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Multidisciplinary, multiple risk factor cardiovascular disease primary prevention programme in community pharmacy: A feasibility study

A thesis presented in partial fulfilment of the requirements for the degree of

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Abstract

Background: Community pharmacy cardiovascular disease (CVD) primary prevention interventions, led by pharmacists, are effective. However, the majority of these have targeted single CVD risk factors and most have not adequately assessed the impact of dietary and physical activity behaviour. A multidisciplinary and multi-risk factor approach that involves collaboration between dietitians (dietary and physical activity consultations) and pharmacists (pharmacological treatment) may provide additional risk reduction benefits for participants.

Objective: To assess the feasibility of implementing a community pharmacy-based CVD primary prevention programme using a multidisciplinary approach to motivate lifestyle behaviour change in participants at risk of CVD. The primary outcome was change in estimated five-year CVD risk.

Methods: A 16-week single cohort pre- and post-test study was undertaken in two community pharmacies with twelve participants aged 40-74 years who had risk factors associated with increased CVD. Participants received dietary and physical activity advice at baseline and every four weeks by a student dietitian as well as pharmacological management assessment at baseline, 16 weeks and as needed by a pharmacist. Biochemical (blood lipids, blood pressure, HbA1c) and anthropometric (body composition, weight, height, waist and hip circumference) measures were compared at baseline, eight and 16 weeks. Behavioural measures (diet, physical activity and medication use) were compared between baseline and 16 weeks.

Results: Eleven participants (68±5.2 years) completed the programme. Significant reductions from baseline to 16 weeks were observed for mean systolic and diastolic blood pressure (-5.47, $p = 0.04$ and -4.06mmHg, $p = 0.01$ respectively) and mean total cholesterol reduced significantly from baseline to eight weeks, (-0.43mmol/L; $p = 0.005$) but not between baseline and 16 weeks. The average diet quality score significantly improved by 12.6% from 65.9 to 74.2 out of 100 during the intervention period ($p = 0.007$). Other CVD risk factor measures showed a trend towards improvement. Five-year CVD risk did not significantly improve.

Conclusions: Results are comparable to existing literature on interventions to reduce CVD in the community pharmacy setting. Findings within this small cohort, particularly the improvements seen in diet, support the inclusion of dietitians for the primary prevention of CVD in community pharmacies. A larger scale, controlled study will help in determining the extent of efficacy with this approach.

Acknowledgements

Dedicated to my Grandfathers for giving this thesis meaning
and to my Mother for inspiring it all.

Thank you to:

The sun who enlivened me
My family who grounded me
My parents who seeded me
My siblings who staked me
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My teachers who nourished me
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And my van who sheltered me.

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and the participants for their warm hearts.

Contents

Tables

Abbreviations

ADIME: assessment, diagnosis, intervention and monitoring and evaluation
ANOVA: analysis of variance
BIA: bioelectrical impedance analysis
BMI: body mass index
BP: blood pressure
CHD: coronary heart disease
CI: confidence interval
cm: centimeter
CVD: cardiovascular disease
d: day
DASH: Dietary Approaches to Stop Hypertension
DBP: diastolic blood pressure
DHA: docosahexaenoic acid
DM: diabetes mellitus
EPA: eicosapentaenoic acid
g: gram
GP: general practitioner
HbA1c: glycated haemoglobin
HC: hip circumference
HDL-C: high density lipoprotein-cholesterol
HHFI: Healthy Heart Food Index
IHD: ischaemic heart disease
IPAQ: International Physical Activity Questionnaire
kcal: kilocalorie
kg: kilogram
kg/m^2: kilogram per square meter
LDL-C: low density lipoprotein-cholesterol
MET: metabolic equivalent
mm/Hg: millimetre of mercury
mmol/L: millimole per litre
mmol/mol: millimole per mol
MUFA: monounsaturated fatty acids

N: number
NZ: New Zealand
OR: odds ratio
PA: physical activity
PAART: Pharmacist Assessment of Adherence, Risk and Treatment in Cardiovascular Disease study
PUFA: polyunsaturated fatty acids
RR: relative risk
SBP: systolic blood pressure
SD: standard deviation
SFA: saturated fatty acids
TC: total cholesterol
TFA: trans fatty acid
TG: triglycerides
WC: waist circumference
WHR: waist to hip ratio
wk: week

Chapter One

Introduction

Burden of cardiovascular disease in New Zealand

Despite improvements in diagnosis and treatment, cardiovascular disease (CVD) remains a leading cause of morbidity and mortality in New Zealand (NZ). It accounted for 31% of all deaths in 2016 (Ministry of Health, 2019b), and 17% (185,640 disability-adjusted life years) of the total health loss in 2013 (Ministry of Health, 2016b). Further, statistical modelling by Blakely et al. (2019) showed 12.5% ($4,801 million NZD) of the total health expenditure from 2007-2014 could be attributed to CVD. In addition, NZ has high prevalence rates of CVD risk factors including being overweight or obese, high blood pressure, high cholesterol and diabetes (Ministry of Health, 2019c). It is estimated that over one quarter of the NZ population has a 5% or greater five-year risk of CVD, indicating medium to high risk (Pylypchuk et al., 2018).

Cardiovascular disease risk factors

Risk factors for CVD include those that are non-modifiable such as family history, genetics, age, sex, and ethnicity; and those that are modifiable which can be grouped as being metabolic or behavioural (Benjamin, Muntner & Bittencourt, 2019). Metabolic risk factors are comprised of dyslipidaemia, hypertension, elevated blood glucose levels/diabetes, and being overweight or obese (Benjamin et al., 2019). Behavioural risk factors include lifestyle factors such as unhealthy diet, physical inactivity, tobacco smoking and medication misuse (Benjamin et al., 2019). It is estimated that behavioural risk factors contribute up to 80% of the risk of CVD (World Health Organization, 2011).

Behavioural risk factors

Diet

Dietary guidelines aimed at reducing the risk of CVD have begun to emphasise overall dietary patterns and food-based recommendations as opposed to recommendations focused on single nutrients, as has been the traditional approach in nutrition research (Fardet & Rock, 2014; Bhupathiraju & Tucker, 2011; Macini & Stamler, 2004). It is now acknowledged that food and nutrients are intrinsically linked and changes to the overall dietary pattern consumed may have a greater impact on CVD risk compared to a focus on alterations in the consumption of individual nutrients alone (Jacobs & Tapsell, 2007; Hu, 2002; Bowen et al., 2018).

There are a number of examples of cardioprotective eating patterns. These include the Dietary Approaches to Stop Hypertension (DASH) diet (National Heart Lung and Blood Institute, 2018), the Mediterranean diet (Bach-Faig et al., 2011), and dietary guidelines produced by national cardiovascular interested organisations (American Heart Association, 2018b; National Heart Foundation of New Zealand, 2018b). Cardioprotective diets still include recommendations for single nutrients such as saturated fatty acids (SFA), sodium, sugar and alcohol, but they are now placing the majority of their emphasis on the overall dietary pattern. Most cardioprotective diets align with the following dietary recommendations (Krauss et al., 2000; Hu & Willett, 2002; Mente et al., 2009; National Heart Foundation of New Zealand, 2018b; National Heart Lung and Blood Institute, 2018; Bach-Faig et al., 2011):

- Being rich in fruits and vegetables
- Eating wholegrain carbohydrates as opposed to refined carbohydrates, and limiting sugar
- Unsaturated fats as the predominant form of dietary fat, choosing low fat dairy products, adequate omega-3 fatty acids, using vegetable oils as opposed to animal fats, and limiting SFA and trans fatty acids (TFA)
- Choosing lean protein sources, such as fish, legumes and nuts, and reducing processed meats
- Reducing salt intake
- Moderating alcohol intake

- Matching energy intake to overall energy needs.

Adherence to the dietary recommendations above can lead to favourable changes to blood pressure (BP) (Hartley et al., 2013; Mozaffarian & Wu, 2011; He & MacGregor, 2002; Xin et al., 2001), blood lipids (Hartley et al., 2013; Brown et al., 1999; Mozaffarian & Wu, 2011; Mensink et al., 2003) blood glucose levels (Foster-Powell, Holt & Brand-Miller, 2002) and weight (Seal, 2006; Anderson, Smith & Gustafson, 1994; Boeing et al., 2012).

Physical activity

It is recommended to do ≥150 minutes/week of moderate-intensity aerobic activity or 75 minutes/week of vigorous aerobic activity for cardiovascular health (American Heart Association, 2018a; National Heart Foundation of New Zealand, 2018a). Physical activity (PA) has been found to be beneficial for BP (Pescatello et al., 2004; Fagard & Cornelissen, 2007), small weight reductions (Swift et al., 2014), and high density lipoprotein-cholesterol (HDL-C) and triglyceride (TG) levels (Durstine et al., 2001; Kodama et al., 2007). In their meta-analysis Kyu et al. (2016) reported that this recommended level of physical activity is associated with a 16% reduction in the risk of ischaemic heart disease and ischaemic stroke, and that higher activity levels can provide even more benefit.

Medication use

Medications can successfully reduce BP (Ettehad et al., 2016; Law, Morris & Wald, 2009), produce more favourable blood lipids (Baigent et al., 2010), control blood glucose levels and diabetes (Nathan et al., 2009), and aid in smoking cessation (Cahill et al., 2013). Individuals who are at high risk of CVD often take more than one medication to lower their risk with lipid lowering and BP lowering medications being commonly taken (Armstrong et al., 2014). Medication adherence (i.e. taking medications as prescribed), therefore plays an important behavioural role in the prevention of CVD. However, in one meta-analysis, adherence to cardiovascular medications was shown to be only 50% at two years (Naderi, Bestwick & Wald, 2012).

CVD primary prevention

Primary prevention (the prevention of onset) and secondary prevention (the prevention of progression after onset) strategies utilise individualised CVD risk assessment to identify those who are at high risk and to guide risk-reducing interventions (Ministry of Health, 2018a). New Zealand uses an individual's five-year absolute CVD risk assessment, which estimates the likelihood of a cardiovascular event over five years (Pylypchuk et al., 2018; New Zealand Guidelines Group, 2012). Lifestyle changes to modify the behavioural and metabolic risk factors of CVD are recommended no matter the degree of overall CVD risk (Ministry of Health, 2018a). Primary preventative educational and counselling interventions aimed at reducing CVD risk factors through lifestyle changes display the most effectiveness when they are targeted towards individuals who are at high risk (Ebrahim et al., 2011; Alageel et al., 2017).

CVD primary prevention in pharmacy

It can be argued that the community pharmacy setting is well placed for the primary prevention of CVD. Pharmacies are local hubs where the public has easy access to trained health professionals. Internationally, screening for people at risk of CVD within community pharmacies has been successful. This includes the targeting of harder to reach groups such as males, ethnic minorities and lower socio-economic communities (Liu et al., 2008; Donyai & Van den Berg, 2009; Horgan, Blenkinsopp & McManus, 2010; Snella et al., 2006).

Additionally, community pharmacy-based interventions have been effective at managing individual risk factors of CVD. A recent systematic review of 27 studies on such interventions found reductions in systolic BP, glycated hemoglobin (HbA1c), and total cholesterol (TC) (Ifeanyi et al., 2015). However, the reviewed studies tended to focus on single CVD risk factors as opposed to multiple risk factors, the latter of which is known to be more effective (Eckel et al., 2014; Ministry of Health, 2018a). They also varied in study design, type of intervention and participant disease severity. Only a handful of studies that addressed multiple risk factors of CVD in a community pharmacy setting have been undertaken (Krass et al., 2007; McNamara et al., 2012a;

Tsuyuki et al., 2016a; Clifford et al., 2005; Ali et al., 2012). Most of these studies found some improvements for five- or ten-year CVD risk, BP, HbA1c, body mass index (BMI), medication adherence and smoking. Those that included dietary and physical activity interventions focused on adherence to general national dietary and physical activity guidelines or guidelines specific to CVD (McNamara et al., 2012a; Clifford et al., 2005; Krass et al., 2007). It is unclear what the extent of dietary and physical activity training pharmacists received was, how much a focus it was in interventions, and the degree of behaviour change produced. Results from McNamara et al. (2012a) suggest that some improvement can be made in these areas through pharmacist intervention. No interventions in NZ pharmacies have targeted multiple risk factors.

Pharmacists are experts on the pharmacotherapy management of CVD risk factors such as medications that control BP, lipids, thrombosis, diabetes and nicotine addiction. However, their depth of knowledge, expertise and confidence with other modifiable behavioural risk factors such as diet, weight management and physical activity have been reported to be low or less than adequate (Chang et al., 2008; Pearce & Cross, 2013; Newlands, Watson & Lee, 2011; Dastani, Brown & O'Donnell, 2004; Fakih, Marriott & Hussainy, 2015; Dirks-Naylor, Griffiths & Bush, 2018; Persky, 2009). Given that diet, physical activity and weight management all play a significant role in CVD risk, a stronger emphasis on these factors alongside medication management could produce even greater reductions in risk than what has been observed in pharmacist-led interventions. A dietitian's scope of practice is much more aligned to these elements (Andersen et al., 2018; Dietitians Board, 2017). Dietitians can provide comprehensive dietary and anthropometric assessment and individualised dietary and physical activity advice, while a pharmacist can additionally provide medication management to complete a lifestyle intervention. Multidisciplinary approaches that are collaborative and patient-centred have been demonstrated to improve control of chronic diseases (McGill & Felton, 2007; Omboni & Caserini, 2018; Bischoff et al., 2017; Houle, Chatterley & Tsuyuki, 2014; Jennings & Astin, 2017; Taylor et al., 2005; Carter et al., 2009). A multidisciplinary approach to lifestyle change for CVD primary prevention between dietitians and community pharmacists could provide greater efficacy in reducing overall CVD risk. This could be effective in a NZ context due to the high number of people who are at medium-to-high risk of developing CVD.

Purpose

The purpose of this research is to ascertain the feasibility of developing and implementing a community pharmacy-based programme to address the risk of CVD in individuals who are at medium-to-high risk. It will do this by employing a multidisciplinary approach between dietitians and pharmacists to motivate lifestyle change over a 16-week period. Participants will receive lifestyle consultations focusing on diet and physical activity by a student dietitian every four weeks, and medication management by a community pharmacist at the beginning, the end and as needed during the programme. Participants will have risk factors measured and interpreted at baseline, week eight and week 16. These will include estimated five-year CVD risk assessment, BP, lipids, HbA1c, weight and body composition.

Aim

To assess the feasibility of implementing a multidisciplinary CVD primary prevention programme that employs dietitians and pharmacists within a community pharmacy setting.

Primary objective:

- To measure changes in estimated five-year CVD risk between baseline, week eight and week 16 of the intervention.

Secondary objectives:

- To measure changes to metabolic CVD risk factors; BP, lipid profile, HbA1c, and anthropometric measures between baseline, week eight and week 16.
- To measure changes in behavioural CVD risk factors; diet quality, physical activity and medication adherence between baseline and week 16.
- To examine programme related measures; participant recruitment, retention, and evaluation of the programme by participants.

Structure of the thesis

The thesis has been structured into four chapters. Chapter one introduces concepts covered in this study and highlights the purpose of the research. The second chapter is a review of the literature, covering the modifiable risk factors of CVD, the primary prevention of CVD with particular focus on the community pharmacy setting, and identifying the gaps in the literature. Chapter three presents a complete research study manuscript on the implementation of a community pharmacy based CVD primary prevention feasibility study. This includes an abstract, introduction, methods, results, and discussion. The fourth and final chapter details the study conclusions, strengths, limitations and recommendations for future research.

Researcher's contributions

Table 1: Researcher's contributions.

Researcher	Role	Contributions
Dave Alsford	Lead Researcher	Study design, developing study materials, ethics proposal, participant recruitment, programme implementation, dietetic consultations, data collection, analysis, and interpretation, thesis writing.
Dr Cheryl Gammon	Primary Supervisor	Study design, developing and reviewing study materials, reviewing proposals, results interpretation, reviewing thesis and approval.
Dr Kathryn Beck	Secondary Supervisor	Study design, reviewing study materials, reviewing proposals, results interpretation, reviewing thesis.
Nico Bejcek	Research Assistant	Assistance with consultation set up and data collection.

Chapter Two

Literature Review

Introduction

The literature review provides the evidential background for the design, development, implementation and appraisal of a cardiovascular disease primary prevention programme based in the community pharmacy setting. The review investigates the literature relating to the risk factors associated with CVD and the primary prevention approaches that have been employed to reduce CVD risk. Particular focus is placed on the modifiable risk factors for CVD and primary preventative trials and programmes that have been implemented in a community pharmacy setting.

The online databases Web of Science, PubMed and Google Scholar were systematically searched for relevant literature from 1960-2019 as well as manual searches using reference lists of articles to identify other pertinent publications.

Cardiovascular disease

Cardiovascular disease is a general term for a group of conditions that affect the blood circulatory system many of which occur as a result of atherosclerosis, the thickening of the arterial walls (World Health Organization, 2019). These include:

- Hypertension - raised BP higher than defined limits
- Coronary heart disease (CHD) also known as ischaemic heart disease (IHD) - insufficient blood and oxygen flow to the heart muscle
- Cerebrovascular disease - insufficient blood flow to the brain
- Peripheral artery disease – insufficient blood flow to the periphery.

The term also encompasses conditions which result from other causes, such as rheumatic heart disease and heart failure however, for the purpose of this review, only CVD due to atherosclerosis will be discussed.

Cardiovascular disease in New Zealand

Although the rate of death from CVD in NZ has been declining since its peak in the late 1960s, CVD remains a leading cause of morbidity and mortality in NZ. The most recent statistics show that in 2016 CVD accounted for 31% of all deaths (9,806 people) (Ministry of Health, 2019b). Results from the 2017/18 NZ Health Survey estimated that 180,000 all adults have been diagnosed with IHD and 58,000 have had a stroke. The health loss and health cost of CVD in NZ is significant. Health loss in 2013 from CVD (including diabetes) measured in disability-adjusted life years (DALY) is estimated to be 17% (185,640 DALYs) of the total health loss from all causes (Ministry of Health, 2016b). In their report, Blakely et al. (2019) estimate that CVD contributed $4,801 million NZ dollars to NZ public health costs, accounting for 21.2% of the total non-communicable disease public health spend, or 12.5% of total health expenditure.

It is estimated that 80-85% of CVD burden in NZ is attributable to six established risk factors; high blood cholesterol, high BP, smoking, obesity, physical inactivity and diabetes (the former three accounting for at least 70%) (Tobias et al., 2005). From 1980 to 2004 it was estimated that 80% of the decrease in CHD mortality resulted from decreases in population systolic BP, total blood cholesterol and smoking prevalence, with each factor contributing 42, 36 and 22% respectively to this decrease (Tobias et al., 2008). However the most recent NZ health survey showed, a significant proportion of the NZ population have been diagnosed with and take medications for high BP (16.5%), high cholesterol (10.9%) and diabetes (6.5%) (Ministry of Health, 2019c). The survey also estimates that 34.6% of the population are overweight and 32.2% of the population are obese.

Cardiovascular disease risk factors

Risk factors for CVD are conditions or states that increase the chance of developing CVD. Risk factors are either non-modifiable - they cannot be changed, or modifiable - there is the ability to change them. The modifiable risk factors can be further categorised as metabolic or behavioural. Risk factors for CVD are listed in table 2 and the ones relevant to this review will be discussed in more detail below.

Table 2: The cardiovascular disease risk factors.

Non-modifiable	Modifiable		Other
	Metabolic	Behavioural	
Family history and genetics	Dyslipidaemia	Diet	Socioeconomic
Age	Hypertension	Physical activity	Chronic kidney disease
Sex	Elevated blood glucose/diabetes	Medication use	Atrial fibrillation
Ethnicity	Overweight/obesity	Cigarette smoking	Rheumatoid arthritis

Non-modifiable risk factors

Non-modifiable risk factors for CVD are briefly discussed as this review focuses on the modifiable risk factors. Family history of CVD is associated with a one and a half to two times higher risk of CVD events compared to those with no family history. Family history also predicts heritability of CVD risk factors, such as elevated BP and BMI (Lloyd-Jones et al., 2004; Jousilahti et al., 1996; Bertuzzi et al., 2003; Benjamin et al., 2017). With increasing age, independent of the acquisition of other CVD risk factors, CVD risk increases up until about midlife then the risk begins to decrease (Lloyd-Jones et al., 2006; Dhingra & Vasan, 2012). Females may have approximately 20% lower risk than males for cardiovascular outcomes and cardiovascular death and at any age, and females tend to have more CVD related measures at ideal levels than do males (Kappert et al., 2012). The presence of ideal cardiovascular health also varies by ethnicity which could be due to genetic variations or factors in the environment (Forouhi & Sattar, 2006). For example mortality rates per 100,000 are two times higher for Māori compared to non-Māori (191 and 94.2 respectively) (Ministry of Health, 2019b; Riddell & North, 2003).

