Psychological determinants of risk behaviours and cardio-metabolic health in a higher risk regional cohort

André Leao Rodrigues
BSc (Hons)

A thesis submitted for the degree of

Doctor of Philosophy (Med)

At Monash University in February, 2019

Department of Epidemiology and Preventative Medicine

Faculty of Medicine, Nursing and Health Sciences

MONASH University
“People who believe they have the power to exercise some measure of control over their lives are healthier, more effective and more successful than those who lack faith in their ability to effect changes in their lives.”

“Behaviour, cognitive, and other personal factors, and environmental influences all operate interactively as determinants of each other.”

Albert Bandura  
David Starr Jordan Professor Emeritus of Social Science in Psychology  
Stanford University
General Declaration

MONASH University

Declaration for thesis based or partially based on conjointly published or unpublished work. In accordance with Monash University Doctorate Regulation 17 Doctor of Philosophy and Research Master’s regulations the following declarations are made:

I hereby declare that this thesis contains no material which has been accepted for the award of any other degree or diploma at any university or equivalent institution and that, to the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

This thesis includes 1 original paper published in a peer reviewed journal and 3 submitted publications. The core theme of the thesis is the association between psychological constructs, specifically behaviour-specific self-efficacy and locus of control, health behaviours and cardio-metabolic disease risk. The ideas, development and writing of all the papers in the thesis were the principal responsibility of myself the student, working within the Pre-Clinical Disease and Prevention Unit.
under the supervision of **Associate Professor Melinda J. Carrington**, **Professor Ralf Schwarzer** and **Associate Professor Chantal Ski**.

In the case of **Chapters 3 to 6** my contribution to the work involved the following:

<table>
<thead>
<tr>
<th>Thesis Chapter</th>
<th>Publication Title</th>
<th>Status</th>
<th>Nature and % of student contribution</th>
<th>Co-author(s) Nature and % of co-author’s contribution*</th>
<th>Co-author(s) Monash student Y/N*</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>A systematic review and meta-analysis of primary prevention programmes to improve cardio-metabolic risk in non-urban communities.</td>
<td>Published</td>
<td>Conception and design; acquisition of data; analysis and interpretation of data; drafting of the article and revision (70%)</td>
<td>1) Jocasta Ball, Data analysis, input into manuscript 10% 2) Simon Stewart, input into manuscript 5% 3) Chantal Ski, input into manuscript 5% 4) Melinda Carrington, input into manuscript 10%</td>
<td>N</td>
</tr>
<tr>
<td>4</td>
<td>Contemporary risk factor management in two regional populations: Screening results of the Management to Optimize Diabetes and metabolic syndrome Risk reduction via Nurse-led Intervention (MODERN) randomized controlled trial.</td>
<td>Submitted</td>
<td>Interpretation of data: drafting of the article and revision, (50%)</td>
<td>1) Paul Zimmet, input into manuscript 10% 2) Melinda Carrington, data analysis, input into manuscript 40%</td>
<td>N</td>
</tr>
<tr>
<td>5</td>
<td>Relationship between cardio-metabolic disease risk, health beliefs and perceptions: a regional perspective.</td>
<td>Submitted</td>
<td>Conception and design, analysis and interpretation of data; drafting of the article and revision, (70%)</td>
<td>1) Ralf Schwarzer, data analysis, input into manuscript 10% 2) Chantal Ski, input into manuscript 5% 3) Melinda Carrington, input into manuscript 15%</td>
<td>N</td>
</tr>
<tr>
<td>6</td>
<td>Determinants of cardiovascular risk factors and health behaviour change in the ‘Management to Optimize Diabetes and metabolic syndrome Risk reduction via Nurse-led Intervention (MODERN)’ randomised controlled trial.</td>
<td>Submitted</td>
<td>Conception and design, analysis and interpretation of data: drafting of the article and revision, (70%)</td>
<td>1) Ralf Schwarzer, data analysis, input into manuscript 10% 2) Chantal Ski, input into manuscript 5% 3) Melinda Carrington, input into manuscript 15%</td>
<td>N</td>
</tr>
</tbody>
</table>
I have not renumbered sections of submitted or published papers in order to generate a consistent presentation within the thesis.

Student signature: Date: 21 / 02 / 2019

The undersigned hereby certify that the above declaration correctly reflects the nature and extent of the student’s and co-authors’ contributions to this work. In instances where I am not the responsible author I have consulted with the responsible author to agree on the respective contributions of the authors.

Main Supervisor signature: Date: 21 / 02 / 2019
Copyright notice

Under the Copyright Act 1968, this thesis must be used only under the normal conditions of scholarly fair dealing. In particular no results or conclusions should be extracted from it, nor should it be copied or closely paraphrased in whole or in part without the written consent of the author. Proper written acknowledgement should be made for any assistance obtained from this thesis.

I certify that I have made all reasonable efforts to secure copyright permissions for third-party content included in this thesis and have not knowingly added copyright content to my work without the owner’s permission.
Acknowledgement

Well, here it is. Five years of relentless persistence, patience and ultimately, faith. From hiking Lion’s Head in Cape Town, South Africa to providing health assessments to impoverished-indigenous communities off the coast of Queensland, Australia, I could not have imagined the doors that would be opened to me during my candidature.

Firstly, I wish to thank my supervisors, Associate Professor Melinda Carrington, Professor Ralf Schwarzer and Associate Professor Chantal Ski. Thank you for the opportunity to undertake a PhD under your supervision, Mel. It has not been without its challenges, however over the last five years I have learned and grown so much under your direction and guidance, and for that I will always be grateful. Ralf and Chantal, thank you for all of your valuable insights, advice and for always being available despite predominantly being located on the other side of the globe.

As most students will attest, while it may take a village to raise a child, it undoubtedly takes a lab to raise a PhD candidate! To Dr. Stephanie Yiallourou, Dr. Jocasta Ball and Glynis Cacavas, thank you for all of the unwavering support and your friendship over the last few years. I will miss working with all of you!

To my dear parents Sev and Monica, thank you for taking the leap of faith all those years ago and migrating your young family to Australia, from Brazil. This PhD is one of many examples of the amazing opportunities that have been afforded to me because of your sacrifices, and I hope it goes some way to showing how grateful I am that you always strived to provide the best for us. I love you, both. To my dear parents-in-law, Dean and Angi, thank you for your unwavering support and constant encouragement and love during my journey towards this milestone. I could not have asked for anything more.

To my darling wife Becky. I did it! Thank you for believing in me when I could not believe in myself. I would not be writing this if not for you. As my girlfriend all those years ago, then as my wife, and now as the mother of our future
child, your support and patience with me has been relentless. I love you, this was for you.

Finally, thank you Jesus, for making a way. For the opportunities, the challenges, the growth, the knowledge and for those I have encountered along the way. Thank you for the strength and patience needed to get me through, I know they were not my own.

I have learnt a great deal on this journey, extending beyond the prevention of cardio-metabolic disease and health behaviour change, to maternal and fetal medicine, psychology and clinical trials. I believe that with my experiences over the last half-decade, I am prepared for what is next.

“For I know the plans I have for you, plans to prosper you and not to harm you, plans to give you hope and a future.” Jeremiah 29:11

Andre L. Rodrigues
February 12th, 2019
Publications produced during candidature relevant to this thesis


Conference presentations given during candidature relevant to this thesis

**Cardiac Society of Australia and New Zealand 65th Annual Scientific Meeting, Perth – August 2017**

Rodrigues AL, Carrington MJ. Health Perceptions, Behaviours and Cardio-metabolic Risk - A Regional Perspective.

**Mary MacKillop 2nd International Scientific Research Symposium - Melbourne - Feb 2017**

Rodrigues AL, Carrington MJ. Relationship between cardio-metabolic disease risk and health beliefs, perceptions and behaviours in regional adults.

**ACU Health Research Symposium - Melbourne – December 2016**


**Mary MacKillop 1st International Scientific Research Symposium - Melbourne - March 2016**

Rodrigues AL, Carrington MJ. Self-efficacy and locus of control as determinants of health behaviours and cardio-metabolic disease risk.
Awards presented during candidature

2014  NMRC Centre of Research Excellence to Reduce Inequality in Heart Disease PhD Scholarship (March 2014 – March 2017)

2016  Mary MacKillop Institute for Health Research Symposium 1st Prize Travel Award

2017  Mary MacKillop Institute for Health Research Symposium 2nd Prize Travel Award

2017  NMRC Centre of Research Excellence to Reduce Inequality in Heart Disease PhD Scholarship, 6-month extension (April 2017 – September 2017)

2017  Cardiac Society of Australia and New Zealand 65th Annual Scientific Meeting, Perth – Complimentary Student Registration Award
Abstract

Introduction
Cardiovascular disease (CVD) is a global killer, being the attributable cause of death for more than 17.7 million people in 2015. A disturbing 95% of Australian adults have at least one risk factor for CVD and around two thirds have two or three concurrent biomedical or behavioural risk factors. The most common combinations of risk factors incorporate obesity, elevated blood pressure (BP), lipids or glucose; any three or more of these occurring simultaneously form a dangerous cluster of risk factors representing Metabolic Syndrome (MetS). The burden of disease caused by cardio-metabolic diseases is more pronounced with increasing remoteness. Factors such as limited access to healthcare, poorer education and lower socio-economic status contribute to residents of regional or remote areas faring worse in key health behaviours and risk factors. Despite this, evidence suggests that MetS can be reversed and type 2 diabetes mellitus (T2DM) and CVD are also largely preventable by effective lifestyle and/or pharmacological management. Although CVD and T2DM prevention aimed at health and lifestyle modification have been effective in urban residents, their administration by nurse intervention in non-urban settings, where cardio-metabolic risk is typically elevated, is relatively unknown.

Facilitating positive health behaviour change is paramount for disease prevention and health promotion. Self-efficacy and health locus of control have been identified as significant determinants of health behaviour change. Self-efficacy refers to one’s belief in their ability to overcome challenging barriers and demands. Choices of behaviour that individuals make are
influenced by self-efficacy. Health locus of control relates to how strongly a person believes they have control over the situations and experiences that may affect their health. Three locus of control dimensions vary as being determined by: their own actions (internal); (powerful) others; and luck or chance.

The aim of this thesis was to explore the associations between behaviour-specific self-efficacy, health locus of control, behaviour changes and cardiometabolic risk status in a high-risk regional cohort.

Methods

A systematic review and meta-analysis was conducted to assess the characteristics of an effective primary prevention program aiming to reduce key risk factors for CVD/T2DM in adults residing in non-urban locations, thereby informing the design of the Management to Optimise Diabetes and mEtabolic syndrome risk reduction via Nurse-led intervention (MODERN) Study. MODERN is a 24-month, multi-centre, appropriately powered and pragmatic randomised controlled trial of an innovative, nurse-led intervention. Nurse-led clinics were established in two regional locations (in Greater Shepparton and Colac Otway, Victoria, Australia) to conduct non-invasive clinical examinations and consultations targeting risk factor and health behaviour modification. All residents aged 40-70 years who presented to the clinic were screened (between October 2014 and March 2016) for MetS and eligibility into the study. Anthropometry, BP, blood lipids, glycated hemoglobin (HbA1c) and health behaviours including smoking, diet and physical activity data were collected at baseline. Self-efficacy for nutrition, physical activity, alcohol consumption and smoking and health locus of control were also assessed via
validated questionnaires. Two hundred and seventy-six adults aged 40-70 years with MetS were eligible and were enrolled into either nurse-led intervention or usual care. The association between health beliefs and behaviours and cardio-metabolic disease risk were investigated at baseline and after 12 months to determine interventional effects.

**Results**

Key findings of the systematic review showed community-based studies, pre/post designs and short-term interventions to be most effective in reducing overall cardio-metabolic disease risk.

Key findings from the screened cohort of 853 individuals indicated that more women than men were assessed for study eligibility (n = 520, 61%). Average age was 56.4 ± 7.9 years. In contrast to recommended guidelines, the majority were overweight or obese (77%). Around 4 in 5 individuals had total cholesterol ≥4.5 mmol/L (86%) and low-density cholesterol ≥2.5 mmol/L (78%) and one third had HbA1C levels ≥5.7%, indicating pre-diabetes. More men than women consumed excess alcohol (44 vs. 22%, p <0.001) whereas more women were physically inactive (40 vs. 32%, p =0.013). Overall, 32% of individuals screened met the criteria for MetS primarily via the triad of increased waist circumference, BP and triglyceride (achieved by 47%); waist circumference, BP and HbA1C (25%) and; waist circumference, triglyceride and HbA1C (17%). Higher internal health locus of control scores were associated with low and moderate CVD and T2DM risk. Higher chance health locus of control scores were associated with high risk for CVD compared to low and moderate CVD risk groups who were no different to each other. People with higher powerful others health locus of control scores had a higher
estimated CVD \( (p < 0.05) \) and T2DM \( (p < 0.001) \) risk than people with lower powerful others scores. There were no significant associations between any behaviour specific self-efficacy and CVD or diabetes risk.

Key findings at 12 months follow-up revealed improvements in both groups in key cardio-metabolic risk factors which were exacerbated within the intervention group compared to usual care. Individuals in the nurse-led intervention group had a greater reduction in systolic BP (-7.6mmHg ± 14.6mmHg v -1.7mmHg ± 13.9mmHg, \( p < 0.001 \)), waist circumference (-2.8cm ± 5.4cm v 1.5cm ± 5.0cm, \( p = 0.046 \)) and absolute CVD risk (1.5 ± 2.4 v -0.6 ± 2.6, \( p = 0.002 \)) over 12 months compared to usual care. There were significant reductions in nutrition, physical activity and alcohol self-efficacy, with no between-group differences. Powerful others health locus of control was increased in the usual care group. Dietary data revealed a reduction in daily kilojoules, carbohydrates and sodium in both groups, while the intervention group saw reductions in fat, cholesterol, and protein intake at 12 months compared to baseline. There were reductions in average daily steps at 12 months in both groups, with the intervention group showing reduced average daily energy expenditure. After adjustment for potential confounders, sex, baseline levels of risk and an internal health locus of control were consistently found to predict 12-month cardio-metabolic risk values.

**Conclusions**

The findings of this thesis highlight the effectiveness of a nurse-led intervention to reduce cardio-metabolic disease risk over 12-months through risk factor modification in a regional cohort. There was less evidence for a role of nurses to positively modify self-efficacy or health locus of control and a potential for
participants to overestimate their self-efficacy for key health behaviours at the beginning of an intervention. Extended follow-up to 24 months will reveal sustainability of health behaviour changes and longer-term interventional effects on self-efficacy, health locus of control and their relationship with cardiometabolic risk.
# Contents

General Declaration............................................................................................................. 3  
Copyright notice.................................................................................................................... 6  
Acknowledgement.............................................................................................................. 7  
Publications produced during candidature relevant to this thesis ................................. 9  
Conference presentations given during candidature relevant to this thesis..... 10  
Awards presented during candidature ............................................................................ 11  
Abstract ................................................................................................................................ 12  
List of Figures.......................................................................................................................... 20  
List of Tables........................................................................................................................... 22  
List of Abbreviations............................................................................................................ 23  
Chapter 1 ............................................................................................................................... 24  
   General Introduction .......................................................................................................... 24  
      1.1 Cardiovascular Disease and Type 2 Diabetes Mellitus ........................................... 26  
         1.1.1. Cardiovascular Disease ..................................................................................... 26  
         1.1.2. Diabetes ............................................................................................................... 28  
         1.1.3. Metabolic Syndrome ............................................................................................ 31  
         1.1.4. Non-Urban Risk - a Health Disparity ................................................................. 33  
         1.1.5. Absolute Cardiovascular Disease Risk ............................................................... 37  
         1.1.6. Diabetes Risk Assessment ................................................................................... 37  
         1.1.7. Prevalence and Epidemiology ............................................................................ 38  
         1.1.8. Management and Prevention .............................................................................. 42  
         1.1.9. Nurse-led Management ....................................................................................... 44  
         1.1.10. Tailoring Management ......................................................................................... 45  
         1.1.11. Summary ............................................................................................................. 47  
      1.2. Health Behaviour Change ......................................................................................... 48  
         1.2.1. Self-efficacy ............................................................................................................ 48  
         1.2.2. Health Locus of Control ...................................................................................... 52  
         1.2.3. Self-efficacy, locus of control and behaviour change ........................................... 54  
         1.2.4. The 5As model ...................................................................................................... 58  
         1.2.5. Motivational Interviewing ..................................................................................... 60  
         1.2.6. Summary ................................................................................................................. 60  
Chapter 2 Research Aims & Hypotheses ........................................................................... 62
2.1. Aims and Hypothesis ........................................................................................................ 64
  2.1.1. Thesis Outline ........................................................................................................... 64
  2.1.2. The MODERN Study ............................................................................................... 65
  2.1.3. Aims and Hypotheses .............................................................................................. 68
Chapter 3 Non-Urban Cardio-metabolic Disease Interventions ............................................ 71
Chapter 4 Research Platforms & Methods ............................................................................. 89
Chapter 5 Self-efficacy, Health Locus of Control and Cardio-metabolic Disease Risk: Baseline ...................................................................................................................... 118
Chapter 6 Self-efficacy, Health Locus of Control and Cardio-metabolic Disease Risk: Nurse-led Intervention .................................................................................................................. 146
Chapter 7 General Discussion and Conclusion ...................................................................... 180
  _7.1. Thesis Summary ........................................................................................................ 182
  _7.2. Research Findings ..................................................................................................... 184
  _7.3. Limitations ................................................................................................................ 187
  _7.4. Future Research ....................................................................................................... 188
  _7.5. Conclusions .............................................................................................................. 189
Bibliography .......................................................................................................................... 191
Thesis References ................................................................................................................... 191
Appendices .............................................................................................................................. 209
Appendix 1 .............................................................................................................................. 209
List of Figures

Figure 1. Atherosclerotic process in the artery | Page 27

Figure 2. Type 2 diabetes pathophysiology | Page 29

Figure 3. Risk factor rates classified by area of residence | Page 35

Figure 4. Australian health-care expenditure by disease classification | Page 39

Figure 5. Australia’s CVD expenditure from 2000-01, 2004-05 and 2008-09 | Page 40

Figure 6. Australia’s diabetes expenditure, 2000-01, 2004-05 and 2008-09 | Page 41

Figure 7. Standardised rate ratios of CVD prevalence, by region and disease type, 2004-05 | Page 42

Figure 8. Behaviour-specific self-efficacy scales | Page 51

Figure 9. Wallston Multidimensional Health Locus of Control (MHLC) Scales | Page 53

Figure 10. The influences of behaviour-specific self-efficacy and health locus of control | Page 56

Figure 11. The 5As model to help change health behaviours | Page 59
Figure 12. Two clinics established to conduct MODERN, located in Shepparton and Colac, VIC, Australia | Page 66

Figure 13. Thesis project schema within the MODERN study | Page 67

Figure 14. Thesis outline | Page 70
List of Tables

**Table 1.** Criteria for clinical diagnosis of the metabolic syndrome | Page 32

**Table 2.** Services received by remoteness – as a proportion of services received in metropolitan cities | Page 34

**Table 3.** Individuals employed in health occupations by remoteness, as a percentage of national totals | Page 34

**Table 4.** Predominant globally utilised absolute cardiovascular disease risk scores | 36

**Table 5.** GARDIAN classification guide | Page 46
List of Abbreviations

ACVDR - Absolute Cardiovascular Disease Risk

CHD - Coronary Heart Disease

CVD - Cardiovascular Disease

GARDIAN - Green, Amber, Red Delineation of Risk and Need

GP - General Practitioner

HDL-C - High-density lipoprotein cholesterol

HF - Heart Failure

HLC - Health Locus of Control

LDL-C - Low-density Lipoprotein Cholesterol

MetS - Metabolic Syndrome

MI - Motivational Interviewing

MODERN - Management to Optimise Diabetes and mEtabolic syndrome risk reduction via Nurse-led intervention

NHMRC - National Health and Medical Research Council

PBS - Pharmaceutical Benefit Scheme

T2DM - Type 2 Diabetes Mellitus

VIC - Victoria
Chapter 1
General Introduction
Chapter 1 describes the pathophysiology, incidence, prevalence and prevention of cardiovascular disease, type 2 diabetes mellitus and metabolic syndrome and highlights the disparities between urban and regional, remote and rural populations. Widely investigated psychological constructs, self-efficacy and locus of control, are discussed as mediators of behaviour change and as potential determinants of cardio-metabolic disease risk.
1.1 Cardiovascular Disease and Type 2 Diabetes Mellitus

Cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) are chronic cardio-metabolic diseases and are a major public health concern; they rank highly in global mortality rates, are predictable and largely preventable. To further understand the burden and causalities of these non-communicable diseases, their largely overlapping pathophysiology is discussed.

1.1.1. Cardiovascular Disease

CVD is an all-encompassing term referring to diseases of the blood vessels and the heart; inclusive of coronary heart disease (CHD), stroke and peripheral vascular disease [1]. CHD, also commonly referred to as ischaemic heart disease, is the predominant form of heart disease [1]; other types of heart disease that are common include cardiomyopathy, heart failure (HF) and atrial fibrillation. The two major manifestations of heart disease are angina and heart attack (myocardial infarction - MI) [1]. Angina is a chronic condition caused by acute shortages of blood supply to the heart, resulting in short bouts of periodic chest pain. Although angina is not typically life-threatening, it has been associated with an elevated risk of heart attack [1]. Blockages (by cholesterol or fatty substances) of arteries that deliver blood to the peripheral body parts such as the arms and legs is referred to as PVD, a disease which in severe circumstances can bring about thrombosis (or rupture) of the arterial wall [1]. Stroke, a form of cerebrovascular disease can similarly occur when blood supply to the brain is hampered by thrombosis or rupture in an artery; often resulting in permanent impairment or in some cases, death [1].
The predominant causal mechanism of CVD is atherosclerosis in the blood[2]. Atherosclerosis is a chronic inflammatory disorder that develops in the walls of blood vessels over many years[3, 4], as seen in Figure 1. It has been shown that this process can be instigated by a plethora of conditions such as high blood pressure (hypertension), high blood sugar (hyperglycaemia) and high blood fats (hyperlipidaemia)[3]. The stiffening of blood vessel walls associated with atherosclerosis results in plaque formation whereby blood flow is limited and often leads to an inadequate supply of blood (ischemia) to associated tissues[3]; this can lead to heart disease, stroke or PVD. Plaques, when unstable, can rupture to activate the accumulation of platelets, triggering acute blockages[4, 5].

