9.87</td>
<td>-6.8%</td>
<td>5.098</td>
<td>&lt;0.001</td>
<td>0.45</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>1(&lt;39yrs)</td>
<td>67</td>
<td>4.84 1.05</td>
<td>4.24 1.01</td>
<td>0.59 0.72</td>
<td>-12.7%</td>
<td>6.721</td>
<td>&lt;0.001</td>
<td>0.56</td>
</tr>
<tr>
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<td>2(40-49)</td>
<td>105</td>
<td>5.02 1.02</td>
<td>4.20 0.76</td>
<td>0.82 0.73</td>
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<td>-15.9%</td>
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<td>Risk Factor</td>
<td>Age categories</td>
<td>Number in each category</td>
<td>Pre - intervention Mean (SD)</td>
<td>Post Intervention Mean (SD)</td>
<td>Mean change (SD)</td>
<td>Percentage mean change</td>
<td>t</td>
<td>p</td>
<td>Cohen's d</td>
</tr>
<tr>
<td>-----------------------------------</td>
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</tr>
<tr>
<td>Low density lipoprotein (mmol/L)</td>
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<tr>
<td>1(&lt;39yrs)</td>
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<td>2.90 0.91</td>
<td>2.51 0.84</td>
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<td>0.73</td>
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<td>0.72 0.60</td>
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<td>0.89</td>
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<td>0.63 0.60</td>
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<td>15.147</td>
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<td>High density lipoprotein (mmol/L)</td>
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</tr>
<tr>
<td>1(&lt;39yrs)</td>
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<td>0.72 0.19</td>
<td>1.22 0.34</td>
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<td>1.20 0.35</td>
<td>0.07 0.26</td>
<td>-5.5%</td>
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<td>0.006</td>
<td>0.18</td>
</tr>
<tr>
<td>3(50-59)</td>
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<td>1.32 0.35</td>
<td>1.19 0.29</td>
<td>0.13 0.18</td>
<td>-10.6%</td>
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<td>1.18 0.30</td>
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<td>-9.2%</td>
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<td>1.35 0.33</td>
<td>1.24 0.33</td>
<td>0.11 0.17</td>
<td>-8.1%</td>
<td></td>
<td>5.939</td>
<td>&lt;0.001</td>
<td>0.33</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1(&lt;39yrs)</td>
<td>67</td>
<td>1.42 1.01</td>
<td>1.17 0.71</td>
<td>0.24 0.77</td>
<td>-17.0%</td>
<td></td>
<td>2.612</td>
<td>0.011</td>
<td>0.27</td>
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<td>2(40-49)</td>
<td>105</td>
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<td>0.29 1.10</td>
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<td>2.665</td>
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<td>3(50-59)</td>
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<td>1.36 0.72</td>
<td>0.15 0.62</td>
<td>-9.9%</td>
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<td>3.205</td>
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<td>1.30 0.63</td>
<td>0.21 0.93</td>
<td>-13.9%</td>
<td></td>
<td>3.178</td>
<td>0.002</td>
<td>0.24</td>
</tr>
<tr>
<td>5(70+)</td>
<td>88</td>
<td>1.39 0.64</td>
<td>1.24 0.48</td>
<td>0.16 0.45</td>
<td>-11.5%</td>
<td></td>
<td>3.256</td>
<td>0.002</td>
<td>0.27</td>
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<tr>
<td>Fasting plasma glucose (mmol/L)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1(&lt;39yrs)</td>
<td>67</td>
<td>5.09 1.23</td>
<td>4.76 0.62</td>
<td>0.32 1.26</td>
<td>-6.3%</td>
<td></td>
<td>2.107</td>
<td>0.039</td>
<td>0.34</td>
</tr>
<tr>
<td>2(40-49)</td>
<td>105</td>
<td>5.42 1.56</td>
<td>5.10 1.08</td>
<td>0.39 1.19</td>
<td>-7.2%</td>
<td></td>
<td>3.389</td>
<td>0.001</td>
<td>0.24</td>
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<td>3(50-59)</td>
<td>172</td>
<td>5.79 1.82</td>
<td>5.29 1.00</td>
<td>0.50 1.37</td>
<td>-8.6%</td>
<td></td>
<td>4.826</td>
<td>&lt;0.001</td>
<td>0.34</td>
</tr>
<tr>
<td>4(60-69)</td>
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<td>5.67 1.49</td>
<td>5.36 1.16</td>
<td>0.31 1.01</td>
<td>-5.4%</td>
<td></td>
<td>4.431</td>
<td>&lt;0.001</td>
<td>0.23</td>
</tr>
<tr>
<td>5(70+)</td>
<td>89</td>
<td>5.38 0.91</td>
<td>5.17 0.80</td>
<td>0.22 0.72</td>
<td>-4.0%</td>
<td></td>
<td>2.842</td>
<td>0.006</td>
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</table>
Figure 5.3 Percentage changes in blood lipid profile across age categories.