Modifiable risk factors

Modifiable risk factors can be divided into two groups, metabolic risk factors and behavioural risk factors. Metabolic risk factors refer to changes in the body's normal function via biochemical processes that increase the risk of diseases (Grundy, 2016). Behavioural risk factors are lifestyle habits or actions that pose an increased risk to an individual's health, generally over the long-term. To a certain extent, they may be

under the individual's control. Behavioural risk factors can influence the metabolic risk factors by effecting the biochemical processes in the body, as such, approximately 80% of CHD and CVDs have been linked to behavioural risk factors (World Health Organization, 2011).

Metabolic risk factors

Dyslipidaemia

Dyslipidaemia refers to an abnormal amount of lipids (TG, cholesterol and phospholipids) in the blood. Lipids are transported in the blood by complexing with proteins forming lipoproteins (Hegele, 2009). These lipoproteins are classified based on their density, a measure of the amount of lipid in the molecule, the more lipids the less dense the lipoprotein. Lipoproteins are implicated with CVD due to the atherogenic effect of cholesterol. Thus, the lipoproteins that contain high concentrations of cholesterol, low-density lipoprotein (LDL), intermediate-density lipoprotein and very low-density lipoprotein, are associated with atherogenesis. They can enter and accumulate overtime in the sub-endothelium, causing immune and inflammatory responses leading to the formation of plaque (Shah, 2019).

Low Density Lipoprotein

Low-density lipoproteins carry the majority of cholesterol in the blood. The relationship between LDL and CVD has had the greatest research interest, and evidence is comprised of epidemiology, laboratory, animal, genetic and interventional studies. High blood LDL particle levels are considered a key contributor and causal to the initiation and progression of atherosclerosis due to increasing the chance of infiltration of the particles into the arterial endothelium and initiating the corresponding immune response (Ference et al., 2017; Badimon & Vilahur, 2012). LDL-cholesterol (LDL-C), the total amount of cholesterol contained in LDL particles, is generally measured in clinical practice and used as an estimate of LDL particle concentration. As a result, LDL-C has become the focus for research and estimating CVD risk. Evidence from inherited disorders of lipid metabolism, epidemiologic studies, Mendelian randomisation studies, and randomised controlled trials show that high LDL-C increases ones risk of developing CVD and that lowering LDL-C reduces ones risk (Ference et al., 2017).

High Density Lipoprotein

High density lipoprotein, the smallest of the lipoproteins, is considered to be atheroprotective (Badimon & Vilahur, 2012). Similarly to LDL-C, HDL-C is generally measured in clinical practice as an estimate of HDL particle concentration. It is estimated that for every 0.1mmol/L increase in HDL-C, CHD risk reduces by 8-15% (Gordon et al., 1989, Turner et al., 1998). A number of mechanisms have been investigated with the main being promotion of cholesterol efflux from the arterial wall and delivering it to the liver for processing. Other mechanistic properties of HDL include being an antioxidant, anti-inflammatory, anti-thrombotic, and promoting endothelial function (Badimon & Vilahur, 2012).

Triglycerides

High TG levels have a long history of being associated with CVD risk. However, a review of the evidence by the American Heart Association concluded that TGs are not directly atherogenic but are an important biomarker of CVD risk due to their association with other metabolic risk factors such as discordant lipoproteins, obesity, visceral adipose tissue, insulin resistance and diabetes (Miller et al., 2011).

Total Cholesterol/HDL-C ratio

Total cholesterol was the first lipid related measure associated with CVD, initially researched by studies such as Ancel Keys' Seven Country Study (Keys & Fidanza, 1960; Keys et al., 1984). Total cholesterol is a non-specific measure of all the cholesterol contained in the lipoproteins inclusive of the pro-atherogenic and anti-antherogenic particles. The ratio between TC and HDL-C is considered to be a stronger predictor of CVD risk than TC or LDL-C alone (Arsenault et al., 2009; Lemieux et al., 2001; Kastelein et al., 2008; Ridker et al., 2005). A large meta-analysis involving data from almost 900,000 adults that investigated a number of indices including TC, HDL-C and TC/HDL-C, determined that the TC/HDL-C ratio was the strongest predictor of IHD mortality, and more than twice as informative than the least informative measure, which was TC (Lewington et al., 2007).

Blood lipid reference levels

New Zealand blood lipid reference levels for those with known CVD or a CVD risk >15% or diabetes are as follows:

Table 3: NZ reference levels for blood lipids of adults with known CVD or a CVD risk >15% or diabetes.

Lipid	Recommendations for those with known CVD or a CVD risk >15% or diabetes
TC (mmol/L)	<4.0
LDL-C (mmol/L)	<1.8
HDL-C (mmol/L)	≥1.0
TC/HDL-C ratio	<4.0
TG (mmol/L)	<1.7

Abbreviations: HDL-C: high density lipoprotein-cholesterol, LDL-C: low density lipoprotein-cholesterol, TC: total cholesterol, TC/HDL-C: total cholesterol/high density lipoprotein-cholesterol, TG: triglyceride.
References: (Ministry of Health, 2018a; New Zealand Guidelines Group, 2012).

Hypertension

Hypertension (the chronic elevation of BP) is the force per unit area on the walls of blood vessels exerted by circulating blood. Blood pressure is usually measured in the brachial artery where systolic blood pressure (SBP) is the maximal pressure in the artery after left ventricle contraction and diastolic blood pressure (DBP) is the lowest pressure during ventricular relaxation (Shahoud & Aeddula, 2019). Hypertension is classified as followed based on severity:

Table 4: Classification of blood pressure in adults aged 18 years or older.

BP classification	Systolic (mmHg)		Diastolic (mmHg)
Normal	<120	and	<80
Prehypertension	120-139	or	80-89
Stage 1 (mild) hypertension	140-159	or	90-99
Stage 2 (moderate) hypertension	160-180	or	100-120
Stage 3 (severe) hypertension	>180	or	>120

Abbreviations: BP: blood pressure.
References: (Best Practice Advocacy Centre New Zealand, 2013; National Institute for Health and Care Excellence, 2019).

Blood pressure is controlled by many complex systems that influence vasomotor control and sodium-water retention. Systems include neurogenic control via the autonomic nervous system and hormonal signalling through mediators such as the renin-angiotensin system, atrial natriuretic peptide, eicosanoids and nitric oxide (Foëx & Sear, 2004). Long-term hypertension can cause left ventricular hypertrophy,

coronary artery disease, and organ damage. These conditions result in an increased risk of CVD through promoting myocardial infarction, heart failure and stroke (Foëx & Sear, 2004).

A meta-analysis of 61 studies showed that CVD risk doubles for individuals aged 40-70 years for every 20mmHg increase in SBP or 10mmHg increase in DBP (Lewington et al., 2002). Conversely drug trials of BP lowering medications have shown that every 10mmHg reduction in SBP significantly reduces the risk of major CVD events by 20% (Ettehad et al., 2016). Management strategies of stage one and two hypertension include drug therapy and lifestyle modification, whereas for the management of prehypertension lifestyle modification is the first line treatment.

Elevated blood glucose levels and diabetes

The Framingham Heart Study in the 1970s showed that diabetes is associated with a two to three times increased risk of CHD due to the effect of diabetes on the macrovascular system (Kannel & McGee, 1979). A meta-analysis of 102 prospective studies showed that diabetes incurs a 2.00, 2.27 and 1.84 fold increased risk of CHD, ischaemic stroke and haemorrhagic stroke respectively (Sarwar et al., 2010). The main mechanism that promotes the increased risk is thought to be via atherosclerosis as diabetes is associated with hyperglycaemia, elevated TG, low HDL-C, increased LDL-C and higher BP which together amplify endothelial dysfunction, vascular smooth muscle dysfunction and impaired platelet function (Beckman, Creager & Libby, 2002). Diabetes is independently associated with CVD risk even when hypertension and dyslipidaemia are controlled for (Wingard & Barrett-Connor, 1995; Stamler et al., 1993).

As hyperglycaemia increases so does cardiovascular event risk. This begins at a level of HbA1c below the diabetic threshold, indicating that hyperglycaemia before the development of diabetes is a risk factor for CVD (Turner et al., 1998; Mario et al., 1999). Glucose reacts with different proteins, such as haemoglobin and lipoproteins, forming glycation end products which are structural alterations that impair protein function. The glycated proteins then promote the formation of atherosclerosis (Selvin et al., 2004; Schmidt et al., 1999). Every 1% increase of HbA1c incurs an 11-18% increase risk of CVD (Selvin et al., 2004; Turner et al., 1998). Likewise every 1% reduction in HbA1c

is associated with a risk reduction of 21% for any diabetes-related mortality and 14% for myocardial infarction (Stratton et al., 2000). HbA1c is now used as an assessment tool for CVD risk and is diagnostic for diabetes (Best Practice Advocacy Centre New Zealand, 2012). Table 5 shows the recommended HbA1c reference ranges.

Table 5: Recommended HbA1c reference ranges for adult New Zealanders.

HbA1c range	Result	Diagnosis
≤40 mmol/mol	Normal range	Normal risk of Diabetes and CVD
41 – 49 mmol/mol	Elevated	Prediabetes and increased risk of CVD
≥50 mmol/ mol	High	Diabetes and increased risk of CVD

Abbreviations: CVD: cardiovascular disease, HbA1c: glycated haemoglobin.
Reference: (Best Practice Advocacy Centre New Zealand, 2012).

Overweight and obesity

Overweight and obesity are characterised by having an excess of body fat. The Framingham Heart Study identified obesity as an independent risk factor for CVD in the early 1980s (Hubert et al., 1983). Obesity also plays a role in increasing an individual's susceptibility to other CVD risk factors including dyslipidaemia, hypertension, impaired glucose tolerance and type 2 diabetes (Perez et al., 2007; Van Gaal, Mertens & De Block, 2006). Although obesity is a significant CVD risk factor, the risk is related more directly to excess visceral fat (intra-abdominal adipose tissue) as opposed to total body fat (Tchernof & Despres, 2013; Van Gaal et al., 2006).

Body mass index is one of the most commonly used measures for determining overweight and obesity. Being overweight is defined as having a BMI ≥25 kg/m^2 and obese as having a BMI ≥30 kg/m^2 (Stanley, 2003). Body mass index is easy to measure but does not necessarily provide an accurate measure of the amount of body fat an individual has. Other body measurements can give an indication of visceral fat, such as waist circumference (WC) and the ratio between WC and hip circumference (HC), the waist to hip ratio (WHR). These have been shown to be better predictors of CVD risk than BMI, with WHR demonstrating the most predictability (Tchernof & Despres, 2013; Ashwell, Gunn & Gibson, 2012). A WC of >102cm for men and >88cm for women and a WHR of ≥0.90cm for men and ≥0.85cm for women indicate increased risk of CVD (World Health Organization, 2008). Another tool that can be used to assess body composition is bioelectrical impedance analysis (BIA) which measures the resistance

of the different body tissues to electrical current to estimate the bodies components. Bioelectrical impedance analysis scales are portable and are a commonly used, non-invasive and low cost approach to calculate body composition (Khalil, Mohktar & Ibrahim, 2014).

Behavioural risk factors

Diet

Poor diet is a major risk factor for CVD. Addressing nutrition has been a key public health measure to reduce the burden of CVD both internationally and in NZ. Traditional epidemiological research and nutrition interventions focused on adjusting the intake of single nutrients in the diet (Fardet & Rock, 2014). For example, the Seven Countries Study demonstrated a three-way relationship between SFA intake, serum cholesterol and CVD risk (Bhupathiraju and Tucker, 2011, Mancini and Stamler, 2004). However, it is now acknowledged that food and nutrients are intrinsically related and changes to the food pattern consumed may have a greater impact on CVD risk compared to the consumption of individual nutrients (Jacobs & Tapsell, 2007; Hu, 2002). For example, macronutrient distributions and their influence on CVD risk change accordingly depending on the replacement nutrient. Replacing SFA with polyunsaturated fatty acids (PUFA) reduces CVD risk, whereas no reduction is seen when it is replaced with monounsaturated fatty acids (MUFA) (Hooper et al., 2015).

Cardioprotective dietary patterns

Dietary guidelines to reduce or prevent CVD are increasingly recognising the importance of overall dietary patterns and food-based recommendations as opposed to recommendations focused on single nutrients (Bhupathiraju & Tucker, 2011). Dietary patterns refers the quantity and combination of foods and drinks consumed over time (Weichselbaum, 2013). An example of a pattern based guideline is the new the National Heart Foundation of New Zealand dietary guidelines 'Eating for a healthy heart' (National Heart Foundation of New Zealand, 2018b). The guideline describes the types of foods that should be eaten and their relative quantities; mostly vegetables and fruit; some grain foods and starchy vegetables; some legumes, fish, seafood, eggs, lean poultry and meat; some reduced-fat milk, yoghurt and cheese; some healthy oils, nuts and seeds; and cutting back on junk foods and takeaways.

However dietary guidelines usually still include recommendations for key single nutrients such as saturated fat, sodium, sugar and dietary cholesterol. These nutrients are associated with increased risk of CVD and are often consumed in high amounts by many people.

There are a number of examples of cardioprotective dietary eating patterns including the Dietary Approaches to Stop Hypertension (DASH) diet (National Heart Lung and Blood Institute, 2018), Mediterranean diet (Bach-Faig et al., 2011) and dietary guidelines produced by national cardiovascular interested not for profits such as the National Heart Foundation of New Zealand and the American Heart Association (National Heart Foundation of New Zealand, 2018b; Krauss et al., 2000; American Heart Association, 2018b) (table 6). These guidelines are produced to meet the needs of those at risk of CVD, those who have CVD as well as the general population. Although there are minor differences between these dietary guidelines in regard to things such as the number of serving sizes recommended. Key areas where they align include:

- Being rich in fruits and vegetables
- Eating wholegrain carbohydrates as opposed to refined carbohydrates, and limiting sugar
- Unsaturated fats as the predominant form of dietary fat, choosing low fat dairy products, adequate omega-3 fatty acids, using vegetable oils as opposed to animal fats, limiting SFA and TFA
- Choosing lean protein sources, including fish, legumes and nuts and reducing processed meats
- Reducing salt intake
- Moderating alcohol intake
- Matching energy intake to overall energy needs.

Table 6: Examples of dietary patterns for cardiovascular health.

Eating pattern	American Heart Association guidelines*	National Heart Foundation of New Zealand guidelines	DASH Diet*	Mediterranean Diet	Examples of Serving Size
Grains and starchy vegetables	6s/d (≥half wholegrain/high dietary fibre)	3s/d (wholegrains)	6-8s/d (mostly wholegrains)	Grains 1-2s/meal (preferably wholegrain) Starchy vegetables ≤3s/wk	1 slice bread ½c cooked pasta, cereal or rice
Vegetables	5s/d	3-4s/d	4-5s/d	2+s/meal	1c raw leafy vegetables, ½c cooked vegetables
Fruit	4s/d	3-4s/d	4-5s/d	1-2s/meal	1 medium fruit ½c frozen or canned fruit ¼c dried fruit
Fat-free or low-fat milk and milk products	3s/d	2-3s/d	2-3s/d	2s/d	1c milk (AHA, HFNZ, DASH), Yogurt: 1c (AHA), 150g (HFNZ) Cheese: 20-30g (HFNZ), 40g (DASH)
Lean meats, poultry, eggs and fish	8-9s/wk Fish 2-3s/wk (preferably oily fish)	Lean meats/poultry 1-1.5s/d Eggs ≤6/wk Fish 2-3s/wk (especially oily fish) Legumes 4-5s/wk	≤6s/d	Red meat <2s/wk Processed meat ≤1s/wk Fish 2+s/wk White meat 2s/wk Eggs 2-4s/wk Legumes ≥2s/wk	1 egg (AHA, HFNZ, DASH) Cooked lean meat: 85g (AHA), 100-120g (HFNZ), 30g (DASH) Cooked fish: 85g (AHA), 150g tuna, 85-95g salmon (HFNZ), 30g (DASH) 1c cooked beans (HFNZ)
Nuts, seeds and legumes	5s/wk	6+s/d (NB: includes fats and oils and not legumes which are included above)	4-5s/wk	1-2s/d (NB legumes included under meat)	Nuts: 2Tb (AHA), 1Dsp (HFNZ), 40g (DASH) Nut butter: 1Tb (AHA), 1Dsp (HFNZ), 2Tb (DASH) Seeds: 2Tb (AHA, DASH), 1Tb (HFNZ)

					Cooked legumes: 1/4c (AHA), 1/4c (DASH)
Fats and oils	3s/d (preferably unsaturated)		2-3s/d	1s/meal (olive oil)	Oil/margarine: 1Tb (AHA, MD), 1tsp (HFNZ, DASH)
Fluid	Not stated	6-8 cups/d	Not stated	6-8cups/d	250ml water, tea
Added sugars	≤half of the daily discretionary calories allowance	Limit	≤5s/wk	≤2s/wk	1Tb sugar or jam (DASH)
Sodium	≤2,300mg/d, ideally 1,500mg/d	Limit	≤2,300mg/d, ideally 1,500 mg/d	Not stated	
Alcohol	Men: 1-2s/d Women: 1s/d	Men: ≤2-3s/d, ≤15s/wk Women: ≤1-2s/d, ≤10s/wk	Men: ≤2s/d Women: ≤1/d	Men: ≤2s/d (wine) Women: ≤1/d	Alcohol: 14g (AHA, DASH), 10g (HFNZ)

*Based on 2000 calorie or 8400 kilojoule/d energy intake
Abbreviations: AHA: American Heart Association, c: cup, d: day, DASH: Dietary Approaches to Stop Hypertension, Dsp: dessertspoon, g: grams, HFNZ: National Heart Foundation of New Zealand, MD: Mediterranean Diet, mg: milligrams, NB: note, s: serves, Tb: tablespoon, tsp: teaspoon, wk: week.
References: (Bach-Faig et al., 2011; American Heart Association, 2018b; National Heart Foundation of New Zealand, 2018b; National Heart Lung and Blood Institute, 2018).

Rich in fruits and vegetables

The CVD protective effect of fruits and vegetables is largely based on observational epidemiological studies. A review of the available epidemiological evidence up to 2010 concluded that the consumption of fruits and vegetable intake displays convincing evidence for the reduction of the risk of hypertension, CHD and stroke (Boeing et al., 2012). The number of servings seems to provide cumulative protection as shown by a meta-analysis of prospective cohort studies; each additional serving of fruit or vegetables/day provided a 5% and 4% decrease in CVD mortality respectively (Wang et al., 2014). However, a different review by Dauchet, Amouyel & Dallongeville (2009) found only four of seven cohort studies displayed a significant association between fruit and vegetable intake and CVD events. Factors which make it difficult to reach conclusions using cohort studies include participant compliance, accuracy and lack of comparability in how diets are assessed, and other healthy lifestyle factors which are hard to control e.g. physical activity, rates of smoking, and poor diet (Dauchet et al., 2009; Joshipura et al., 1999).

Shorter, controlled studies examining the effect of fruit and vegetable intake on CVD risk factors such as plasma lipids and BP can provide additional insight. A Cochrane Review of four studies of at least three months duration which isolated fruit and vegetable consumption from other lifestyle modifications found favourable effects on BP and LDL-C at six months (Hartley et al., 2013). Pooled analysis of the two studies that investigated BP showed a significant reduction in SBP (mean difference -3.0 mmHg) (Smith-Warner et al., 2000; John et al., 2002). Pooled analysis of the two studies that investigated LDL-C and HDL-C showed a non-significant reduction in LDL-C (-0.17 mmol/L) and no effect on HDL-C (Smith-Warner et al., 2000; Djuric et al., 2006).

A number of mechanisms for the cardioprotective effect of fruit and vegetables have been suggested. Fruit and vegetables:

- Contain a significant amount of antioxidants, such as phenolic flavonoids, lycopene, carotenoids and glucosinolates which could help to reduce oxidative stress particularly in the vascular endothelium (Kaur & Kapoor, 2001).
- Are a good source of potassium which helps to control BP (Appel et al., 2006; Sacks et al., 2001).

- Reduce levels of homocysteine (an independent risk marker for cardiovascular disease), due to increased intake of vitamins B6, B12, and folate (Eikelboom et al., 1999).
- Are low in energy density and help with weight management (Boeing et al., 2012).
- Are high in fibre (discussed below).