![Atherosclerotic process in an artery](image)

**Figure 1.** Atherosclerotic process in an artery[6]
1.1.2. Diabetes

Glucose is a cell’s main source of energy. The regulation and metabolism of glucose are fundamental to our existence. The metabolism of glucose comprises an interplay of intricately coordinated physiological processes that mediate the movement of glucose from the digestive tract to the liver for circulation around the body, providing energy to muscle and fat cells\[7\]. Cells within the pancreas secrete insulin, the hormone responsible for the metabolism and regulation of glucose. Insulin aids in the suppression of glycogen (the main form of stored glucose) from the liver after a meal. The release of insulin into circulation assists in the maintenance of glucose homeostasis; the circulating blood glucose is taken up by the muscle and fat cells. In the instances where fat and muscle cells cease responding adequately to insulin or the production of insulin itself is reduced from the pancreas, diabetes develops\[7\].

Diabetes is defined by elevated blood glucose levels. It is characterised by a decreased production of, or sensitivity to, insulin\[8\]. While type 1 diabetes often affects the β-cells of the pancreas in younger individuals, type 2 diabetes mellitus\[9\] (T2DM) is the more common form of diabetes; characterised by elevated insulin resistance, with insulin secretion becoming compromised with disease progression (see Figure 2). Often coexisting with other metabolic disorders such as high blood lipids (hyperlipidaemia) and high blood pressure (hypertension), T2DM is strongly associated with physical inactivity and overweight and obesity\[9\].
If uncontrolled, T2DM can lead to deleterious health complication such as blindness, autonomic dysfunction, renal failure and paraesthesia (“pins and needles”)\[9\]. CVD is the most common cause of death among individuals with T2DM[9], not only due to the microvascular complications associated with long term elevated blood glucose levels but due to the commonalities in risk factors.

On average, those diagnosed with T2DM die eight years earlier than individuals without diabetes, predominantly due to CVD[11, 12]; the trio of hyperlipidaemia, hyperglycaemia and hypertension bring rise to the increased CVD risk associated with T2DM[13]. As such, controlling this trio can be an effective measure in preventing CVD in those with diabetes[14] given that an individual with T2DM has a 2- to 4-fold elevated risk for CVD[15]. These chronic diseases are closely associated, with over half of all individuals with T2DM prematurely dying of CVD[16, 17]. Additionally, around 3 in 10 people

Figure 2. Type 2 Diabetes pathophysiology[10]
with CHD have diabetes (30%) [17-19]. Furthermore, people with diabetes and CHD are predisposed to double the risk for mortality than individuals without diabetes, rendering them a group at very high risk for CVD death [20, 21].

Results from experimental research has revealed that independent of any other risk factors such as hypertension and dyslipidaemia, dysfunctional glucose metabolism elevates the risk of macrovascular disease due to its disruption of regular endothelial functionality, accelerating the formation of plaque in atherosclerosis, resulting in subsequent plaque rupture and eventual thrombosis [11-13]. Furthermore, there is an association between excess levels of insulin (hyperinsulinaemia), insulin resistance, and impaired glucose metabolism (all risk factors for macrovascular disease). Impaired glucose metabolism has also been known to compound the CVD risk attributed to other risk factors such as dyslipidaemia and hypertension [11].

The cause for the elevated risk for CVD in individuals diagnosed with diabetes is not yet fully understood, although T2DM has been shown to influence the development of a number of dangerous modifications to the arterial wall [10]. The endothelial dysfunction associated with diabetes, along with the potential thrombus development and elevated inflammatory activation, are all contributing factors that lead to atherosclerosis [1] and CVD. Lipid accumulation in the blood vessels is also known to be influenced by the insulin resistance associated with T2DM, with the excess LDL-cholesterol in those with diabetes leading to inflammatory responses and inhibition of nitric oxide production, a vasodilator [14]. Vagal nerve activity is often subsequently hampered, contributing to an elevated heart rate and blood pressure [14]. Along with reduced nitric oxide production, angiotensin-II and endothelin-I
production (vasoconstrictors) are elevated, while hyperglycaemia alone is thought to impact the integrity of the vascular wall via excessive oxidative stress resulting in endothelial dysfunction[1].

1.1.3. **Metabolic Syndrome**

A cluster of risk factors for T2DM and CVD, which commonly occur together, have been termed the metabolic syndrome (MetS). These risk factors include central obesity, elevated fasting glucose, elevated blood pressure and dyslipidaemia (reduced high-density lipoprotein cholesterol [HDL-c] and elevated triglycerides). The presence of three out of five risk factors qualifies an individual for the MetS. Table 1 reflects the International Diabetes Federation definition for MetS.

The MetS predisposes individuals to the development of CVD and T2DM. A concerning 2 in 5 Australian adults have three or more risk factors for CVD[22], forming the cluster of the most dangerous risk factors that typifies MetS. An insurmountable amount of evidence exists to link MetS with an elevated risk of CVD and T2DM; in individuals with MetS, CVD morbidity and mortality is 3-fold and 2-fold higher, respectively, and the T2DM risk is 5-fold greater[15].
Criteria for Clinical Diagnosis of the Metabolic Syndrome

<table>
<thead>
<tr>
<th>Measure</th>
<th>Categorical Cut Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated waist circumference</td>
<td>≥94cm for men;</td>
</tr>
<tr>
<td></td>
<td>≥80cm for women</td>
</tr>
<tr>
<td>Elevated triglycerides (drug treatment for elevated triglycerides</td>
<td>≥1.7 mmol/L</td>
</tr>
<tr>
<td>is an alternate indicator)</td>
<td></td>
</tr>
<tr>
<td>Reduced HDL-C (drug treatment for reduced HDL-C is an alternate indicator)</td>
<td>&lt;1.0mmol/L in males;</td>
</tr>
<tr>
<td></td>
<td>&lt;1.3mmol/L in females</td>
</tr>
<tr>
<td>Elevated blood pressure (antihypertensive drug treatment in a patient</td>
<td>Systolic ≥130 mmHg and/or</td>
</tr>
<tr>
<td>with a history of hypertension is an alternate indicator)</td>
<td>diastolic ≥85 mmHg</td>
</tr>
<tr>
<td>Elevated fasting glucose (drug treatment of elevated glucose is</td>
<td>≥5.6mmol/L</td>
</tr>
<tr>
<td>an alternate indicator)</td>
<td></td>
</tr>
</tbody>
</table>

Certain risk factors have been recognised as increasing the risk of developing CVD. Whilst sex, age and family history are ‘non-modifiable’ risk factors, behavioural risk factors including physical inactivity, poor diet, smoking and excessive alcohol consumption are considered ‘modifiable’. These health behaviours can influence biomedical risk factors such as high blood pressure, overweight/obesity, and blood glucose and lipid levels[22].

A large case-control study (The INTERHEART Study) of over fourteen-thousand participants with acute myocardial infarction found that approximately 9 in 10 of all cardiovascular events could be explained by nine risk factors in both male and female individuals [24]. These include diabetes, obesity/overweight, psychosocial condition, dyslipidaemia, hypertension, physical inactivity, smoking, poor diet, and excessive alcohol consumption[24]. Interestingly the familial predispositions to heart attacks were also considered, however this genetic susceptibility explained only an additional one percent of the sample attributable-risk when combined with the other risk factors[24]. Similarly,
another study recruited over eighty thousand healthy nurses free from diabetes and CVD who were followed for sixteen years[25]. Individuals with normal weight, diets rich in polyunsaturated fat and fibre, regular physical activity, no smoking and moderate alcohol consumption had an over 90% lower incidence of T2DM than individuals without those characteristics and behaviours[25]. Given that the increasing incidence of cardio-metabolic diseases can be largely attributed to modifiable risk factors, reducing these via health programs focussing on behaviour change is a rational approach to preventing these chronic diseases.

1.1.4. Non-Urban Risk - a Health Disparity

People living in rural, regional and remote (non-urban) locations have poorer health compared to their metropolitan counterparts[26] with mortality rates rising with greater remoteness[27]. Non-urban residing individuals are renowned for having higher levels of antecedent risk for CVD, and this observation is paralleled around the globe from Africa[28] and Europe[29], to USA[30] and Australia [31]. This disparity may be attributed to several underlying differentials. Access to and utilisation of health-related services such as general practitioners (GP), specialist and allied health services reduce with increasing remoteness in contrast to metropolitan areas (See Table 2). Furthermore, there are still shortages of all types of health professionals currently practicing within non-urban areas, with greater shortages in more remote areas (See Table 3).
Table 2. Services received by remoteness – as a proportion of services received in metropolitan cities[31]

<table>
<thead>
<tr>
<th>Service Type</th>
<th>Inner Regional</th>
<th>Outer Regional</th>
<th>Remote</th>
<th>Very Remote</th>
</tr>
</thead>
<tbody>
<tr>
<td>GP Services</td>
<td>84%</td>
<td>79%</td>
<td>71%</td>
<td>54%</td>
</tr>
<tr>
<td>Specialist Services</td>
<td>74%</td>
<td>59%</td>
<td>38%</td>
<td>30%</td>
</tr>
<tr>
<td>Allied Health Services</td>
<td>75%</td>
<td>45%</td>
<td>24%</td>
<td>9%</td>
</tr>
</tbody>
</table>

Table 3. Individuals employed in health occupations by remoteness, as a percentage of national totals[31]

<table>
<thead>
<tr>
<th>Occupation</th>
<th>Metropolitan</th>
<th>Inner Regional</th>
<th>Outer Regional</th>
<th>Remote</th>
<th>Very Remote</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Practitioners</td>
<td>38%</td>
<td>21%</td>
<td>17%</td>
<td>16%</td>
<td>8%</td>
</tr>
<tr>
<td>Medical Imaging Workers</td>
<td>40%</td>
<td>27%</td>
<td>19%</td>
<td>11%</td>
<td>3%</td>
</tr>
<tr>
<td>Pharmacists</td>
<td>35%</td>
<td>24%</td>
<td>21%</td>
<td>14%</td>
<td>6%</td>
</tr>
<tr>
<td>Allied Health Workers</td>
<td>34%</td>
<td>25%</td>
<td>19%</td>
<td>16%</td>
<td>6%</td>
</tr>
</tbody>
</table>

On average, metropolitan residents earn higher incomes than those residing in rural, regional or remote areas. The mean age amongst non-urban communities is greater than that of their metropolitan counterparts and there are more residents with a disability in non-urban areas[32]. There are also education disparities between urban and non-urban communities, with only 40% of very remote residents completing Year 12, compared to the
national average of 72%. This contributes to the underrepresentation of non-urban residents in higher education, making up less than a fifth of tertiary students in Australia; with only 12% of tertiary education being provided in non-urban areas[32]. Disparities also extend to infrastructure and communications, with local governments being responsible for local infrastructure directly related to lifestyle and wellbeing, funding among non-urban councils is minimal. While over two-thirds of homes in major cities have access to the internet, only 42% of non-urban residences do. Education and access to the internet can not only play large roles in health knowledge but may lead to different thinking and decision-making patterns concerning health behaviours[33]. Alarmingly, the National Institute of Industry and Economic Research reported that it costs non-urban individuals 2 to 10 times more to access education, health services and other essential services such as aged care[31].

Figure 3. Risk factor rates classified by area of residence[31]
The Australian National Health Survey for 2014/15 revealed that in comparison to metropolitan individuals, residents of non-urban communities reported higher rates of smoking, and were 32% more likely to drink excessive amounts of alcohol and 40% more likely to report excessive sedentary behaviour\[31\]. A total of while 61% of major city residents were overweight or obese compared to around 70% of those living in regional, rural or remote locations (see Figure 3) \[31\].

**Table 4.** Predominant globally utilised absolute cardiovascular disease risk scores

<table>
<thead>
<tr>
<th>Name</th>
<th>Study, Region</th>
<th>Year</th>
<th>Age</th>
<th>Sex</th>
<th>Chol</th>
<th>LDL</th>
<th>HDL</th>
<th>CRP</th>
<th>T2DM</th>
<th>HbA1c</th>
<th>Smoking</th>
<th>Family History</th>
<th>BMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Framingham[37]</td>
<td>USA</td>
<td>1998</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>PRO-CAM[38]</td>
<td>Germany</td>
<td>2002</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>QRISK[39]</td>
<td>UK</td>
<td>2007</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Reynolds (M)[40]</td>
<td>USA</td>
<td>2008</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Reynolds (W)[41]</td>
<td>USA</td>
<td>2007</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>EURO-SCORE[42]</td>
<td>Europe</td>
<td>2003</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td></td>
</tr>
</tbody>
</table>
Non-urban dwelling residents tend to be from lower socioeconomic backgrounds (reflected by occupational and educational status\textsuperscript{[34]}), engage in sub-optimal dietary and lifestyle behaviours\textsuperscript{[27]} and have overall poorer attitudes toward health\textsuperscript{[35]}. These key differentials culminate to elevate the burden of health risk, particularly cardio-metabolic disease risk and suggest that a one-size-fits-all approach to preventive health may not always suffice.

1.1.5. \textbf{Absolute Cardiovascular Disease Risk}

Absolute cardiovascular disease risk (ACVDR) refers to the likelihood of an individual developing CVD based on the presence and level of selected risk factors\textsuperscript{[36]}. It takes into account the interaction and cumulative impact of numerous risk factors. The clinical benefit of determining an individual's ACVDR is to direct management leading to improved health outcomes for those who may benefit most. Many ACVDR scores have been developed and validated and are in use today; varying on risk factors and covariates. Key examples are shown in Table 4. In Australia, the Framingham score is the preferred ACVDR score, recommended by the National Vascular Disease Prevention Alliance.

1.1.6. \textbf{Diabetes Risk Assessment}

Similar to CVD, there are various risk calculators used to estimate the likelihood of developing T2DM. The most widely acknowledged T2DM risk scores are the (Finnish) Diabetes Risk Score\textsuperscript{[43]} and AUSDRISK\textsuperscript{[44]}, the latter which is validated and widely utilised in Australia. The AUSDRISK assessment tool uses demographic, lifestyle and anthropometric measures to determine the risk of diabetes within the next five years. Age, sex, family history, smoking status, waist
circumference, physical inactivity, BMI, use of antihypertensive medications and history of high blood glucose are recorded to elucidate T2DD risk.

1.1.7. Prevalence and Epidemiology

Over the last few decades, there has been an epidemiological shift globally from mortality dominated by communicable diseases such as malnutrition and infectious diseases, towards non-communicable diseases and lifestyle oriented conditions\[45\]. What began in high-to-middle-income nations has now stretched to lower-income parts of the globe. Non-communicable diseases account for around 65% of deaths worldwide\[45, 46\], with CVD and diabetes accounting for 48% and 3.5% respectively\[46\]. CVD devastatingly remains the major cause of morbidity and premature mortality globally, representing around 31% of all deaths\[47\]. In Australia, CVD accounted for 30% of all deaths in 2013, and although the number of deaths by CVD in Australia has dropped by 8% since 2004\[48\], prevalence remains on the rise and one in six Australians are affected. The ageing population has had a large impact on the rising CVD prevalence; the elderly were the most affected with 62% of individuals aged over 75 diagnosed with a cardiovascular condition compared to 5% of the population under the age of 45\[49\]. Mortality rates were higher in males than in females for the majority of cardiovascular conditions\[50\]. However, more females die from CVD than men due to typically longer female life-expectancies\[46\].

From a primary care perspective, in Australia, one in ten primary care consultations in 2009-10 were a direct result of CVD\[51\]. Over 1 in 20 hospitalisations received a principle CVD diagnosis, with a further 1 in 20 involving a secondary diagnosis of CVD. CVD is the main cause of
hospitalisations in Australia, with around 475,000 directly related hospitalisations each year. The direct disease health care costs associated with CVD exceeds that of any other, with almost $8 billion spent on CVD in 2008-09, making up around 12% of Australia’s healthcare expenditure (Figure 4)[52]. In the same year, an Australian Statistics on Medicine publication revealed spending in excess of $2 billion on Pharmaceutical Benefits Scheme items for CVD[53]. CHD accounted for over $2 billion dollars, with stroke costing health care over $600 million in 2008-09 and the average hospitalisation costing $9,982 for males, and $8,634 for females. Between 2001 and 2009, there was a 55% rise in annual CVD-related health care expenditure52 (Figure 5).

![Figure 4. Australian health-care expenditure by disease classification, 2008-09][52]
Similarly, T2DM poses a significant burden globally with one in eleven adults having diabetes, and only half of these diagnosed\cite{8}. Globally, it is estimated diabetes accounts for over 5 million deaths every year while over $670 billion is directly spent on diabetes healthcare (Figure 6) annually\cite{52}. Alarmingly, over 1.6 million Australians have diabetes, with around 280 being newly diagnosed daily. The burden to the Australian economy caused by diabetes, particularly T2DM, is estimated to be in excess of $14 billion\cite{50}; an 86% increase since 2001-02. In 2008-09, it is estimated that diabetes accounted for 2.3% of Australia’s health expenditure, amounting to $1.5 billion, with another $150 million spent on government diabetes programs and initiatives.
The prevalence of CVD increases with remoteness (Figure 7): compared to major cities, inner and outer regional areas of Australia have 11% greater incidence of CVD while the prevalence in remote and very remote regions are even greater (15%) [54]. Not surprisingly, mortality rates and hospitalisations from CVD showed similar patterns with greater remoteness [54]. Similarly, the prevalence of T2DM increases with remoteness, with 6.7% of individuals living in remote areas and 6.0% in inner regional areas diagnosed with T2DM, compared to 4.7% of urban residents [31]. These disparities in prevalence and outcomes show that the need for effective treatment and management of risk factors and primary prevention of CVD increases with greater distance from major cities.
1.1.8. Management and Prevention

While CVD can strike abruptly (particularly in the form of a stroke or sudden heart attack), these occurrences are typically preceded by a history of risk factors for CVD. T2DM also has a long ‘incubation’ period and many people have elevated glucose levels and other risk factors for an extended period of time prior to diagnosis. A combination of risk factors can contribute to the development of CVD and T2DM; 2 in 3 Australians have three or more of these\(^{49}\). Poor diet, physical inactivity, abdominal obesity, psychosocial factors, smoking, elevated blood pressure, diabetes and impaired blood lipid levels account for around 90% of the risk of a heart attack (myocardial infarction) observed worldwide\(^{13}\). Primary health care is fundamental in the management of T2DM and CVD, particularly given disease progression over
an extended time frame and the mild severity characterising its early stages. Prevention of CVD and T2DM treatment and monitoring of risk factors is paramount in primary health care. Primary care guidelines both locally and internationally emphasise detailed risk assessment to allow efficacious management of modifiable health behaviours or risk factors via lifestyle changes (e.g. exercise, weight loss) and pharmacotherapy (e.g. blood pressure lowering medication)\[56-62\].

Recent efforts to elucidate the impact of lifestyle modification on the risk of T2DM or CVD have shown such impressive risk reductions\[63\] that many now recommend behaviour modification as the primary mechanism for the prevention of cardio-metabolic disease\[64, 65\]. Despite the overwhelming amount of data revealing the benefits of behavioural changes for T2DM and CVD, individuals are increasingly succumbing to the burdens associated with poor dietary habits, low levels of physical activity, and excess body fat.

While pharmacological approaches using therapies such as acarbose\[66\] and metformin\[67\] have shown to prevent the onset of T2DM in those at risk of the disease, conclusive evidence has been provided elucidating the benefits of behavioural and lifestyle modification, particularly those targeting common risk factors, namely diet, physical inactivity, smoking and excessive alcohol consumption\[68\]. Continual positive health behaviour changes can delay or even prevent the onset of CVD and diabetes\[68-71\].

Many studies (predominantly randomised controlled trials) assessing the effectiveness of lifestyle modification via education and counselling in the absence of pharmacological treatments to improve key cardiovascular risk factors, have been repeatedly undertaken in a primary care setting and
reviewed to show that even slight behavioural and lifestyle changes (such as improved diet, physical activity, smoking cessation and reducing alcohol consumption) can significantly reduce cardiovascular risk and morbidity and mortality\[68]. Several large-scale studies have also emphasised the positive effect of lifestyle and health behaviour changes on diabetes risk\[72-75]. The Greater Green Triangle (GGT) randomised controlled trial of 237 individuals 40-75 years of age, for example, reported improvements in body weight, glucose, total cholesterol, low-density lipoprotein cholesterol (LDL), triglycerides and blood pressure after six, 90-minute nurse-led sessions over eight months\[73]. Given many of the positive results of lifestyle modifications accumulate over time, adhering to these changes over the long term increase both population and individual benefits\[68]. Despite interventions targeting physical activity and diet showing positive rates of initial behavioural changes, difficulties associate with maintaining new habits mean these are often not translated into long-term lifestyle changes\[76]. More focus on the maintenance of health behaviours post-intervention is needed. To address the rise in risk factors for CVD and T2DM, systems of care need to be implemented.