Figure 5.4 Percentage changes in FPG, BMI and blood pressure across age categories
Marital status

The participants were classified according to their marital status into single, married, divorced or widowed categories. There were 63 (9%) CHIP participants who were single, 519 (77.9%) married, 46 (6.9%) divorced and 38 (5.7%) widowed.

At program entry, there was no significant difference in TG, FPG, BMI and HDL between the various marital status categories. There were significant differences between SBP, DBP, TC and LDL, although the differences were not large enough to be identified through post hoc analysis.

Over the 30-day intervention, the married participants had the largest reductions in BMI, DBP and TG, FPG and HDL. The divorced category had the largest reductions in TC and LDL. The single participants had the greatest reduction in SBP. The percentage differences in biometrics between the marital categories can be seen in Figures 5.5 and 5.6.
Table 5.3 Changes in the participants' biometric risk factors stratified for marital status.

<table>
<thead>
<tr>
<th>Factor</th>
<th>N</th>
<th>Pre intervention Mean (SD)</th>
<th>Post intervention Mean (SD)</th>
<th>Change mean</th>
<th>SD</th>
<th>% mean change</th>
<th>t statistic</th>
<th>p value</th>
<th>Cohen's d</th>
</tr>
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<tbody>
<tr>
<td><strong>Body mass index (kg/m²)</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 single</td>
<td>55</td>
<td>32.35 9.20</td>
<td>31.30 8.78</td>
<td>1.05</td>
<td>0.88</td>
<td>-3.3%</td>
<td>9.120</td>
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<td>503</td>
<td>30.80 7.43</td>
<td>29.63 7.10</td>
<td>1.16</td>
<td>0.80</td>
<td>-3.8%</td>
<td>32.413</td>
<td>&lt;0.001</td>
<td>0.16</td>
</tr>
<tr>
<td>3 divorced</td>
<td>45</td>
<td>30.50 5.97</td>
<td>29.47 5.63</td>
<td>1.03</td>
<td>0.88</td>
<td>-3.4%</td>
<td>7.829</td>
<td>&lt;0.001</td>
<td>0.17</td>
</tr>
<tr>
<td>4 widowed</td>
<td>34</td>
<td>32.00 6.98</td>
<td>31.04 6.73</td>
<td>0.95</td>
<td>0.63</td>
<td>-3.0%</td>
<td>8.762</td>
<td>&lt;0.001</td>
<td>0.14</td>
</tr>
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<td><strong>Systolic blood pressure (mmHg)</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 single</td>
<td>59</td>
<td>128.15 16.20</td>
<td>120.12 15.93</td>
<td>8.03</td>
<td>14.10</td>
<td>-6.3%</td>
<td>4.376</td>
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<td>12.385</td>
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<td>1.76</td>
<td>24.41</td>
<td>-1.3%</td>
<td>0.483</td>
<td>0.632</td>
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<td>34</td>
<td>136.76 21.20</td>
<td>129.74 12.26</td>
<td>7.03</td>
<td>20.51</td>
<td>-5.1%</td>
<td>1.999</td>
<td>0.054</td>
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<td><strong>Diastolic blood pressure (mmHg)</strong></td>
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<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
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<td>59</td>
<td>77.66 10.61</td>
<td>74.47 11.32</td>
<td>3.19</td>
<td>8.96</td>
<td>-4.1%</td>
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<td>-5.0%</td>
<td>9.250</td>
<td>&lt;0.001</td>
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<td>45</td>
<td>79.13 10.99</td>
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<td>10.35</td>
<td>-1.7%</td>
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<td>11.27</td>
<td>-4.5%</td>
<td>1.750</td>
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<td>0.27</td>
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<td>Factor</td>