Eating wholegrain carbohydrates as opposed to refined carbohydrates, and limiting sugar
Wholegrain consumption is found to be protective of CHD and CVD by providing at least a 20% and up to 40% reduction in risk for those who regularly eat wholegrains compared to those who eat them rarely (Flight & Clifton, 2006). Wholegrains provide more complex carbohydrate, vitamins, minerals and fibre than refined carbohydrate. The structure of wholegrains compared to their refined derivatives have a lower glycaemic index which is associated with a lower glycaemic response (Foster-Powell et al., 2002), increased satiety and better weight control (Seal, 2006; Anderson et al., 1994).

Fibre, the indigestible component of plant matter, has been researched extensively in relation to its effects on CVD risk factors. Adequate intakes of dietary fibre have been shown to improve serum lipids, lower BP, provide better blood glucose control, appetite control and weight management (Slavin, 2013; Reynolds et al., 2019). Wholegrains, vegetables, fruits, legumes, and nuts are good sources of fibre (Van Horn, 1997). Soluble fibres modestly reduce TC and LDL-C levels beyond those achieved by a diet low in saturated fat and cholesterol (Theuwissen & Mensink, 2008). Soluble fibre at 2-10g/day is associated with small but significant decreases in TC and LDL-C (-0.045mmol/L and -0.057mmol/L per gram of soluble fibre consumed respectively). No effect has been shown on TG or HDL-C (Brown et al., 1999; Theuwissen & Mensink, 2008).

Sugar, particularly sugar added to foods during processing or preparation is linked with higher blood glucose levels, elevated TGs, greater energy intake, higher body weight and lower intake of essential nutrients (Johnson et al., 2009). This is contrasted to recommended foods such as fruit, where sugar is intrinsically part of the food product. These foods contain dietary factors such as fibre that minimise the negative

effect of the sugar in that food on the body, as well as providing other additional nutritional benefits.

Unsaturated fats as the predominant form of dietary fat, choosing low fat dairy products, adequate omega-3 fatty acids, using vegetable oils as opposed to animal fats, limiting SFA and TFA

For a long time diets low in SFA, TFA and cholesterol have been shown to reduce the risk of CVD, in large part through their effects on LDL-C levels (Mozaffarian et al., 2006; Hegsted et al., 1993; Keys et al., 1986). Animal fats such as full fat milk products (butter, cream and milk), fatty meats and lard provide a large contribution of dietary SFA. Some recent studies have challenged the link between SFA and CVD outcomes (De Souza et al., 2015; Siri-Tarino et al., 2010), but have received criticism regarding their methodology (Stamler, 2010). A recent meta-analysis showed a decrease in SFA had no effect on reducing the risk of death from CVD, but produced a 17% reduction in the risk of having cardiovascular events such as stroke and heart attack (Hooper et al., 2015). The risk level also depends on the type of nutrient substitution, for example replacing SFA with PUFA provides a lowered risk of CVD events (up to 27%) compared to replacement with MUFA (no risk change) or carbohydrates (increased risk) (Jakobsen et al., 2009; Mozaffarian, Micha & Wallace, 2010; Hooper et al., 2015). When considering the effect of SFA replacements on TC/HDL-C a meta-analysis by Mensink et al. (2003) demonstrated that replacement with unsaturated fats produced favourable changes whereas replacement with carbohydrates produced no change to the TC/HDL-C ratio. Hence, to reduce CVD risk the recommendation is to replace SFA containing foods with PUFA or MUFA containing foods.

Fish, especially oily fish, contains relatively high amounts of the omega-3 PUFAs; eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Modest consumption of fish (e.g. one to two servings/week) reduces risk of coronary death by 36% with intake of 250mg/day of EPA and DHA appearing sufficient for primary prevention (Mozaffarian & Rimm, 2006). Eicosapentaenoic acid and DHA positively impact CVD risk factors; TG, resting heart rate, and BP and might also improve myocardial filling and efficiency, lower inflammation, and improve vascular function (Mozaffarian & Wu, 2011). The evidence for the efficacy of EPA and DHA supplementation is mixed leading to recommendations to consume these PUFAs through food if possible (Evangelos et al., 2012).

Choosing lean protein sources, including fish, legumes and nuts and reducing processed meats

Lean protein sources contain less total fat and less SFA than their non-lean and processed counterparts which are favourable for control of energy intake and blood cholesterol. Fish, legumes and nuts are lean sources of protein that contain other benefits such as omega-3 PUFAs in the case of fish, and fibre in the case of legumes and nuts. A systematic review and meta-analysis in 2010 showed that consumption of processed meats, but not red meats, is associated with a higher incidence of CVD and diabetes (Micha, Wallace & Mozaffarian, 2010). On average, processed meats contain approximately 400% more sodium and 50% more nitrates per gram than non-processed meats, with sodium accounting for about two-thirds of the risk difference (Micha, Michas & Mozaffarian, 2012).

Reducing salt intake

On average, as a person's intake of dietary salt increases, their BP also increases. A meta-analysis showed that a reduction in salt intake by 4.6g/day (78mmol/day sodium) in people with hypertension and 4.4g/day in people without hypertension over four or more weeks lowered SBP and DBP by 5.0 and 2.7mmHg and 2.0 and 1.0mmHg respectively (He & MacGregor, 2002). Dose response analysis also displays significant, direct, progressive dose response relationships, the greater the salt reduction the greater the drop in BP (Sacks et al., 2001; He & MacGregor, 2002). In a more recent meta-analysis, the authors concluded that when salt intake was <5.12g/day compared to higher intakes, SBP and DBP was lower by 3.47mmHg and 1.81mmHg respectively (Aburto et al., 2013). They also found that increased sodium intake was associated with an increased risk of stroke (relative risk (RR) 1.24), stroke mortality (RR 1.63), and CHD mortality (RR 1.32). Evidence suggests there is some variability to the sensitivity of the BP lowering effects of salt between individuals, this seems to result from individuals that tend to have a less responsive renin-angiotensin-aldosterone system (Appel et al., 2006).

Moderating alcohol intake

Moderate intake is defined as up to one standard drink (15g alcohol)/day for women and one-two standard drinks/day for men. Earlier studies found a J-shaped curve in relation to alcohol intake with CVD outcomes and all-cause mortality (O'Keefe, Bybee & Lavie, 2007; Corrao et al., 2000; O'Keefe et al., 2014). For example, the meta-analysis by Corrao et al. (2000) showed that CHD risk decreased from 0-20g/day (RR = 0.80)

with a protective effect up to 72g/day (RR = 0.96). Increased risk was demonstrated at 89g/day (RR = 1.05) or greater. Females displayed protection and harmful effects at lower levels (10g/day and 52g/day respectfully) compared to males (25g/day and 114g/day) (Corrao et al., 2000). Heavy drinkers on the other hand can reduce their BP and TG levels through reduction in alcohol consumption (Xin et al., 2001; Pejic & Lee, 2006). Alcohol is also an energy dense nutrient (7kcal/g) and can contribute to weight gain, especially if consumed in excess of energy requirements (Suter & Tremblay, 2005; Wannamethee & Shaper, 2003).

Reasons for the decreased risk at moderate doses has been explored with regards to CVD risk factors. It has been shown that 30g/day increases HDL-C, apolipoprotein A1 and lowers concentrations of fibrinogen which are considered cardioprotective (Rimm et al., 1999; Ronksley et al., 2011). More recently, a meta-analysis questioned the protective effect against all-cause mortality at moderate doses because of flaws in grouping former and occasional drinkers, once this misclassification was corrected they concluded that reduced mortality in low-volume alcohol drinkers was no longer found and a more linear curve relationship was formed (Stockwell et al., 2016). When isolating the effect of moderate alcohol consumption on the type of CVD event stroke and IHD are not protected against (Ronksley et al., 2011; O'Keefe et al., 2014). Additionally there is a lack of randomised trials data using alcohol for improving clinical outcomes (O'Keefe et al., 2007). For these reasons and the other harms drinking alcohol can produce, guidelines generally recommend moderate intake only for those who already consume alcohol (Lichtenstein et al., 2006).

Matching energy intake to overall energy needs

Maintaining an energy balance is important for controlling body weight to prevent becoming overweight and obese (discussed in the metabolic risk factor section). The energy density of the macronutrients differ per gram, fat: ~9kcal/g, carbohydrate and protein: ~4kcal/g, and alcohol 7kcal/g. Limitation of energy dense and low nutrient dense foods is an effective way to reduce total energy intake (Krauss et al., 2000). This typically involves reducing intake of foods high in fat, sugar and alcohol. Foods that are favourable for minimising energy intake while maximising nutrient intake are vegetables, fruits, wholegrains and lean proteins. Overall energy needs can be attenuated with increased energy expenditure by remaining physically active.

Methodologies to assess dietary intake

Dietary assessment methods can help to assess the link between dietary intake and health outcomes (Biro et al., 2002; Shim, Oh & Kim, 2014). Food records, food frequency questionnaires, diet recall, diet histories and diet quality indices have been used to measure assess dietary intake. Each method has strengths and weaknesses and it is important to choose an assessment method that meets the objectives of a given study (Biro et al., 2002; Shim et al., 2014). Diet quality indices are a dietary assessment method used to effectively comprehend dietary patterns. Respondents are scored on how closely their diet aligns to a specific nutritional guideline or criteria, thus giving an indication of diet quality (Hu, 2002; Newby et al., 2003). Three diet quality indices have been developed internationally that specifically relate to cardiovascular health. These are the Dietary Quality Score (Toft et al., 2007), the Diet Quality Tool (O'Reilly & McCann, 2012), and the Food Pyramid Index (Massari et al., 2004). Recently a diet quality index, the Healthy Heart Food Index, was produced to compare an individual's dietary intake with recommendations made by the National Heart Foundation of New Zealand (National Heart Foundation of New Zealand, 2019a). It has subsequently been validated (Beck, 2019).

Physical activity

Physical activity is usually defined as low, moderate or intense and includes many domains; recreation, transportation, household chores, and/or occupation. Physical activity has been found to be beneficial for BP (Pescatello et al., 2004; Fagard & Cornelissen, 2007), small weight reductions (Swift et al., 2014), increasing HDL-C and decreasing TG with generally no impact on TC and LDL-C (Durstine et al., 2001; Kodama et al., 2007). Comparisons of high physical activity versus low physical activity in cohort studies demonstrated a 35% risk reduction in CVD mortality (Nocon et al., 2008). There are differences in the effect the type of physical activity has, which may be due to the differing intensity of the type of exercise, for example Li & Siegrist (2012) found that high levels of leisure time physical activity produced a greater CHD risk reduction (20-30%) than high levels of occupational physical activity (10-20%). When controlling for energy expenditure epidemiologic studies find a greater reduction in risk of CVD, BP and glucose glucose with vigorous compared to moderate intensity physical activity (Swain & Franklin, 2006).

The amount of exercise required to achieve results has been investigated with interest. Investigations into low intensity physical activity such as walking state that 5,000 steps/day is indicative of sedentary behaviour, whereas greater than 8,000 steps suggests a more active lifestyle (Swift et al., 2014). A meta-analysis of studies where pedometers were used as a motivational device showed that a 1.3kg weight loss is possible over a median of 16 weeks (Richardson et al., 2008). Additionally, a systematic review reported that over 18 weeks significant reductions in BMI (−0.38 kg/m^2) and SBP (−3.8 mmHg) can be achieved, but no significant reductions in cholesterol, TG, or fasting glucose levels (Bravata et al., 2007).

A recent meta-analysis by (Kyu et al., 2016) suggests that for disease prevention total physical activity needs to be several times higher than the current recommended minimum level, 150 minutes moderate activity/week, the equivalent of 600 metabolic equivalent (MET) minutes/week (a measure of PA). These researchers found that the risk of IHD and ischemic stroke reduced in a dose response manner to total PA. The greatest benefit of IHD risk reduction would be ~4500 MET minutes and ~2500 MET minutes for ischemic stroke. Table 7 summarises current physical activity recommendations for heart health.

Table 7: Examples of physical activity recommendations for heart health.

	American Heart Association guidelines	National Heart Foundation of New Zealand guidelines	WHO guidelines*	Mediterranean Diet
Type and amount	≥150min/wk moderate-intensity aerobic activity or 75min/wk vigorous-aerobic activity or combination of both throughout the week. Moderate- to high-intensity muscle-strengthening ≥2d/wk Spend less time sitting	150min/wk moderate intensity activity	≥150min/wk moderate-intensity aerobic activity or ≥75min/wk vigorous-intensity aerobic activity or combination of both throughout the week Muscle-strengthening ≥2d/wk Aerobic activity performed in bouts of ≥10min duration.	≥30min/d moderate physical activity throughout the day
Additional health benefits	≥300 min/wk		300min/wk moderate-intensity aerobic activity or 150min/wk vigorous-intensity aerobic activity	

*for general health.

Abbreviations: d: day, min: minutes, wk: week.

References: (American Heart Association, 2018a; Bach-Faig et al., 2011; National Heart Foundation of New Zealand, 2018a; World Health Organization, 2016).

Medication use

Medications can successfully be used to reduce BP, produce more favourable blood lipids, control blood glucose levels and diabetes, and aid in smoking cessation. A large meta-analysis of statin clinical trials showed that after one year of statin treatment LDL-C decreased by a mean of 1.09mmol/L, by five years the effect had reduced to a mean reduction of 0.80mmol/L. The authors suggested this reflected non-adherence to the medication over time (Baigent et al., 2010). The same study concluded that reducing LDL-C by 1mmol/L in individuals treated with statins is associated with a 22% reduction in the risk of major CVD events. Clinical trials using BP lowering medications have also been shown to be effective. Blood pressure lowering medications have shown that every 10mmHg reduction in SBP significantly reduces the risk of major CVD events by 20% (Ettehad et al., 2016). It is common for patients at risk of CVD to be taking more than one medication to lower their risk. Adherence to a four-pronged treatment with aspirin, statin, and angiotensin-converting enzyme inhibitors and stopping smoking produced a 40% reduction in major cardiovascular events (Armstrong et al., 2014).

Medication adherence usually refers to whether patients take their medications as prescribed. Factors that influence adherence have been described by the World Health Organisation as including patient characteristics, condition type, therapy complexity, socioeconomic status and health system-related design (World Health Organization, 2003). Patients' beliefs about their medicines is a strong predictor of medication adherence such as how necessary they believe medicines are for maintaining health or beliefs regarding the potential for adverse side effects. With cardiac patients the concern about side effects tended to outweigh the necessity for maintaining health, leading to less adherence than other classes of medications (Horne & Weinman, 1999).

Levels of measured medication adherence in studies varies, for example Vrijens et al. (2008) found that about half of all patients prescribed antihypertensive medications stopped taking them within one year of the initial prescription, in contrast Bramley et al. (2006) found approximately 75% of patients taking antihypertensive medication were adherent. A meta-analysis of adherence to primary preventative cardiovascular medications showed 50% adherence at two years (Naderi et al., 2012). High adherence to antihypertensive medications was associated with better BP control compared with

those who had medium or low levels of adherence (Granger et al., 2005). Similarly, each 25% increase in the number of days of statin medication use was associated with an approximately 0.1mmol/L reduction in LDL-C (Ho et al., 2006).

Smoking cessation

Cigarette smoking is a major risk factor for CVD (Doll & Peto, 1976; Wolf et al., 1988; Willett et al., 1987) and one of the most avoidable (Pechacek et al., 2003). Smoking is associated with a greater risk of non-fatal myocardial infarction (odds ratio (OR) 2·95) compared with never smoking, with an increase in risk for every additional cigarette smoked (Teo et al., 2006). Quitting smoking can help to reduce an individual's risk of CVD substantially. However, risk does not return to the same level as people who have never smoked. The major mechanism through which smoking contributes to CVD is by influencing many stages of atherosclerosis including endothelial dysfunction, inflammation, dyslipidaemia, oxidation, and thrombosis (Ambrose & Barua, 2004). Over a three year period atherosclerosis progresses at a rate 50% greater in active smokers relative to never smokers (Howard et al., 1998).

CVD risk assessment

Individualised CVD risk assessment is viewed as an appropriate prevention strategy to identify those who are at high risk and to provide an appropriate level of intervention to lower their risk (Ministry of Health, 2018a). In NZ, CVD treatment is based on a individuals five-year absolute CVD risk (the likelihood of a cardiovascular event, heart attack, stroke or angina, over five years) (New Zealand Guidelines Group, 2012). It is recommended CVD risk assessments are undertaken every five years for men aged 45-74 years and women aged 55-74 years, and 10 years earlier for people of Māori, Pacific and Indian descent (Ministry of Health, 2018a). The overall goal is to reduce five-year CVD risk to less than 15% (New Zealand Guidelines Group, 2012). Recently the NZ risk assessment calculations for people aged 30-74 years were updated based on the New Zealand PREDICT study (Pylypchuk et al., 2018).

The PREDICT study developed a NZ specific CVD risk prediction model using over 400,000 NZ participants. Risk assessment for people aged over 74 years is outside the range for the PREDICT equations but the tool is potentially useful as an estimation.

Variables used in the equations include demographics, family history, deprivation quintile, medical history, BP, TC/HDL-C, and CVD medication use. The study also produced national CVD risk estimations for the eligible population showing that 74% have a less than 5% five-year risk of CVD, 24% have a 5–14% risk, and 2% have a risk of 15% or higher (Pylypchuk et al., 2018).

Internationally it is generally accepted that for a five-year risk below 5% it is not recommended to begin CVD medications due to the potential harms from treatment compared to the expected benefits (Ministry of Health, 2018a). Lifestyle advice including diet, weight management, physical activity and smoking cessation, which is tailored to the individual is recommended for all levels of risk. The potential benefits of lipid lowering and BP lowering drug treatment should be considered for people above 5% risk of CVD and antiplatelet therapy for those above 15% (Ministry of Health, 2018a).

CVD primary prevention interventions

The wide area of CVD primary prevention includes but is not limited to interventional programmes that focus on a reduction in CVD risk factors through the use of lifestyle changes in diet, physical activity, smoking cessation and/or medication use and adherence. Outcome measures can include quantifying changes in primary outcomes - CVD events, mortality or CVD risk over a given timeframe, or secondary outcomes – CVD risk factors (cholesterol levels, BP levels, levels of glycaemic control, body composition, diet, physical activity levels/fitness). Primary outcomes can be hard to examine largely due to the short time frame of these studies and the length of time it takes to develop cardiovascular diseases. Educational and counselling interventions aimed at reducing CVD risk factors in addition to, or instead of, pharmacological treatment are believed to be cost-effective and more beneficial with the greater degree of risk factor control that is achieved (Appel, 2004; Puska et al., 1985; Farquhar et al., 1990). However, reviews of the evidence for the effectiveness of CVD primary prevention that involve counselling and education to reduce CVD have shown mixed results for both primary outcomes and secondary outcomes (Daviglus, Lloyd-Jones & Pirzada, 2006; Ebrahim & Smith, 1997).

A 2011 Cochrane Review examined the effectiveness of 55 randomised controlled trials of at least six months follow up (median 12 months) that targeted multiple CVD risk factors using education and counselling intervention strategies (Ebrahim et al., 2011). The types of interventions were varied and included workshops, lectures, individual sessions, personal counselling, provision of written material, assignments, shopping tours and cooking sessions. These were provided by a variety of health professionals including physicians, nurses, nutritionists, dietitians, nurses, exercise trainers, cooks, psychotherapists and physiotherapists. Intervention number ranged from four to 54 sessions over periods of time ranging from two weeks to three years and most targeted diet, exercise, weight loss, salt intake, alcohol use, stress management, smoking cessation, adherence to medication or specific clinical regimens. The meta-analysis found no strong evidence for the reduction in all-cause mortality (OR 1.00), coronary heart disease mortality (OR 0.99), weak evidence for fatal and nonfatal events (RR 0.84), and favourable evidence for stroke mortality (RR 0.75) when used in general populations. However, interventions where patients were recruited with hypertension or diabetes were more effective at lowering all-cause mortality (RR 0.78 and RR 0.86 respectfully) and fatal and non-fatal CVD events (OR 0.71). Those who were taking antihypertensive or lipid-lowering drugs also lowered their all-cause mortality (RR 0.86). Changes to individual risk factors were small, SBP (-2.71mmHg), DBP (-2.13mmHg), TC (-0.24mmol/L), and smoking prevalence (OR 0.87). Additionally, the studies with the highest baseline DBP, TC and smoking prevalence demonstrated larger reductions at follow up. Overall the findings suggested that lifestyle interventions have limited use in the general population but can be more effective when targeting individuals who are at high risk of CVD. Another recent meta-analysis that also evaluated controlled behaviour change interventions aimed at reducing CVD risk by targeting multiple CVD risk factors showed similar results (Alageel et al., 2017).