1.1.9. Nurse-led Management

Given the difficulty associated with health behaviour changes, support and advice can prove to be important in achieving risk reductions. A nurse-led management and prevention approach that is ‘user-friendly’, more accessible, and that encourages and motivates whilst forming a rapport with individuals can be effective. There is evidence elucidating the benefits of nurse-led interventions on significant risk factors \[77]. Also, nurse-led
management has been effective in a secondary prevention context and chronic disease management to reduce recurrent hospital stays (particularly in heart failure[78-84]), as well as in primary prevention to minimise risk levels[85]. For example, The Protecting Healthy Hearts Program[85] was a 6-month pre/post observational study involving 530 adults aiming to reduce risk factors for CVD and T2DM via a nurse-led intervention. The nurse-led program was a multifactorial intervention with a 6-month follow-up, with individualised management and self-care plans established with participants. Clinically significant changes in 326 individuals were recorded, with improvements observed in blood pressure, weight, total cholesterol, BMI and overall ACVDR.

1.1.9. Tailoring Management

The GARDIAN (Green, Amber, Red Delineation of Risk and Need) management system represents an assessment of individual risk and need that informs the intensity and frequency of a future healthcare intervention[86]. Level of need for ongoing support and intervention can be determined via three risk levels; green (low risk), amber (intermediate risk) and red (high risk) according to three key domains. 1) Clinical stability refers to any limitations to functionality or chronic symptoms related to CVD or any related conditions. 2) Gold-standard management is assessed by comparing all prescribed and recommended treatments according to gold-standard guidelines for CVD prevention. 3) significant social, psychological and behavioural factors that may impact on a person’s health is defined as their Holistic profile; this can range from their knowledge and awareness of disease to their cognitive function, mental health and self-care abilities. The GARDIAN system can be applied from a chronic disease management, secondary
prevention or primary prevention stand-point. Table 5 is a GARDIAN classification guide from a primary prevention perspective.

<table>
<thead>
<tr>
<th>RISK/DOMAIN</th>
<th>Clinical stability</th>
<th>Gold-standard management</th>
<th>Holistic profile</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LOW (GREEN)</strong></td>
<td>No extreme risk factors and low-to-moderate CVD risk prediction</td>
<td>Ideal risk factor control, appropriate treatment, and lifestyle/dietary behaviours to prevent a primary CVD event</td>
<td>Optimal self-care behaviours and knowledge, medication adherence, and optimal social support. Factors likely to positively influence health</td>
</tr>
<tr>
<td><strong>INTERMEDIATE (AMBER)</strong></td>
<td>As above</td>
<td>Suboptimal risk factors, lack of gold-standard treatment, and/or management to prevent CVD</td>
<td>Social, cognitive, or mental health problems, suboptimal self-care behaviours and knowledge, and/or medication nonadherence</td>
</tr>
<tr>
<td><strong>HIGH (RED)</strong></td>
<td>Extreme risk factors present and/or high CVD risk prediction in older individual or family history of premature CVD</td>
<td>As above</td>
<td>As above</td>
</tr>
</tbody>
</table>

Table 5. GARDIAN classification guide[86]
1.1.10. Summary

The incidence and prevalence of CVD and diabetes are increasing both locally and globally. Primary prevention is paramount and largely modifiable behavioural risk factors can be targeted to reduce risk and mortality. The challenge remains to translate these behavioural changes into long-term lifestyle habits, particularly in non-urban communities where compared to urban populations, individuals are at higher risk. There are many barriers and challenges posed to adopting and maintaining new health behaviours over the long term which may be explained by individual psychological determinants.
1.2. Health Behaviour Change

An individual’s health and well-being are strongly influenced by lifestyle factors and behaviours such as physical activity, diet, drinking and smoking - all of which have the potential to be modified. The effectiveness of an intervention is extremely reliant on an individual’s adherence to prescribed behaviours or lifestyle modifications, which is typically poor [87]. Facilitating behaviour change is paramount for disease prevention and health promotion. Perceived behaviour-specific self-efficacy and multidimensional health locus of control, as psychological constructs, have both been identified as significant determinants of health behaviour change. Self-efficacy (or perceived self-efficacy) refers to one’s belief in their ability to succeed in specific situations [88]. Health locus of control is related to how strongly a person believes they have control over the situations and experiences that may affect their health. In addition, behavioural counselling (therapy aiming to assist in changing behaviour(s)) can have a significant influence on health behaviour change [88]. Elucidating the individual barriers to health behaviour change may be important to combat cardio-metabolic disease. Many individuals have to make multiple health behaviour changes and few studies test or apply health behaviour theory or behaviour change principles.

1.2.1. Self-efficacy

Albert Bandura introduced self-efficacy as a key concept of Social Cognitive Theory [89]. Self-efficacy is widely utilised in health behaviour research to explain and predict human behaviour [90-92]. It reflects an
individual’s optimistic belief in one’s ability to overcome obstacles associated with performing an action in a particular context [88], such as performing 30-minutes of physical activity every day, despite a busy schedule. The theory suggests that an individual, their environment and the emotional and cognitive processes particular to that individual all interact to regulate behaviour[88]. It has been argued that specific behavioural choices are influenced by judgements of behaviour-specific self-efficacy. (i.e. individuals are more likely to adopt behaviours they believe they can perform and avoid behaviours they feel exceeds their abilities). Furthermore, it has been highlighted that the effort and persistence an individual applies in adopting a new behaviour is dependent on their self-efficacy for the behaviour. An individual with a high self-efficacy may be more likely to adopt a particular novel positive health behaviour[88].

Some individuals may have low confidence in their ability to maintain novel health behaviours but are relatively confident in taking the initiative and setting goals. Contrastingly, others may have more confidence in their abilities to maintain a desired behaviour but are not so confident when it comes to getting started. Given this phase-specific nature, self-efficacy is often divided into two specific function stages. Action self-efficacy constitutes the pre-actional phase, the period prior to the adoption of a novel behaviour[93]; people high in self-efficacy will take initiatives in order to succeed, picturing successful scenarios and strategies. Those who doubt themselves and procrastinate, while also often imagining negative scenarios, have a low self-efficacy. Coping self-efficacy refers to the post-actional phase of the adoption of a new health behaviour[93]. Those high
in self-efficacy are able to deal with obstacles and barriers that they are faced with during the maintenance period of a behaviour. They are able to respond with greater resistance, persistence confidence, various strategies and effort. Those with low self-efficacy, are often not able to recover and maintain the behaviour when setbacks ensue, however. It is possible to be high in action self-efficacy but low in coping self-efficacy, or conversely low in action self-efficacy and high in coping self-efficacy [93]. Self-efficacy varies on scales of magnitude, generality and strength and as such should be assessed using these dimensions. Generalised self-efficacy can be assessed although can be domain specific[94]. For example, an individual who may have a high self-efficacy for engaging in physical activity may not necessarily have a high self-efficacy for engaging in healthy eating. As such, self-efficacy assessments are specific to behaviour, e.g. physical activity, nutrition, alcohol consumption and smoking,[95] (and will be assessed in this manner as part of this thesis).

In health behaviour research, behaviour-specific self-efficacy scales (Figure 8) have been developed and widely utilised[96].
Nutrition

It is always hard to change. I am sure I can improve my daily nutrition, even if...
...I have to force myself to start immediately
...I like to snack and indulge

It is important to maintain good dietary behaviours. I am sure I can eat healthily in the long run, even if...
...I feel a strong temptation to snack and indulge.
...I feel tense or stressed out.

In spite of good intentions, smaller or larger relapses may occur. Imagine you indulged in tempting foods for some time. How confident are you about resuming a healthy diet? I am sure I can resume a healthy diet, even if...
...I have already relapsed several times.
...I feel weak when facing temptations or feel unable to pull myself together

Physical Activity

It is always hard to change. I am sure I can do more regular physical activity, even if...
...I have to force myself to start immediately.
...I prefer not to exercise.

It is important to stay physically active. I am sure I can keep being physically active regularly in the long run, even if...
...I feel a strong temptation not to be active
...I feel tense or stressed out

In spite of good intentions, smaller or larger relapses may occur. Imagine you stopped exercising for some time. How confident are you about resuming exercising? I am sure I can be physically active again regularly, even if...
...I have already paused for several times.
...I feel weak and unable to pull myself together

Alcohol Consumption

It is always hard to change. I am sure I can control myself to...
...reduce alcohol consumption
...not to drink alcohol at all

It is important to drink sensibly. I am sure I can maintain this in the long run even if...
...I feel a strong temptation to drink.
...I feel tense or stressed out

In spite of good intentions, smaller or larger relapses may occur. How confident are you about stopping drinking again? I am sure I know...
...how to deal with relapses into my old drinking habits
...when to especially watch out in order not to drink alcohol again

Smoking

Some situations make it hard to quit smoking. I am sure I can stop smoking, even if...
...I have to force myself to quit immediately
...I get little support

It is important not to smoke, I am sure I can be smoke-free in the long run, even if...
...I feel a strong temptation or urge to smoke
...I am around friends and colleagues who smoke

In spite of good intentions, smaller or larger relapses may occur. How confident are you about quitting smoking again? I am sure I can quit smoking again, even if...
...I have failed several times
...I have to start all over again several times until I succeed

Figure 8. Behaviour-specific self-efficacy Scales. Schwarzer and Renner[96]
1.2.2. Health Locus of Control

Health locus of control\[97\] is a causal attribution construct that relates to whether an individual has an internal, powerful others or chance health belief; that is, does a person regard health outcomes as personally determined, or not.

It is generally assumed that individuals who believe that they have control over their health may be more likely to engage in health-enhancing behaviours and avoid health-compromising behaviours and as a result, may have better health outcomes\[98\]. It can be further assumed that individuals who believe that their health is mainly determined by healthcare professionals may also be more likely to engage in health-enhancing behaviours, that is, they may be more receptive to advice and translate it into action\[99\]. However, evidence for this is scarce; reliance on others (e.g. a family member or health professional) can also undermine one’s own action towards health. Individuals who feel their health is mediated by luck and chance may be less likely to consciously engage in health-enhancing behaviours. Findings utilising the Rotter’s Locus of Control scales and the Multidimensional health locus of control scales have shown that individuals who have a high internal locus of control behave in a more healthy way in relation to a number of important cardiovascular health-related behaviours\[100, 101\]. When assessing health locus of control, the Wallston Multidimensional Health Locus of Control scale\[102\] has been a popular choice amongst health behaviour researchers (Figure 9). The scale consists of 18 items, aiming to reveal the
degree to which an individual sees their health as personally determined or not.

<table>
<thead>
<tr>
<th>Statement</th>
<th>STRONGLY AGREE</th>
<th>SOMEWHAT AGREE</th>
<th>SLIGHTLY AGREE</th>
<th>SLIGHTLY DISAGREE</th>
<th>SOMEWHAT DISAGREE</th>
<th>STRONGLY DISAGREE</th>
</tr>
</thead>
<tbody>
<tr>
<td>If I get sick, it is my own behaviour which determines how soon I get well again.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>No matter what I do, if I am going to get sick, I will get sick.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Having regular contact with my physician is the best way for me to avoid illness.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Most things that affect my health happen to me by accident</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Whenever I don’t feel well, I should consult a medically trained health professional</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>I am in control of my health</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>My family has a lot to do with my becoming sick or staying healthy</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>When I get sick, I am to blame</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Luck plays a big part in determining how soon I will recover from an illness</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Health professionals control my health</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>My good health is a matter of good fortune</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>The main thing which affects my health is what I myself do</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>If I take care of myself, I can avoid illness.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Whenever I recover from an illness it’s usually because other people (for example, doctors, nurses).</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>No matter what I do, I’m likely to get sick.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>If it’s meant to be, I will stay healthy.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>If I take the right actions, I can stay healthy.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Regarding my health, I can only do what my doctor tells me to do.</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
<td>O</td>
</tr>
</tbody>
</table>

**Figure 9.** Wallston Multidimensional Health Locus of Control (MHLC) Scales [104]
It is important to assert the differences between self-efficacy and health locus of control; while one may have an impact on the other, they remain distinct. Internal locus of control is part of the self-efficacy construct. Self-efficacy pertains to one’s conviction that one has the capability to overcome challenges which is equivalent to an internal causal attribution of one’s future behaviour. Attributions to chance or powerful others are incompatible with self-efficacy. A difference between locus of control and self-efficacy lies in the time perspective: self-efficacy is always prospective, whereas locus of control can also be retrospective. Moreover, self-efficacy is an operative construct, and relates to future behaviour (e.g. physical exercise next week), whereas locus of control can apply to external past events (e.g. an accident or death of a loved one). Despite this, Bandura suggested that the conviction that one’s health is mediated by one’s own health behaviour (internal locus of control) can have a variety of influences upon behaviour-specific self-efficacy and subsequent behaviour\cite{89}. While self-efficacy is task-specific, locus of control remains specific to a domain (i.e. health).

1.2.3. Self-efficacy, locus of control and behaviour change

Self-efficacy can be a useful predictor of health behaviour\cite{88}. For smoking, it was identified that self-efficacy could be used as a predictor of both smoking cessation and maintenance of cessation\cite{103-107}. Self-efficacy has been shown to be a powerful predictor of other health behaviours such as physical activity\cite{108}, cardiac rehabilitation\cite{109, 110}, diet and weight loss\cite{111, 112}. For example, a 2014 study by Smit et al., found that individuals with a higher self-efficacy in regards to smoking
have significantly lower drop-out rates\cite{103} compared to those with a lower self-efficacy. Self-efficacy has also been shown to be predictive of interventional response and outcome\cite{88}. An internal health locus of control has been associated with a reduced ACVDR\cite{112}. Wallston suggested that self-efficacy may only be predictive for those who have an internal health locus of control.

Although a 1994 study by Winkleby\cite{113} et al. revealed self-efficacy scores to be lower among individuals who did not respond well to a heart disease intervention than those who did, to our knowledge no other studies have investigated the association between cardio-metabolic risk and the psychological constructs self-efficacy or health locus of control, particularly in a primary prevention scenario. Although a range of studies have revealed the association between self-efficacy, health locus of control, and many common health behaviours, little work has focused on elucidating the influence on cardio-metabolic disease risk. As shown in Figure 10 this is the aim of this thesis work.
Figure 10. The influences of behaviour-specific self-efficacy and health locus of control on health behaviour and potential associations with cardio-metabolic disease risk

Self-efficacy can be ‘improved’ via frequently repeating practices which are positively experienced and even by verbal persuasion its enhancement is associated with subsequent health behaviour modifications [100]. Given the potential of these constructs to aid in the prediction of health behaviour adoption and adherence, and the known
impact health behaviour change can have on cardio-metabolic risk, further investigation into the potential influence (Figure 10) an individual’s self-efficacy and locus of control has on one’s subsequent cardio-metabolic health is needed.

The amount of effort exerted and persistence required to overcome and strive in the face of setbacks and barriers to behaviour change is largely mediated by self-efficacy. Improving one’s self-efficacy in a particular domain can prove beneficial. Self-efficacy is derived from four sources[114], all of which can be enhanced. Primarily, self-efficacy may be enhanced by achieving a high level of competency or mastery for a given behaviour; the individual may feel this success can be repeated, particularly if they feel internally responsible for the success. Personal mastery is the strongest source of self-efficacy. Vicarious experience can also result in an enhanced self-efficacy; a successful second-hand mastering of behaviour or a situation can result in a social comparison between a ‘model person’ and the individual. Verbal persuasion by others and emotional arousal can also prove valuable in enhancing self-efficacy, where an individual may feel no anxiety or fear in a difficult situation and feel a greater ability to master a situation.

The enhancement of self-efficacy in physical activity has been observed in interventions focusing on vicarious experience, past performances of the participant, or others[115]. Verbal persuasion was associated with significantly higher levels of physical activity, and self-efficacy [116]. Similarly, nutritional or dietary self-efficacy improved via interventions utilising performance feedback, self-monitoring, reviewing of intentions
and success rewards[117]. Smit et al. note that self-efficacy was the most effective forecaster of a successful quitting attempt for smoking[118]. Evidence suggests that self-efficacy can be enhanced, whether this leads to any improvements in health behaviour is unclear[103].

Wallston indicated that self-efficacy may only be predictive if an individual holds an internal health locus of control and that investigators and/or educators should focus on training participants toward an internal health locus of control[119]. Ultimately, training responsible individuals with an internal health locus of control who acknowledge the advice of a health professional as a resource, also recognise actions as their own, may lead to better health outcomes.

1.2.4. The 5As model

The 5As model[120] consists of behavioural counselling to help individuals to change multiple health behaviours. Typically conducted by a health professional, the objective is to aid participants or patients to develop a personal action plan by systematically applying a series of five interrelated behaviour change principles (the 5As). Figure 11 highlights a personal action plan mediated by the 5As: assess, advise, agree, assist and arrange. The 5As model begins with an evaluation of [assess] an individual’s current state of knowledge, current health behaviours and lifestyle along with their perceptions and beliefs regarding their health and behaviours. The individual is then advised [advise] on the certain health risks associated with their behaviours and the benefits of changing behaviours. Goals are then set collaboratively [agree], concurring with the individual’s interests, confidence and self-efficacy for a change of
behaviour. The individual is then aided in identifying barriers, problems, and the formulating of strategies for achieving behaviour change [**assess**]. Finally, a follow-up plan is established [**arrange**], entailing face-to-face visits, phone calls or simple reminders; establishing consistent and timely follow-up cultivates trust between a nurse and patient[119]. The 5As model is rooted in behaviour change theory, particularly self-efficacy enhancement[120].

---

**Assess** Beliefs, Behaviour & Knowledge. | **Advise** Provide specific information about health risks and benefits of change | **Agree** Collaboratively set goals based on patient’s interest and confidence in their ability to change the behaviour | **Assist** Identify personal barriers, strategies, problem-solving techniques and social/environmental support | **Arrange** Specify plan for follow-up (e.g. visits, phone calls, reminders etc.)

**Figure 11.** The 5As model to help change health behaviours
1.2.5. Motivational Interviewing

Motivational interviewing (MI) is a well-established method of interacting with individuals to enhance and promote (health) behaviour change[121]. A review of motivational interviewing in improving cardiovascular health found strong evidence to suggest that MI is an effective approach in stimulating motivation for health behaviour change[122]. It depends on elucidating and mobilising the patient’s inherent goals and values to stimulate and provoke behaviour and lifestyle change, acknowledging that motivation to take on new behaviours is elicited from the patient. The premise of MI is to reveal and resolve uncertainty and to focus on the perceived positive results associated with behaviour change. Continual interaction with a health professional in the context of MI can establish a readiness to change, a trait not typical of most individuals. While MI inherently pertains to a set of counselling styles and techniques, it is a directive and therapeutic relationship with acknowledgement of the individual’s autonomy, ultimately aiming to elicit health-promoting behaviour change. The support of self-efficacy is a fundamental principle of the MI approach; enhancing a patient’s belief in their ability to change health behaviours is paramount for success.

1.2.6. Summary

Eliciting health behaviour change is difficult. Behaviour-specific self-efficacy and health locus of control as psychological constructs may prove insightful in investigating the barriers and enablers of behaviour change, and subsequent health risks. MI and other behavioural
counselling techniques may aid in eliciting health behaviour change that can, in turn, improve health outcomes, including concerning cardio-metabolic health. Despite the evidence revealing the enhancement of behaviour-specific self-efficacy in many domains and the malleability of health locus of control, analyses indicating that changes in these constructs mediate health behaviour change and subsequent health outcomes are scarce. As such, the associations between behaviour-specific self-efficacy and health locus of control, and their influence on health behaviour change and subsequent cardio-metabolic health outcomes will be further investigated as part of this thesis.
Chapter 2
Research Aims & Hypotheses
Chapter 2 outlines the main aims, hypotheses and research streams pertaining to this thesis work. The rationale, thesis structure and research publications derived from this research program are also summarised.
2.1. Aims and Hypothesis

2.1.1. Thesis Outline

This thesis describes a program of research exploring the associations between behaviour-specific self-efficacy, health locus of control, behaviour changes and cardio-metabolic risk status. It forms a sub-study of the larger Management to Optimise Diabetes and mEtabolic syndrome risk reduction via Nurse-led intervention (MODERN) Study. As described in Chapter 1, CVD and T2DM pose a significant burden on global health and the economy with risks increasing with greater remoteness from metropolitan areas. Irrespective of contextual factors however, this first chapter also highlights the psychological factors that may be important barriers and enablers to health behaviour change. Changing health behaviours can reduce cardio-metabolic disease risk. Although behaviour change is a fundamental vehicle of many interventions, little research has focussed on the correlation between self-efficacy and health locus of control and cardio-metabolic risk status, and whether self-efficacy and health locus of control can be adapted to positively impact health outcomes. In Chapter 2, the aims and hypotheses of this thesis project are described. Chapter 3 presents a systematic review and meta-analysis of a broad range of primary prevention programs aiming to improve cardio-metabolic risk in non-urban communities. This review was important to help understand effective characteristics and mechanisms of a successful intervention to reduce CVD and T2DM risk in non-urban populations. This has been published as the following peer-reviewed manuscript:

The methodology, study design and screening results of the MODERN study are presented in Chapter 4. Chapter 5 reports the results of the first study that examines the association between behaviour-specific self-efficacy, health locus of control and cardio-metabolic risk status. In Chapter 6, the effect of a nurse-led intervention on behaviour-specific self-efficacy, health locus of control and cardio-metabolic risk status is described. Chapter 7 summarises the overall findings of this thesis and discusses the implications and limitations of this work. General conclusions are presented and possible future directions of this research are commented upon.

Primary prevention interventions, particularly nurse-led models of care, can aid in the control of risk factors for CVD and T2DM. Despite this, a limited number of interventions have targeted higher risk non-urban populations. An increased number of strategies that influence the psychological determinants of health are recommended.