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<td>Post intervention Mean (SD)</td>
<td>Change mean</td>
<td>SD</td>
<td>% mean change</td>
<td>t statistic</td>
<td>p value</td>
<td>Cohen’s d</td>
</tr>
<tr>
<td>-------------------------------</td>
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</tr>
<tr>
<td><strong>Total cholesterol (mmol/L)</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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<td>59</td>
<td>4.87 (0.96)</td>
<td>4.26 (0.89)</td>
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<td>0.61</td>
<td>-12.7%</td>
<td>7.734</td>
<td>&lt;0.001</td>
<td>0.66</td>
</tr>
<tr>
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<td>496</td>
<td>5.13 (1.10)</td>
<td>4.35 (0.98)</td>
<td>0.79</td>
<td>0.69</td>
<td>-15.4%</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
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<tr>
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<td>-18.5%</td>
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<td>0.66</td>
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<td>-19.4%</td>
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<tr>
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<td>59</td>
<td>1.35 (0.35)</td>
<td>1.25 (0.37)</td>
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<td>0.31</td>
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<td>-9.2%</td>
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<td>3.771</td>
<td>0.001</td>
<td>0.46</td>
</tr>
<tr>
<td><strong>Triglycerides (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 single</td>
<td>59</td>
<td>1.40 (0.84)</td>
<td>1.24 (0.73)</td>
<td>0.16</td>
<td>0.61</td>
<td>-11.4%</td>
<td>2.013</td>
<td>0.049</td>
<td>0.20</td>
</tr>
<tr>
<td>2 married</td>
<td>496</td>
<td>1.52 (1.00)</td>
<td>1.32 (0.69)</td>
<td>0.20</td>
<td>0.79</td>
<td>-13.2%</td>
<td>5.755</td>
<td>&lt;0.001</td>
<td>0.23</td>
</tr>
<tr>
<td>3 divorced</td>
<td>45</td>
<td>1.52 (0.78)</td>
<td>1.45 (0.80)</td>
<td>0.07</td>
<td>0.59</td>
<td>-4.6%</td>
<td>0.818</td>
<td>0.418</td>
<td>0.09</td>
</tr>
<tr>
<td>Factor</td>
<td>N</td>
<td>Pre intervention Mean (SD)</td>
<td>Post intervention Mean (SD)</td>
<td>Change mean</td>
<td>SD</td>
<td>% mean change</td>
<td>t statistic</td>
<td>p value</td>
<td>Cohen’s d</td>
</tr>
<tr>
<td>---------------</td>
<td>----</td>
<td>---------------------------</td>
<td>-----------------------------</td>
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<td>------</td>
<td>---------------</td>
<td>-------------</td>
<td>---------</td>
<td>-----------</td>
</tr>
<tr>
<td>4 widowed</td>
<td>34</td>
<td>1.51</td>
<td>1.57</td>
<td>0.26</td>
<td>1.56</td>
<td>-17.2%</td>
<td>0.974</td>
<td>0.337</td>
<td>0.22</td>
</tr>
<tr>
<td>Fasting plasma glucose (mmol/L)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 single</td>
<td>59</td>
<td>5.50</td>
<td>1.40</td>
<td>5.30</td>
<td>0.20</td>
<td>-3.6%</td>
<td>0.916</td>
<td>0.363</td>
<td>0.12</td>
</tr>
<tr>
<td>2 married</td>
<td>494</td>
<td>5.51</td>
<td>1.36</td>
<td>5.22</td>
<td>0.29</td>
<td>-5.3%</td>
<td>7.276</td>
<td>&lt;0.001</td>
<td>0.24</td>
</tr>
<tr>
<td>3 divorced</td>
<td>45</td>
<td>5.88</td>
<td>2.24</td>
<td>5.28</td>
<td>0.60</td>
<td>-10.2%</td>
<td>1.850</td>
<td>0.071</td>
<td>0.37</td>
</tr>
<tr>
<td>4 widowed</td>
<td>34</td>
<td>5.56</td>
<td>1.43</td>
<td>5.15</td>
<td>0.41</td>
<td>-7.4%</td>
<td>1.830</td>
<td>0.076</td>
<td>0.36</td>
</tr>
</tbody>
</table>
Figure 5.5 Percentage change in blood lipid profile for marital status categories.