Laws et al. (2013) determined that the biggest barriers to attendance of lifestyle interventions for CVD prevention were work commitments and poor physical access to the programmes. These external factors had more influence than internal factors such as the person's individual health risk or readiness to change. Endorsement of programmes by general practitioners or medical practices also encouraged participation (Laws et al., 2013).

CVD primary prevention in the community pharmacy

The community pharmacy setting is well placed for the prevention of diseases and improvement of health outcomes because they offer the public accessibility to trained health professionals with the convenience of locality, extended open hours and without the need for an appointment. Pharmacists are able build relationships with patients who are on regular prescriptions and are often a patient's first or only point of contact with a health professional (Gray, Chamberlain & Morris, 2016; Pharmaceutical Services Negotiating Committee, 2010). They are intimately involved with the management of a patient's medication and provide health and lifestyle advice in an informal manner. A rural Australian survey revealed that people with CVD or at risk of developing CVD had the same to more interactions with a community pharmacist compared to a general practitioner within a 12-month period (McNamara et al., 2012b). For these reasons community pharmacies and their pharmacists may provide a useful additional service point to identify patients who are at risk of CVD as well as providing an environment for the primary prevention of CVD.

Identification of individuals at risk of CVD

It has been shown that community pharmacists are successful at CVD risk screening within the pharmacy setting including the measurement of risk factors required for CVD risk calculation: height, weight, BP, TC and HDL-C level (Liu et al., 2008; Donyai & Van den Berg, 2009; Horgan et al., 2010). Screening programmes in community pharmacies are also able to effectively target harder to reach groups such as males, ethnic minorities and lower socio-economic communities (Snella et al., 2006; Horgan et al., 2010).

Interventions targeting individual CVD risk factors

Studies of pharmacist involvement in managing CVD related risk factors has been investigated. Through medication management, disease education and patient counselling pharmacists can produce greater outcomes compared to standard care for hypertension (Tsuyuki et al., 2015; Hirsch et al., 2014; Lee, Grace & Taylor, 2006; Santschi et al., 2014; McLean et al., 2008; Neto et al., 2011), dyslipidaemia (Ellis et al., 2000; Faulkner et al., 2000; Mazzolini et al., 2005; Tsuyuki, Rosenthal & Pearson, 2016b;

Tsuyuki et al., 2002; Neto et al., 2011; Spence et al., 2014), diabetes management (Machado et al., 2007; Hayward, Krein & Vijan, 2005; Coast-Senior et al., 1998; Rothman et al., 2005; Fazel et al., 2017; Spence et al., 2014), anticoagulation management (Gray, Garabedian-Ruffalo & Chretien, 1985; Wilt et al., 1995; Conte et al., 1986) and secondary prevention of CHD and heart failure (Altowaijri, Phillips & Fitzsimmons, 2013). Pharmacists can also provide effective behavioural and medication support for smoking cessation (Augustine et al., 2016). There is limited data on the success of pharmacist-driven weight management. Some studies demonstrate modest weight loss (O'Neal & Crosby, 2014; Gordon, Watson & Avenell, 2011; Boardman & Avery, 2014).

A meta-analysis of 30 randomised controlled trials using pharmacist lead interventions showed benefits for BP control (-8.1/-3.8mmHg), TC (-0.45mmol/L), LDL-C (-0.35mmol/L), and a 23% reduction in the risk of smoking (Santschi et al., 2011). Most (87%) of the studies in this analysis included counselling about medications or lifestyle, distribution or use of educational material, or patient educational workshops. In a recent review of the literature on pharmacist interventions for the primary and secondary prevention of CVD, Omboni & Caserini (2018) identify similar clinical outcomes. A systematic review by Ifeanyi et al. (2015) of specifically community pharmacy based interventions to reduce CVD risk included 27 pharmacist led studies and reported similar reductions in SBP (7.8-17.3mmHg), TC (0.47-0.70mmol/L) and HbA1c (5-24mmol/mol) to Santschi et al. (2011).

All three reviews report that the quality of most the research is poor in regard to heterogeneity in a number of domains; design, intervention, setting (community, hospital, pharmacist only, multidisciplinary) and participant disease severity. Studies varied in type and extent interventions including medication management (most common), education, adherence assessment, referral to other health professionals, and some were co-interventional. This makes it difficult to directly compare studies and thus clearly identify the type of intervention that is most effective in the management of CVD risk factors. Additionally, the majority of the studies reviewed focused on one or only a couple of the risk factors of CVD as opposed to multiple risk factor interventions, especially multiple lifestyle behavioural factors. This shows that there is a lack of standardisation and consensus on the most effective type of intervention design and outcomes to explore.

Multiple CVD risk factor reduction in pharmacy settings using lifestyle interventions

Two studies utilising pharmacists in a community pharmacy setting and one using pharmacists within a medical center were found that aimed to reduce overall CVD risk and multiple CVD risk factors using lifestyle behaviour interventions. These are summarised in table 8. Additionally, the table includes three community pharmacy studies that used similar lifestyle-based approaches to impact type 2 diabetes outcomes but also included CVD risk endpoints. No studies of this kind could be found within the NZ context.

Table 8: Multiple CVD risk factor reduction interventions in pharmacy setting.

Study, reference, country	Study aim	Population & setting	Recruitment	Intervention	Outcome measures	Results	Other relevant information
RxEACH Trial (Tsuyuki et al., 2016a) Canada	Evaluate the effectiveness of a community pharmacy-based case finding & intervention programme on CVD risk.	N=723, 56 community pharmacies M/F adults ≥18 yrs, at high risk for CVD: those with DM, CKD, atherosclerotic vascular disease, or multiple risk factors with a 10-year CVD risk >20% ≥ one uncontrolled risk factor	Proactive case-finding strategy by pharmacists to identify participants taking meds that match disease states & smokers Advertising e.g. newspaper or other advertising outlets, or pharmacy heart health clinics 913 screened, 827 eligible, 723 enrolled, 29 drop out. 53 were primary prevention.	3-month RTC: Intervention group (n=370): follow up every 3-4wks for 3 months with pharmacist. Patient assessment – BP, WC, Ht, Wt, HbA1c, fasting lipids, estimated GFR, ACR. Individualised intervention: CVD risk calculation, explanation of risk, targets for intervention, healthy lifestyle options, treatment recommendations for CVD risk factors, prescription adaptation, communication with patients GP Control group (n=353): Usual pharmacist & physician care for 3 months.	Primary outcome: change in CVD risk between intervention & control group at 3 months Secondary outcomes: change in clinical measures & smoking cessation between intervention & control group at 3 months.	10-CVD risk (%): -5.37, relative decrease 21% (p<0.001). SBP (mmHg): -9.37 (p<0.001) DBP (mmHg): -2.92 (p<0.001) HbA1c (%): -0.92 (p<.001) Smoking (%): -20.2 (p<0.001) LDL-C: -0.2mmol/L (p=0.001)	Pharmacist training: online training program based on current Canadian Guidelines (modules on case finding, CVD risk calculation, & patient communication of CVD risk, CKD, HTN, dyslipidaemia, DM, smoking cessation, diet & lifestyle management).
PAART CVD Pilot Project (McNamara et al., 2012a)	Assess the feasibility of implementing a primary CVD prevention	N=70, 10 community pharmacies M/F, 50-74 yrs, taking	Pre-screening of participants based on the inclusion criteria - obtained from	6 month longitudinal pre- & post-test study single-cohort intervention (n=67)	Primary: change to 5-year CVD risk between baseline & 6 months.	5-year CVD risk (%): -1.7% (25% relative risk reduction) (p<0.001) SBP (mmHg): -11 (p<0.001)	Pharmacist received 2 days training accordance to national guidelines. Health promotion & behavioural change

Study, reference, country	Study aim	Population & setting	Recruitment	Intervention	Outcome measures	Results	Other relevant information
(McNamara et al., 2010) Australia	program in a community pharmacy setting.	med for cholesterol &/or BP who did not already have CVD or DM	dispensing records & patient interview; or participant expression of interest. Participants then received formal assessment of eligibility. 70 recruited, 3 withdrew due to low CVD risk.	5 monthly sessions with pharmacist (initial 30mins, follow up 15-20 mins) for 6 months Lifestyle goals & treatment targets were established collaboratively with patients Baseline & final data taken by research assistants (60-90mins)	Secondary: changes to clinical measures between baseline & 6 months. Self-report questionnaires: medical history, med use, med adherence, health behaviours (smoking, diet, weight management, alcohol intake, PA) & psychosocial health	DBP (mmHg): -5 (p<0.001) TC/HDL-C ratio -0.2 (NS) TC (mmol/L): -0.12 (NS) LDL-C (mmol/L): -0.14 (NS) HDL-C (mmol/L): 0.02 (NS) TG (mmol/L): -0.01 (NS) Wt (kg): -0.75 (p=0.03) BMI (kg/m^2): -0.3 (p=0.04) WC (cm): -1.3 (p=0.01) Med non-adherence (%) -16 (p=0.001) Diet score: +9.6 points (p<0.001) Moderate PA (days 30mins): +0.5 (p=0.009) Patients with a higher baseline risk benefited 2.6 times than low risk patients	CVD risk assessment medicines adherence Medicines management Lifestyle modification (diet, PA, weight management, alcohol consumption, smoking cessation) Antiplatelet use GP engagement Interventions using Health Action Process Approach Equal focus on meds prescribed, med adherence, & lifestyle modification during training Diet assessed using Diet Quality Tool - adherence to CVD dietary guidelines
CRRC model (Taveira et al., 2006) United States of America	Assess the effectiveness of the CRRC model in reducing the overall CVD risk of the referred patients as measured by 10-year CVD risk	N=375 Patients seen in the CRRC Medical Center during the study period, Diagnosed with DM or CVD	Retrospectively review of the electronic medical records of patients enrolled in the CRRC program over 1 year. Enrolled into the CRRC by	Cohort design - each patient had avg of 4.2 visits & 202.2 days of follow up Intervention - conducted by pharmacist Initial (30min) – med adherence, treatment plan to control BP, lipids, DM & smoking	Primary: change in 10-year CVD risk from baseline to discharge or end of study period (1 year) Secondary: change in	Change in 10-year CVD risk: -0.9 (NS) TC (mmol/L): -0.61 (p<0.01) LDL-C (mmol/L): -0.05 (p<0.01) HDL-C (mmol/L): +0.01 (NS) HbA1c (%): -1.1 (p<0.01) BMI (kg/m^2): +0.01 (NS) SBP (mmHg): -7 (p<0.01)	Consultations utilised motivational interviewing techniques to modify behaviour & med titration CRRC pharmacists underwent clinic based training for 12 months with a physician

Study, reference, country	Study aim	Population & setting	Recruitment	Intervention	Outcome measures	Results	Other relevant information
			GP referral if diagnosed with DM or CVD 5.3% lost to follow up.	Creation of individualised diet & PA programs Referral made to nutritionist & physical therapist on an as-needed basis Follow up (30min) every 6-8 weeks Monitor adherence & therapeutic effects, reinforce lifestyle modification, & adjust meds	clinical measures & active smokers	Active smokers (%): -9.6 (p<0.01)	cardiologist. Are certified by local & national DM, lipid & patient assessment programs.
Fremantle Diabetes Study (Clifford et al., 2005) Australia	Examine the effect of a 12-month pharmaceutical care program on vascular risk in type 2 DM.	N=180, setting unclear. M/F adults with type 2 DM from the FDS who were Southern European or Anglo-Celt ethnicity	Recruited during patients annual review. 198 recruited, 180 completed	12 month RTC using a model that could be applied to community pharmacies Intervention group (n=92) conducted by pharmacist Standard FDS assessment. Biochemical tests Assessment at baseline & 6 weekly intervals by telephone & face-to-face at 6 & 12 months (avg 15mins) Pharmacotherapy management, biochemical targets, PA, smoking cessation, diet advice & educational handouts based on National Heart	Primary: change in HbA1c from baseline to 12 months Secondary: change in 10-year CVD risk, BMI, BP, fasting plasma glucose, lipids, urinary albumin/creatinine Self-reported: PA, med use	HbA1c (%): -0.5 vs 0 (p=0.002) 10-year CVD risk – decrease from 25.1% to 20.3% (p=0.002) (for those without CVD history). No change in controls BMI (kg/m^2): -0.6 vs +0.1 (p=0.005) BG (mmol/L): -0.8 vs +0.4 (p<0.001) SBP (mmHg): -14 vs -7 (p=0.002) DBP (mmHg): -5 vs -2 (p=0.043) TC (mmol/L): -0.3 vs -0.2 (NS) HDL-C (mmol/L): +0.03 vs -0.02 (NS) TG (mmol/L): -0.6 vs 0 (NS)	Use of the telephone to provide an inexpensive means of communication between face to face visits Individualised patient education. Goals set for intervention components.

Study, reference, country	Study aim	Population & setting	Recruitment	Intervention	Outcome measures	Results	Other relevant information
				Foundation of Australia recommendations Control group (n=88) Standard FDS assessment Biochemical tests. Baseline & 6 months		PA: no change in duration or intensity Med use: NS increase in antihypertensive, lipid lowering med & sig increase in ACE inhibitor/angiotensin 2 receptor blocker med (p=0.032)	
Pharmacy Diabetes Care Program (Krass et al., 2007) Australia	Assess the impact of a community pharmacy DM service model on patient outcomes in type 2 DM.	N=289, 28 community pharmacies Type 2 DM with HbA1c ≥7.5%, taking at least one oral glucose-lowering med or insulin; HbA1c ≥7.0%, taking at least one oral glucose-lowering med or insulin & on at least one anti-hypertensive, angina or lipid-lowering drug.	Advertisement within pharmacy, or in local papers. Eligibility verified by requesting HbA1c, BP & lipids from GP Over 400 expressed interest. 335 recruited. 84% of intervention & 88% of control patients completed.	6 month RTC Intervention group (N=149) conducted by pharmacist 5 visits in 6 months Daily BG measurement used to inform consultations Adherence support, med review, DM self-management Lifestyle information – PA & Wt loss using 'National PA Guidelines for Australians', 'Dietary Guidelines for Australian Adults' Individual goals set each visit Referral to GP if required Control group (N=140) 2 visits -baseline & end. Usual care	Primary: change in HbA1c between groups over 6 months Secondary change in clinical measures & QoL score between groups over 6 months Changes in BG within intervention group over 6 months	HbA1c (%): -0.97 vs -0.27 (p<0.01) BMI (kg/m^2): -0.4 vs +0.2 (NS) SBP (mmHg): -2.2 vs +2.6 (NS) DBP (mmHg): -2.4 vs -1.3 (NS) TC (mmol/L): -2.1 vs -2.1 (NS) TG (mmol/L): -0.3 vs -0.1 (NS) QoL (utility score): -0.04 vs -0.02 (NS) QoL (health state) +5.3 vs +1.1 (p=0.02) BG (mmol/L): 9.4 baseline vs 8.5 end (p<0.01) BG (proportion in normal range): 39% first baseline 51% end (p<0.01)	Pharmacists received a DM education manual for self-directed learning & attended 2-day workshop Workshop inc lectures on DM, pharmacotherapy, dietary management, & role-playing exercises; training on the use of the DM devices & BP measurement Average intervention given to participants: 33% hypo & hyper control 31% med adherence 29% foot care & lifestyle (PA, nutrition, alcohol, smoking) 4% med history.

Study, reference, country	Study aim	Population & setting	Recruitment	Intervention	Outcome measures	Results	Other relevant information
				For both groups clinical data was sources from GP records at baseline & end.			Overall 92% participants received lifestyle interventions.
Impact of community pharmacy diabetes monitoring (Ali et al., 2012) United Kingdom	Evaluate the impact of a pharmacist-led patient education & DM monitoring programme on HbA1c & other CVD risk factors in the community setting.	N= 46, 2 community pharmacies Type 2 DM on oral med Not on insulin Above 18 years No sig co-morbidity Not involved in any other study Able to attend regular visits HbA1c ≥ 53 mmol/mol	Advertising displayed in pharmacies. Screening computerised patient med records held in the pharmacies GP referral. Patients were invited to take part by letter or at med-dispensing opportunities 216 patients approached. 48 eligible & participated, 2 withdrawn	12 month RTC Intervention group (N=23) - conducted by pharmacist 6 consultations (20-30min) - every month for first 2 months, then every 3 months until 12 months Clinical measurements - every visit: BMI, BP, BG. Months 0, 5 & 12: HbA1c, lipids & questionnaires Pharmacist carried out targeted medicine use review & lifestyle modification counselling. Referral to GP or other healthcare professional when appropriate Control group (N=23) Received usual care by GP & community pharmacy. Seen by pharmacist at 0 & 12 months for clinical measurements & questionnaires	Primary: Change in clinical measures between groups over 12 months Secondary = Questionnaires: DM QoL, beliefs & concerns about meds, satisfaction with information received about meds, health status, DM knowledge test.	Intervention vs control HbA1c (mmol/mol): -17 vs -6 (p<0.001) BMI (kg/m^2): -3.86 vs -1.09 (NS) SBP (mmHg): -20.09 vs +2.95 (p=0.012) DBP (mmHg): -6.09 vs -3.95 (NS) BG (mmol/L): -1.92 vs -0.49 (p<0.001) LDL-C (mmol/L): -0.38 vs -0.56 (p<0.001) HDL-C (mmol/L): +0.27 vs +0.05 (p=0.041) TC (mmol/L): -0.03 vs -0.52 (p<0.001) TG (mmol/L): +0.17 vs +0.34 (NS) DM QoL: -6.33 vs -2.65 (NS) Beliefs about meds Necessity: +2.61 vs -3.43 (p<0.001) Concern: -4.15 vs + 4 (p<0.001) Satisfaction with information received about meds: +3.43 vs -2.74 (p<0.001) Health status: +13.48 vs -3.51 (p<0.001)	Pharmacists undertook 8hr training programme involving workshops with consultant diabetologist & DM nurse specialist, updates on DM management & referrals, overview of diagnostic equipment.

Study, reference, country	Study aim	Population & setting	Recruitment	Intervention	Outcome measures	Results	Other relevant information
						DM knowledge test: +2.24 vs -1.39 (p<0.001)	

Abbreviations: ACR: albumin creatinine ratio, avg: average, BG: blood glucose, BMI: body mass index, BP: blood pressure, CKD: chronic kidney disease, CRRC: Cardiovascular Risk Reduction Clinic, CVD: cardiovascular disease, DBP: diastolic blood pressure, DM: diabetes mellitus, FDS: Fremantle Diabetes Study, GFR: glomerular filtration rate, GP: general practitioner, HbA1c: glycated haemoglobin, HDL-C: high density lipoprotein-cholesterol, Ht: height, LDL-C: low density lipoprotein-cholesterol, HTN: hypertension, mins: minutes, NS: not significant, PA: physical activity, PAART: Pharmacist Assessment of Adherence, Risk and Treatment in Cardiovascular Disease, QoL: quality of life, RTC: randomised controlled trial, RxEACH: Alberta Vascular Risk Reduction Community Pharmacy Project, SBP: systolic blood pressure, TC: total cholesterol, TG: triglycerides, WC: waist circumference, wk: week, Wt: weight, yrs: years.
References: (Krass et al., 2007; McNamara et al., 2012a; Tsuyuki et al., 2016a; Taveira et al., 2006; Clifford et al., 2005; Ali et al., 2012).

The studies summarised in table 8 indicate that lifestyle behaviour modification interventions based in the community pharmacy, implemented by pharmacists can be effective in reducing CVD risk. Specifically, most of the studies found benefit in estimated five- or ten-year CVD risk assessments, BP, HbA1c, BMI and the lifestyle behaviours of medication adherence and smoking.

It is unclear what the extent of training pharmacist received was, specifically for dietary and physical activity lifestyle behaviour change. Three of the six studies specifically mentioned that the pharmacists had received training in lifestyle modification that was included in their overall study participation training module, these ranged from an online course to a two-day workshop (Krass et al., 2007; McNamara et al., 2012a; Tsuyuki et al., 2016a). It is possible that during training as well as when conducting participant consultations greater interventional focus could have been on the areas that pharmacists feel more comfortable within their role; including medicine management and compliance, and smoking cessation. Krass et al. (2007) indicated that over two thirds of participant interventions were focused on blood glucose monitoring, adherence to medications and medication history, 31% of interventions were dedicated to lifestyle behaviours (PA, nutrition, alcohol consumption, smoking and foot-care). This is indicative of a modest amount of attention given to lifestyle behaviours though not equally distributed.