2.1.2. The MODERN Study

The MODERN study is funded by the National Health and Medical Research Council (NHMRC) and aims to develop a cost-effective regional health care program to optimise the reduction of risk factors in individuals with the MetS with/without T2DM. The main study consists of a 24-month, multi-centre, appropriately powered and pragmatic randomised controlled trial of an innovative, nurse-led intervention. Two regional clinics were established to conduct this research study, located in Shepparton and Colac, Victoria,
Australia (Figure 12). Figure 13 represents a study schema of the MODERN study indicating the available data and timepoints used for this PhD dissertation.

**Figure 12.** Two clinics established to conduct MODERN, located in Shepparton and Colac, VIC, Australia
In brief, all residents of Colac or Shepparton who presented to the clinic were screened for MetS and eligibility into the study (refer to Appendix 1 for published study protocol and detailed inclusion criteria). Following baseline assessment, eligible participants were subsequently randomised to receive either nurse-led intervention or usual care. The nurse-led intervention consisted of risk assessments and recommended behaviour changes using MI strategies, personal action plans, community program connection, goal setting, educational resources, follow-ups and health professional referrals. The
intervention was facilitated by registered nurses; as previously described, nurse-led interventions have been shown to be effective in reducing cardio-metabolic disease risk. Those randomised into usual care were provided with a report comparing their measured risk factors across time and in comparison to recommended target levels within a brief nurse education session. No additional intervention was delivered as part of the intervention although the participants usual health care providers could be utilised if needed.

Arising from this trial, this thesis will report the baseline results from all screened participants (Chapters 4 and 5) and the 12-month results of intervention and usual care groups in Chapter 6 (Figure 13).

2.1.3. Aims and Hypotheses

The primary aim of this thesis was to examine the association between behaviour-specific self-efficacy and health locus of control, health behaviours and cardio-metabolic disease risk.

Utilising the baseline data of the MODERN study cohort (Chapter 5) it was hypothesised that a higher self-efficacy for key health behaviours (physical activity, nutrition, smoking cessation and alcohol consumption) would be associated with lower cardio-metabolic disease risk, while a lower self-efficacy would be associated with a high cardio-metabolic disease risk.

It was also hypothesised that an internal health locus of control would be associated with a lower cardio-metabolic disease risk and an external (powerful others, chance) health locus of control would be associated with a higher cardio-metabolic risk status.
As shown in Figure 14, this has been submitted as the following peer-reviewed manuscript:


**Chapter 6** aimed to examine the effect of nurse-led management on behaviour-specific self-efficacy and health locus of control and cardio-metabolic risk factors after 12-months follow-up within the MODERN study. It was hypothesised that the self-efficacy for key health behaviours and health locus of control profile would be enhanced in individuals randomised to the nurse-led intervention and that there would be a concomitant reduction in cardio-metabolic risk, compared to usual care.

Figure 14 highlights that this has been submitted as the following peer-reviewed manuscript:


**Chapter 7** summarises the overall findings pertaining to this thesis work and discusses the implications of this research. General conclusions are presented and the limitations of this work are also discussed. Future directions of this work are also highlighted.

**Figure 14** represents the thesis structure and corresponding publications.
Figure 14. Thesis Outline
Chapter 3

Non-Urban Cardio-metabolic Disease Interventions
Chapter 3 summarises the findings of a systematic analysis review pertaining to non-urban primary prevention programmes aimed at reducing T2DM or CVD risk. The manuscript describes interventions conducted in rural, regional or remote settings. Specifically, data were pooled to conduct meta-analyses to identify the most effective characteristics of a successful non-urban primary prevention programme to reduce cardio-metabolic risk.

This Chapter includes the following published, peer-reviewed manuscript:

Preface

As described in Chapter 1, individuals residing in regional, rural or remote communities fare poorer in cardio-metabolic disease outcomes compared to their metropolitan counterparts. Many primary prevention programmes targeting urban populations have been effective in ameliorating chronic disease risk by promoting positive health behaviour change. Contrastingly, little is known regarding what characteristics pertain to an effective primary prevention programme in a non-urban context. Therefore, a comprehensive review of non-urban primary prevention programmes aiming to reduce T2DM and/or CVD risk was conducted. This systematic review and meta-analyses aims to highlight the effective components of a non-urban primary prevention programme aiming to reduce the risk of type 2 diabetes mellitus and/or cardiovascular disease risk.
A systematic review and meta-analysis of primary prevention programmes to improve cardio-metabolic risk in non-urban communities

Andre L. Rodrigues a,b, Jocasta Ball b, Chantal Ski b, Simon Stewart a,b, Melinda J. Carrington a,b,*

a Dept. of Epidemiology and Preventive Medicine, Monash University, Melbourne, Australia
b Mary MacKillop Institute for Health Research, Australian Catholic University, Melbourne, Australia

Abstract

Introduction. Although cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) prevention programmes have been effective in urban residents, their effectiveness in non-urban settings, where cardio-metabolic risk is typically elevated, is unknown. We systematically reviewed the effectiveness of primary prevention programmes aimed at reducing risk factors for CVD/T2DM, including blood pressure, body mass index (BMI), blood lipid and glucose, diet, lifestyle, and knowledge in adults residing in non-urban areas.

Methods. Twenty-five manuscripts, globally, from 1990 were selected for review (seven included in the meta-analyses) and classified according to: 1) study design (randomised controlled trial [RCT] or pre-/post-intervention); 2) intervention duration (short [≤12 months] or long term [≥12 months]), and; 3) programme type (community-based programmes or non-community-based programmes).

Results. Multiple strategies within interventions focusing on health behaviour change effectively reduced cardio-metabolic risk in non-urban individuals. Pre-/post-test design studies showed more favourable improvements generally, while RCTs showed greater improvements in physical activity and disease and risk knowledge. Short-term programmes were more effective than long-term programmes and in pre-/post-test designs reduced systolic blood pressure by 4.02 mm Hg (95% CI −6.25 to −1.79) versus 3.63 mm Hg (95% CI −7.34 to 0.08) in long-term programmes. Community-based programmes achieved good results for most risk factors except BMI and (glycated haemoglobin) HbA1c.

Conclusion. The setting for applying cardio-metabolic prevention programmes is important given its likelihood to influence programme efficacy. Further investigation is needed to elucidate the individual determinants of cardio-metabolic risk in non-urban populations and in contrast to urban populations.

© 2016 Elsevier Inc. All rights reserved.

Keywords: Cardiovascular disease Diabetes Prevention Rural Intervention

Contents

1. Introduction ...............................................................23
2. Methods .................................................................23
3. Inclusion and exclusion criteria .........................................23
4. Definitions ..............................................................23
4.1. Non-urban .............................................................23
4.2. Primary CVD/T2DM prevention programmes .................24
4.3. Investigated outcomes ..............................................24
4.4. Data extraction and analyses. ......................................24
5. Results .................................................................24
5.1. Description of publications .......................................30
5.2. Programme approaches and components ..................30
6. Biomedical risk factors ...............................................30
6.1. Blood pressure ....................................................30
The burden of cardiovascular disease (CVD), and its common precursor type 2 diabetes mellitus (T2DM), extends globally but differentially according to location and population profile (Mendis et al., 2011).

Globally, CVD accounts for over 30% of deaths per year (Mendis et al., 2011) and a significant proportion of the population have risk factors that contribute to the development of CVD (ABS, 2011). While CVD can be attributed to risk factors that cannot be modified, many cases are caused by risk factors that can be detected and treated; including but not limited to, elevated blood cholesterol and sugar, overweight/obesity, hypertension, smoking and physical inactivity (Mendis et al., 2011). Individuals with multiple risk factors, as in the case of metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) are at increased risk of CVD (Mendis et al., 2011). Many risk factors are shared between diabetes and CVD such that diabetes risk reduction programmes are associated with a reduced incidence of CVD- and all-cause-mortality, as well as diabetes after 23-years follow-up (Li et al., 2014). Hence, application of public health and clinical interventions to reduce the major risk factors for these diseases, while differing in their focus (e.g. dietary intervention to reduce saturated fats for CVD versus carbohydrates for diabetes), could substantially reduce the disease burden.

People living in rural, regional and remote (non-urban) locations have worse health compared to their metropolitan counterparts (Department of Health, Victoria, 2008) with mortality rates rising with greater remoteness (ABS, 2011). Non-urban residing individuals (Department of Health, Victoria, 2008), with mortality rates rising with greater remoteness (ABS, 2011) and a significant proportion of the population have risk factors that contribute to the development of CVD (ABS, 2011). While CVD can be attributed to risk factors that cannot be modified, many cases are caused by risk factors that can be detected and treated; including but not limited to, elevated blood cholesterol and sugar, overweight/obesity, hypertension, smoking and physical inactivity (Mendis et al., 2011). Individuals with multiple risk factors, as in the case of metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) are at increased risk of CVD (Mendis et al., 2011). Many risk factors are shared between diabetes and CVD such that diabetes risk reduction programmes are associated with a reduced incidence of CVD- and all-cause-mortality, as well as diabetes after 23-years follow-up (Li et al., 2014). Hence, application of public health and clinical interventions to reduce the major risk factors for these diseases, while differing in their focus (e.g. dietary intervention to reduce saturated fats for CVD versus carbohydrates for diabetes), could substantially reduce the disease burden.

People living in rural, regional and remote (non-urban) locations have worse health compared to their metropolitan counterparts (Department of Health, Victoria, 2008) with mortality rates rising with greater remoteness (ABS, 2011). Non-urban residing individuals (Department of Health, Victoria, 2008), with mortality rates rising with greater remoteness (ABS, 2011) and a significant proportion of the population have risk factors that contribute to the development of CVD (ABS, 2011). While CVD can be attributed to risk factors that cannot be modified, many cases are caused by risk factors that can be detected and treated; including but not limited to, elevated blood cholesterol and sugar, overweight/obesity, hypertension, smoking and physical inactivity (Mendis et al., 2011). Individuals with multiple risk factors, as in the case of metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) are at increased risk of CVD (Mendis et al., 2011). Many risk factors are shared between diabetes and CVD such that diabetes risk reduction programmes are associated with a reduced incidence of CVD- and all-cause-mortality, as well as diabetes after 23-years follow-up (Li et al., 2014). Hence, application of public health and clinical interventions to reduce the major risk factors for these diseases, while differing in their focus (e.g. dietary intervention to reduce saturated fats for CVD versus carbohydrates for diabetes), could substantially reduce the disease burden.

People living in rural, regional and remote (non-urban) locations have worse health compared to their metropolitan counterparts (Department of Health, Victoria, 2008) with mortality rates rising with greater remoteness (ABS, 2011). Non-urban residing individuals (Department of Health, Victoria, 2008), with mortality rates rising with greater remoteness (ABS, 2011) and a significant proportion of the population have risk factors that contribute to the development of CVD (ABS, 2011). While CVD can be attributed to risk factors that cannot be modified, many cases are caused by risk factors that can be detected and treated; including but not limited to, elevated blood cholesterol and sugar, overweight/obesity, hypertension, smoking and physical inactivity (Mendis et al., 2011). Individuals with multiple risk factors, as in the case of metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) are at increased risk of CVD (Mendis et al., 2011). Many risk factors are shared between diabetes and CVD such that diabetes risk reduction programmes are associated with a reduced incidence of CVD- and all-cause-mortality, as well as diabetes after 23-years follow-up (Li et al., 2014). Hence, application of public health and clinical interventions to reduce the major risk factors for these diseases, while differing in their focus (e.g. dietary intervention to reduce saturated fats for CVD versus carbohydrates for diabetes), could substantially reduce the disease burden.

People living in rural, regional and remote (non-urban) locations have worse health compared to their metropolitan counterparts (Department of Health, Victoria, 2008) with mortality rates rising with greater remoteness (ABS, 2011). Non-urban residing individuals (Department of Health, Victoria, 2008), with mortality rates rising with greater remoteness (ABS, 2011) and a significant proportion of the population have risk factors that contribute to the development of CVD (ABS, 2011). While CVD can be attributed to risk factors that cannot be modified, many cases are caused by risk factors that can be detected and treated; including but not limited to, elevated blood cholesterol and sugar, overweight/obesity, hypertension, smoking and physical inactivity (Mendis et al., 2011). Individuals with multiple risk factors, as in the case of metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) are at increased risk of CVD (Mendis et al., 2011). Many risk factors are shared between diabetes and CVD such that diabetes risk reduction programmes are associated with a reduced incidence of CVD- and all-cause-mortality, as well as diabetes after 23-years follow-up (Li et al., 2014). Hence, application of public health and clinical interventions to reduce the major risk factors for these diseases, while differing in their focus (e.g. dietary intervention to reduce saturated fats for CVD versus carbohydrates for diabetes), could substantially reduce the disease burden.

People living in rural, regional and remote (non-urban) locations have worse health compared to their metropolitan counterparts (Department of Health, Victoria, 2008) with mortality rates rising with greater remoteness (ABS, 2011). Non-urban residing individuals (Department of Health, Victoria, 2008), with mortality rates rising with greater remoteness (ABS, 2011) and a significant proportion of the population have risk factors that contribute to the development of CVD (ABS, 2011). While CVD can be attributed to risk factors that cannot be modified, many cases are caused by risk factors that can be detected and treated; including but not limited to, elevated blood cholesterol and sugar, overweight/obesity, hypertension, smoking and physical inactivity (Mendis et al., 2011). Individuals with multiple risk factors, as in the case of metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) are at increased risk of CVD (Mendis et al., 2011). Many risk factors are shared between diabetes and CVD such that diabetes risk reduction programmes are associated with a reduced incidence of CVD- and all-cause-mortality, as well as diabetes after 23-years follow-up (Li et al., 2014). Hence, application of public health and clinical interventions to reduce the major risk factors for these diseases, while differing in their focus (e.g. dietary intervention to reduce saturated fats for CVD versus carbohydrates for diabetes), could substantially reduce the disease burden.
Definitions of these terms vary depending on purpose or policy; often in health research, the term rural represents small towns, isolation or low population density (compared to those residing in more densely populated areas) (Hart et al., 2005). Studies were included if literature searches identified ‘rural’, ‘regional’ or ‘remote’ as a respective keyword or if sample population characteristics were concordant with respective national definitions concerning rurality and remoteness set by national statistical offices; including distance from urban health centres, population size and density, geographical size and location.

4.2. Primary CVD/T2DM prevention programmes

Primary prevention pertains to preventing the onset of a disease or condition (in contrast to secondary prevention that aims to reduce the impact of a disease or injury that has already occurred). A primary prevention programme was defined as an intervention targeting dietary, lifestyle and behavioural modifications to reduce the likelihood of developing T2DM and/or CVD. Lifestyle modification for physical activity (PA), diet, smoking and alcohol consumption could be achieved via support that includes goal-setting (Artinina et al., 2010), education (Staten et al., 2004), regular follow-up of participants (Green et al., 2002), incentives (Jeffery et al., 1998), print- or media-delivery strategies (Marcus et al., n.d.), or PA and diet demonstrations (Artinina et al., 2010). Prevention programmes can be administered in either group or one-on-one sessions by trained research personnel, health professionals, nurses, diabetes educators and/or exercise physiologists. Counselling methods such as cognitive behavioural therapy and motivational interviewing can assist participants in overcoming barriers to positive health behaviour changes (Gulliksson et al., 2011; Rollnick and Miller, 1995). Pharmacological treatments upon referral may also be a feature of some prevention programmes.

4.3. Investigated outcomes

Widely recognised biomedical risk factors for CVD and T2DM reported in this review were all objectively measured (i.e. not self-reported). They included: systolic and diastolic blood pressure (SBP and DBP), body mass index (BMI), body weight, lipids including triglycerides, total-, high-density lipoprotein-(HDL), and low-density lipoprotein (LDL) cholesterol, fasting blood glucose (FBG) and glycated haemoglobin (HbA1c). Self-reported knowledge and behaviours: knowledge about CVD or diabetes, PA, diet, smoking and alcohol consumption.