Figure 5.6. Percentage change in FPG, BMI and BP for marital status categories.
Predictors of the change in the respective risk factors

Multiple regression analyses, using a backward stepwise approach, was undertaken to explore the potential relationships between the changes in the biometric risk factors and the participants’ age and gender. Marital status was not included in the model as it was neither dichotomous or linear. Family history of associated diseases, the baseline level of the biometric risk factor being examined, and the participants’ baseline and change in BMI were also included in the model.

Initial screening of the data showed some significant correlations (at the 0.01 level) between the independent variables, therefore tests for multi-collinearity were conducted. The tolerance index was calculated for each independent variable and no values were found to be less than 0.10, which suggests that multi-collinearity was not a major concern with this data set (Ho, 2006). Residual analysis was used to test the assumptions required for multiple regression analysis. A normal plot of the regression-standardized residual was generated to test for normality of the residual distribution. The respective residual plots were consistent with the assumption of near normality. Coakes & Steed (2003) have suggested that mild deviations from the multivariate assumptions are not serious. The scatter plot of the regression-standardised residual against the regression standardised predicted value was used to test the assumption of linearity. No obvious patterns of clustering were observed for each of the respective dependent variables (BMI TC, HDL, LDL, TG, SBP, DBP and FGP), supporting the assumption of linearity.

As shown in Table 5.4, the R squared values for the regression models ranged from 22.4% for HDL to 67% for TG. The baseline level of the biometric was the strongest predictor of change for all of the biometrics. BMI was also a strong predictor of change in all of the biometrics except for blood pressure and HDL. However, those participants with the lowest initial BMI levels achieved greater changes in TC, LDL, TG and FPG compared to those participants with higher BMI initial levels.

Gender was also a significant predictor for change in BMI, TC, LDL and HDL. Males
had a larger change in their BMI, TC, TG and LDL levels than females. Age was a significant predictor of change for DBP and FPG. A family history of CVD or diabetes was not a significant predictor of change for any of the biometrics.
Table 5.4 Summary of Predictors of Change for TC, HDL, LDL, TG, FPG, BMI, SBP and DBP.

<table>
<thead>
<tr>
<th>Predictors</th>
<th>BMI</th>
<th>TC</th>
<th>LDL</th>
<th>HDL</th>
<th>TG</th>
<th>FPG</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>R Square</td>
<td>0.225</td>
<td>0.328</td>
<td>0.05</td>
<td>0.224</td>
<td>0.667</td>
<td>0.404</td>
<td>0.344</td>
<td>0.248</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-0.095</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>0.448</td>
<td>-0.154</td>
<td>-0.123</td>
<td>-0.106</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline BMI</td>
<td>-0.186</td>
<td>-0.147</td>
<td>-0.104</td>
<td></td>
<td>-0.253</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI change</td>
<td></td>
<td>-0.285</td>
<td>0.231</td>
<td>0.087</td>
<td>0.124</td>
<td></td>
<td>-0.119</td>
<td></td>
</tr>
<tr>
<td>Initial biometric level at program entry</td>
<td>0.483</td>
<td>0.562</td>
<td>0.500</td>
<td>0.822</td>
<td>0.665</td>
<td>0.498</td>
<td></td>
<td>0.623</td>
</tr>
<tr>
<td>Family history of cardiovascular disease mortality</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.046</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history of diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.075</td>
</tr>
</tbody>
</table>

Note: For gender, a negative standardised beta difference indicates the males had greater changes compared to females.

For age, the negative sign standardised beta indicates that greater changes occurred in the younger age groups.
Discussion

Regardless of gender, age and marital status, the 30-day CHIP lifestyle education program reduced all biometrics across all categories, indicating a reduced chance of CVD for these participants. However, the males, the 50-59 years age category and the married individuals had greater reductions than the other categories in response to the CHIP lifestyle education program.

Gender

The results of the blood tests taken at program entry indicated that the males had higher levels than the females in TG, SBP and DBP; there were no significant differences in BMI and FPG. Changes in the participants’ biometrics from the CHIP intervention are impacted by the initial condition of these participants. The differences between the male and the female initial conditions are minor except for HDL and should not mask the gender difference in change between pre- and post-intervention biometric readings.