Reported dietary interventions focused on adherence to general national dietary guidelines or dietary guidelines specific to CVD (McNamara et al., 2012a; Clifford et al., 2005; Krass et al., 2007) or creating individualised diet programs (Taveira et al., 2006). Diet quality was only investigated by McNamara et al. (2012a) using a diet quality questionnaire which showed a significant 11.4% increase in adherence to CVD dietary guidelines compared to baseline. Significant areas of improvement were saturated fat and added salt scores. Although there was an increase in dietary quality, overall average scores for both baseline and end indicate a moderate level of compliance with CVD dietary guidelines. Physical activity interventions that were reported in these studies included using national guidelines as resources (Clifford et al., 2005; Krass et al., 2007), setting goals such as 30 minutes of exercise ≥3 times a week (Clifford et al., 2005), or creating individualised exercise programs (Taveira et al., 2006). Physical activity levels were assessed by McNamara et al. (2012a), Clifford et al. (2005) and Krass et al. (2007), with the former showing an increase of 0.5 days of

30 minutes or more moderate activity/week and the latter two no change in physical activity duration or intensity.

Overall it is uncertain what the total extent of dietary and physical activity training received, intervention focus, and the effectiveness of these interventions made by pharmacists in these studies was. Results from McNamara et al. (2012a) suggest that some improvement can be made in these areas with pharmacist intervention. Additional to note is referrals to dietitians, nutritionists or physical therapists were an option for pharmacists if they perceived it was required in three of the studies, indicating a potential need for more expertise with regard to these lifestyle factors (McNamara et al., 2012a; Taveira et al., 2006; Ali et al., 2012).

The role of the pharmacist

Pharmacists are experts when it comes to the pharmacotherapy management of CVD risk factors i.e. BP lowering, lipid lowering, anti-thrombotic, diabetic and smoking cessation medications and their adherence. However, pharmacists' depth of knowledge about other risk factors may be less in comparison i.e. diet, weight management and PA. General nutritional knowledge of pharmacists has been demonstrated to be low but can increase with training (Chang et al., 2008; Pearce & Cross, 2013). However even with training Pearce & Cross (2013) demonstrated that Australian pharmacy students overall nutritional knowledge was less than the general population. This was specifically for general dietary recommendations, knowledge of food sources related to nutrients and ability to choose appropriate everyday foods. Nutrition knowledge with respect to diet-disease relationships was comparable with the general population. One possible explanation of this difference is that the course these students attended covered key nutritional aspects that a pharmacist needs to know in practice, not specifically general nutrition knowledge that the questionnaire tested. Additionally, a systematic review of pharmacist knowledge of dietary supplements concluded that knowledge was poor and there is significant scope for improvement (Waddington et al., 2015).

With weight management there is a perceived lack of expertise, knowledge, skills and self-confidence among pharmacists (Newlands et al., 2011; Dastani et al., 2004; Fakih et al., 2015), however this has been shown to increase with training (Um et al., 2016).

Most weight management programmes provided in Australian pharmacies are product related (Fakih et al., 2015; Rieck, Clifford & Everett, 2006). Dietitians were the most selected health professional (55.3%) to provide weight loss advice by the general public in a questionnaire about weight management and the role of pharmacists. Pharmacists were selected 1.2% of the time (George et al., 2010). Pharmacists' confidence in providing physical activity recommendations to individuals has been demonstrated to be low, including to those with CVD, but again has been shown to improve with training (Dirks-Naylor et al., 2018; Persky, 2009). Together these studies indicate pharmacists may have less than adequate nutritional and physical activity knowledge and confidence to undertake lifestyle behaviour modification interventions in this area. Consultations involving dietary and physical activity behaviour change may be more suited to the expertise of a dietitian.

A role for the dietitian

A greater emphasis on diet, weight management and physical activity could produce greater reductions in CVD risk than have been observed in pharmacist led CVD interventions. In NZ the Scope of Practice for the dietitian is to work in partnership with individuals, whānau, communities and populations, in states of health and disease, to support optimal health and well-being. They do this by evaluating the scientific evidence of food and nutrition and translating it into practical strategies (Dietitians Board, 2017). Dietitians follow the internationally standardised Nutrition Care Process to provide high quality care that includes nutrition assessment, nutrition diagnosis, nutrition intervention and nutrition monitoring and evaluation (also called the ADIME approach) (Andersen et al., 2018). Dietitians are trained to interpret test results related to nutritional status e.g. blood measures, BP, anthropometrics (height, weight, BMI, WC and body composition) (Andersen et al., 2018).

Dietetic prevention and management of weight, hypertension, dyslipidaemia and diabetes is statistically and clinically effective, including success at improving metabolic risk factors discussed earlier in this review (Briggs & Stanley, 2018). For example, two to 12 consultations with a dietitian can reduce LDL-C by 0.39mmol/L to 1.22mmol/L (Briggs & Stanley, 2018). Energy expenditure is crucial when managing weight as well as having many benefits for chronic disease reduction. Thus dietitians need to be skilled and knowledgeable about recommended guidelines of

physical activity and how to implement it in an individual's lifestyle (Raynor & Champagne, 2016).

Motivating behaviour change is an important skillset that dietitians bring to the clinical field. Training and expertise using a patient centered approach and behaviour change paradigms such as the stages of change model, cognitive behaviour therapy, motivational interviewing and acceptance and commitment therapy can lend dietitians to being effective at implementing lifestyle behaviour interventions (Raynor & Champagne, 2016). The ability to individualise nutritional interventions is key to intervention success (Briggs & Stanley, 2018).

A combined approach – dietitians in the community pharmacy setting

For these reasons, a dietitian could provide comprehensive dietary and physical activity interventions that are tailored to the individual's needs and risk factors. This may have a greater impact on motivating behaviour change than that of a pharmacist in this domain. At the same time a community pharmacist could additionally provide valuable medication management to complete the lifestyle intervention. From the research conducted with this literature review there was no evidence that this dietitian-pharmacist approach to CVD primary prevention has been trialed before.

An encompassing model of lifestyle behaviour change intervention would require a multidisciplinary approach and collaboration between pharmacists and dietitians. Evidence demonstrates that a collaborative and patient-centred model of care is beneficial to improving control of chronic diseases by linking the different aspects of healthcare to the health and wellness of the individual (McGill & Felton, 2007; Omboni & Caserini, 2018; Bischoff et al., 2017; Houle et al., 2014; Jennings & Astin, 2017; Taylor et al., 2005; Carter et al., 2009). The assistance of dietitians in multidisciplinary teams to reduce the risk of CVD specifically is recommended because of the significant influence lifestyle factors have on CVD risk (Masana et al., 2017; Liebson & Amsterdam, 1999; Millen et al., 2014). Through collaboration dietitians and pharmacists may be able to provide more effective lifestyle interventions targeting CVD risk factors, and therefore produce better clinical outcomes than have been reported in the literature.

Conclusion

Cardiovascular disease is a major contributor to the morbidity and mortality of New Zealanders as well as placing a significant impact on the NZ health system. Many factors influence a person's chance of developing CVD, but behavioural and metabolic factors can be successfully adjusted to lower risk. These include modifying diet, physical activity and medication related lifestyle behaviours to improve BP, blood lipids, blood glucose and weight. Community pharmacies are well placed to be settings for the identification and management of CVD. Internationally, programmes led by pharmacists in community pharmacy settings have adopted lifestyle behaviour change approaches and have shown promising results. However, the extent and effectiveness of dietary and physical activity intervention alongside medication management warrants further investigation as pharmacists are not experts in this area. Dietitians offer specialist competence in regard to diet, physical activity and lifestyle behaviour change and could produce improvements in risk factors additional to what has been observed. An opportunity exists to examine the impact of a multidisciplinary approach between dietitians and pharmacists in the community pharmacy setting to implement a CVD primary prevention programme using lifestyle behaviour change. To the researcher's knowledge this has not been undertaken previously in NZ or abroad.

Chapter Three

Manuscript

Abstract

Background: Community pharmacy CVD primary prevention interventions, led by pharmacists, are effective. However, the majority of these have targeted single CVD risk factors and most have not adequately assessed the impact of dietary and physical activity behaviour. A multidisciplinary and multi-risk factor approach that involves collaboration between dietitians (dietary and physical activity consultations) and pharmacists (pharmacological treatment) may provide additional risk reduction benefits for participants.

Objective: To assess the feasibility of implementing a community pharmacy-based CVD primary prevention programme using a multidisciplinary approach to motivate lifestyle behaviour change in participants at risk of CVD. The primary outcome was change in estimated five-year CVD risk.

Methods: A 16-week single cohort pre- and post-test study was undertaken in two community pharmacies with twelve participants aged 40-74 years who had risk factors associated with increased CVD. Participants received dietary and physical activity advice at baseline and every four weeks by a student dietitian as well as pharmacological management assessment at baseline, 16 weeks and as needed by a pharmacist. Biochemical (blood lipids, BP, HbA1c) and anthropometric (body composition, weight, height, waist and hip circumference) measures were compared at baseline, eight and 16 weeks. Behavioural measures (diet, physical activity and medication use) were compared between baseline and 16 weeks.

Results: Eleven participants (68±5.2 years) completed the programme. Significant reductions from baseline to 16 weeks were observed for mean systolic and diastolic BP (-5.47, $p = 0.04$ and -4.06mmHg, $p = 0.01$ respectively) and mean TC reduced significantly from baseline to eight weeks, (-0.43mmol/L; $p = 0.005$) but not between baseline and 16 weeks. The average diet quality score significantly improved by 12.6% from 65.9 to 74.2 out of 100 during the intervention period ($p = 0.007$). Other CVD risk factor measures showed a trend towards improvement. Five-year CVD risk did not significantly improve.

Conclusions: Results are comparable to existing literature on interventions to reduce CVD in the community pharmacy setting. Findings within this small cohort, particularly the improvements seen in diet, support the inclusion of dietitians for the primary prevention of CVD in community pharmacies. A larger scale, controlled study will help in determining the extent of efficacy with this approach.

Introduction

Despite improvements in diagnosis and treatment, CVD remains a leading cause of morbidity and mortality in NZ. It is estimated that over one quarter of the NZ population has a 5% or greater five-year risk of CVD (Pylypchuk et al., 2018). The primary prevention of CVD occupies an important of portion of public health efforts to reduce the incidence of chronic disease. Education and counselling interventions are believed to be cost-effective and beneficial to CVD prevention and can provide a greater degree of risk factor control than medication alone (Appel, 2004; Puska et al., 1985; Farquhar et al., 1990). Further, evidence suggests that targeting individuals who are at high risk of CVD may be the most efficacious approach (Ebrahim et al., 2011).

The community pharmacy setting is well placed for the primary prevention of CVD because the pharmacy offers the public accessibility to trained health professionals without an appointment, locality and extended open hours. Internationally community pharmacies have been shown to be successful at screening individuals to identify those at risk of developing CVD (Liu et al., 2008; Donyai & Van den Berg, 2009; Horgan et al., 2010; Snella et al., 2006). Interventions in this setting to reduce CVD risk have also been shown to be effective. A recent systematic review of 27 studies on interventions to reduce CVD risk implemented in community pharmacy settings found reductions in SBP (7.8-17.3mmHg), HbA1c (5-24mmol/mol), and TC (0.47-0.70mmol/L) (Ifeanyi et al., 2015). The researchers acknowledged that the quality of many of the studies was generally poor due to heterogeneity of design and intervention, and participant disease severity. Additionally, the majority of studies reviewed focused on only one or two of the risk factors for CVD as opposed to targeting multiple risk factors, which is recognized as being more effective at reducing overall CVD risk (Eckel et al., 2014; Ministry of Health, 2018a).

Only a handful of studies that address multiple risk factors of CVD with lifestyle modification in a community pharmacy setting have been conducted (Krass et al., 2007; McNamara et al., 2012a; Tsuyuki et al., 2016a; Clifford et al., 2005; Ali et al., 2012). All of the interventions were led by pharmacists and most found improvements in estimated five- or ten-year CVD risk, BP, HbA1c, BMI and the lifestyle behaviours of medication adherence and smoking. The only study that measured diet found an increase in diet quality (McNamara et al., 2012a). One study reported an improvement in physical activity while two demonstrated no change (McNamara et al., 2012a; Clifford et al., 2005; Krass et al., 2007). It is unclear how much training the pharmacists received regarding dietary and physical activity, the extent to which these were a focus during interventions, and the degree of behaviour change produced.

Pharmacists are experts when it comes to the pharmacotherapy management of CVD risk factors. However, their depth of knowledge, expertise and confidence with other modifiable behavioural risk factors such as diet, weight management and physical activity has been reported to be low or less than adequate (Chang et al., 2008; Pearce & Cross, 2013; Newlands et al., 2011; Dastani et al., 2004; Fakih et al., 2015; Dirks-Naylor et al., 2018; Persky, 2009). Given that diet, physical activity and weight management play a major role in CVD risk, a stronger emphasis on these factors alongside medication management could produce improved outcomes. One way to achieve this can be through personalised dietary and physical activity advice and feedback provided by a dietitian, who's scope of practice is more aligned to these elements (Andersen et al., 2018; Dietitians Board, 2017). A multidisciplinary approach including dietetic consultations alongside pharmacist pharmacological management tailored to an individual's needs and risk factors would provide a complete lifestyle intervention. This could provide greater efficacy at reducing overall CVD risk than pharmacist led intervention alone (Jennings & Astin, 2017).

This feasibility study investigates the efficacy of using a multidisciplinary approach between dietitians and pharmacists to reduce the risk of CVD in a community pharmacy setting. The aim of the study was to motivate lifestyle behaviour change to produce a measurable effect on five-year CVD risk as well as on individual CVD risk factors.

Methods

Study design

The programme was a 16 week pre- and post-test feasibility study with a single cohort of participants at risk of CVD. Two community pharmacies in the greater Auckland area served as the setting for recruitment and intervention. The study was approved by the Massey University Human Ethics Committee SOA 18/63. All participants provided informed written consent.

Participants

Each pharmacy was asked to recruit participants aged between 40 to 74 years who were taking cholesterol and/or lipid lowering medication but did not have established CVD or a complex medical condition such as organ damage or cognitive impairment. Patients were also required to be available to attend monthly consultations in the pharmacy between May and September, live independently and be proficient speakers of English. Prospective participants were identified and approached by dispensary staff to discuss the programme and given a flyer (Appendix 1.1). Posters (Appendix 1.2) were displayed in waiting areas within the pharmacies. Prospective participants who expressed an interest were given the study information sheet (Appendix 1.3) and the screening questionnaire (Appendix 1.4) to access their eligibility against the participation criteria. The screening questionnaire could be completed online using Qualtrics online survey software, on a printed form, or via phone conversation. Participants meeting the screening criteria were invited to attend the baseline assessment which included biochemical measurement (BP, TC and HDL-C) to assess their five-year CVD risk using the NZ-based PREDICT equations (Pylypchuk et al., 2018). If participants had less than 5% CVD risk, they were ineligible for the study and were instead provided with National Heart Foundation of New Zealand resources related to lifestyle-based CVD prevention (National Heart Foundation of New Zealand, 2019b). If their risk was 5% or greater they were invited to participate in the programme.

The intervention

A student dietitian in their second year of a two year Master's Degree to gain dietetic practice registration requirements led the intervention components over the 16 weeks. Participants were given lifestyle consultations related to CVD every four weeks for a total of five visits. An outline of the intervention procedures is provided in table 9.

Primary outcome

The primary outcome was the mean change to five-year risk of CVD between baseline, week eight and week 16.

Secondary outcomes

Changes to metabolic CVD risk factors (BP, lipid profile, HbA1c, and anthropometric measures) between baseline, week eight and week 16 and changes in behavioural CVD risk factors (diet quality, physical activity and medication adherence) between baseline and week 16. Programme related measures (participant recruitment, retention, and participant evaluation of the programme) were also examined.

Biochemical and anthropometry measurements

Biochemical and anthropometric measurements were taken at baseline, week eight and week 16. Five-year CVD risk was also recalculated at these stages. Participants were provided their results in real time as well as interpretation and education from the student dietitian. Results were reviewed in relation to National recommendations and the participants' previous results.

A finger prick blood sample was used to give blood lipid profile measurements (TC, LDL-C, HDL-C, TG, TC/HDL-C ratio) and an HbA1c level, using an Afinion™ 2 Analyser point of care meter in accordance with instructions provided by the manufacturer. Blood pressure was measured using Riester ri-champion® N digital BP monitor following American Heart Association Guidelines (Pickering et al., 2005).

Height, waist and hip circumferences were measured in accordance with the World Health Organization protocol using a portable stadiometer and Lukfin tape (World Health Organization, 2008). Weight and body composition (fat mass, skeletal muscle

mass) were measured using a Biospace InBody230 Bioelectrical Impedance Analysis scales. Body mass index and WHR ratio were then calculated.

Questionnaires

Participants completed the health and demographic questionnaire (Appendix 1.5) during the first visit, and the short form International Physical Activity Questionnaire (IPAQ) (IPAQ Group, 2012) and the Healthy Heart Food Index (HHFI) during their first visit and at week 16. The HHFI was developed and validated (Beck, 2019) to compare an individual's dietary intake with recommendations made by the National Heart Foundation of New Zealand (National Heart Foundation of New Zealand, 2019a). It measures consumed amounts of fruit and vegetables, carbohydrates and wholegrains, protein, dairy, healthy oils, unhealthy fats, sweet foods, salty foods, and takeaways. If possible, this questionnaire was completed online before the first session so it could be reviewed by the student dietitian before the dietetic consultation. During the last visit, participants completed an evaluation questionnaire (Appendix 1.6) to ascertain the impact of the programme from the participant's point of view.

Pharmacist consultation

A pharmacist consultation was conducted at baseline, with consultations lasting between five to 15 minutes depending on the complexity of the participant. They were facilitated by completing a medication usage questionnaire (Appendix 1.7) to assess compliance to medication and identify any issues participants might have that related to their medicine usage. Participants received information on medications if required. The same pharmacist consultation was also competed at week 16. Participants could also consult with the pharmacist as needed throughout the programme.

Dietetic and physical activity consultation

The dietetic and physical activity consultation was administered by the student dietitian (see appendix 1.8 for consultation assessment form). The initial consultation (week 0) took approximately 45 minutes for each participant using an individualised Nutrition Care Process approach (Andersen et al., 2018). An emphasis was placed on establishing motivations for change, identifying areas of concern related to CVD risk factors, and education on relevant aspects of lifestyle and CVD risk. Areas where dietary and physical activity changes could be made to reduce risk were identified and discussed. At the end of the consultation, lifestyle goals were set for the

programme. This included two nutrition goals and one physical activity goal. The National Heart Foundation of New Zealand resources were provided at the end of the first session and referred to throughout the programme.

Participants were also given a Fitbit (Flex 2™) as a means of recording daily step count and to provide motivation for doing PA. The Fitbit was set up on the participants smart phone and they were educated on how to use it during the first session. Fitbit results were reviewed at each follow up session. Step counts were calculated using the median daily step count every four weeks. This was to account for fluctuations in device use such as forgetting to wear and low battery.

Follow up consultations (weeks 4, 8, 12 and 16) ran for approximately 30 minutes. Again consultations followed the ADIME approach. Previous goals were reviewed and adjusted if required, further education was provided, and new goals were determined. Up to three lifestyle goals were set each session for the duration of the programme, depending on the requirements and progress of the participant. Participants were asked to complete a weekly diary to record any major changes to their health and weekly routine. Any changes noted were discussed with the participant (Appendix 1.9).

Table 9: Outline and order of intervention procedures.

Week 1 (baseline) 125min	**Week 4 45min**	**Week 8 (mid) 80min**	**Week 12 45min**	**Week 16 (end) 110min**
Study introduction (4min)	Weekly diary review (5min)	Weekly diary review (5min)	Weekly diary review (5min)	Weekly diary review (5min)
Consent form (1min)	Goal review (5min)	Goal review (5min)	Goal review (5min)	Goal review (5min)
Biochemical measurement (20min)		Biochemical measurement (20min)		Biochemical measurement (20min)
Five-year CVD risk calculation (5min)		Anthropometry measurement (10min)		Anthropometry measurement (10min)
HDQ (5min) IPAQ (5min) HHFI (5min)				
Anthropometry measurement (10min)		Five-year CVD risk calculation (5min)		Five-year CVD risk calculation (5min)
Pharmacist consultation (5-15min)				Pharmacist consultation (5-15min)
Dietary & PA consultation (45min)	Dietary & PA consultation (30min)	Dietary & PA consultation (30min)	Dietary & PA consultation (30min)	Dietary & PA consultation (30min)
Goal setting (5min)	Goal setting (5min)	Goal setting (5min)	Goal setting (5min)	
Fitbit setup (5min)				IPAQ (5min) HHFI (5min) PES (10min)

Abbreviations: CVD: cardiovascular disease, HHFI: Healthy Heart Food Index, HQQ: health and demographic questionnaire, IPAQ: International Physical Activity Questionnaire, min: minutes, PA: physical activity, PES: participant evaluation survey.