4.4. Data extraction and analyses

Accepted publications were classified according to 3 criteria: 1) study design (randomised controlled trial [RCT] or pre-/post-intervention studies); 2) intervention duration (short [<12 months] or long...
<table>
<thead>
<tr>
<th>Study</th>
<th>Design/duration</th>
<th>Participants</th>
<th>Control groups (C)/intervention (I)</th>
<th>Between group biomedical outcomes</th>
<th>Between group behavioural outcomes</th>
</tr>
</thead>
</table>
| **Davis et al., (2010)** | Community based RCT/12-months | N = 165; adult with HbA1c over 7%; BMI over 25; aged over 35 years | C: One 20 min diabetes education session conducted individually  
I: Delivered by health professionals to groups: education classes, education material, face-to-face counselling, dietary advice, video-conferencing component. Follow-up ≥ once per month. | Systolic: I: −3.2 mm Hg v C: −3.7 mm Hg;  
Diastolic: I: −2.5 mm Hg v C: −1.6 mm Hg;  
BMI: I: −0.2 v C: −0.2;  
LDL: I: −13.3 mg/dl v C: +0.6 mg/dl;  
HbA1c: I: −1.2 v C: −0.26 | ... |
| **Ko et al., (2004)**  | RCT/12-months    | N = 180; Chinese diabetic adults; aged 25–70 years | C: Received a doctor consultation every 3 months, usual care  
I: Delivered by health professionals to individuals: education classes, face-to-face counselling, dietary and physical activity advice. Follow-up ≤ once per month. | % Change  
Systolic: I: 0.7 v C: 1.0;  
Diastolic: I: −3.2 v C: −0.4  
BMI: I: −0.2 v C: 0.3;  
Triglycerides: I: −6.8 v C: −11.9;  
Total cholesterol: I: −0.3 v C: −10.0;  
LDL: I: −12.2 v C: −15.4;  
HDL: I: 1.1 v C: 0.2;  
Glucose: I: −0.9 v C: −1.4;  
HbA1c: I: 6 v C: −2.1;  
Smoking (quit or reduced): I: −16.7% v C: −5.7% | Systolic: I: 0.7 v C: 1.0;  
Diastolic: I: −3.2 v C: −0.4  
BMI: I: −0.2 v C: 0.3;  
Triglycerides: I: −6.8 v C: −11.9;  
Total cholesterol: I: −0.3 v C: −10.0;  
LDL: I: −12.2 v C: −15.4;  
HDL: I: 1.1 v C: 0.2;  
Glucose: I: −0.9 v C: −1.4;  
HbA1c: I: 6 v C: −2.1;  
Smoking (quit or reduced): I: −16.7% v C: −5.7% |
| **Lupton et al., (2003)** | Community based RCT/6-years | N = 1324; Norwegian adults; 50.4% male | C: Reference community/usual care  
I: Delivered by researchers to groups: education classes, physical activity demonstrations, educational material. Follow-up ≤ once per month. | Men  
Systolic: I: −0.1 mm Hg v C: +2.2 mm Hg;  
Diastolic: I: −2.1 mm Hg v C: +0.8 mm Hg;  
BMI: I: +1.5 kg/m² v C: +1.1 kg/m²;  
Total cholesterol | Men  
Systolic: I: −0.1 mm Hg v C: +2.2 mm Hg;  
Diastolic: I: −2.1 mm Hg v C: +0.8 mm Hg;  
BMI: I: +1.5 kg/m² v C: +1.1 kg/m²;  
Total cholesterol |
| **Majumdar et al., (2003)** | RCT/6-months | N = 379; type 2 diabetic adults from North Alberta; 58.4% male | C: Three bimonthly visits from the Canadian Diabetes Association (CDA) Resource Programme  
I: Delivered by health professionals to groups and individuals: education classes, face-to-face counselling. Follow-up ≤ once per month. | Blood pressure: I: 42% saw 10% decrease or more;  
C: 25% saw 10% decrease or more;  
HbA1c | Blood pressure: I: 42% saw 10% decrease or more;  
C: 25% saw 10% decrease or more;  
HbA1c |
| **Mayer-Davis et al., (2004)** | RCT/12-months | N = 105; diabetic adults; aged over 45 years; BMI over 25 kg/m² | C: Usual care  
I: Delivered by health professionals, educators and researchers to individuals: education classes, educational material, face-to-face counselling, dietary and physical activity advice, goal setting. Follow-up ≤ once per month. | Systolic: I: −3.31 mm Hg v C: −9.52 mm Hg;  
Diastolic: I: −0.49 mm Hg v C: −2.65 mm Hg;  
BMI: I: −0.97 kg/m² v C: 0.16 kg/m²;  
Triglycerides: I: 0.87 mg/dl v C: 0.91 mg/dl;  
Total cholesterol | Physical activity;  
Diet;  
Smoking;  
Alcohol |
<table>
<thead>
<tr>
<th>Study</th>
<th>Design/duration</th>
<th>Participants</th>
<th>Control groups (C)/intervention (I)</th>
<th>Between group biomedical outcomes</th>
<th>Between group behavioural outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miller et al., (2001)</td>
<td>RCT/24-months</td>
<td>N = 277; Premenopausal Women; aged over 18 years</td>
<td>C: Usual care &lt;br&gt; I: Intervention delivered by health professionals to groups: face-to-face counselling, dietary and physical activity advice. Follow-up 2x once per month</td>
<td>HbA1c: I: $-1.56%$ v C: $-1.12%$&lt;br&gt; BMI: I: $-0.1$ v C: $0.0$</td>
<td>Diet: % of calories from fat: I: $-2.8%$ v C: $-1.3%$&lt;br&gt; Knowledge: I: $&gt; C$</td>
</tr>
<tr>
<td>Nguyen et al., (2012)</td>
<td>Community based RCT/36-months</td>
<td>N = 4650; Vietnamese adults; aged over 25 years</td>
<td>C: Routine Conventional Primary Healthcare &lt;br&gt; I: Delivered by disease educators to groups and individuals: education classes, physical activity demonstrations, educational material, and dietary advice. Follow-up ≥ once per month.</td>
<td>BMI: I: $-0.1$ v C: $0.0$&lt;br&gt; HbA1c: I: $-1.56%$ v C: $-1.12%$&lt;br&gt; Diet: % of calories from fat: I: $-2.8%$ v C: $-1.3%$&lt;br&gt; Knowledge: I: $&gt; C$</td>
<td>Women Physical activity: I: $+6%$ v C: $-0.4%$&lt;br&gt; Diet: Salt intake: I: $-7.4%$ v C: $-2.4%$&lt;br&gt; Smoking: I: $-0.6%$ v C: $-0.0%$&lt;br&gt; Alcohol: I: $-0.2%$ v C: $-0.2%$&lt;br&gt; Men Physical activity: I: $+7.4%$ v C: $+2.6%$&lt;br&gt; Diet: Salt intake: I: $-8.2%$ v C: $-1.2%$&lt;br&gt; Smoking: I: $-2.3%$ v C: $-2.1%$&lt;br&gt; Alcohol: I: $-10.4%$ v C: $-10.6%$&lt;br&gt; Knowledge: I: $+25%$ v C: $+1%$</td>
</tr>
<tr>
<td>Pieber et al., (1995)</td>
<td>RCT/6-months</td>
<td>N = 108; diabetic adults; mean age 63.9 years</td>
<td>C: Usual care &lt;br&gt; I: Delivered by investigators to groups: education classes, physical activity advice. Follow-up 2x once per month.</td>
<td>Systolic: I: $-16.6$ mm Hg v C: $-7.2$ mm Hg&lt;br&gt; Diastolic: I: $-11.1$ mm Hg v C: $-5.4$ mm Hg&lt;br&gt; BMI: I: $-0.99$ kg/m$^2$ v C: $+0.11$ kg/m$^2$&lt;br&gt; Triglycerides: I: $-0.63$ mmol/l v C: $+0.17$ mmol/l&lt;br&gt; Total cholesterol: I: $1.04$ mmol/l v C: $-0.45$ mmol/l&lt;br&gt; HbA1c: I: $-0.46%$ v C: $+0.26%$&lt;br&gt; BMI: I: $0.37$ kg/m$^2$ v C: $0.71$ kg/m$^2$ (men)&lt;br&gt; I: $0.38$ kg/m$^2$ v C: $0.63$ kg/m$^2$ (women)&lt;br&gt; Total cholesterol: I: $0.04$ mmol/l v C: $-0.08$ mmol/l (women)&lt;br&gt; HDL: I: $-0.09$ mmol/l v C: $0.17$ mmol/l (women)&lt;br&gt; Men&lt;br&gt; HDL: I: $-0.09$ mmol/l v C: $0.17$ mmol/l (women)&lt;br&gt; Men&lt;br&gt; Glucose: I: $-0.09$ mmol/l v C: $0.17$ mmol/l (women)&lt;br&gt; Men&lt;br&gt;</td>
<td>Women Systolic: I: $-10.3$ mm Hg v C: $-8.6$ mm Hg&lt;br&gt; Diastolic: I: $-5.0$ mm Hg v C: $-1.0$ mm Hg&lt;br&gt; Weight: I: $+0.6$ kg v C: $-1.0$ kg&lt;br&gt; BMI: I: $0.0$ kg/m$^2$ v C: $-0.6$ kg/m$^2$&lt;br&gt; Physical activity: I: $+6%$ v C: $-0.4%$&lt;br&gt; Diet: Salt intake: I: $-7.4%$ v C: $-2.4%$&lt;br&gt; Smoking: I: $-0.6%$ v C: $-0.0%$&lt;br&gt; Alcohol: I: $-0.2%$ v C: $-0.2%$&lt;br&gt; Men Physical activity: I: $+7.4%$ v C: $+2.6%$&lt;br&gt; Diet: Salt intake: I: $-8.2%$ v C: $-1.2%$&lt;br&gt; Smoking: I: $-2.3%$ v C: $-2.1%$&lt;br&gt; Alcohol: I: $-10.4%$ v C: $-10.6%$&lt;br&gt; Knowledge: I: $+25%$ v C: $+1%$</td>
</tr>
<tr>
<td>Schuit et al., (2006)</td>
<td>Community based RCT/5-years</td>
<td>N = 2414; adults from Maastricht Holland; aged 30–70 years</td>
<td>C: Usual care &lt;br&gt; I: Delivered by investigators to groups: education classes, cooking demonstrations, physical activity groups, educational material, dietary advice. Follow up ≥ once per month.</td>
<td>BMI: I: $0.37$ kg/m$^2$ v C: $0.63$ kg/m$^2$ (men)&lt;br&gt; I: $0.38$ kg/m$^2$ v C: $0.63$ kg/m$^2$ (women)&lt;br&gt; Total cholesterol: I: $-0.08$ mmol/l v C: $-0.08$ mmol/l (women)&lt;br&gt; HDL: I: $-0.09$ mmol/l v C: $0.17$ mmol/l (women)&lt;br&gt; Men&lt;br&gt; Glucose: I: $-0.09$ mmol/l v C: $0.17$ mmol/l (women)&lt;br&gt; Men&lt;br&gt;</td>
<td>BMI: I: $0.37$ kg/m$^2$ v C: $0.71$ kg/m$^2$ (men)&lt;br&gt; I: $0.38$ kg/m$^2$ v C: $0.63$ kg/m$^2$ (women)&lt;br&gt; Total cholesterol: I: $-0.08$ mmol/l v C: $-0.08$ mmol/l (women)&lt;br&gt; HDL: I: $-0.09$ mmol/l v C: $0.17$ mmol/l (women)&lt;br&gt; Men&lt;br&gt; Glucose: I: $-0.09$ mmol/l v C: $0.17$ mmol/l (women)&lt;br&gt; Men&lt;br&gt;</td>
</tr>
<tr>
<td>Shahar et al., 2012</td>
<td>RCT/6-months</td>
<td>N = 42; Malaysian adults with metabolic syndrome; mean age 66.3 years, 28.6% diabetic; 73.8% hypertensive</td>
<td>C: General Health Education Package &lt;br&gt; I: Delivered by investigators to groups: education classes, cooking demonstrations, physical activity groups, educational material. Follow-up 2x once per month.</td>
<td>Systolic: I: $-15.8$ mm Hg v C: $-5.0$ mm Hg&lt;br&gt; Diastolic: I: $-4.1$ mm Hg v C: $-4.7$ mm Hg&lt;br&gt; Weight: I: $-0.8$ kg v C: $+0.7$ kg&lt;br&gt; BMI: I: $3.1$ kg/m$^2$ v C: $0.3$ kg/m$^2$&lt;br&gt; Triglycerides: I: $-0.1$ mmol/l v C: $-0.0$ mmol/l&lt;br&gt; Total cholesterol: I: $-0.0$ mmol/l v C: $+0.9$ mmol/l&lt;br&gt; LDL: I: $-0.1$ mmol/l v C: $+0.8$ mmol/l&lt;br&gt; HDL: I: $-0.1$ mmol/l v C: $-0.0$ mmol/l</td>
<td>Triglycerides: I: $-0.1$ mmol/l v C: $-0.0$ mmol/l&lt;br&gt; Total cholesterol: I: $-0.0$ mmol/l v C: $+0.9$ mmol/l&lt;br&gt; LDL: I: $-0.1$ mmol/l v C: $+0.8$ mmol/l&lt;br&gt; HDL: I: $-0.1$ mmol/l v C: $-0.0$ mmol/l</td>
</tr>
</tbody>
</table>

* Not significant.  
** p < 0.001.
Table 2
Findings of pre/post studies to improve cardio-metabolic risk factors in rural adults.

<table>
<thead>
<tr>
<th>Study</th>
<th>Design/duration</th>
<th>Participants</th>
<th>Control groups (C)/intervention (I)</th>
<th>Within group biomedical outcomes</th>
<th>Within group behavioural outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balagopal et al., (2008)</td>
<td>Community based/7-months</td>
<td>N = 585; adults from Almarathupatti, India; mean age 40.3 years; 41% male; 5.1% diabetic</td>
<td>I: Delivered by health professionals, disease educators to individuals: education classes, cooking demonstrations, physical activity groups, face-to-face counselling, and dietary and physical activity advice. Unspecified follow up intervals</td>
<td>Systolic: −1.07 mm Hg⁎; Diastolic: −5.9 mm Hg⁎⁎⁎; BMI: +0.2 kg/m²; Glucose: −3.2 mg/dl</td>
<td>Physical activity⁎; Diet: Protein intake − 0.7 g/day⁎⁎; Knowledge: T2DM knowledge improved⁎⁎⁎</td>
</tr>
<tr>
<td>Balagopal et al., (2012)</td>
<td>Community based/6-months</td>
<td>N = 1638; adults from Gujarat, India; mean age 41.9 years; 44.2% smokers; 7.1% diabetic</td>
<td>I: Delivered by health professionals to groups and individuals: education classes, cooking demonstrations, physical activity demonstrations, face-to-face counselling, and dietary advice. Follow-up 2 once per month</td>
<td>Systolic: −7.37 mm Hg⁎⁎⁎; Diastolic: −3.24 mm Hg⁎⁎⁎; BMI: −0.46 kg/m²; Glucose: −1.28 mg/dl</td>
<td>Physical activity: 11.6% improved; self-reported⁎⁎⁎; Diet: Fruit intake + 0.4 servings/day; vegetables intake + 0.19 servings/day⁎⁎⁎; Knowledge: T2DM and CVD improved⁎⁎⁎</td>
</tr>
<tr>
<td>Carrington and Stewart, (2014)</td>
<td>Community based/6-months</td>
<td>N = 530; Australia adults; mean age 54 years; 62% female; high CVD risk status</td>
<td>I: Delivered by health professionals and researchers to individuals: education classes, physical activity groups, educational, material, face-to-face counselling, dietary and physical activity advice, goal setting. Follow-up 2 once per month</td>
<td>Systolic: −4.0 mm Hg⁎⁎⁎; Diastolic: −1.3 mm Hg⁎⁎⁎; Weight: −0.8 kg⁎⁎⁎; Triglycerides: −0.4 mmol/l⁎⁎⁎; Total cholesterol: −0.6 mmol/l⁎⁎⁎; LDL: −0.3 mmol/l⁎⁎⁎; HDL: −0.1 mmol/l⁎⁎⁎; Glucose: −0.6 mmol⁎⁎</td>
<td>Physical activity: − 36 MET⁎⁎⁎; Diet: Saturated Fat intake decreases⁎⁎⁎</td>
</tr>
<tr>
<td>Chockalingam and Fodor, (1990)</td>
<td>Pilot study/6-month intervention/12-month follow-up</td>
<td>N = 41; adults from fishing communities in Newfoundland, Canada; 30% smokers; 54% overweight</td>
<td>I: Delivered by health professionals to groups: educational material, face-to-face counselling, dietary advice, goal setting. Follow-up intervals not specified</td>
<td>Systolic: −4.38 mm Hg⁎; Diastolic: −2.56 mm Hg⁎; BMI: −0.34 kg/m²⁎</td>
<td>...</td>
</tr>
<tr>
<td>Daniel et al., (1999)</td>
<td>Community based/24-months</td>
<td>N = 295; Indian adults from Okanagan region; aged over 35 years;</td>
<td>I: Delivered by disease educators and researchers to groups and individuals: education classes, cooking demonstrations, physical activity demonstrations, and physical activity groups. Follow-up − once per month</td>
<td>...</td>
<td>Diet⁎; Smoking⁎; Knowledge⁎</td>
</tr>
<tr>
<td>Farag et al., (2010)</td>
<td>Community based/6-months</td>
<td>N = 187; employees from public school system in Oklahoma; mean age 45 years</td>
<td>I: Delivered by educators to groups: Advice on physical activity. Follow-up – once per month</td>
<td>Systolic: −3 mm Hg⁎⁎⁎; Diastolic: −0.0 mm Hg⁎⁎⁎; LDL: −0.0 kg/m²⁎⁎⁎; Triglycerides: −2.0 mg/dl⁎⁎⁎; Total cholesterol: −12 mmol/l⁎⁎⁎; HDL: −0.0 mmol/l⁎⁎⁎; Glucose: −0.0 mmol/l⁎⁎⁎; HbA1c: −0.5%⁎⁎⁎</td>
<td>Physical activity⁎⁎; Smoking⁎⁎; Alcohol⁎⁎</td>
</tr>
<tr>
<td>Franks and Gold, (1990)</td>
<td>Community based/24-months</td>
<td>N = 625; adults from Meredith and Kortright communities; mean age 47.6 years</td>
<td>I: Delivered by health professionals to individuals: education classes, face-to-face counselling, and dietary advice. Follow-up intervals not specified</td>
<td>Systolic: I: −3.1 mm Hg⁎</td>
<td>...</td>
</tr>
<tr>
<td>Giampaoli et al., (1991)</td>
<td>Community based/36-months</td>
<td>N = 3585; adults aged 20–69 years; 46.9% male</td>
<td>I: Delivered by educators to groups: Dietary and physical activity advice. Follow-up – once per month</td>
<td>I: Men Systolic: +0.9 mm Hg⁎; Diastolic: +0.5 mm Hg⁎; BMI: +0.31 kg/m²⁎; Cholesterol: +0.6 mg/dl⁎</td>
<td>I: Men Smoking: # of cigarettes − 22.6% (male)⁎⁎⁎; I: Women Smoking: # of cigarettes − 33.3% (female)⁎⁎⁎</td>
</tr>
</tbody>
</table>

(continued on next page)
### Table 2 (continued)

<table>
<thead>
<tr>
<th>Study</th>
<th>Design/duration</th>
<th>Participants</th>
<th>Control groups (C)/intervention (I)</th>
<th>Within group biomedical outcomes</th>
<th>Within group behavioural outcomes</th>
</tr>
</thead>
</table>
| Huang et al., (2011)         | Community based/36-months | N = 1636; Chinese adults; aged over 35 years | I: Delivered by investigators to groups: education, educational material, and advice on physical activity. Follow-up once per month | I: Women  
Systolic: $-3.2$ mm Hg<sup>**</sup>;  
Diastolic: $+2.6$ mm Hg<sup>***</sup>;  
BMI: $+0.4$ kg/m<sup>2</sup>;  
Cholesterol: $-3.5$ mg/dl<sup>a</sup>  
I: Systolic<sup>c</sup>;  
Diastolic<sup>c</sup>;  
Weight<sup>c</sup>;  
BMI<sup>c</sup>  
I: Percent with salty diet $-30.0%$<sup>a</sup>;  
Fat intake reduced in $-9.1%$;  
Percent smoking $-3.5%$;  
Percent consuming alcohol $-4.2%$;  
Percent exercising $+16.4%$;  
Knowledge increased by $28.5%$  
C: Percent with salty diet $-5.5%$;  
Fat intake reduced in $-3.1%$;  
Percent smoking $16.6%$;  
Percent consuming alcohol $4.2%$;  
Percent exercising $+16.4%$;  
Knowledge increased by $15.5%$ | I: Physical activity: $+11.6%$<sup>a</sup>;  
Physical activity: I: Mean pedometer count +2573<sup>***</sup>  
...                                                                 |
| Laatikainen et al., (2007)   | Longitudinal/12-months | N = 237; non-diabetic adults at high risk of diabetes; 27.4% male | I: Delivered by health professionals to groups: education classes, face-to-face counseling, dietary advice, goal setting. Follow-up intervals not specified | Systolic: $-1.01$<sup>c</sup>;  
Diastolic: $-2.6$<sup>c</sup>;  
Weight: $-2.7%$;  
BMI: $-2.8%$;  
Triglycerides: $-7.6%$;  
Total cholesterol: $-5.1%$<sup>c</sup>;  
LDL: $-7.3%$;  
HDL: $-4.8%$<sup>c</sup>  
I: Systolic<sup>c</sup>;  
Diastolic<sup>c</sup>;  
Weight<sup>c</sup>;  
BMI<sup>c</sup>;  
Triglycerides<sup>c</sup>;  
Total cholesterol<sup>c</sup>;  
LDL<sup>c</sup>;  
HDL<sup>c</sup>  
I: Physical activity: fruit and vegetable intake increased $+26.5%$;  
Knowledge: Increased by $15.5%$  
...                                                                 |
| Nafriger et al., (2001)      | Hospital-based/5-years | N = 548; adults from Otsego and Schoharie Counties, USA; aged 20–79 years | I: Delivered by educators to groups: education classes, cooking demonstrations, educational material, dietary advice. Follow-up once per month | Systolic: $-0.5$ mg/dl<sup>a</sup>;  
Diastolic: $-1.13$ mg/dl<sup>a</sup>;  
Weight: $-0.05$ kg<sup>a</sup>;  
BMI: $-0.25$ kg/m<sup>2</sup>;  
Total cholesterol: $-0.158$ mg/dl<sup>a</sup>;  
LDL: $-0.2$ mg/dl<sup>a</sup>;  
HDL: $-0.101$ mg/dl<sup>a</sup>  
I: Systolic<sup>c</sup>;  
Diastolic<sup>c</sup>;  
Weight<sup>c</sup>;  
BMI<sup>c</sup>;  
Total cholesterol<sup>c</sup>;  
LDL<sup>c</sup>;  
HDL<sup>c</sup>  
I: Physical activity: $+11.6%$<sup>a</sup>;  
Smoking: $-8.9%$<sup>a</sup>  
...                                                                 |
| Richardson et al., (2006)    | Community based/10-years | N = 313; overweight adults; aged 45–64 years | I: Delivered by educators to individuals: educational material with referrals. Follow-up once per month | Systolic: $-2.88$ mm Hg<sup>***</sup>;  
Diastolic: $-0.158$ mm Hg<sup>c</sup>;  
Weight: $-0.184$ kg<sup>c</sup>;  
BMI: $-0.101$ mmol/l<sup>c</sup>;  
Total cholesterol: $-0.158$ mmol/l<sup>c</sup>;  
LDL: $-0.2$ mmol/l<sup>c</sup>;  
HDL: $-0.101$ mmol/l<sup>c</sup>  
I: Systolic<sup>c</sup>;  
Diastolic<sup>c</sup>;  
Weight<sup>c</sup>;  
BMI<sup>c</sup>;  
Total cholesterol<sup>c</sup>;  
LDL<sup>c</sup>;  
HDL<sup>c</sup>  
I: Physical activity: $+26.5%$;  
Diet: fruit and vegetable intake increased $+26.5%$;  
Smoking: smoking decreased;  
Alcohol: consumption decreased | Physical activity<sup>c</sup>;  
Diet: fruit and vegetable intake increased $+26.5%$;  
Smoking: smoking decreased;  
Alcohol: consumption decreased |...                                                                 |
| Sherman et al., (2007)       | Female study/6-months | N = 61; Women; mean age 42.5 years; at least one risk factor for CVD; 78% overweight/obese | I: Delivered by researchers to individuals: physical activity demonstrations, physical activity groups, educational material, physical activity advice. Follow-up once per month | Systolic: $-0.12$ mm Hg<sup>c</sup>;  
Diastolic: $-0.03$ mm Hg<sup>c</sup>;  
Weight: $-0.184$ kg<sup>c</sup>;  
BMI: $-0.101$ mmol/l<sup>c</sup>;  
Total cholesterol: $-0.158$ mmol/l<sup>c</sup>;  
LDL: $-0.2$ mmol/l<sup>c</sup>;  
HDL: $-0.101$ mmol/l<sup>c</sup>  
I: Systolic<sup>c</sup>;  
Diastolic<sup>c</sup>;  
Weight<sup>c</sup>;  
BMI<sup>c</sup>;  
Total cholesterol<sup>c</sup>;  
LDL<sup>c</sup>;  
HDL<sup>c</sup>  
I: Physical activity: $+2573$<sup>***</sup>  
...                                                                 |
| Vadheim et al., (2010)       | Community based/6-months | N = 65; Overweight Adults; mean age 50.5 years; 88% female; 66% dyslipidemia — paid to participate in intervention | I: Delivered by health professionals to groups: cooking demonstrations, physical activity demonstrations, face-to-face counseling, and dietary and physical activity advice. Follow-up once per month | Systolic: $-0.12$ mm Hg<sup>c</sup>;  
Diastolic: $-0.03$ mm Hg<sup>c</sup>;  
Weight: $-0.184$ kg<sup>c</sup>;  
BMI: $-0.101$ mmol/l<sup>c</sup>;  
Total cholesterol: $-0.158$ mmol/l<sup>c</sup>;  
LDL: $-0.2$ mmol/l<sup>c</sup>;  
HDL: $-0.101$ mmol/l<sup>c</sup>  
I: Systolic<sup>c</sup>;  
Diastolic<sup>c</sup>;  
Weight<sup>c</sup>;  
BMI<sup>c</sup>;  
Total cholesterol<sup>c</sup>;  
LDL<sup>c</sup>;  
HDL<sup>c</sup>  
I: Physical activity: $+2573$<sup>***</sup>  
...                                                                 |
| Weinheall et al., (2001)     | Community based/10-years | I: Delivered by health professionals to groups and individuals: face-to-face counselling, and dietary and physical activity advice. Follow-up once per month | Men  
Systolic: $-6.7$ mm Hg<sup>***</sup>;  
Diastolic: $-2.9$ mm Hg<sup>***</sup>;  
Total cholesterol: $-0.83$ mmol/l<sup>***</sup>  
I: Systolic<sup>c</sup>;  
Diastolic<sup>c</sup>;  
Total cholesterol<sup>c</sup>  
I: Percent with salty diet $-30.0%$;  
Fat intake reduced in $-9.1%$;  
Percent smoking $-3.5%$;  
Percent consuming alcohol $-4.2%$;  
Percent exercising $+16.4%$;  
Knowledge increased by $28.5%$  
C: Percent with salty diet $-5.5%$;  
Fat intake reduced in $-3.1%$;  
Percent smoking $16.6%$;  
Percent consuming alcohol $4.2%$;  
Percent exercising $+16.4%$;  
Knowledge increased by $15.5%$ | I: Smoking<sup>c</sup>  
...                                                                 |

<sup>a</sup> Not significant  
<sup>⁎</sup> p < 0.05  
<sup>⁎⁎</sup> p < 0.01  
<sup>⁎⁎⁎</sup> p < 0.001
term [≥12 months]), and; 3) programme type (community-based programmes which utilised existing community resources (McLeroy et al., 2003) [e.g. local fitness, health groups] as part of their intervention or non-community-based programmes [interventions constructed and applied internally within studies]). Results were summarised as between-group differences (for RCTs) and within-individual changes (for pre-/post-test study designs).