Both genders had significant reductions in all biometrics at the conclusion of the CHIP intervention, however the males appeared to have larger reductions than the females. Although the literature is limited in this area, there are some CHIP studies that have also found that males have larger reductions in their biometrics than females after implementation of the CHIP lifestyle changes (Diehl, 1998; Englert et al., 2004; Pischke et al., 2007; Roberts & Barnard, 2005). This may indicate a significant clinical advantage for males in the reduction of risk factors associated with CVD.

As can be seen from the results of this study the men lost weight more quickly in the 30-day intervention. Males appear to have a physiological advantage in fat burning as they have significantly greater muscle mass, which is associated with a faster metabolism (Alway, Grumbt, Gonyea, & Stray-Gundersen, 1989). As was
indicated in the literature review, all of the CVD risk factors are interrelated. For example obesity is related to an elevation in all other CVD risk factors (Gregg et al., 2005; Klein et al., 2004). Thus with the males’ advantage of quicker weight loss they also appear to have the advantage in quicker reductions in other CVD risk factors.

Another study, not related to CHIP has also found men tend to lose weight faster than women and can more quickly reduce other CVD risk factors (Wing & Jeffery, 1995). Interestingly, the regression analysis in this study showed that, although not gender specific, change in BMI was a strong predictor for change in all of the biometrics except for blood pressure and HDL.

There are also differences between men and women in their engagement in exercise and its intensity. It has been found that women generally have less confidence when it comes to exercise, exercise less, have less exercise capacity and lower fitness levels than men (Koertge et al., 2003; Willis et al., 2012). It is possible therefore that the men engaged in more exercise than females in the CHIP programs studied and thus experienced greater benefits. Data relating to exercise levels and intensity has not been recorded in this study but the relationship between gender, weight loss and CVD risk factors may be an area for further investigation.

Men also seem to have some behavioural advantages in that they receive more support from their partner than vice versa (Pischke et al., 2007). It is feasible that the men attended the CHIP intervention with their spouses and therefore benefited from the ensuing household changes made by the women.

Secondly whilst it may take longer for men to commit to lifestyle education programs like the CHIP program, once they do, they appear to be more compliant to the lifestyle changes (Bautista-Castano et al., 2004; Prochaska & DiClemente, 1982).
Age

At baseline the CHIP participants that were under 39 years of age had lower levels of TC, LDL and FPG levels than those between the age of 50 and 59 years. Also at baseline SBP levels increased as age increased.

The 50-59 years age category had significantly greater changes compared to the under 39 years for TC, FPG and LDL. The benefits to the younger age category, under 39 years, are smaller as they have biometrics in the lower risk categories at baseline. By starting the CHIP education program at this younger age they have more chance of maintaining their biometrics in the low risk category. Fraser and colleagues (2001) suggest, that maximum benefit from changes to lifestyle habits are obtained if the changes are made before the age of 30 years (Fraser & Shavlik, 2001). This assertion is supported by Lloyd–Jones et al (2006) who found that the absence of CVD risk factors at 50 years of age is associated with low lifetime risk for CVD. Conversely, there is a high lifetime risk of CVD for those with intermediate or high CVD risk factors at 50 years of age. Thus prevention of CVD risk factors through the CHIP lifestyle education program should be encouraged in young individuals, although significant benefits can be gained at any age.

Marital Status

The married couples in the group appeared to gain quicker reductions over the 30 days in most of their CVD risk factors compared to those in the other categories. Data on the incidence of married couples attending the program together was not recorded, however the informal reports from the CHIP coordinators suggest that the majority of married participants were doing the program as a couple. The divorced group had the largest reductions in TC and LDL, but note they they started out with significantly higher levels at baseline, thus it would be expected that they have greater reductions in the 30 days.
Social and spouse support has been found to increase compliance to lifestyle education programs (Pischke et al., 2007). Other studies have also shown that married couples do better in achieving lifestyle change, and have attributed the improved results of this category to the support offered by the partner (Pischke et al., 2007; Verheijden & Kok, 2005).