Statistical methods

All study data was entered into Microsoft Excel, with subjects only identified by their unique study number. Analysis was performed using IBM SPSS Statistics Version 22.0 (IBM Corp, 2013). Before statistical tests were carried out, the variables were tested for normality using the Kolmogorov–Smirnov test and normality plots. Normally distributed data was summarised as the mean and standard deviation (SD). Non-normally distributed data was log transformed into approximately normal distributions and reported as geometric mean and 95% confidence interval (CI). Categorical data was expressed as frequencies.

Descriptive statistics were used to describe the subjects' characteristics at baseline. Participant socioeconomic status was estimated using the NZ Index of Multiple Deprivation based on home address (University of Auckland, 2013). Changes in anthropometric, biochemical variables were tested using repeated measures ANOVA. Greenhouse-Geisser correction was used if a variable failed sphericity testing. Significant repeated measures ANOVA results were further tested by post hoc analysis with Fisher's Least Significant Difference correction. Paired sample *t*-tests were used to measure changes in behavioural variables. The level of significance used in all tests was $p<0.05$.

Results

Programme participation

Twenty-four people expressed interest in enrolling in the programme from which twelve participants were recruited. People who expressed interest were lost to recruitment because they declined to participate ($n= 3$), they did not reply to contact ($n= 1$), they did not meet the eligibility criteria ($n= 6$), or participant numbers were met ($n= 2$). Table 13 (Appendix 2) displays the stages of the programme with the number of people that completed each stage. One participant withdrew at eight weeks (due to illness) and was excluded from data analysis. Two other participants also withdrew early (week 12 and week 15; due to travel), but their results have been included in the analysis.

Participant characteristics

Baseline demographic information of the 11 participants who completed the programme are outlined in table 10. The mean age was 68 (±5.2) years (range= 60-74 years). Of the participants three were taking lipid lowering medication, three were taking BP medication and five were taking both.

Table 10: Baseline demographic characteristics of participants.

Age, years, mean (*SD*)	68 (5.2)
Sex, *n* (%)	
Male	6 (54.5)
Female	5 (45.5)
Ethnicity, *n* (%)	
European	11 (100)
Deprivation quintile, *n* (%)	
1 least deprived	2 (18.2)
2	1 (9.1)
3	5 (45.5)
4 most deprived	3 (27.3)
Smoking status, *n* (%)	
Never smoked	8 (72.7)
Ex-smoker	3 (27.3)
Consumes alcohol, *n* (%)	
No	3 (27.3)
Yes	8 (72.7)
Family history of CVD, *n* (%)	6 (54.5)
Relevant medical conditions, *n* (%)	
Type 2 diabetes	1 (9.1)
Atrial fibrillation	2 (18.2)
Medications, *n* (%)	
Lipid lowering medication	8 (72.7)
Blood pressure medication	8 (72.7)
Antithrombotic medication	5 (45.5)

Abbreviations: n: number, SD: standard deviation.

Metabolic risk factors

Table 11 shows that baseline average five-year CVD risk was 9.29%, indicating intermediate risk (5-15%). The average TC was high (>4.0mmol/L) at 5.13mmol/L (range 3.34-7.87). However, nine participants (seven on lipid lowering medication) had a TC/HDL-C ratio less than four (recommended level for those with known CVD or a CVD risk >15% or diabetes. The average BP was 140.2/80mmHg. One (not on blood pressure medication (BPM)) was normal (<120/<80mmHg), four (three on BPM) were prehypertensive (120-139/80-89) and six (five on BPM) were stage one

hypertensive (140-159/90-99mmHg). The majority of participants can be classified as overweight (n=6, BMI 25-29.9kg/m^2) or obese (n=3, (≥30kg/m^2). Two participants had a HbA1c in the prediabetic range (41-49mmol/mol) and one in the diabetic range (>50mmol/mol).

Changes to the metabolic risk factors are reported in table 11. Geometric mean TC differed between measurements [F (2, 20) = 5.229, p = 0.015]. A significant reduction of 0.43mmol/L (p = 0.005) from baseline to middle time points, but an insignificant decrease of 0.36mmol/L (p = 0.061) from baseline to end was found. Mean SBP [F (2, 20) = 3.545, p = 0.048] and geometric mean DBP [F (2, 20) = 5.353, p = 0.014] also differed between measurements. Post hoc tests revealed they both reduced by a mean of 5.47mmHg (p = 0.041) and 4.06mmHg (p = 0.01) respectively from baseline to end time points. Improvements were also observed for most other metabolic risk factors, however were not statistically significant. These included reductions in weight (-1.12kg), BMI (-0.22kg/m^2), waist circumference (-1.6cm), hip circumference (-1.1cm), fat mass (-1.01kg, -0.81%), and LDL-C (-0.24mmol/L). The majority of the reduction in weight (91.8%) can be attributed to a loss in fat mass rather than muscle mass. Five-year CVD risk percentage decreased by 0.12 between baseline and 16 weeks, though not significantly.

Table 11: Comparison of metabolic measures between baseline, middle and end.

Metabolic measures	Baseline (week 0)	Middle (week 8)	End (week 16)	Change: baseline - end[c]	*p* value
Anthropometry					
Weight (kg)[a]	84.9 (15.7)	84.0 (14.9)	83.8 (14.2)	-1.12 (-2.98, 0.75)	0.197
BMI (kg/m^2)[b]	28.8 (1.17)	28.6 (1.16)	28.6 (1.15)	-0.22 (-1.27, 0.54)	0.414
WC (cm)[a]	101.2 (13.7)	100.7 (12.8)	99.6 (9.61)	-1.60 (-5.19, 1.99)	0.381
HC (cm)[b]	108.2 (1.12)	108.1 (1.12)	107.0 (1.11)	-1.13 (-3.06, 0.31)	0.183
WHR	0.93 (0.07)	0.93 (0.07)	0.93 (0.06)	0.00 (-0.03, 0.02)	0.845
Fat mass (%)	35.3 (8.34)	34.5 (7.81)	34.5 (8.65)	-0.81 (-0.03, 0.01)	0.473
Fat mass (kg)[b]	28.8 (22.9, 36.1)	27.9 (22.6, 34.4)	27.7 (22.5, 34.3)	-1.01 (-4.04, 1.07)	0.349
Skeletal muscle mass (%)	36.0 (4.67)	36.1 (4.60)	36.1 (5.19)	+0.17 (-1.20, 1.50)	0.932
Skeletal muscle mass (kg)	30.4 (5.77)	30.3 (5.75)	30.2 (6.14)	-0.14 (-1.18, 0.90)	0.943
Biochemical					
TC (mmol/L)[b]	4.94 (4.08, 5.99)	4.51 (3.77, 5.39)	4.59 (3.79, 5.55)	-0.36 (-0.81, 0.02)	0.015*
HDL-C (mmol/L)	1.62 (0.47)	1.56 (0.43)	1.50 (0.40)	-0.11 (-0.26, 0.03)	0.122
LDL-C (mmol/L)[b]	2.58 (1.94, 3.42)	2.30 (1.76, 3.00)	2.34 (1.77, 3.10)	-0.24 (-0.64, 0.08)	0.074
TC/HDL-C	3.30 (0.96)	3.12 (0.97)	3.27 (0.95)	-0.03 (-0.28, 0.22)	0.306
TG (mmol/L)	1.57 (0.43)	1.36 (0.53)	1.57 (0.44)	0.00 (-0.28, 0.28)	0.234
HbA1c (mmol/mol)[b]	39.0 (1.15)	39.2 (1.14)	39.4 (1.13)	+0.43 (-0.85, 1.46)	0.693
Blood pressure (mmHg)					
Systolic	140.2 (10.52)	136.7 (10.64)	134.7 (10.72)	-5.47 (-10.6, 0.27)	0.048**
Diastolic[b]	79.8 (1.08)	79.5 (1.07)	75.7 (1.11)	-4.06 (-6.51, -2.38)	0.014**
Five-year CVD risk (%)	9.29 (4.93)	9.11 (5.11)	9.17 (4.99)	-0.12 (-0.43, 0.19)	0.530

*Significant difference between baseline and middle, **significant difference between baseline and end.

Abbreviations: BMI: body mass index, CVD: cardiovascular disease, HbA1c: glycated haemoglobin, HC: hip circumference, HDL-C: high density lipoprotein-cholesterol, LDL-C: low density lipoprotein-cholesterol, TC: total cholesterol, TC/HDL-C: total cholesterol/high density lipoprotein-cholesterol, TG: triglyceride, WC: waist circumference, WHR: waist to hip ratio.
Values: Mean (SD) unless otherwise indicated, [b]geometric mean (95% CI), [C]mean (95% CI).
Analysis: Repeated measures ANOVA with [a]Greenhouse-Geisser correction for weight and waist circumference.

Behavioural risk factors

At baseline (table 12) participants' dietary quality scored 66 out of 100. Participants engaged in more than the recommended amount of moderate intensity physical activity (>600 MET-min/week), and total physical activity was 2569 MET-min/week. Medication compliance was 100% in all but one participant, whose score was 75%.

Changes in the measured behavioural risk factors are reported in table 12. Participants significantly increased their diet quality score by 8.24 points (t (10) = -3.341, p = 0.007), an increase of 12.6% from baseline to end. Individual diet quality measures improved but were non-significant. These were the consumption of fruit, wholegrains, protein, healthy oils, unhealthy fats, dairy, sugary and salty foods, and takeaways. Other behavioural risk factor measures improved but not significantly. These were: self-reported total, moderate and vigorous physical activity (+199, +138, and +160 Met-min/week respectfully), and alcohol consumption (-0.63 drinks/week). Average steps taken by participants peaked at a mean of 10,374 steps/day between baseline and week four and were lowest at 8,988 during weeks eight to 12 (table 14, Appendix 2). Baseline step count was not recorded. Medication compliance was 100% for all participants at the end of the programme.

Table 12: Comparison of behavioural measures between baseline and end.

Behavioural measures	Baseline (week 0)	End (week 16)	Change[a]	*p* value
Diet (score)	65.9 (8.26)	74.2 (8.17)	+8.24 (2.75, 13.7)	0.007*
Fruit consumption	7.72 (4.10)	9.09 (2.02)	+1.36 (-1.28, 4.00)	0.277
Vegetable consumption	8.18 (2.52)	6.36 (2.51)	-1.36 (-3.54, 0.81)	0.192
Carbohydrate consumption	3.86 (1.81)	3.30 (1.40)	-0.57 (-1.83, 0.70)	0.341
Choosing wholegrains	7.73 (1.75)	8.86 (1.31)	+1.14 (-0.43, 2.71)	0.138
Protein consumption	8.00 (4.22)	10.0 (0.00)	+2.00 (-1.02, 5.02)	0.168
Healthy oil consumption	2.95 (2.18)	3.64 (2.34)	+0.68 (-0.84, 2.20)	0.341
Unhealthy fat consumption	3.18 (2.52)	4.55 (1.51)	+1.36 (-0.21, 2.93)	0.082
Dairy consumption	6.36 (3.93)	8.64 (2.34)	+2.27 (-0.87, 5.41)	0.138
Sugary food consumption	4.55 (1.51)	5.23 (2.36)	+0.68 (-1.17, 2.54)	0.432
Salty food consumption	4.09 (1.69)	4.32 (1.62)	+0.23 (-0.28, 0.73)	0.341
Takeaway consumption	9.09 (3.02)	10.0 (0.00)	+0.91 (-1.12, 2.93)	0.341
Physical Activity (MET-min/week)				
Walking	1293 (1141)	1194 (557)	-99.0 (-780, 582)	0.75
Moderate	840 (1743)	978 (1030)	+138 (-657, 357)	0.71
Vigorous	436 (1278)	596 (903)	+160 (-281, 933)	0.44
Total	2569 (2607)	2769 (1930)	+199 (-935, 1333)	0.70
Sedentary (hours)	3.59 (1.80)	3.86 (1.07)	+0.27 (-0.78, 1.33)	0.58
Medication Compliance (%)	97.7 (7.54)	100 (0.00)	+2.27 (-2.78, 7.34)	0.17
Alcoholic drinks/week (number)	4.37 (4.27)	3.75 (2.82)	-0.63 (-3.39, 2.14)	0.61

*Significant differences between baseline and end.
Values: Mean (SD) unless otherwise indicated, [a]mean (95% CI).
Analysis: Paired samples t-test.

Programme evaluation

Responses to the evaluation survey questionnaire are reported in table 15, Appendix 2. The majority of the participants rated the programme as excellent (n=9) and all said they would recommend the programme to someone else. All participants believed they acquired new knowledge and made improvements to their diet and most (n=8) to their PA. Participants strongly agreed (n=7) and agreed (n=4) that they will continue with the lifestyle changes they had made. Most (n=10) stated that their medication knowledge and compliance hadn't changed. Participants were asked how much they would pay for a programme such as this and responses ranged from $0 to $200/session, with the average being $58. The most valuable aspects of the programme identified by participants was the information provided, an increase in motivation and receiving regular test results.

Discussion

To our knowledge this is the first study to examine the clinical effects of a multiple CVD risk factor reduction intervention in community pharmacies utilising the expertise of dietitians and pharmacists. The findings suggest it is feasible to implement such a lifestyle change programme within this setting and it has the potential for clinical benefits. The main outcome factor, five-year CVD risk, did not significantly change when comparing baseline results to the middle and end of the programme. However, participants significantly improved modifiable CVD risk factors including diet quality, BP and TC levels by the end of the programme. Other measures that showed improvements included total, moderate and vigorous physical activity, weight, WC, HC, fat mass, and LDL-C, though they were not significant. Participants that completed the programme stated that it was very beneficial and believed they had improved their knowledge of diet and physical activity and had sustainably changed their lifestyle.

Participants in this study were identified as being at an intermediate risk of CVD with an average five-year risk of 9.29%. During the programme this risk percentage decreased but not significantly (-0.12). The variability in this measure was large, ranging from a risk percentage decrease of 1.11 to an increase of 0.46. The modifiable risk factors that influence the five-year CVD risk calculation are SBP and the

TC/HDL-C ratio. In this study SBP decreased significantly whereas the TC/HDL-C ratio did not. No change in TC/HDL-C ratio likely contributed to the insignificant change that was seen for five-year CVD risk. Most participants met the recommended level for the TC/HDL-C ratio at baseline which may have prevented a meaningful degree of reduction with this measure. In comparison, McNamara et al. (2012a) found a significant 1.7 decrease in five-year CVD risk percentage with a similar but longer six-month intervention. They also found no change in TC/HDL-C ratio but observed a greater reduction in SBP, which gave rise to the significant decrease in CVD risk.

Blood pressure improved significantly throughout the programme (mean change -5.47mmHg systolic/-6.25mmHg diastolic) which moved the average BP classification from stage one hypertension at baseline to prehypertension at end. Three participants improved their BP classification in this way. Total cholesterol significantly decreased from baseline to eight weeks by 0.43mmol/L and insignificantly decreased from baseline to end by 0.36mmol/L. No significant change was seen in HbA1c levels, however, one participant moved from being classified as prediabetic at baseline to having normal glycaemia.

The improvements seen in SBP and TC are slightly lower than those reported by Ifeanyi et al. (2015) in their review of community pharmacy-based CVD reduction studies that mostly used single risk interventions (SBP range 7.8-17.3mmHg, and TC range 0.47-0.70mmol/L). When compared to interventions that have addressed multiple CVD risk factors in the community pharmacy setting, the current study reports similar to lower reductions in SBP (range 2.2-20.1mmHG) and similar to greater reductions in TC (range 0.12-2.1mmol/L) (Tsuyuki et al., 2016a; McNamara et al., 2012a; Krass et al., 2007; Ali et al., 2012; Clifford et al., 2005; Taveira et al., 2006). The most similar analysis to the current study, the PAART (Pharmacist Assessment of Adherence, Risk and Treatment in Cardiovascular Disease) pilot study conducted in Australia, achieved similar reductions in anthropometric measures, including weight, BMI, and WC. However, in their study with a larger patient population, these changes reached statistical significance (McNamara et al., 2012a). It is encouraging that the results of this small sampled feasibility study are comparable to the outcomes of much larger interventions.

The major difference between the current study and other multiple CVD risk factor interventions based in the pharmacy setting was the use of a dietitian to consult on dietary and lifestyle behaviours. Diet quality increased significantly by 12.6%. Improvements in the consumption of fruit, wholegrains, protein (particularly oily fish), healthy oils, unhealthy fats, dairy (particularly low fat dairy), sugary and salty foods, and takeaways contributed to the increase in dietary score. If maintained these changes could be expected to further positively impact metabolic risk factors. The PAART also measured diet quality. They found an equivalent dietary score improvement (11.4% increase) which assessed diet quality in terms of cardiovascular health (McNamara et al., 2012a). Specifically, their participants improved in regard to only saturated fat and salt intake and not fruit and vegetable, omega-3 or fibre intake. This may reflect a greater breath of nutritional intervention when administered by a dietitian in comparison to a pharmacist.

Other lifestyle behaviours also improved in the current study but not significantly such as self-reported moderate, vigorous and total PA. Inference is difficult as the variability between participants was large, but participants may have transitioned walking time into increased moderate and vigorous PA. Only three studies had measured physical activity, the PAART study found a significant increase of 0.5 days of 30 minutes or more moderate physical activity / week whereas the two other studies found no change (Clifford et al., 2005; Krass et al., 2007; McNamara et al., 2012a). The extent of physical activity change in the current study is directionally greater than the PAART study. Given a greater population size and less variability, the current study may have shown a greater improvement in PA. Changes in medication compliance were difficult to assess because baseline compliance was 100% for all but one participant. This participant had improved their compliance by the end of the programme.

Examining programme related measures found that half of all that expressed interest ended up participating, and three quarters of these completed all 16 weeks of the programme. Advertisement posters in the pharmacy did generate some interest in the study, but it was the active involvement of and targeting efforts made by dispensary staff that generated the majority of the expressions of interest. Other studies have used screening of pharmacy databases to identify those who may be eligible, this can be explored in the future (Tsuyuki et al., 2016a; McNamara et al., 2012a). Positive

feedback was also received from the staff of both pharmacies that took part in the study. The participant evaluation questionnaire found the majority of participants (n=8) thought the length of the programme and time between sessions was 'about right'. Those that did complete the programme stated that they had increased their knowledge and behaviour regarding diet and physical activity, and this is supported by the results. Importantly all participants 'strongly agreed' (n=7) or 'agreed' (n=11) that they would continue to follow the lifestyle changes they had made, indicating the changes made may be sustainable, and that benefits seen over the 16-week programme may continue to improve. Six participants went on holiday during the intervention period and two participants suffered back injuries towards the end of the programme. These factors could have a negative impact on the results but also may be indicative of the target demographic and should be considered for future intervention designs.

Strengths and limitations

The results support the notion that community pharmacy-based interventions to reduce CVD is a viable avenue to investigate further. Cardiovascular disease is a major cause of morbidity and mortality in NZ, and programmes such as this can be a targeted strategy to support other public health initiatives to respond to this health issue. Valuable aspects of the programme were the multidisciplinary approach to the reduction of chronic disease - a model that produces enhanced outcomes (Bischoff et al., 2017; Houle et al., 2014; Jennings & Astin, 2017; Taylor et al., 2005). Point of care devices made it possible to conduct the various measures that were used in this study. They provided participants real time feedback along with health professionals' interpretation of the results. Participants identified this as an important aspect of the study as well as being motivational. This has also been suggested by other studies (Brown et al., 2004; Laurence et al., 2010; Gialamas et al., 2009). Almost all participants expressed that they found using the Fitbit to be informative and motivational, and this has been shown previously with pedometers (Richardson et al., 2008; Bravata et al., 2007). Continuity of care throughout the programme resulted in positive relationships between participants and health professionals and likely had an influence on motivating sustainable behaviour change (Bundy, 2004; Parchman et al., 2002).

The feasibility study lays the groundwork for the possible implementation of a more substantial study that can test the methodology with a greater and more diverse number of participants over a larger geographical range of community pharmacies. The current study is limited by its sample size and insufficient power which restricts the interpretability and generalisation of the results. A larger study could also utilise a randomised control design in order to ascertain the magnitude of the effect the intensive intervention has compared to standard care, especially because aspects of the current design relied on self-reporting for the lifestyle behaviours. The self-reporting of diet, physical activity (IPAQ questionnaire) and medication use may have added bias to the results. The measures could have been influenced by social desirability, exaggeration or memory. The use of clinical measures helps to validate the self-reported lifestyle changes.