Mean differences and 95% confidence intervals (CIs) were calculated where there were a significant number of publications for meta-analyses. This applied to SBP, DBP, BMI, LDL cholesterol and HbA1c for

Fig. 2. Overall effect size (represented as a black diamond) for mean differences in SBP and DBP for short and long term programmes.
selected programmes via direct comparison meta-analysis. Pre- and post-test results for each respective outcome and standard errors calculated for each resulting mean difference were entered into Review Manager (RevMan) 5.1.7. A random effects model was used due to heterogeneity between the included studies. Effect sizes of the change in risk factors as a result of the programme were calculated using the generic inverse variance method and the I² statistic was utilised to estimate the proportion of variance as a result of heterogeneity.

5. Results

5.1. Description of publications

Ten of the manuscripts included RCTs (Table 1) and fifteen were pre-/post-test designs (Table 2). Publications were included from Asia (Ko et al., 2004; Nguyen et al., 2012; Shahar et al., 2012; Balagopal et al., 2008; Balagopal et al., 2012; Huang et al., 2011), North America (Davis et al., 2010; Majumdar et al., 2003; Mayer-Davis et al., 2004; Miller et al., 2001; Chockalingam and Fodor, 1990; Daniel et al., 1999; Farag et al., 2010; Franks and Gold, 1990; Nafziger et al., 2001; Sherman et al., 2007; Vadheim et al., 2010), Europe (Lupton et al., 2003; Pieber et al., 1995; Giampaoli et al., 1991; Richardson et al., 2008; Weinhehall et al., 2001) and Australia (Carrington and Stewart, 2014; Laatikainen et al., 2007), to enable a broad inclusion of studies. The length of intervention included 10 short (<12 months) (Majumdar et al., 2003; Pieber et al., 1995; Shahar et al., 2012; Balagopal et al., 2008; Balagopal et al., 2012; Carrington and Stewart, 2014; Chockalingam and Fodor, 1990; Farag et al., 2010; Sherman et al., 2007; Vadheim et al., 2010) and 15 long-term (≥12 months) (Davis et al., 2010; Ko et al., 2004; Lupton et al., 2003; Mayer-Davis et al., 2004; Miller et al., 2001; Nguyen et al., 2012; Schuit et al., 2006; Daniel et al., 1999; Franks and Gold, 1990; Giampaoli et al., 1991; Huang et al., 2011; Laatikainen et al., 2007; Nafziger et al., 2001; Richardson et al., 2008; Weinhehall et al., 2001) prevention programmes. A considerable number (64%) were community-based programmes (Davis et al., 2010; Lupton et al., 2003; Nguyen et al., 2012; Schuit et al., 2006; Balagopal et al., 2008; Balagopal et al., 2012; Carrington and Stewart, 2014; Daniel et al., 1999; Farag et al., 2010; Franks and Gold, 1990; Giampaoli et al., 1991; Huang et al., 2011; Nafziger et al., 2001; Richardson et al., 2008; Vadheim et al., 2010; Weinhehall et al., 2001).

5.2. Programme approaches and components

The majority of studies included in this systematic review delivered their programme to groups of individuals (Lupton et al., 2003; Miller et al., 2001; Pieber et al., 1995; Schuit et al., 2006; Shahar et al., 2012; Balagopal et al., 2008; Daniel et al., 1999; Farag et al., 2010; Giampaoli et al., 1991; Huang et al., 2011; Laatikainen et al., 2007; Nafziger et al., 2001) while a lesser number administered it individually (Ko et al., 2004; Mayer-Davis et al., 2004; Franks and Gold, 1990; Richardson et al., 2008; Sherman et al., 2007), some studies combined both group and individual delivery (Davis et al., 2010; Majumdar et al., 2003; Balagopal et al., 2012; Daniel et al., 1999; Vadheim et al., 2010; Weinhehall et al., 2001). Eleven studies had infrequent (<1 per month) follow-up contact either by telephone or face-to-face while nine studies contacted participants regularly (Davis et al., 2010; Majumdar et al., 2003; Mayer-Davis et al., 2004; Miller et al., 2001; Nguyen et al., 2012; Pieber et al., 1995; Balagopal et al., 2012; Sherman et al., 2007; Vadheim et al., 2010) (≥1 per month). Studies that had infrequent contact between investigators and participants (Ko et al., 2004; Lupton et al., 2003; Schuit et al., 2006; Daniel et al., 1999; Farag et al., 2010; Giampaoli et al., 1991; Nafziger et al., 2001; Richardson et al., 2008; Weinhehall et al., 2001) were not as effective in lowering CVD or T2DM risk compared to programmes where participants were regularly contacted (≥1 per month). Programmes were conducted by health professionals, disease educators and researchers. The programmes reported consisted of disease and risk education classes, cooking- and PA-demonstrations, PA groups, distribution of educational material, and face-to-face counselling. Advice on diet modification and exercise benefits were also included while one study utilised a videoconferencing component to deliver part of the programme (Balagopal et al., 2008). Individual goal setting was also implemented as a motivational tool in some interventions and while it was not widely utilised, the few studies that incorporated goal setting were effective, all exhibiting statistically significant improvements across all respective outcomes targeted (Carrington and Stewart, 2014; Daniel et al., 1999; Laatikainen et al., 2007; Vadheim et al., 2010).

6. Biomedical risk factors

6.1. Blood pressure

Of the 25 publications selected, 20 reported SBP changes comprising 8 RCTs and 12 pre-/post-test studies. Overall, 3 RCTs reported a decrease in SBP in favour of the intervention group and 9 pre-/post-test studies reported a decrease within individuals. The remaining 8 papers reported no statistically significant changes in SBP. Six out of 8 short term and 6 of 12 long term studies reported decreases in SBP. Thirteen community-based studies reported on SBP; 11 found a decrease in SBP and 2 found no statistically significant changes. Of the 7 non-community-based programmes, 2 found a decrease in SBP and 5 found no change.

Nineteen publications reported changes in DBP including 8 RCTs and 11 pre-/post-test studies. Four RCTs and 8 pre-/post-test studies reported a decrease in DBP with the remaining 7 papers reporting no statistically significant changes. Six of 8 short term studies and 6 of 11 long term studies reported a decrease in DBP. Nine out of twelve community-based programme studies found a decrease in DBP and 3 showed no difference. Seven non-community-based programmes reported on DBP; four found a decrease and 3 found no change. Neither SBP nor DBP increased in any of the included studies.

The mean difference in BP in short-term, pre-/post-test programmes (Fig. 2A and D) was −4.0 mm Hg (95% CI −6.3 to −1.8) and −2.6 mm Hg (95% CI −5.1 to −0.0) for SBP and DBP respectively. Effect sizes were significant for SBP (p = 0.0004), although most variation was due to heterogeneity (I² = 91%). The mean difference in BP in long-term, pre-/post-test programmes (Fig. 2B and E) was −3.6 mm Hg (95% CI −7.34 to 0.08) and −1.0 mm Hg (95% CI −2.12 to 0.10) for SBP and DBP respectively. Effect sizes were not significant (p = 0.05 and 0.07 respectively) and heterogeneity was high for SBP (I² = 87%) but lower for DBP (I² = 42%). The mean difference in SBP and DBP in long-term RCT programmes (Fig. 2C and F) was −3.1 mm Hg (95% CI −4.2 to −2.0) and −2.5 mm Hg (95% CI −3.1 to −1.8) respectively. Effect sizes were significant (p < 0.001) and heterogeneity was low for SBP and DBP (I² = 0% for both).

6.2. Body mass index

Eighteen studies reported BMI changes; 9 RCTs and 9 pre-/post-test design. A total of 3 RCTs found a reduction in BMI in favour of the intervention group and 4 pre-/post-test studies reported a decrease within individuals. Two RCTs and 2 pre-/post-test studies found an increase in BMI. Seven papers reported no statistically significant changes. Four of seven short-term studies and 3 of 11 long term studies reported a decrease in BMI. Eleven community-based studies reported on BMI; 5 found a decrease, 4 found an increase and 2 showed no change in BMI. Of 7 non-community-based programmes, 5 found a decrease in BMI and 2 found no change.

The mean difference in BMI in long-term RCT programmes (Fig. 3) was −0.2 kg/m² (95% CI −0.60 to 0.17). Effect size was not significant (p = 0.27) and heterogeneity was 0.
6.3. Lipids

Fourteen studies measured changes in total cholesterol; 7 RCTs and 7 pre-/post-test studies. Only 1 RCT and 5 pre-/post-test studies reported a decrease in total cholesterol with the remaining 8 papers finding no difference. Two of 5 short term studies and 4 of 9 long term studies found a decrease in total cholesterol. Eight community-based studies measured total cholesterol; 5 showed a decrease and 3 found no significant change. Six non-community-based programmes reported on total cholesterol; 1 reported a decrease and 5 reported no change. In fewer studies, LDL cholesterol followed the same pattern of results as total cholesterol (data shown in Tables 1 and 2).

The mean difference in total cholesterol in long-term pre-/post-test programmes (Fig. 4) was −0.2 mmol/l (95% CI −0.30 to 0.01). Effect size was not significant (p = 0.07) and heterogeneity was relatively high (I² = 66%).

Nine studies reported HDL cholesterol changes; 4 RCTs and 5 pre-/post-test studies. Two pre-/post-test studies found an increase in HDL cholesterol and 3 pre-/post-test studies found a decrease. The remaining RCTs reported no change. Two of three short term studies and 1 of 6 long-term studies reported a decrease in HDL cholesterol. Three of 5 community-based studies showed an increase, another reported a decrease and the other showed no change in HDL cholesterol. Four non-community-based programmes reported HDL cholesterol; 1 found an increase and 3 reported no change.

6.4. Glucose and HbA1c

Seven studies measured FBG changes; 2 RCTs and 5 pre-/post-test studies. One RCT and 4 pre-/post-test studies found a decrease in FBG and the other 2 found no change. Three of 4 short term studies and 2 of 3 long term studies showed a decrease in FBG levels. Five of 6 community-based studies showed a decrease in FBG and the remaining showed no change. Both non-community-based programmes showed a reduction in FBG. In fewer studies, HbA1c followed the same pattern of results as FBG (data shown in Tables 1 and 2).

7. Behavioural risk factors

7.1. Physical activity

Of the 25 publications selected, 11 studies included self-reported PA changes; 3 RCTs and 8 pre-/post-test studies. Two RCTs reported an increase in PA in favour of the intervention group and 3 pre-/post-test studies reported an increase in individuals. The other 6 papers reported no statistically significant changes. Two of 5 short term studies showed increases in PA, while 3 of 6 long term studies reported increases. Four of 8 community-based studies reported increases in PA. Only one of 2 non-community-based programmes found an increase in PA. No studies found a decrease in PA.

7.2. Diet

Dietary change included self-reported fruit and vegetable intake, fibre content, fat intake and/or salt intake. One of 4 RCTs reported a reduction in dietary fat-intake while 5 of 6 pre-/post-test studies reported dietary improvements. All short term studies and 3 of 6 long term studies found dietary improvements. Of 8 community-based studies incorporating dietary change, 6 found improvements and 2 found no changes. Both non-community-based programmes showed no significant changes in diet.

7.3. Smoking

Eleven studies assessed smoking status changes (i.e. cigarettes per day); 4 RCTs and 7 pre-/post- test studies. One RCT and 2 pre-/post-test studies reported a decrease in the number of cigarettes smoked per day. The other 8 papers reported no change. The only short term study which reported on smoking did not report an improvement, while 3 of 10 long term studies reported reductions in cigarettes smoked per day. Eight community-based studies reported on smoking but only 2 showed improvements. Two non-community-based programmes reported on smoking rates with only one finding a decrease in cigarettes smoked per day.

7.4. Disease and risk knowledge

Six manuscripts assessed disease and risk knowledge; 2 RCTs and 4 pre-/post-test studies; both RCTs and 3 pre-/post-test studies showed increased knowledge. All short term studies and 2 of 3 long term studies showed an improvement in knowledge. Three of 4 community-based studies showed improvements while both non-community-based programmes reported enhanced knowledge.

8. Discussion

To our knowledge, this is the first systematic review to investigate the effectiveness of primary prevention programmes to improve cardio-metabolic risk in non-urban communities. In a total of 25 studies, we reaffirmed the overall potential for primary prevention programmes to reduce cardio-metabolic risk through improved risk factor control. We found that slightly more pre-/post-test design studies were associated with better outcomes, with the exception of BMI. More RCTs however were only associated with improved physical activity and disease and risk knowledge. Longer study durations showed to be of lesser value overall predominantly due to difficulties in sustaining health promoting behaviours, with programmes of less than 12 months duration shown to be more effective than longer-term interventions (≥ 12 months) in improving all biomedical outcomes. Community-based programmes were more effective than non-community-based programmes in improving all biomedical parameters with the exception of BMI and HbA1c.

Although more pre-/post-test design programmes than RCTs showed improvements in risk factors, differences were marginal. Pre-/post-design interventions have also been known to over-estimate effects due to confounding by factors external to the intervention (Robinson and Doueck, 1994). Appropriately designed and powered RCTs remain the gold standard for evaluating the effectiveness of interventions by virtue of randomization and having a comparator group who may similarly be affected by extraneous confounders. Participants within a control group may also modify their behaviour as a result of being involved in research (the well-known Hawthorne effect (Adair, 1984)). This would have the effect of reducing group differences making (greater) change in the intervention arm more onerous to achieve.

Our findings indicated more obvious changes in risk factor levels and behaviour over a shorter than longer intervention period. This may be due to difficulty associated with maintaining health behaviours over time. A study by Matthews et al. (2007) elicited the challenges associated with maintaining health related behavioural improvements in the long term, particularly concerning diet and PA, that may be counteracted by regular and frequent follow-up (Marcus et al., 1998). A growing body of evidence suggests that short-term programmes including regular follow-up of participants are generally more effective (Yanek et al., 2001; Appel et al., 2003; Stevens et al., 2001; McNabb et al., 1997; Coates et al., 1999; Rosal et al., 2005; Simkin-Silverman et al., 2003; Toobert et al., 2005; Yeh et al., 2003) as frequently contacting subjects also cultivates trust between the participant and study staff (Yanek et al., 2001). As an intervention eases over time or contact is reduced, a participant’s adherence to novel behaviour change often declines in accordance with a reduction in reinforcement of health goals (Wadden et al., 2005). On this basis, it is imperative that regular
follow-up with a health professional is maintained to assess diabetes and cardiovascular risk and initiate pharmacotherapy where needed. Community-based programmes were shown to be more effective than non-community-based programmes in improving all biomedical risk factors with the exception of BMI and HbA1c. These programmes utilised existing community resources (McLeroy et al., 2003) such as local fitness and health groups, as part of their intervention. Health behaviours are influenced by a range of factors including cultural, political and socioeconomic factors (Schooler et al., 1997; Stokols, 1992). Sourcing suitable programmes that an individual can relate to within the community in which they belong or identify with may be important in instigating and promoting health behaviour change (McLeroy et al., 2003). Although some health behaviour change is a representation of an individual's recognition of risk and their decision to minimise that risk, community-based programmes may exude an effect whereby individuals adopt novel behaviours characteristic of their friends, family and social networks.

Significant heterogeneity in the elements of the intervention amongst the programmes in this review made it difficult to delineate the particular aspect(s) responsible for the beneficial effect. Most programmes however were multi-faceted in implementation. Various modes of contacts have achieved effective results, including telephone, electronic mail, and face-to-face communication (Yeh et al., 2003; McManus et al., 2001; Tate et al., 2003; Elliot et al., 2007). Both group and individual intervention delivery have been effective in urban studies targeting smoking cessation and reducing alcohol consumption (Wadden et al., 2005; Schooer et al., 1997) and disease and risk education classes have been valuable in reducing the risk of diseases (Farquhar et al., 1977; Luepker et al., 1994). Goal setting at the outset of a programme is associated with positive health related behaviour change (Cullen et al., 2001; Streccher et al., 1995) and can lead to better results in comparison to vague, or no goals (Bodenheimer and Handley, 2009). It is unknown whether the effectiveness of prevention intervention programmes (or factors thereof) differs by the setting within which it is applied. While a comparison between (matched) urban and non-urban individuals of the benefit of cardio-metabolic primary prevention interventions and their components was not the focus of this review, there is reason to believe that the context may indeed be important. A previous study in one location in Australia provided some evidence that the socio-economic gradient in cardiovascular health is not due to location of residence (Tideman et al., 2013). There may therefore be barriers to health behaviour change in non-urban settings that do not apply in metropolitan locations such as geographical isolation causing residents to drive short distances and less autonomy to engage in physical activities such as walking/riding to work or shopping due to no safe walking and bicycle paths. Furthermore, there may be urban and non-urban disparities in health that have a differential impact in developing compared to developed countries; urban areas in developing countries can lead to unsanitary environments, poorer working and living conditions and are associated with poorer health than non-urban locations (McManus et al., 2001). Despite this, the interventions conducted in developing countries included in this review (Nguyen et al., 2012; Shahar et al., 2012; Balagopal et al., 2008; Balagopal et al., 2012; Huang et al., 2011) showed that individuals living in urban communities enjoyed better health.

9. Limitations

There are a number of limitations that require comment. To enable a sufficient number of manuscripts to be used in this review, older studies worldwide were included spanning a period of socioeconomic and technological change. Despite this however, there has been no significant change in the attributable burden of cardio-metabolic risk factors which have remained the highest ranked of the top 15 and shown upwards shift, not an improvement, since 1990 (Lim et al., 2012). The tradeoff was that extensive meta-analyses may not have been possible due to heterogeneity, variation in the presentation of data, variable outcome measures and inconsistent follow-up intervals. As such, the data presented were predominantly descriptive. Despite broad investigation efforts, all relevant manuscripts may not have been located; the included studies however likely capture the current status of this field of research.
Although there was variance in the strength and direction of reported findings across the included studies, we cannot rule out a bias effect against programmes with results that were not significant. Some prominent programme approaches are discussed, however a comprehensive breakdown of all the elements of interventions was beyond the scope of this review and any potential pharmacological influence on outcomes was not assessed. Additionally, it is also important to note that some studies included in this review had near-normal base-line values which may have diminished intervention effects on some risk factors; as a result, the effect of some interventions may have been underestimated.

10. Summary, implications and future direction

This review has highlighted the significance of primary prevention health behaviour programmes, particularly those utilising community and health resources within study populations in order to promote health behaviour change in adults of a non-urban community. The context of the setting in the development of CVD and/or T2DM prevention programmes is potentially important given the likelihood to influence a programme’s efficacy. Further investigation is needed to elucidate the key individual determinants of cardio-metabolic risk in these non-urban populations and in contrast to urban populations.

Conflict of interest statement

The authors declare that there are no conflicts of interest.

Transparency document

The Transparency document associated with this article can be found, in online version.

Acknowledgements

MC, JB and SS are supported by the National Health and Medical Research Council of Australia (1069043 (MC), 1044897 (SS), 1112829). JB is supported by the Heart Foundation (100950 (JB)).

References

Summary

This manuscript drew attention to the effective characteristics of an intervention designed to reduce non-urban population cardio-metabolic disease risk via health behaviour change. Information from 25 studies confirmed the benefit of health modification programs in preventive health. It identified that pre/post study designs were marginally more effective, but this may be due to overestimation/confounding as well as any potential ‘Hawthorne effect’ occurring within randomised controlled trials, whereby participants (particularly in control group) may respond to an intervention simply by virtue of participating. The difficulties associated with maintaining new health behaviours over time may have led to short-term interventions being identified as more effective than longer term interventions while community-based interventions were also particularly effective at reducing cardio-metabolic disease risk, likely due to utilisation of community constructs to deliver their intervention.

To our knowledge, this report provided insight into the effectiveness of primary prevention programmes to improve cardio-metabolic risk in non-urban populations. It highlights the need for more interventions for people in regional, rural and/or remote communities, where risk is exacerbated due to contextual factors. Further research to identify the particular aspect(s) of an intervention that successfully reduce cardio-metabolic risk in non-urban populations is needed to inform any future tailored interventions.
Chapter 4
Research Platforms & Methods
Chapter 4 introduces the research framework on which this thesis work was conducted – the Management to Optimise Diabetes and mEtabolic syndrome risk reduction via Nurse-led intervention (MODERN) Study. The aims, methods and baseline characteristics of MODERN are presented, and the methodology used for this research work is outlined.

This Chapter includes the following submitted manuscript:

Preface

The effective characteristics of primary prevention interventions aimed to reduce cardio-metabolic disease risk in non-urban populations have been identified. As previously described, MODERN is a pragmatic, RCT of a nurse-led health and lifestyle intervention targeting risk factor management to prevent CVD and T2DM in a regional cohort. A broad screening process was undertaken to identify individuals living in the community with MetS.