Other Predictors of Change

The regression analysis showed that whilst the different gender, age and marital status categories had some effect on the biometrics, by far the biggest impact on the amount of biometric change was the participants initial biometric levels. Those participants in the highest risk category achieved the greatest amount of change. This trend has been demonstrated in professionally delivered CHIP interventions (Aldana et al., 2008; Diehl, 1998; Englert, Dhiel, & Greenlaw, 2004).

The regression models developed in this study explained between 5 and 67 percent of the variance. Clearly, there were other factors not included in the models, particularly dietary and exercise changes that would have a substantive effect on the change in the biometrics. It is a limitation of this study that data on these factors were not included in the study, because this was outside the scope of the project. Further research is needed in this area and more information about the participants’ lifestyle changes throughout the program should be recorded in future studies.

Conclusion

The results of this study indicate that the participants’ age, gender and marital status has an influence on their responsiveness to the 30-day CHIP. The males, the 50 – 59 years of age and the married couples had better outcomes from the program compared to those in other categories.
Chapter 6: Summary and Conclusions

Study limitations

Caution in the interpretation of the findings of this study is necessary because of a number of limitations. Firstly the participants were self-selected. Volunteering to participate in the program indicates an interest in the program and possible readiness to change on the part of the individual. A study may be needed to compare the results conducted of individuals with no prior knowledge or interest in the program to the volunteers’ results achieved in this study.

Secondly, as this study has had no control group, the extent to which regression to the mean explains the observed outcomes cannot be determined. However similar results to the present study were obtained in several professionally delivered randomised control trials of CHIP performed in the United States (Englert et al., 2007). Further study using a randomised control design in Australasia should be undertaken.

Finally, the Hawthorne effect may also have been a confounder of the outcomes observed in this study. The pre- and post-intervention blood tests may have had an influence on the participants’ levels of adherence to the program. Whilst it is likely that these pre- and post-blood measures motivated the participants to comply to the program, it can be argued that these blood tests are a part of the CHIP intervention. Furthermore it would be difficult to measure the effectiveness of a program without the accountability measures.

Summary and conclusions; research question 1

Can a 30-day lifestyle education program (CHIP) improve risk factors for CVD in Australasia?
Significant reductions in CVD risk factors were observed as a result of the 30-day, volunteer-delivered CHIP in Australasia, with those most at risk making the largest reductions. Thus improvements in diet, exercise and other lifestyle factors can reduce CVD risk factor levels into low risk categories. The improvements in the CVD risk factors were comparable to those observed in a large CHIP cohort in the United States.

The results from the educationally intensive CHIP presented by volunteers in Australasia have compared favourably with statin treatments and other drug interventions. Furthermore there are no known significant side effects of a low fat diet.

The use of trained volunteers appears to be a successful method of presenting the program, as results of this CHIP study were not dissimilar to results presented by professionally delivered CHIP interventions. This practice is beneficial in reducing costs of the program and in making it accessible to the community at large.

**Summary and conclusions; research question 2**

How do age, gender and marital status influence the responsiveness of participants to the CHIP lifestyle education program?

Although all of the participants had significant reductions in their biometrics, there were categories that did receive better outcomes in the 30 days compared to others. The males, the 50 -59 years age category and the married couples received better results than those in other categories.

From the literature addressing age and CVD, it appears that 50 – 59 years of age may be an important age for determining lifetime risk of CVD. Although there were benefits to all the age categories in this study, the 50 -59 years of age were
more successful at decreasing their risk factors than the other age categories. More research is needed to establish why this age bracket received better results and whether this age is important to focus on for decreasing one’s lifetime risk of CVD.

The marital category also had larger reductions in their risk factors than the other categories most likely illustrating the importance of support for lifestyle change.

Lack of information on the gender, age and marital status categories is a limiting factor of this study. The differences between gender, age and marital status groups may need further research to ascertain whether different treatment approaches and different treatment intensities may be more suitable to different participant groups.

**Directions for further research**

Despite the limitations of this study, the results indicate that lifestyle education programs such as CHIP may be useful in the management of CVD within the Australasian context. To investigate the long term reductions in the CVD risk factors and the sustainability of the lifestyle choices encouraged by CHIP longitudinal, studies are warranted. In addition an examination of the specific nutrition requirements of the recommended CHIP diet is required. Finally, to optimise the effect of the program on the CVD risk factors of CHIP participants, more research is needed on participant factors and program properties such as structure, content and facilitation.
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