Opportunities exist to specifically target the population of people who have a high CVD risk (>15%) as this can produce greater outcomes (Ebrahim et al., 2011). Additionally, focusing on those with high modifiable risk factors as opposed to using overall risk (which includes non-modifiable risk) may improve clinical end points. An enhanced pharmacological intervention from pharmacists could be used to educate and refer participants to their general practitioner who may qualify for additional pharmacological management. This can increase the number of risk reduction strategies available and was implemented successfully by Tsuyuki et al. (2002). Additionally, a cost benefit analysis is an important consideration as well as investigating funding strategies available for such programmes, whether it be government, industry, out of pocket, or a mix. Finally, long-term follow-up could be investigated to determine the sustainability of the lifestyle behaviours made as well as the changes in clinical measures over time.

Collaboration between dietitians and pharmacists to improve lifestyle behaviours in a community pharmacy setting is a viable and potentially effective initiative to reduce the burden of CVD in New Zealand. Further investigation into this approach with a larger study is warranted to establish the significance of achievable health outcomes as well as their cost.

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Conflict of interest: None reported.

Chapter Four

Conclusion and recommendations

Overview

The community pharmacy CVD primary prevention feasibility programme was implemented to positively influence participants' five-year CVD risk. It aimed to do this over a 16-week period through a collaborative approach between dietitians and pharmacists to motivate lifestyle behaviour change in diet, physical activity and medication compliance. The programme provided participants monthly consultations where measurements were taken and explained, information was provided, and goals were set.

The primary objective was to measure changes in five-year CVD risk. For this objective there was a small decrease over the study duration, however it was not significant. The secondary objectives examined changes to the modifiable CVD risk factors – metabolic and behavioural, and assessing the programme's implementation feasibility. Blood pressure, TC and diet quality score displayed significant meaningful improvements and a majority of the other metabolic and behavioural measures produced improvements though not significantly. Nine of the 12 participants completed the entire 16 weeks, with 11 completing for the majority of the duration. Participant evaluation showed that participants viewed the programme as valuable and had made sustainable lifestyle behaviour changes.

Relevance

Cardiovascular disease is still a major cause of morbidity and mortality in NZ therefore it is important that new initiatives for preventing its burden on the NZ people and health system are investigated and developed. Initiatives that are available and accessible to the public, and that are based on improving the overall lifestyle and health of the population offer promise to not only reduce the incidence of CVD, but also that of other chronic diseases. This programme is intended to meet this need while supporting the greater national public health environment as a variety of

strategies are needed to reduce CVD. Other public health strategies include initiatives by the government and not for profit organisations that target healthy eating and physical activity in families, schools and communities These include Green Prescriptions, Project Energize, Food for Thought and Healthy Families NZ (Ministry of Health, 2016a; Ministry of Health, 2018b; National Heart Foundation of New Zealand, 2019c). There are also other policy and advertising strategies aimed at smoking cessation, alcohol reduction and healthy eating (Ministry of Health, 2019a; Ministry for Primary Industries, 2019; Health Promotion Agency, 2019). This programme is viewed as being additional to these by providing targeted individualised care directly to those who are identified at high risk, and to the researcher's knowledge it is the first of its kind to be investigated in NZ.

Being a feasibility study the programme provides a test run for the potential implementation of a larger study with a more diverse cohort of participants and greater regional spread. The study showed that it is feasible to recruit, run and retain a small cohort of participants in two community pharmacies. It has been valuable to identify aspects that were successful as well as areas in need of improvement. A follow up study would have to deal with greater funding requirements, consistency of approach between study locations and multiple dietitians. A lack of space and the availability of the consultation room was an issue encountered with one pharmacy that was approached to participate and could be a problem for other pharmacies.

The programme also advocates for both dietitians and pharmacists in providing quality services to prevent or delay chronic diseases as per their Scope of Practice (Andersen et al., 2018; Pharmacy Council, 2019). The study highlights that by collaboration these skills can be used to motivate positive lifestyle changes in the population. Dietitians are experienced and proficient in motivating behaviour change and individualising interventions to meet the patient's needs (Raynor & Champagne, 2016; Briggs & Stanley, 2018). Diet and physical activity are significant contributors to an individual's overall lifestyle and their propensity to develop chronic diseases, not just CVD. Dietitians therefore possess a desirable skillset to help control and prevent these diseases in the community and it is hoped this research can help to create new avenues where dietitians can use their expertise.

Strengths and limitations

A valuable aspect of this programme was the multidisciplinary approach (McGill & Felton, 2007; Omboni & Caserini, 2018; Bischoff et al., 2017; Houle et al., 2014; Jennings & Astin, 2017; Taylor et al., 2005; Carter et al., 2009). The participants received continuity of care by the team of health professionals involved. Each participant saw the same student dietitian and pharmacist throughout the entirety of the programme. This allowed for relationship and rapport building between the participant and health professional and also with the local community pharmacy as a whole. It is believed that establishing strong relationships and continuity of care are important moderators of sustained behaviour change (Bundy, 2004; Parchman et al., 2002).

Using point of care anthropometric and biochemical testing allowed for a variety of health indicators to be explored with participants. Providing participants with the results as well as interpretation was highly valued. It aided motivation to see the impact their behaviour changes were having over time. Other studies have also suggested that point of care testing can be a motivation and improve health outcomes (Brown et al., 2004; Laurence et al., 2010; Gialamas et al., 2009). Most participants also found knowing their daily steps motivational and often aimed to increase these. The Fitbit provided a discussion point at consultations and goal setting for those who were engaged. Although Fitbit devices have shown moderate to high validity for measuring steps (Sushames et al., 2016; Alharbi et al., 2016; Diaz et al., 2015), application as a study measure is limited because of variations in appropriate use i.e. wearing and charging. Smartphones also have the capacity to record steps so these could be used in future studies as opposed to providing an additional device.

The feasibility pretest posttest study design was chosen to develop the methodology for implementing the intervention and to provide insight into the effectiveness of the intervention. A major limitation because of this is the study population size. To reach adequate power to show a change in CVD risk requires approximately 80 participants, as referenced in the PAART study (McNamara et al., 2010). Being a feasibility study, the low participant number produced an underpowered study design. While statistically significant changes in measured variables were found, the power of the design to accurately gauge these changes is inadequate. Future studies using a similar design should determine participant numbers to produce an adequate power.

Additionally, statistical significance was difficult to achieve due to the large variability between the small number of individuals. This is evident by the wide range in clinical response to the intervention experienced by participants. However, because it is a feasibility study it is useful to look at the data trends, which are promising in this case. The size also impacts the programme's ability to be generalised to the national population especially to Māori and Pacific people (groups known to be at increased CVD risk) as well as to different pharmacy configurations and geographical areas. Although all were of European ethnicity, almost three quarters of the participants resided in the two most deprived socioeconomic quintiles. Significant socioeconomic inequities exist in relation to the incidence and mortality of CVD in NZ (Riddell & North, 2003).

Beyond feasibility, future studies would benefit from having a control group. Without a control group it is uncertain whether the results can be attributed to the intervention or to some other variable. Having a control group can help to ascertain the difference between giving the comprehensive care provided in this study compared to current practice or another design. For example, a study with a control group can compare the effect of an intensive lifestyle intervention against providing only a CVD risk health check and a lifestyle change handout. Finally, dietetic consultations were undertaken by a student dietitian during their final year of study to become a registered dietitian. It is plausible that an experienced dietitian could motivate further behaviour change than what was observed in this study and therefore greater change in measured outcomes.

Future recommendations

The following points should be considered in future research.

- Research shows that targeting high risk individuals is more effective than those at low risk (Ebrahim et al., 2011). Therefore, focusing specifically on populations where CVD risk is high and groups who traditionally have poor access could give greater gains.
- The calculation for overall CVD risk is based on modifiable and non-modifiable risk factors. The sole use of a modifiable risk calculation as an eligibility criterion would be more aligned to lifestyle intervention as non-modifiable risk

factors cannot be changed. For example, in the current study, it was easier for older, male participants to meet the eligibility criteria. Some younger females were declined because they did not meet the threshold, but they potentially had high modifiable risk. The PREDICT calculation can be adapted to calculate modifiable risk independently.

- A larger study may find people who meet the criteria for CVD medication who have are not actively under the care of a GP. Pharmacists can educate them on the pros and cons of the medication then refer them to their GP if appropriate. Participants will benefit from the added intervention. This approach was successfully tested by Tsuyuki et al. (2002) where participants were encouraged by pharmacists to visit their GP for cholesterol lowering medication. In the current study there were participants who met the requirements for additional CVD medication, but this was being closely monitored by their GP. Smoking was not an issue in the current study but with a larger cohort, pharmacists if needed can provide nicotine replacement therapy to those who require it.
- Long term follow-up would be useful to investigate the sustainability of the behaviour changes as well as the continued change these have on the modifiable CVD risk measures. Participants thought the programme length was appropriate, but regular follow -up sessions, for example six and 12 months may provide more sustained benefits.
- Future research should consider a resource and cost analysis to determine the costs of implementation and the health benefits achieved; as well as options for long term funding.

The feasibility study, the first of its kind in NZ, has provided a starting point for a multidisciplinary and multi-risk factor approach to CVD primary prevention. With the improvements outlined above it is predicted that the intervention will be more targeted and effective, the efficacy of the intervention will be more accurately realised, and the requirements and costs of large-scale implementation will be determined. Collaboration between dietitians and pharmacists to improve lifestyle behaviours in a community pharmacy setting is a viable and potentially effective initiative to reduce the burden of CVD in NZ.

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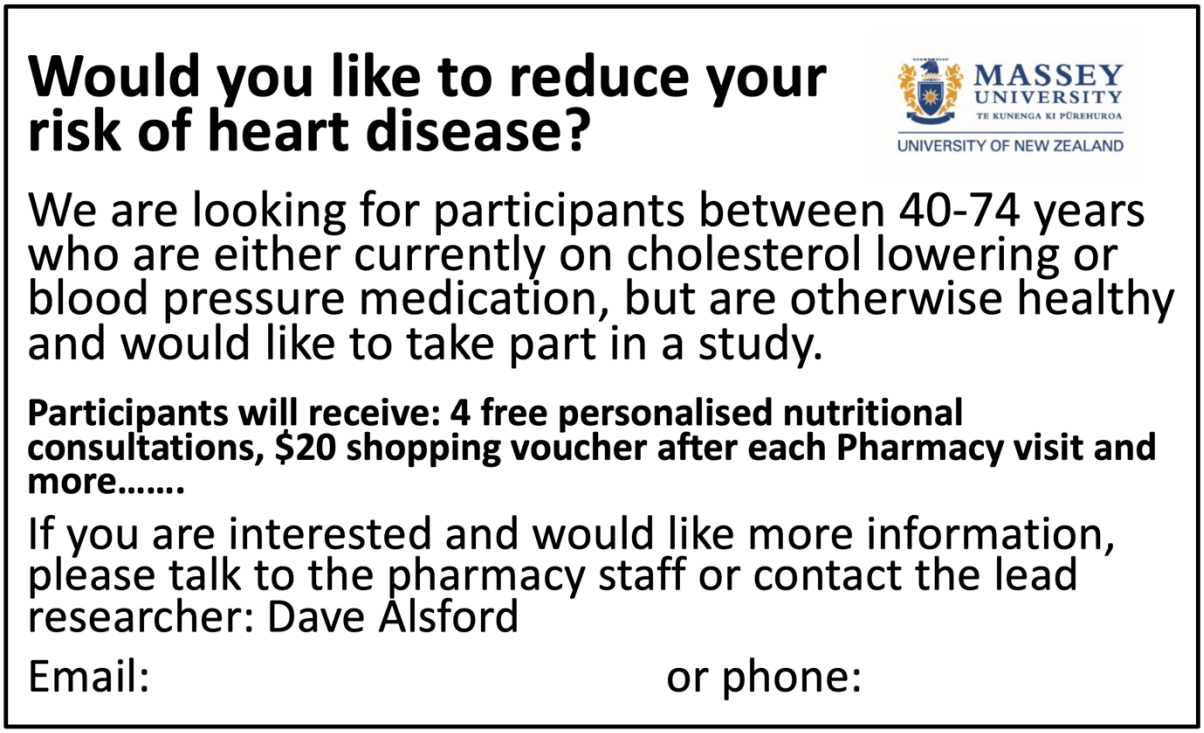
1.0 Questionnaires and materials

1.1 Study flyer



1.2 Study poster

Are you on cholesterol lowering or blood pressure medication?

Would you like to reduce your risk of heart disease?

Changes to your lifestyle can lower the chance of getting heart disease and help you live a healthier, longer life.

The Schools of Sport, Exercise & Nutrition and Health Sciences at Massey University are running a lifestyle program (nutrition and exercise advice) to see if we can improve cholesterol & blood pressure levels.

We are looking for participants 40-74 years taking cholesterol lowering or blood pressure medication, but who are otherwise healthy.

You need to be able to make monthly visits to Life Pharmacy in Glenfield or Warkworth for consultations between May & September.

All participants will receive:

- **Four free personalised** nutrition consultations with a student dietitian
- **$20** shopping voucher after each Pharmacy visit (**$100** in total)
- **Free** body composition & blood pressure measures
- **Free** cholesterol & HbA1c* measures – using just a finger prick of blood

***a measure of glucose levels in the blood**

If you would like more information, please talk to the pharmacy staff or contact the lead researcher:

Dave Alsford
Email:
Phone:

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

This study has been approved by Human Ethics Committee: Southern A reference no. 18/63

1.3 Participant information sheet

Community Pharmacy Cardiovascular Disease Risk Reduction Program

INFORMATION SHEET

Researchers Introduction

We would like to invite you to take part in the Community Pharmacy Cardiovascular Disease Risk Reduction Program. The purpose of the program is to assess the design of a community pharmacy-based lifestyle modification intervention that aims to reduce cardiovascular disease (CVD) risk. It adopts a collaborative approach between pharmacists and dietitians, looking at medication use, diet and exercise. This study is being conducted by a group of researchers from Massey University.

Please read this Information Sheet carefully before deciding whether or not to participate. You are under no obligation to participate in this program.

The lead researchers for this study are Dave Alsford and Dr Cheryl Gammon.

Dave Alsford
Student Dietitian
School of Sport Exercise and Nutrition
College of Health
Massey University
Email:
Phone:

Dr Cheryl Gammon
Lecturer
School of Health Sciences
College of Health
Massey University
Email:
Phone:

Project Description and Invitation

Despite improvements in identification and treatment, cardiovascular disease (CVD) remains a leading cause of illness and death in New Zealand. Many factors can contribute to CVD risk and many of the factors are modifiable. They include high cholesterol, high blood glucose levels/diabetes, high blood pressure, being overweight or obese, tobacco smoking, physical inactivity, and poor diet. Prevention of CVD has been shown to be successful and cost effective. Guidelines on CVD prevention highlight importance of overall CVD risk assessment and the management of multiple risk factors. Therefore, treatments focus on medication management and dietary and physical activity intervention.

Community pharmacies are well placed for providing heath advice in the prevention of diseases, with convenient accessibility to trained health professionals without an appointment and extended opening hours. Internationally, the community pharmacy has also been shown to be effective at managing the individual risk factors of CVD, but few studies have targeted multiple risk factors, especially with dietary advice from a trained dietitian.

This program employs a lifestyle intervention administered by pharmacists and dietetic health professionals that includes medication use, diet and physical activity.

Participant Identification and Recruitment

Who are we looking for?
We are looking for 12 male and female volunteers to participate in this study. To take part in this study you should:

- Be between 40-74 years of age
- Be on blood pressure or cholesterol lowering medication
- Be free from established CVD, diabetes or cardiovascular events
- Have a 5% or greater CVD risk over five years
- Be living independently (not requiring assistance with daily activities or 24/7 skilled nursing)
- Be proficient in English
- Not have a complex medical condition e.g. organ damage, cognitive impairment

You are welcome to bring a family member or support person with you at any time during the program.

Project Procedures

What is going to happen?

The program is 16 weeks long requiring a visit to the pharmacy every 4 weeks. The first visit will be approximately 2 hours, the following 3 visits will be 30-45 minutes and the final visit will be approximately 1 hour in duration. You will also be asked to fill in a brief diary every week that will take about 2 minutes to complete. You will be giving about 5 and a half hours of your time for the whole program over the 16 weeks.

After you have read and had time to consider the information contained in this information sheet, and you decide to take part in this study, you will be asked to complete a short screening questionnaire (10 minutes) to ensure that you meet the criteria for participation. If you meet the inclusion criteria you will be invited to take part. A researcher will then make an appointment with you to visit the pharmacy for your first session. At this appointment you will first be asked to sign a consent form for participating in the study and you will have the opportunity to ask any questions about the study.

During the first session we will ask you to:

- Complete a health and demographic questionnaire.
- Complete questionnaires to assess your medication use, dietary intake and physical activity levels
- Have height, weight, waist and hip circumference measured.
- Have percentage body composition measured using bioelectrical impedance analysis.
- Have your blood pressure measured.
- Provide two small finger prick blood samples which will be taken by a trained researcher for the measurement of blood cholesterol (total cholesterol, HDL-cholesterol, triglycerides), and HbA_1C.

You will also be consulted about your medicine use by a pharmacist and your dietary intake and physical activity by a student dietitian. Goals for lifestyle changes related to CVD risk will be made for the program. You will be provided a Fitbit for the duration of the study to keep track of your physical activity and be asked to keep a diary of any changes to your daily life e.g. sickness, holidays.

Visits every four weeks for the next 16 weeks will include a revision of set goals and progress of your lifestyle changes by the student dietitian. If medications have changed or you have questions a pharmacist will be available for consultation. During the third and last visit (weeks 8 and 16) we will take the body measurements and finger prick tests again. During the final visit (week 16) we will ask you to complete an evaluation survey.

What are the benefits and risks of taking part in this study?

Benefits

By participating in this study, you will receive four free consultations with a student dietitian. You will also receive feedback by the student dietitian on your individual blood (Total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, HbA_1C), blood pressure and body composition results.

The principal benefit of taking part in this study is receiving education and advice on how to reduce your CVD risk and to help you live a healthier life. You will also be contributing to testing the feasibility of implementing programs like this or similar programs in a community pharmacy setting. You will receive a brief report summarising the main findings of the project via mail or email.

To cover any travel expenses, you will be given a $20 supermarket voucher for completing each visit.

Risks
Some people may have a fear of having a finger prick blood sample taken or experience discomfort when the finger prick blood samples are taken. Occasionally a slight bruising of the finger can result. The bruising usually disappears within a day or two. There may be social or cultural discomfort from having body composition measurements taken, however, privacy will be ensured, and you will be treated with respect. The height measurement involves standing upright, and the researcher bringing a head plate gently onto the top of your head and compressing the hair if necessary. We will explain all measurements and ask for your permission prior to undertaking these measurements. You may also be accompanied by a support person at any time during the program. Every effort will be made to ensure your comfort and respect during your participation.

We will use a bioelectrical impedance machine (BIA) to estimate body composition. BIA is a commonly used method for estimating body composition, and in particular body fat. When you stand on the BIA, a very low, safe electrical signal is sent from four metal electrodes through your feet to your legs, abdomen and arms to the hand-held electrodes. The electrical signal passes quickly through water that is present in hydrated muscle tissue but meets resistance when it hits fat tissue. This resistance, known as impedance, is measured and input into scientifically validated equations to calculate body composition measurements. Body composition measurements are provided in under 20 seconds.

Data Management

The data will be used only for the purposes of this project and no individual will be identified. Only the investigators and administrators of the study will have access to personal information, and this will be kept secure and strictly confidential. Participants will be identified only by a study identification number. Results of this project may be published or presented at conferences or seminars. No individual will be able to be identified.

At the end of this study the list of participants and their study identification number will be disposed of. Any raw data on which the results of the project depend will be retained in secure storage for up to 10 years, after which it will be destroyed.

Who is funding the research?

Massey University.

Participant's Rights

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study at any time;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Project Contacts

If you have any further questions or concerns about the project, either now or in the future, please contact either Dave Alsford or Dr Cheryl Gammon (contact details above). The other member of the research team is Dr Kathryn Beck (School of Sport Exercise and Nutrition, College of Health, Massey University).

Compensation for Injury

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic, and your claim will be assessed by ACC in accordance with the Accident Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements, and must help you access those entitlements. Entitlements may include, but not be limited to, treatment costs, travel costs for rehabilitation, loss of earnings, and/or lump sum for permanent impairment. Compensation for mental trauma may also be included, but only if this is incurred as a result of physical injury.

If your ACC claim is not accepted, you should immediately contact the researcher. The researcher will initiate processes to ensure you receive compensation equivalent to that to which you would have been entitled had ACC accepted your claim.

Thank you for considering participating in this study!