The following manuscript (Chapter 4) describes the MODERN study design and baseline screening results.
Cardiometabolic risk characteristics and adherence to risk factor targets in regional communities: Screening results of the Management to Optimise Diabetes and mEtabolic syndrome Risk reduction via Nurse-led intervention (MODERN) randomized controlled trial

Andre L Rodrigues, BSc (Hon) 1,2 | andre.rodrigues@baker.edu.au

Paul Zimmet, MD 3 | paul.zimmet@monash.edu

Melinda J Carrington, PhD 1,2 | melinda.carrington@baker.edu.au

1 Pre-Clinical Disease and Prevention Unit, Baker Heart and Diabetes Institute, Victoria, Australia.
2 Department of Epidemiology and Preventive Medicine, Faculty of Medicine, Nursing and Health Sciences, Monash University, Victoria, Australia.
3 Department of Diabetes, Faculty of Medicine, Nursing and Health Sciences, Monash University, Victoria, Australia

Corresponding author:

Associate Professor Melinda Carrington, PO Box 6492, Melbourne, VIC 3004, Australia
Telephone: +61 3 8532 1638; Fax: +61 3 8532 1100; Mobile: +61 447 359 273; Email: melinda.carrington@baker.edu.au

Keywords

Cardiovascular Disease, Diabetes, Prevention, Intervention, Regional
ABSTRACT

Purpose. We measured risk factors to identify individuals with metabolic syndrome (MetS) to recruit for the MODERN (Management to Optimise Diabetes and mEtabolic syndrome risk reduction via Nurse-led intervention) randomised controlled trial. It was hypothesised that high levels of cardio-metabolic risk factors would be evident.

Methods. Nurse-led clinics were established in two regional locations to conduct non-invasive clinical examinations and consultations. Self-selected participants were assessed by study nurses to collect information about anthropometry, blood pressure (BP), blood lipids and glycated hemoglobin (HbA$_1C$), and health behaviours including smoking, diet and physical activity.

Findings. A total of 853 individuals comprising more women than men were screened (n=520, 61%). Average age was 56.4 ± 7.9 years. In contrast to recommended guidelines, the majority were overweight or obese (77%). Around 4 in 5 individuals had total cholesterol ≥4.5 mmol/L (86%) and low-density cholesterol ≥2.5 mmol/L (78%) and one third had HbA$_1C$ levels ≥5.7%, indicating prediabetes. More men than women consumed excess alcohol (44 vs. 22%, p<0.001) whereas more women were physically inactive (40 vs. 32%, p=0.013). Overall, 32% of individuals screened met the criteria for MetS primarily via the triad of increased waist circumference, BP and triglyceride (achieved by 47%); waist circumference, BP and HbA$_1C$ (25%) and; waist circumference, triglyceride and HbA$_1C$ (17%).

Conclusions. High levels of cardio-metabolic risk factors were evident, demonstrating eligibility to participate in a lifestyle modification program with substantial potential to lead longer and healthier lives. This is the aim of the longer-term MODERN intervention trial.
INTRODUCTION

Cardiovascular disease (CVD) is a global killer, being the attributable cause of death for more than 17.7 million people in 2015\(^1\). Despite the marked decline in age-adjusted mortality over the past five decades, CVD kills one Australian every 12 minutes\(^2\). Owing to this, a disturbing 95% of Australian adults (over 18 years of age) have at least one risk factor for CVD and around two thirds have two or three concurrent biomedical or behavioural risk factors\(^3\). The most common combinations of risk factors incorporate obesity, hypertension, dyslipidemia or dysglycemia; any three or more of these occurring simultaneously form a dangerous cluster of risk factors representing Metabolic Syndrome (MetS)\(^4\). Compared to those without MetS, this condition increases the 5 to 10 year risk of type 2 diabetes mellitus (T2DM) 5-fold and the risk of CVD 2-fold\(^5\)\(^6\). MetS also confers a 3 to 4-fold increased risk of myocardial infarction and a 2 to 4-fold increased risk in stroke\(^6\). However, in Australian adults, there is a high prevalence of obesity (3 in 5 are either overweight or obese), elevated blood pressure (BP) (25%), physical inactivity (3 in 4), lipid abnormalities (63% have dyslipidemia) and elevated blood glucose (8.2%)\(^3\)\(^4\). Rising risk factor levels predisposes individuals to T2DM and CVD. Given these associations, the implications on public health are substantial; with around 1 and a half million Australians living with T2DM and around 4.2 million living with CVD\(^7\), the health care costs are exorbitant and are estimated to exceed $22 billion by 2033\(^8\).

The burden of disease caused by cardio-metabolic diseases is more pronounced with increasing remoteness\(^9\). Residents of regional or remote areas fare worse in key health behaviours and risk factors; high BP (24% v 22%), physical inactivity (72% v 64%), overweight or obesity (69% v 61%) and smoking (21% v 13%) rates are all higher in people living in regional locations than in major cities\(^2\). Despite this, evidence
suggests that MetS can be reversed and T2DM and CVD are also largely preventable via means of lifestyle and/or pharmacological intervention\textsuperscript{10}. However, there have been few interventions aimed at reducing cardio-metabolic risk via independent nurse-led clinics situated in higher risk non-metropolitan communities\textsuperscript{11}.

The Management to Optimise Diabetes and mEtabolic syndrome risk reduction via Nurse-led intervention (MODERN) Study is a community-based evaluation of a nurse-led and lifestyle intervention program targeted at individuals with MetS and an increased risk of CVD and T2DM. We report the cardio-metabolic risk factors for screened participants of the MODERN cohort. We hypothesised that there would be high levels of risk factors for CVD and T2DM in this regional cohort.

METHODS

Study design and setting

MODERN is a multi-site randomised trial for individuals aged 40 to 70 years who live in regional Colac or Shepparton, Victoria; the study protocol and design has been published previously\textsuperscript{12}. Nurse-led clinics were established in each location whereby study nurses obtained written consent from participants and non-invasive clinical examinations and consultations were held. The study was approved by the Australian Catholic University Human Research Ethics Committee (Project No. 2014 244V). Upon completion of the study, MODERN will follow CONSORT guidelines depicting a flow diagram displaying the progress of all participants through the trial when reporting the primary outcome.

Study participants
Self-selected participants volunteered in response to community engagement by study personnel who promoted the study at 29 local businesses, 7 community group meetings (e.g. Rotary), 14 schools and 7 neighbourhood markets in addition to broader scale media exposure (16 print, 2 television news stories and approximately 240 radio advertisements), random and direct postal letters of invitation to 3,416 and 1,397 households, respectively, and via social networking and local political leaders’ web sites.

Respondents were initially screened by telephone and scheduled for a clinic assessment to determine study eligibility. Inclusion criteria were: a) age between 40 and 70 years with; b) any three or more measured risk factors for MetS of central obesity (men >94 cm; women >80 cm), dyslipidemia (high triglycerides [≥1.7 mmol/L] or low high density lipoprotein cholesterol [men <1.03 mmol/L; women <1.29 mmol/L]), elevated BP (≥130/85 mmHg) and dysglycemia (HbA1c ≥5.7%) [4] and; c) living in Shepparton or Colac and; d) capable of attending scheduled study clinic visits. Exclusion criteria were: a) non-local residents; b) age <40 or >70 years; c) ≤ 2 risk factors for MetS or; d) previously diagnosed CVD, chronic kidney disease, neurological/cognitive impairment or other chronic disease. Recruitment occurred between September 2014 and April 2016.

**Study variables and data collection procedures**

All potentially eligible participants were comprehensively profiled at baseline study visits. Participants received structured questionnaires in the mail to complete at home and return with them to their visit that was conducted by research nurses. The structured questionnaire collected demographic information (age, sex, ethnicity, relationship status, employment and education level); lifestyle factors including
smoking, diet and alcohol (assessed using the Dietary Questionnaire for Epidemiological Studies (DQES v2)\textsuperscript{13}), sleep, physical activity and sedentary behaviour (measured with the International Physical Activity Questionnaire (IPAQ)\textsuperscript{14}); and data on medications; medical history and family history of CVD and diabetes. A screening assessment collected physical outcomes of height, weight, waist and hip circumference; fasting capillary/fingerstick whole blood tests of total (TC), high (HDL-C) and low (LDL-C) density lipoprotein cholesterol, triglycerides and HbA\textsubscript{1C} with a point of care analyser (Afinion\textsuperscript{TM} AS100, Alere, MA, USA); and sitting BP with an automated BP monitor (Omron HEM-907, Omron Healthcare Co. Ltd, Kyoto, Japan). Participants’ BP, body weight, height, waist and hip circumference and biochemistry tests of TC, HDL-C, LDL-C, triglycerides and HbA\textsubscript{1C} were measured following World Health Organization surveillance guidelines\textsuperscript{15}. For BP, two measurements were taken one minute apart after at least five minutes of rest; we used the mean of these two readings in analyses. If BP varied by $\geq 10/\geq 5$ mmHg, a third reading was taken and the closest two out of the three readings were analysed. Cardiovascular 5-year risk was calculated via the Framingham Risk Equation\textsuperscript{16} based on sex, age, diabetes status, smoking history, systolic BP and TC:HDL ratio. T2DM 5-year risk was calculated via the 10-item AUSDRISK prediction model\textsuperscript{17} based on sex, age, country of birth, family history of diabetes, history of high blood glucose, treatment for hypertension, smoking history, fruit and vegetable intake, physical activity levels and waist circumference.

\textit{Risk factor definitions}

Smoking cessation was encouraged. Intake of alcohol was assessed by alcohol guidelines\textsuperscript{18} using a cut off of no more than 2 standard drinks on any day to reduce the lifetime risk of alcohol-related harm. Physical activity and sedentary behaviours were
compared against the Australian Government Department of Health recommendations to accumulate at least 30 minutes of moderate intensity physical activity on all or most days of the week and if capable, vigorous activity for 30 minutes or more for 3 or 4 days a week, and to limit sedentary behaviour. Healthy weight was assessed by Australian Dietary Guidelines using BMI ranges <24.9 kg/m$^2$ (healthy weight), 25.0 – 29.9 kg/m$^2$ (overweight) and ≥30.0 kg/m$^2$ (obese) and waist circumference <80 cm (women) or <94 cm (men) thresholds. The Royal College of Pathologists of Australasia manual was consulted for optimal lipid levels for reduced risk of coronary artery disease using thresholds <4.5 mmol/L for TC, <2.5 mmol/L for LDL-C, >1.0 mmol/L for HDL-C, and <2.0 mmol/L for triglycerides. Blood sugar levels were assessed by American Diabetes Association guidelines using HbA$_1C$ <5.7% (39 mmol/mol). Elevated BP was classified by National Heart Foundation of Australia guidelines using a cut off value of <140/90 mmHg. Five-year CVD risk scores were categorised in accordance with Australian guidelines using low (≤9%), moderate (10-15%) and high (≥16%) risk levels. Similarly, AUSDRISK scores for 5-year T2DM risk were classified as low (≤5 points), intermediate (6-11 points) or high (≥12 points).

**Intervention study (longer term follow-up over 24 months)**

Eligible participants were invited to participate in a two-year randomised controlled trial of a nurse-led health and lifestyle intervention compared to standard care. This prospective, multi-centre, parallel group trial will determine the effectiveness of a nurse-led clinical program that modulates health and lifestyle factors to reduce cardio-metabolic risk and future development of CVD and T2DM in individuals with MetS. In brief, the MODERN intervention comprised: 1) cardio-metabolic risk factor management according to recommended guidelines, 2) health education and advice, 3)
care planning and, 4) scheduled follow-up by way of individualised face-to-face and telephone sessions at a frequency indicated by the GARDIAN traffic-light management system\textsuperscript{25} based on risk level. Standard care involved generalised risk factor management but less intensive health education and advice in addition to any typical health care an individual may seek as required. Twelve and 24-month follow-up data were collected for all participants. The primary outcome is achievement of MetS risk factor thresholds or minimal clinically meaningful changes for at least three risk factors that characterise MetS over 2-year follow up. All aspects of the MODERN study design, study power, secondary endpoints, detailed procedures and description of the intervention can be found in the published protocol\textsuperscript{11}.

**Statistical analyses**

Descriptive statistics summarised the baseline characteristics of the screening cohort. Continuous variables were assessed by means and standard deviations and discrete variables were assessed by frequencies and percentages. Continuous outcome variables were compared between groups using independent t-tests while categorical variables were analysed with $\chi^2$ tests. Statistical significance was interpreted as two-sided at the level $\alpha=0.05$. Data were analysed using SPSS Statistics 21.0 (SPSS Inc., Chicago, Illinois, USA).

**RESULTS**

**Sex-based differences in cardio-metabolic risk profile**

A total of 853 individuals were assessed for study eligibility. Participant risk factor characteristics according to sex are summarised in Table 1. More women than men were screened ($n = 520, 61\%$). Mean age was $56.4 \pm 7.9$ years, with no difference
according to sex. The proportion of men and women with Caucasian ethnicity and a maximum secondary school education was similar (all $P < .05$). A significantly higher proportion of women than men were living alone (24% vs. 14%, $P < .001$) and not employed (28% vs. 22%, $P = .035$).

Mean BMI was not different for men and women although the majority of participants were in the overweight or obese range (men: 83%; women: 73%). There were more men and fewer women than expected who were classified overweight than healthy weight ($P < .001$). A higher percentage of women had a waist circumference that put them at increased risk of developing chronic diseases (women: 85%; men: 78%, $P = .009$). Mean systolic and diastolic BP was significantly higher in men than women (systolic [men: 131 mmHg; women 126 mmHg, $P < .001$]; diastolic [men: 79 mmHg; women 77 mmHg, $P = .004$]) albeit a similar proportion of participants had elevated BP levels ≥140/90 mmHg ($P = .360$). Mean TC was significantly higher in women than men (5.71 ± 1.10 mmol/L vs. 5.48 ± 1.09 mmol/L, $P = .003$) but the prevalence exceeding recommended levels was similarly elevated (men: 84%; women: 88%, $P = .135$). Women had lower mean LDL-C levels compared to men (3.20 ± 1.00 mmol/L vs. 3.33 ± 0.91 mmol/L, $P = .045$) and a lesser percentage (74% vs. 83%, $P = .001$) had LDL-C levels ≥2.5 mmol/L. Moreover, women had higher mean HDL-C levels compared to men (1.80 ± 0.41 mmol/L vs. 1.42 ± 0.38 mmol/L, $P < .001$) and a smaller percentage (1% vs. 11%, $P < .001$) had HDL-C levels <1.0 mmol/L. Mean triglyceride and HbA1C levels and the proportion of participants exceeding the recommended targets of these risk factors was similar in men and women. The prevalence of MetS was 32% with no sex-based differences shown.

Smoking prevalence was low overall and not different according to sex. In contrast to women, a higher (double) proportion of men consumed alcohol at high risk levels
(22% vs. 44%, \( P < .001 \)) and a lower percentage were physically inactive (40% vs. 32%, \( P = .013 \)). Men had significantly higher mean dietary energy levels than women (p<0.001).

As shown in Table 1, family history of CVD or diabetes was not different for men and women. Low CVD and T2DM risk was more prevalent in women than men resulting in more men and fewer women than expected who had moderate and high CVD and T2DM risk.
Metabolic syndrome characteristics

Table 2 reports participant risk factor characteristics for MetS components and eligibility. Most people screened had an increased waist circumference (80%) with approximately half of individuals having elevated BP (48%). Around a third of all adults had elevated triglyceride (34%) and HbA\textsubscript{1C} (33%) levels, with fewer people having reduced HDL-C levels (11%). Similarly, the majority of eligible participants (97%) satisfied the increased waist circumference criteria specified by the IDF [12]. The remaining additional three conditions of elevated BP, triglyceride and HbA\textsubscript{1C} were met by around 60-70% of participants, with fewer individuals fulfilling the HDL-C criteria to be included in the MODERN randomised controlled trial.

Of the five MetS focus risk factors, the most frequent combination met by nearly half of participants was increased waist circumference, BP and triglyceride (47%) followed by a quarter of participants who had increased waist circumference, BP and HbA\textsubscript{1C} (25%). The next highest ranked triad of risk factors was increased waist circumference, triglyceride and HbA\textsubscript{1C} (17%). Together, these top three combinations reported 89% of study eligibility for people with MetS.

Eligibility characteristics

Of the 853 participants screened, 276 (32%) were deemed eligible to participate in the MODERN intervention study. As shown in Table 3, eligible participants were older and there was a tendency for fewer couples (married or de facto) and more single people living separately to be eligible. Significant differences in eligibility were not found between sex, ethnicity, employment type or level of education (all \( P < .05 \)).
DISCUSSION

High levels of risk factors for T2DM and CVD were common in this regional cohort. There was a high prevalence of overweight/obesity affecting around 80% of participants, dyslipidemia impacting approximately 80% of individuals’ total and LDL-C levels and dysglycemia affecting one third of adults. In addition, health behaviours such as high-risk alcohol consumption and insufficient physical activity were reported by around one third of participants. There were disparities between men and women with respect to several risk factors; overweight/obesity, LDL-C, HDL-C, excess alcohol consumption and dietary energy intake levels were all poorer for men than women. These negative health determinants were manifest in risk prediction scores which were also worse for men. Women however were not free from risk factors that increase the likelihood of cardio-metabolic illness, showing a higher proportion than men with central adiposity and insufficient amounts of physical activity. Consequently, the presence of MetS was high leading to one-third of participants, who were more likely to be older and living alone, being eligible for the MODERN trial. These findings highlight the need for health and lifestyle modification programs targeting biomedical risk factors and health behaviours for adults residing in regional areas of Australia with MetS parameters.

We previously reported high CVD risk factor levels in regional Victoria\(^{36}\) whereby 59% of people from four regional communities were hypertensive, 28% were obese, 65% had elevated TC and 29% did not meet physical activity guidelines. Whilst our present findings document a potential improvement in BP (25%), this discrepancy is likely due to differences in risk factor definitions for primary and secondary risk prevention. Other risk factors such as obesity (39%), elevated TC (86%) and insufficient physical activity (37%) have not shown any significant improvement
however. National statistics\(^2\) showed similar findings where it was reported that 27% of people living in regional areas had elevated BP however 70% did not meet physical activity guidelines; almost twice that reported in our findings. Reasons for this disparity may be attributed to exaggerated self-reported data. Objective measures of physical activity are being utilised in the MODERN randomised controlled trial to more accurately ascertain physical activity levels in this regional population.

Findings from population studies and community settings support targeted risk factor management for improving cardio-metabolic risk factors. The Diabetes Prevention Study\(^{27}\) compared a nurse-facilitated lifestyle intervention with a control group and reported improvements after 12 months in weight (-4.7%), fasting glucose (-0.24mmol/L), total cholesterol (-0.13mmol/L) and BP (-5mmHg). Further, systematic reviews and meta-analyses of nurse-led lifestyle interventions have been shown to improve risk factors in individuals with hypertension\(^{28}\), T2DM\(^{29}\) and MetS\(^9\). Dunkley et al reported the effectiveness of lifestyle interventions for reducing CVD and T2DM risk in individuals with MetS which were almost four-times (OR 3.81; 95% CI 2.47-5.88) more likely to reverse MetS compared with control (no intervention) and 60% (OR 1.59; 95% CI 1.04-2.45) more likely to reverse MetS than pharmacological intervention. In our own experimental studies\(^{30}\), we have also found clinically significant improvements in BP, total cholesterol and BMI through nurse-led management. Whether similarly positive changes persist longer term is unknown and our MODERN study intervention will determine this response over a follow-up length of two years.

There are limitations to be considered to our screening eligibility study. Self-reporting to gather some health behaviour data could not be confirmed and may be subject to potential exaggeration or desirability bias. Further, self-selection of participants from
two regional communities may not be representative when compared to wider population data. Some participants who volunteered may also have been “worried well” and wanting reassurance about their cardio-metabolic risk whereas others self-nominated in search of more intensive management to reduce their elevated risk. Nonetheless, all participants deemed eligible for the longer-term MODERN randomised trial were identified with MetS and thus represent ideal and interested candidates for risk factor modification intervention.

The screening results of the MODERN study have further highlighted the elevated cardio-metabolic disease risk status of individuals from regional communities. These data suggest that there is scope for regional dwelling Australians to attenuate risk for developing chronic diseases such as CVD and T2DM. It will be the aim of the MODERN study, a multi-faceted, nurse-led, lifestyle intervention, to improve the cardio-metabolic risk profile of this regional cohort and advocate for more nurse-led clinics to be established.