The Community Pharmacy Cardiovascular Disease Risk Reduction Program
Research Team

1.4 Screening questionnaire

Community Pharmacy CVD Risk Reduction Program

Health Screening Questionnaire

Thank you for your interest in our research project. To ensure that you fit the inclusion criteria of the study, we would appreciate it if you could answer the questions below. If you have any queries or concerns about the form, please feel free to contact Dave Alsford by email at heartstudy@massey.ac.nz or by phone on 027 276 8891.

When you have completed this form, please email it to Dave Alsford at heartstudy@massey.ac.nz

Name: _____

Gender (Please make a cross): Male ☐ Female ☐ Gender diverse ☐

Date of birth (day/month/year): _____

Address: _____

Contact telephone number: _____

Email address: _____

****

Have you been diagnosed with elevated cholesterol levels?

Yes ☐ No ☐

If yes, please specify what your last total cholesterol and/or LDL-C levels were if you know them (if you don't know – put NA)

_____

Have you been diagnosed with a cholesterol related genetic disorder?

e.g. Familial hypercholesterolaemia

Yes ☐ No ☐

If yes, please specify condition

_____

Have you been diagnosed with elevated blood pressure?

Yes ☐ No ☐

If yes, please specify what your last blood pressure level was if you know it (if you don't know – put NA)

_____

Have you ever been diagnosed with any of the following:

Indicate yes or no

Heart condition e.g. angina, heart failure, myocardial infarction, peripheral vascular disease, atrial fibrillation	
Diabetes or high blood sugar levels	
Kidney or renal function problems	
Disorders of the liver	
Cognitive impairment e.g. dementia, Alzheimer's	

If yes, provide more details please:

_____

Are you taking any prescription medications for your cholesterol?

e.g. atorvastatin (Lorstat®), simvastatin (Simvastatin Mylan®), pravastatin (Apo-Pravastatin®) simvastatin/ezetimibe (Zimybe®), ezetimibe (Ezetimibe Sandoz®), bezafibrate (Bezalip®)

Yes ☐ No ☐

If yes, please specify which tablet, dosage and how long you have been taking them

Tablet	**Dosage & frequency**	**Number of months/years**

Are you taking any natural health products for your cholesterol?

e.g. Blackmores cholesterol health, Go Healthy cholesterol shield, Fish oil capsules

Yes ☐ No ☐

If yes, please specify which products and how long you have been taking them

Product	**Dosage & frequency**	**Number of months/years**

Do you use any sterol enriched spreads to lower cholesterol?

E.g. Flora Pro-Activ®, Meadowlea Logicol®

Yes ☐ No ☐

If yes, please specify which one, how much you use daily and how long you have used it

Product	Amount & frequency	Number of months/years

Are you taking any prescription medications for your blood pressure?

Yes ☐ No ☐

If yes, please specify which tablet, dosage and how long you have been taking them

Tablet	Dosage & frequency	Number of months/years

Are you taking other medications, including traditional or alternative medicines?

Yes ☐ No ☐

Please specify the condition, the medication, the dosage and the length of time taken in the table provided.

Condition	Medication	Dosage & Frequency	Number of months/years

If you are unsuccessful in becoming a participant in this study, would you be interested in hearing about future research projects within the College of Health?

Yes ☐ No ☐

End of questionnaire, thank you

1.5 Health and demographics questionnaire

Community Pharmacy CVD Risk Reduction Program

Health and demographics questionnaire

Please complete the following form. All the information you give us is in confidence and will be used only for the purposes of this study. If you need any help to complete the form, please ask one of the research team.

Which ethnic group(s) do you belong to? Tick whichever applies to you (you may tick more than one box)

☐ European
☐ Māori
☐ Pacific Peoples
☐ Asian
☐ Middle Eastern/Latin American/African
☐ Other (*Please state which other ethnicity or ethnicities you belong to)*

_____

Which country were you born in?

Please state which country _____

If you live in New Zealand but were not born here, when did you first arrive to live in New Zealand?

Month (e.g. February) _____

Year (e.g. 2000) _____

What is your first language? _____

What is your current living arrangement?

☐ Living alone

☐ Living with others

If living with others, how many others do you live with and what is their relationship to you (e.g. Husband, wife, partner, son, daughter, grandson, granddaughter, flatmate, boarder, etc)

_____

What is your highest educational level (choose one)?

☐ No qualifications

☐ Primary school

☐ Secondary school

☐ Post-secondary certificate, diploma, or trade diploma

☐ University degree

Which of the following best describes your current work situation? (please tick as many as apply)

☐ Paid employment

Occupation and number of hours of paid employment per week?

_____

☐ Volunteer work

Position and number of volunteer hours per week?

_____

☐ Fully retired

☐ Semi-retired

☐ Other (e.g. caregiver, studying, homemaker), please describe

During your working life, what was your main occupation?

☐ Labourer (e.g. Cleaner, food packer, farm worker)

☐ Machinery operator/driver (e.g. Machine operator, store person)

☐ Sales worker (e.g. Insurance agent, sales assistant, cashier)

☐ Community or personal service worker (e.g. Teacher aide, armed forces, hospitality worker, care)

☐ Technician/trades worker (e.g. Engineer, carpenter, hairdresser)

☐ Professional (e.g. Accountant, doctor, nurse, teacher)

☐ Manager (e.g. General manager, farm manager)

☐ Other (Please Specify) _____

Do you smoke tobacco?

Yes ☐ No ☐ Former smoker ☐

If yes, approximately how many cigarettes per day: _____

Do you drink alcohol?

Yes ☐ No ☐ I used to drink alcohol, but no longer do ☐

If yes, how often do you usually drink alcohol?

☐ Monthly

☐ Weekly

☐ Daily

How many standard drinks of alcohol do you usually drink in the timeframe selected above?

A standard drink is 1 can of beer (330ml), 1 glass of wine (100ml), 1 Ready to Drink (RTDs), 1 shot/nip of spirits (30ml)

_____

On any one drinking occasion, what is the maximum number of standard drinks you would have?

A standard drink is 1 can of beer (330ml), 1 glass of wine (100ml), 1 Ready to Drink (RTDs), 1 shot/nip of spirits (30ml)

_____

How many alcohol-free days do you usually have per week?

☐ 1 day
☐ 2 days
☐ 3 days
☐ 4 days
☐ 5 days
☐ 6 days
☐ 7 days

Do any close family members (mother, father or sibling) that have a high cholesterol concentration?

Yes ☐ No ☐ Don't know ☐

If yes, please give details

_____

Do any close family members (mother, father or sibling) that have a high blood pressure?

Yes ☐ No ☐ Don't know ☐

If yes, please give details

_____

Have any close family members (parent or sibling) suffered from coronary heart disease or vascular disease?

e.g. heart attack, angina, stroke

Yes ☐ No ☐ Don't know ☐

If yes, please give details, including relationship, age at which they were diagnosed and if they were hospitalised or died before the age of 50 years.

_____

Have any close family members (parent or sibling) with type 2 diabetes?

Yes ☐ No ☐ Don't know ☐

If yes, please give details, including relationship and at what age they were diagnosed.

_____

Do you have any mobility or other problems which would limit your ability to exercise?

Yes ☐ No ☐ Not sure ☐

If yes, please give details

_____

In general, would you say your health is:

☐ Excellent

☐ Very good

☐ Good

☐ Fair

☐ Poor

Has what you eat changed in the past 10 years?

☐ Yes

☐ No

If yes, please describe (e.g. I eat less because my appetite is lower; I eat more vegetables).

_____

_____

_____

_____

_____

Compared with 10 years ago, has your intake of the following foods increased, decreased or stayed the same (tick the box which is most accurate – either eat more, eat less or eat the same)

	Eat more	**Eat the same**	**Eat less**
Fruit			
Vegetables			
Breads, cereals & grains			
Milk, yoghurt, cheese			
Oily fish and seafood (e.g. salmon, tuna, mackerel, herring)			
All fish and seafood			
Red meat & poultry			

Has your level of physical activity changed in the past 10 years?

☐ Yes

☐ No

If yes, please describe.

_____

_____

_____

Would you like a summary of your results forwarded to your General Practitioner?

Yes ☐ No ☐ Not sure ☐

This is the end of the questionnaire, thank you for participating.

MASSEY UNIVERSITY
TE KUNENGA KI PŪREHUROA
UNIVERSITY OF NEW ZEALAND

COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

1.6 Participant evaluation survey

Community Pharmacy CVD Risk Reduction Program
Participant Evaluation Survey

How easy was it to complete this program?	*Very hard* 1	2	3	4	*Very Easy* 5
The length of the entire program was	*Too short* 1	2	3	4	*Too long* 5
The length of time between sessions was	1	2	3	4	5
How would you rate this program overall?	*Poor* 1	2	3	4	*Excellent* 5
The program met my expectations	*Strongly disagree* 1	2	3	4	*Strongly agree* 5
I acquired new knowledge on how to improve my diet to reduce my risk of CVD	1	2	3	4	5
I acquired new knowledge on how to improve my physical activity to reduce my risk of CVD	1	2	3	4	5
I acquired new knowledge on how to improve my medication use to reduce my risk of CVD	1	2	3	4	5
I have made improvements to my diet	1	2	3	4	5
I have increased my weekly physical activity amount	1	2	3	4	5
My medication compliance has improved	1	2	3	4	5
I will continue to follow the lifestyle changes made during this program	1	2	3	4	5
How did you hear about this program?					
Why did you participate in this program?					

What was the most helpful aspect of this program for making lifestyle changes?
If the program was repeated, what should be left out or changed?
If the program was repeated, what could be added to improve it?
What did you like least about the program?
What did you like most about the program?

In what way was this program useful to you?
Would you recommend this program to someone you know? i.e. a family member or a friend.
How much would you pay to attend a program like this?
Any other comments?

End of questionnaire, thank you

1.7 Medication usage questionnaire

Community Pharmacy CVD Risk Reduction Program

Medication usage questionnaire

To be completed in conjunction with the Pharmacist

Doctors name:	
Doctors address:	
Allergies:	
Adverse medicine effects:	

In regard to your medicines	Tick if Applicable	Details
Difficulty reading labels		
Difficulty taking medicines on time		
Difficulty remembering to take medicine doses		
Gaps in knowledge of medicines and their use		
Other issues, please specify		

Current Medications Including OTC and complementary medicines	Do you know why you are using this medicine	Do you take the medicine as prescribed?	Would you like more info. on your medicine	Do you have any adverse effects from your medicine	General comments and other issues
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					

Current Medications Including OTC and complementary medicines	Do you know why you are using this medicine	Do you take the medicine as prescribed?	Would you like more info. on your medicine	Do you have any adverse effects from your medicine	General comments and other issues
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					
Name/dosage/form/strength	Yes No	Yes No If No, specify	Yes No	Yes No If Yes, specify	
Dose					

Other Notes:

1.8 Dietetic and physical activity assessment form

DIETETIC and PHYSICAL ACTIVITY ASSESSMENT FORM

Session number: Date of Assessment:

Name:			Age	DOB	
Smoking status	Family Hx CVD		DepIn		

Anthropometry						
BP					AVG	
Pulse					AVG	
Height		AVG		Weight		BMI
Fat mass	kg %		SMM mass	kg	%	WHR
Waist Cmf					AVG	
Hip Cmf					AVG	
Biochemical						
Total cholesterol: Triglycerides: HDL-C: LDL-C: TC/HDL ratio:			HbA1c: 5 year CVD risk:			
Clinical						
Medical History/Investigations/Mental health:						
Relevant Medications/Supplements:						
Bowels:						
Appetite:						
Social/Family/Support:						
Cooking/Food Shopping:						
Physical Activity: **Ministry of Health Physical Activity Guideline** *2 ½ hours of moderate or 1 ¼ hours of vigorous physical activity spread throughout the week.*						
Other:						

Dietary
History of dieting/restrictive eating/food allergies:
Diet History: **Heart Foundation Healthy Heart guideline** *Eat 3-4 serves each of vegetables and fruit* *Eat 6+ serves a day of breads and cereals including starchy vegetables* *Eat fish twice a week; eat legumes 4-5 times each week; small piece of chicken or lean meat (up to 1-1.5 servings/day) or vegetarian alternative.* Eat 2-3 servings of dairy or diary alternatives each day Eat 6+ servings or oils and nuts a day Cut back on junk foods, takeaways & foods or drinks high in sugar, salt or saturated & trans fats

Summary/Additional Comments

Client Negotiated Interventions/Goals

1.9 Weekly diary

Community Pharmacy CVD Risk Reduction Program

Your Diary

Name: _____

Study ID: _____

Dear Participant,

We very much appreciate your participation in this research project. By committing time and effort to this project we assume that it is just as important to you as it is to us that this project is a success. For this project to be successful we need reliable information and data which will also allow us to publish the findings of the study in a scientific journal.

In order to do this, it is extremely important that you are honest if you do have changes to your diet, physical activity levels or health during this study.

Please complete one sheet for each week of the study. It should only take a few minutes to complete each week.

If you have any further questions, please feel free to contact

Dave Alsford
Tel:

or Dr Cheryl Gammon
Tel:

Email:

Thank you again for taking part in this study.
Kind regards,
The Research Team.

Important dates to remember

Your appointment dates will be written here.

Appointment	Date / Day	Time
1st visit to the Pharmacy (Week 0)		
2nd visit to the Pharmacy (Week 4)		
3rd visit to the Pharmacy (Week 8)		
4th visit to the Pharmacy (Week 12)		
5th visit to the Pharmacy (Week 16)		

Measurements

Visit	Body measurements	Blood Pressure	Blood measurements
Baseline	Weight BMI Hip Circumference Waist Circumference Body fat (kg) Body fat (%) Muscle mass (kg) Muscle mass (%)	Systolic / diastolic Heart rate	TC LDL-C HDL-C TC/HDL-C TG HbA1c 5-year CVD risk
Week 8	Weight BMI Hip Circumference Waist Circumference Body fat (kg) Body fat (%) Muscle mass (kg) Muscle mass (%)	Systolic / diastolic Heart rate	TC LDL-C HDL-C TC/HDL-C TG HbA1c 5-year CVD risk
Week 16	Weight BMI Hip Circumference Waist Circumference Body fat (kg) Body fat (%) Muscle mass (kg) Muscle mass (%)	Systolic / diastolic Heart rate	TC LDL-C HDL-C TC/HDL-C TG HbA1c 5-year CVD risk

Consultation Goals

	Goal	Details
Consultation 1	**1**	
	2	
	3	
Consultation 2	**1**	
	2	
	3	
Consultation 3	**1**	
	2	
	3	
Consultation 4	**1**	
	2	
	3	

WEEKLY DIARY

Week 1 **Date:** _____

Were you ill this week?

O Yes O No

If yes, what was the nature of your illness?

_____

_____

_____

Did you consume any medication for the illness?

O Yes O No

If yes, please provide details of the medication used:

_____

_____

_____

Have you had any changes to your regular medications?

O Yes O No

If yes, please provide more detail:

_____

_____

_____

Did you do anything different this week from your normal routine with regard to physical activity? e.g. went tramping, joined a gym, took part in a competition

O Yes O No

If yes, please provide details of the change to your physical activity:

_____

_____

_____

Have there been any changes in your normal daily routine this week, e.g. eating habits, sleeping habits, alcohol consumption, use of other nutritional supplements, etc?

O Yes O No

If yes, please provide more detail:

_____

_____

_____

Do you have anything else you would like to report?

Extra Notes:

Appendix Two

Supplementary results

Table 13: The number of people completing each stage of the programme.

Stage of programme	*N*	Attrition
Expressions of interest	24	3 declined to participate 1 didn't reply 1 excluded due to reaching study number
Completed screening questionnaire	19	2 excluded due to a precluding medical condition 2 excluded as likely to be less than 5% CVD risk
Meet screening eligibility	15	1 excluded due to reaching study number
Completed initial assessment	14	2 excluded as CVD risk was less than 5%
Participated in the programme	12	1 withdrew at 8 weeks due to the development of a medical condition 1 withdrew at 12 weeks and 1 at 15 weeks due to overseas travel
Completed 16 weeks of programme	9	

Abbreviations: CVD: cardiovascular disease, N: number of people.

Table 14: Average steps taken throughout the program.

	Weeks 0-4	Weeks 5-8	Weeks 9-12	Weeks 13-16
Steps, mean (*SD*)	10374.2 (3950.8)	10102.5 (4247.1)	8987.8 (2526.2)	9950.4 (3931.8)

Abbreviations: SD: standard deviation.

Table 15: Responses to the programme evaluation questionnaire.

Programme evaluation questions	Score, *n* (%)					
	1= very hard, too short, Poor, strongly disagree			5= very easy, too long, excellent, strongly agree		NA
How easy was it to complete this program?			3 (27.3)	5 (45.5)	3 (27.3)	
The length of the entire program was		2 (18.2)	8 (72.7)	1 (9.1)		
The length of time between sessions was			8 (72.7)	1 (9.1)	1 (9.1)	
How would you rate this program overall?				2 (18.2)	9 (81.8)	
The program met my expectations				2 (18.2)	9 (81.8)	
I acquired new knowledge on how to improve my diet to reduce my risk of CVD				2 (18.2)	9 (81.8)	
I acquired new knowledge on how to improve my physical activity to reduce my risk of CVD				4 (36.4)	7 (63.6)	
I acquired new knowledge on how to improve my medication use to reduce my risk of CVD	1 (9.1)	2 (18.2)	3 (27.3)	1 (9.1)	3 (27.3)	1 (9.1)
I have made improvements to my diet				6 (54.5)	5 (45.5)	
I have increased my weekly physical activity amount		1 (9.1)	2 (18.2)	5 (45.5)	3 (27.3)	
My medication compliance has improved	2 (18.2)	1 (9.1)	6 (54.5)		1 (9.1)	1 (9.1)
I will continue to follow the lifestyle changes made during this program				4 (36.4)	7 (63.6)	

Abbreviations: CVD: cardiovascular disease, n: number.

Appendix 3

Photos of intervention

Photo 1: Dietetic consultation.

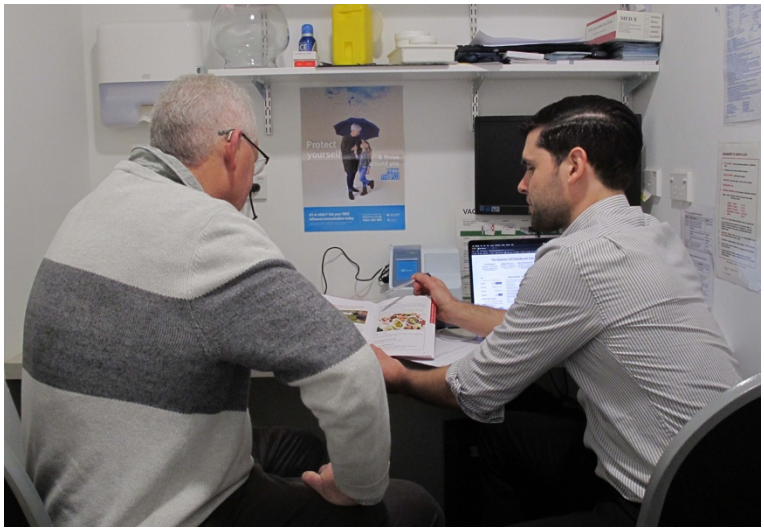


Photo 2: Participant completing questionnaire.

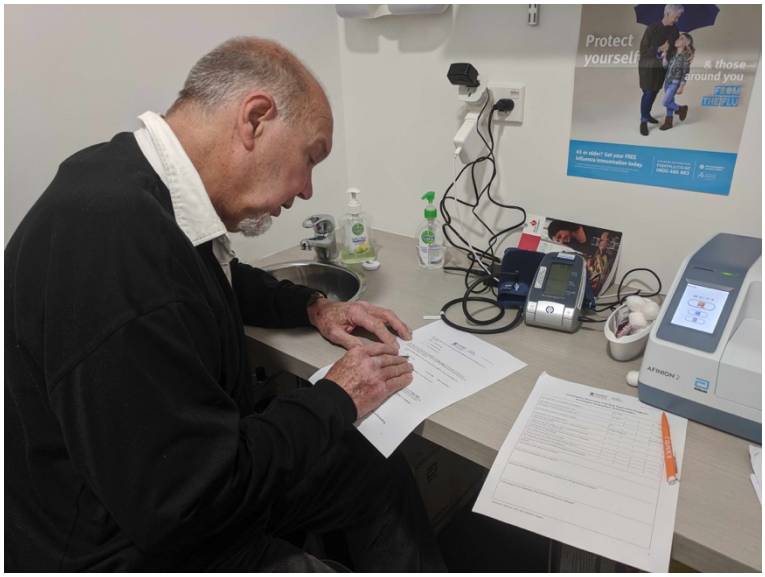


Photo 3: Finger prick point of care blood test.

Photo 4: Bioelectrical impedance analysis.