REFERENCES


Table 1  Risk characteristics of participants assessed for eligibility according to sex

<table>
<thead>
<tr>
<th>Risk factor characteristics</th>
<th>All (n=853)</th>
<th>Men (n=333)</th>
<th>Women (n=520)</th>
<th>p value</th>
<th>All (n=853)</th>
<th>Men (n=333)</th>
<th>Women (n=520)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>X̄ (sd), n(%)</td>
<td>t-test</td>
<td></td>
<td>X̄ (sd), n(%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Demographic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>853</td>
<td>56.4 (7.9)</td>
<td>56.2 (8.1)</td>
<td>56.5 (7.9)</td>
<td>0.656</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian/European ethnicity</td>
<td>853</td>
<td>812 (95%)</td>
<td>314 (94%)</td>
<td>498 (96%)</td>
<td>0.326</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Living alone</td>
<td>852</td>
<td>170 (20%)</td>
<td>46 (14%)</td>
<td>124 (24%)</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not employed</td>
<td>853</td>
<td>218 (26%)</td>
<td>72 (22%)</td>
<td>146 (28%)</td>
<td>0.035</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maximum secondary education</td>
<td>852</td>
<td>459 (54%)</td>
<td>185 (56%)</td>
<td>274 (53%)</td>
<td>0.430</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Biomedical</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>853</td>
<td>29.3 (5.8)</td>
<td>29.0 (4.6)</td>
<td>29.4 (6.5)</td>
<td>0.213</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Healthy weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>194 (23%)</td>
<td>55 (17%)</td>
<td>139 (27%)</td>
</tr>
<tr>
<td>Overweight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>328 (38%)</td>
<td>161 (48%)</td>
<td>167 (32%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------</td>
<td>-------</td>
<td>-------</td>
<td>-------</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>853</td>
<td>853</td>
<td>848</td>
<td>853</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obese</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waist circumference</td>
<td>98.0</td>
<td>102.6</td>
<td>95.0</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men ≥94 cm; Women ≥80 cm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>128</td>
<td>131</td>
<td>126</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>77</td>
<td>79</td>
<td>77</td>
<td>0.004</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure ≥140/90 mmHg</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood lipids</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol ≥4.5 mmol/L</td>
<td>5.62</td>
<td>5.48</td>
<td>5.71</td>
<td>0.003</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL-cholesterol ≥2.5 mmol/L</td>
<td>3.25</td>
<td>3.33</td>
<td>3.20</td>
<td>0.045</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL-cholesterol &lt;1.0 mmol/L</td>
<td>1.65</td>
<td>1.42</td>
<td>1.80</td>
<td>&lt;0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triglyceride ≥2.0 mmol/L</td>
<td>1.70</td>
<td>1.68</td>
<td>1.72</td>
<td>0.603</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HbA1C ≥ 5.7%</td>
<td>5.6</td>
<td>5.6</td>
<td>5.6</td>
<td>0.674</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metabolic syndrome [12]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>853</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Health behaviours</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoking</td>
<td>60</td>
<td>19</td>
<td>41</td>
<td>0.225</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

112
<table>
<thead>
<tr>
<th>Risk factor</th>
<th>N</th>
<th>261 (31%)</th>
<th>147 (44%)</th>
<th>114 (22%)</th>
<th>&lt;0.001</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excess alcohol consumption</td>
<td>853</td>
<td>&gt;2 standard drinks on any day</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical activity</td>
<td>851</td>
<td>&lt;150 minutes/week</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet</td>
<td>840</td>
<td>Energy (kJ/day)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family history</td>
<td>847</td>
<td>Low ≤ 9%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVD</td>
<td>853</td>
<td>Moderate 10-15%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>846</td>
<td>High ≥ 16%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVD risk score</td>
<td>853</td>
<td>T2DM risk score</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Diet                  | 820 | Energy (kJ/day) |
|-----------------------|---|-----------|-----------|-----------|--------|
| Excess alcohol consumption | 853 | >2 standard drinks on any day |
| Physical activity     | 851 | <150 minutes/week |
| Diet                  | 840 | Energy (kJ/day) |
| Family history        | 847 | Low ≤ 9% |
| CVD                   | 853 | Moderate 10-15% |
| Diabetes              | 846 | High ≥ 16% |
| CVD risk score        | 853 | T2DM risk score |

<p>| Diet                  | 820 | Energy (kJ/day) |
|-----------------------|---|-----------|-----------|-----------|--------|</p>
<table>
<thead>
<tr>
<th></th>
<th>-</th>
<th>-</th>
<th>-</th>
<th>-</th>
<th></th>
<th>51 ( 6%)</th>
<th>13 ( 4%)</th>
<th>38 ( 7%)</th>
<th>0.017</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>275 (34%)</td>
<td>95 (30%)</td>
<td>180 (36%)</td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
<td>494 (60%)</td>
<td>208 (66%)</td>
<td>286 (57%)</td>
<td></td>
</tr>
</tbody>
</table>
Table 2  Metabolic syndrome components of participants assessed for eligibility

<table>
<thead>
<tr>
<th>Metabolic syndrome characteristics</th>
<th>Total n=853</th>
<th>Eligible n=276 (32%)</th>
<th>Not eligible n=577 (68%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waist circumference (WC)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men &gt; 94 cm</td>
<td>684 (80%)</td>
<td>268 (97%)</td>
<td>416 (72%)</td>
</tr>
<tr>
<td>Women &gt; 80 cm</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure (BP) ≥ 130/85 mmHg</td>
<td>412 (48%)</td>
<td>216 (78%)</td>
<td>196 (34%)</td>
</tr>
<tr>
<td>Triglyceride ≥ 1.7 mmol/L</td>
<td>290 (34%)</td>
<td>192 (70%)</td>
<td>98 (17%)</td>
</tr>
<tr>
<td>HbA\textsubscript{1C} ≥ 5.7 %</td>
<td>278 (33%)</td>
<td>185 (67%)</td>
<td>93 (16%)</td>
</tr>
<tr>
<td>HDL-cholesterol</td>
<td>95 (11%)</td>
<td>71 (26%)</td>
<td>24 (4%)</td>
</tr>
</tbody>
</table>

**Combinations (ranked)**

| WC, BP, Triglyceride              | 129 (47%)   |
| WC, BP, HbA\textsubscript{1C}     | 69 (25%)    |
| WC, Triglyceride, HbA\textsubscript{1C} | 46 (17%)  |
| WC, BP, HDL-cholesterol           | 11 (4%)     |
| WC, Triglyceride, HDL-cholesterol | 9 (3.5%)    |
| WC, HbA\textsubscript{1C}, HDL-cholesterol | 4 (1%)   |
| BP, Triglyceride, HbA\textsubscript{1C} | 4 (1%)    |
| BP, Triglyceride, HDL-cholesterol | 3 (1%)      |
| BP, HbA\textsubscript{1C}, HDL-cholesterol | 0 (0%)    |
| Triglyceride, HbA\textsubscript{1C}, HDL-cholesterol | 1 (0.5%) |
Table 3  Demographic characteristics of participants assessed for eligibility

<table>
<thead>
<tr>
<th>Demographic characteristics</th>
<th>Eligible</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=276 (32%)</td>
</tr>
<tr>
<td></td>
<td>n=577 (68%)</td>
</tr>
<tr>
<td></td>
<td>p</td>
</tr>
<tr>
<td>Age, years (sd)</td>
<td>57.4 (7.7)</td>
</tr>
<tr>
<td>Sex</td>
<td>Men 106 (32%)</td>
</tr>
<tr>
<td></td>
<td>Women 170 (33%)</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>Caucasian/European 267 (33%)</td>
</tr>
<tr>
<td></td>
<td>Other 9 (22%)</td>
</tr>
<tr>
<td>Relationship status</td>
<td>Married or de facto 200 (29%)</td>
</tr>
<tr>
<td></td>
<td>Never married, separated, divorced or widowed 76 (45%)</td>
</tr>
<tr>
<td>Employment</td>
<td>Full-time, part-time or casual 198 (31%)</td>
</tr>
<tr>
<td></td>
<td>Not employed or retired 78 (36%)</td>
</tr>
<tr>
<td>Education (highest level)</td>
<td>Higher education 115 (29%)</td>
</tr>
<tr>
<td></td>
<td>High school, TAFE or trade 161 (35%)</td>
</tr>
</tbody>
</table>
Summary

It is the aim of the MODERN study, to develop the evidence for improved health of individuals living in the community with clusters of risk factors and the need for more nurse-led clinics to be established in order to help address this need.

To identify eligible participants, this chapter reported the screening results which showed high levels of cardio-metabolic risk factors were evident, demonstrating participants’ eligibility for a lifestyle modification program with substantial potential to lead longer and healthier lives.

This chapter highlighted an obvious need for health and lifestyle modification programs targeting biomedical risk factors and health behaviours. All participants deemed eligible for the MODERN RCT were identified with MetS and thus represented as ideal and interested candidates for risk factor modification intervention.

Understanding the role of self-efficacy and health locus of control as potential mediators of interventional response may aid in the design and implementation of future prevention programs. Whether these psychological constructs are themselves associated with any key health behaviour changes or cardio-metabolic risk status or are modifiable as a result of nurse-led lifestyle intervention will be discussed in Chapters 5 and 6.
Chapter 5
Self-efficacy, Health Locus of Control and Cardio-metabolic Disease Risk: Baseline
Chapter 5 reports the associations between behaviour-specific self-efficacy, health locus of control, health perceptions and cardio-metabolic disease risk at baseline.

This Chapter includes the following submitted manuscript:

Preface

Chapter 4 presented the design and rationale of the overall MODERN study together with the screening results which showed a high level of risk and hence potential for risk factor modification. Behaviour-specific self-efficacy and health locus of control have been widely investigated as mediators of health behaviour adoption and change.

Understanding the degree to which a person believes they are able to adhere to a prescribed novel behaviour (self-efficacy) or the extent to which a person feels they themselves or others have control over their health outcomes (health locus of control) may provide meaningful explanation for their current risk status (Chapter 5) and also be influential in predicting future risk (Chapter 6) for developing CVD or T2DM.

Chapter 5 discusses the associations between these psychological constructs, health perceptions and cardio-metabolic disease risk within the screening cohort of the MODERN study.
Relationship between cardio-metabolic disease risk, health beliefs and perceptions: A regional perspective

Andre L. Rodrigues 1, 2 | andre.rodrigues@baker.edu.au

Ralf Schwarzer 3 | ralf.schwarzer@fu-berlin.de

Chantal Ski 4 | c.ski@qub.ac.uk

Melinda J. Carrington 1, 2 | melinda.carrington@baker.edu.au

1 Pre-Clinical Disease and Prevention Unit, Baker Heart and Diabetes Institute, Victoria, Australia

2 Dept. of Epidemiology and Preventive Medicine, Monash University, Victoria, Australia

3 Dept. of Psychology, Freie Universität Berlin, Berlin, Germany

4 School of Nursing and Midwifery, Queen’s University Belfast, Belfast, Northern Ireland, UK

Corresponding Author:

Andre Leao Rodrigues, PO Box 6492, Melbourne, VIC 3004, Australia

Telephone: +61 3 8532 1749; Fax: +61 3 8532 1100; Mobile: +61 430 781 421;

Email: andre.rodrigues@baker.edu.au
**Introduction:** An individual’s sense of self-efficacy, locus of control and cardiovascular disease (CVD) risk perception may influence health behaviours and health status.

**Methods:** As part of screening for the MODERN randomised controlled trial of a health and lifestyle intervention, we assessed behaviour specific self-efficacy (nutrition, physical activity, alcohol, smoking), locus of control (internal, powerful others, chance) and CVD risk perception as potential modulators for developing CVD and type 2 diabetes mellitus (T2DM) in (n = 853) 40 to 70-year-old adults.

**Results:** Higher internal locus of control scores were associated with low and moderate CVD and T2DM risk. Higher chance locus of control scores were associated with high risk for CVD compared to low and moderate CVD risk groups who were no different to each other. People with higher powerful others locus of control scores had a higher estimated CVD (p<0.05) and T2DM (p<0.001) risk than people with lower powerful others scores. There were no significant associations between any behaviour specific self-efficacy and CVD or diabetes risk.

**Conclusions:** Individuals with an external (chance, powerful others) locus of control were associated with a greater risk of developing CVD or T2DM compared to internals. Health beliefs and perceptions may be modifiable and useful in predicting intervention response.

**Keywords:** cardiovascular disease, diabetes, intervention, prevention, regional
Introduction

Cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) are chronic cardio-metabolic diseases and are a major health concern; they rank highly in global mortality rates, are predictable and largely preventable (1). The prevalence of CVD increases with remoteness (2). Similarly, mortality rates and hospitalisations attributed to CVD and T2DM increase with greater geographical remoteness (2). These disparities in prevalence and outcomes reflect an incremental need for effective treatment and management of risk factors and primary prevention of cardio-metabolic disease with greater distance from metropolitan cities. Initial management for reducing the risk factors for cardio-metabolic disease is lifestyle health behaviour modification, primarily: increasing physical activity; improving diet; smoking cessation; minimising sedentary time; and reducing alcohol consumption (3). When compared to metropolitan locations, residents of non-urban communities reported higher rates of smoking, excess alcohol consumption and sedentary behaviour, (4) and engaged in sub-optimal dietary and physical activity behaviours. These key differentials combined with other disparities such as lower education levels, lower incomes and poorer access to healthcare (4) elevate the overall burden of cardio-metabolic disease risk.

Facilitating positive behaviour change is paramount for disease prevention and health promotion. Perceived self-efficacy and locus of control have been identified as significant determinants of health behaviour change. Self-efficacy refers to one’s belief in their ability to overcome challenging barriers and demands (5). Choices of behaviour that individuals make are influenced by self-efficacy. Health locus of control relates to how strongly a person believes they have control over the situations and experiences that may affect their health (5). Three locus of control dimensions
vary as being determined by (1) their own actions (internal); (2) (powerful) others; and (3) luck or chance.

This research aims to investigate the associations between self-efficacy, locus of control, and health perceptions with risk for developing CVD and T2DM. We hypothesised that there would be an inverse correlation between self-efficacy, locus of control and CVD and T2DM risk, whereby a lower perceived self-efficacy for health behaviours and a more external health locus of control would be associated with increased CVD and T2DM risk. Conversely, a higher perceived self-efficacy for health behaviours and a higher internal locus of control would be associated with a low CVD and T2DM risk. We tested this hypothesis in the Management to Optimise Diabetes and mEtabolic syndrome risk reduction via the Nurse-led intervention (MODERN) Study (6).

Methods

Study Design

Briefly, MODERN is a 24-month, multi-centre, appropriately powered and pragmatic randomised controlled trial of an innovative, nurse-led intervention and has been previously described in detail (6). Two regional clinics (in Greater Shepparton and Colac Otway, Victoria, Australia) were established to conduct this research. All residents aged 40-70 who presented to the clinic were screened (between October 2014 and March 2016) for metabolic syndrome (MetS) and eligibility into the study. Recruitment strategies have been previously reported (7). MetS was characterised by any three or more of: elevated blood pressure (BP), elevated triglyceride levels; low high density lipid cholesterol (HDL), central obesity or hyperglycaemia. Eligible participants were invited to participate and were subsequently randomised to receive
either nurse-led intervention or usual care over the course of the study period. MODERN is registered with the Australian New Zealand Clinical Trials Registry (ACTRN12616000229471) and the study protocol was approved by the Australian Catholic University (ACU) Human Research Ethics committee (2014 244V). All participants provided written informed consent.

Procedures

All participants underwent a comprehensive screening assessment to determine eligibility. This included blood pressure measurement, anthropometric measurements and blood analysis of total-cholesterol, low density lipoprotein cholesterol (LDL-c), HDL-c, triglycerides and glycated haemoglobin (HbA1c) obtained by (8-hour fasted) capillary point of care assessments. Participants had a 5-year absolute CVD risk score computed and were classified into Low (<10% absolute risk of CVD events over 5 years), Moderate (10-15%) or High (>15%) absolute cardiovascular disease risk (ACR) according to Framingham criteria (7). Similarly, 5-year risk for the development of T2DM was calculated using the AUSDRISK risk assessment tool, and participants were classified as Low (scoring ≤5), Intermediate (6-11) or High (≥12) (8).

Screened individuals completed health belief questionnaires consisting of items from the Health Behaviour-Specific Self-Efficacy scales (9) and Wallston Multidimensional Health Locus of Control Scale (10, 11).

Self-efficacy was assessed with 4 x 4 behavior-specific items rated on six-point Likert scales ranging from (1) ‘not at all true’ to (6) ‘exactly true’. Items were adapted from Schwarzer and Renner (9) and had been validated in many studies (12). In the present data, internal consistencies (Cronbach’s α) were .80 for nutrition, .82 for physical
activity, .87 for alcohol consumption, and .94 for smoking cessation self-efficacy. Scoring involved a composite score for each of the four behavioural domains, and then averaging the responses to all items pertaining to a behaviour. Scoring values ranged between 1 and 6. A higher score reflected a higher perceived self-efficacy for that behaviour.

Scoring for the Multidimensional Health Locus of Control Scale involved cumulative scores of 18 items, with values ranging from -3 to +3 per item. The scale was itemised based on the three domains (internal, powerful-others and chance) of six items each. For each item individuals indicated the extent to which they agreed or disagreed, with values ranging from 1 (strongly disagree) to 6 (strongly agree) per item. An updated discussion on the validity of the MHLC scale was published in 2005 (11). Participants were also asked to indicate their perceived CVD risk during their screening visit prior to receiving any results (according to a 5-point scale) “Compared with another person your own age and sex, how would you rate your risk of having a heart attack or stroke in the next 5 years?” to rate their responses whereby 1 indicated ‘Very Low Risk’ to 5 being ‘Very High Risk’.

Data Analyses

For self-efficacy, analysis was carried out separately for each health behaviour (i.e., physical activity, nutrition, alcohol consumption, and smoking cessation) resulting in a mean score for each. Similarly for locus of control, item analysis was carried out separately for each domain (i.e., internal, powerful others and chance) and item scores were summed for each domain. Due to very low numbers of participants indicating Very Low or Very High perceived CVD risk, scoring was recoded into a 3-point scale combining Very Low and Low, and High and Very High categories. Perceived CVD
risk was analysed as an ordinal categorical variable. Descriptive values are presented as mean (± standard deviation [SD]) for continuous variables or a proportion for categorical variables. Group differences were assessed using independent t-tests for normally distributed continuous variables. ANOVA was performed to assess differences in self-efficacy and locus of control between CVD and AUSDRISK groups. Correlations between perceived CVD risk and calculated CVD risk scores were assessed using a chi-squared test ($\chi^2$). For this, moderate and high CVD risk categories were merged due to few individuals classified as high risk. Logistic regression analysis was undertaken to predict the probability of CVD risk category (low vs. moderate or high). A probability value of $p< 0.05$ (two-tailed) was considered statistically significant for all tests. Data were analysed using SPSS v.19.

**Results**

*Screened Cohort*

Eight-hundred and fifty-three individuals were screened for study eligibility, and participant characteristics are displayed in Table 1. The average age was 56 ± 8 years, and 39% of those screened were men. Women had significantly lower HDL-c levels ($p < 0.001$), whereas men had greater waist circumference ($p < 0.001$) and systolic ($p < 0.001$) and diastolic ($p = 0.004$) blood pressure. There were no significant differences in behaviour specific self-efficacy between men and women except for alcohol consumption; women reported a higher self-efficacy for refraining from alcohol compared to men (4.73 v 4.22, $p < 0.001$). There were no significant gender differences across all domains of locus of control or CVD risk perception.

*Self-efficacy*
There were no statistically significant differences in absolute CVD risk for behaviour specific self-efficacies for nutrition, physical activity, alcohol consumption and smoking cessation with respect to CVD risk (Table 2). Similarly, there were no significant differences for nutrition, physical activity, alcohol consumption and smoking cessation self-efficacies with respect to diabetes risk.

*Locus of control*

Internal locus of control scores showed an inverse relationship with CVD and diabetes risk, but did not reach statistical significance (Figure 1a and 2a) for either CVD or T2DM risk. Contrastingly, external (powerful others and chance) scores had a positive relationship with both CVD and diabetes risk. Those with higher powerful others locus of control scores had a higher CVD (Figure 1b) \( (p = 0.025) \) and T2DM (Figure 2b) \( (p < 0.001) \) risk than those with a lower powerful others locus of control scores. Those with a higher chance locus of control also had a higher CVD (Figure 1c) \( (p < 0.001) \) (Figure 2c) risk, although this was not the case for T2DM.

*Risk perceptions*

An association between perceived and estimated calculated absolute CVD risk was observed, \( \chi^2 (2) = 11.37, p = 0.023 \). There were more individuals than expected who perceived their risk to be low who were assessed as being low risk for CVD. Conversely, of people who perceived their risk to be low, there were fewer than expected individuals who had a high calculated CVD. There were fewer individuals than expected who rated their CVD risk as moderate and were assessed as low calculated CVD risk, yet more than expected who had a high calculated CVD risk. A similar proportion of individuals reported a low (40.8%) or average (40.7%) perceived CVD risk.
Correlates of higher CVD risk classification

Independent correlates of CVD risk category were sex, age and level of education. Men were more likely to be in a moderate or higher than in a low CVD risk category, compared to women (OR 11.41, 95% CI 5.16 - 25.23). Individuals who were older (OR 1.17, 95% CI 1.11 – 1.23) and who had a lower level of education were also more likely to be in moderate to high risk CVD category (OR 2.39, 95% CI 1.23 – 4.65).

Discussion

This study was an investigation into the relationship between self-efficacy, locus of control, risk perception and cardio-metabolic risk. The hypothesis that there would be an inverse correlation between self-efficacy, locus of control and CVD and T2DM risk was not met with respect to self-efficacy, but was upheld for locus of control. We found that individuals with a higher external (powerful others and chance) locus of control had a higher risk for developing CVD and T2DM compared to those with lower calculated risk who had lower external locus of control scores. We did not find any association between self-efficacy and risk. People who perceived their CVD risk to be low were generally accurate, although people who perceived their CVD risk to be moderate or high over-generally over-estimated their risk. Increasing age, male gender and a lower level of education were found to be associated with CVD risk classification.

Those with a high risk for developing CVD and T2DM tended to have an external locus of control compared to lower risk individuals who showed a more internal locus of control. People with an external locus of control generally believe that their successes or failures result from external factors beyond their control such as luck, fate or circumstance. In contrast, people with an internal locus of control believe that they
are in control of their own lives and health outcomes. Generally, an internal locus of control has been associated with positive health outcomes (13). A follow-up of a large population-based cohort revealed that an internal locus of control is associated with a reduced risk of heart attack (14). Conversely, individuals with an external locus of control are more likely to develop coronary heart disease (15). The challenge presented to health professionals then is in changing the mindset of an individual with a higher external locus of control (to an internal locus of control) to positively influence health behaviour change or adaptation.

Whilst we did not identify any significant relationship between behaviour specific self-efficacy and CVD risk at baseline, it is important to note that self-efficacy is widely reported as a powerful predictor of future behaviour (5, 16). Self-efficacy has long been known for predicting behaviour change. It has been previously reported that self-efficacy could be used as a predictor of both smoking cessation and maintenance of cessation (17-19). Self-efficacy has also been shown to be a powerful predictor of other health behaviours such as physical activity (20), cardiac rehabilitation (21) diet and weight loss (22-24). For example, a 2014 study by Smit et al, found that individuals with a higher self-efficacy in regards to smoking have significantly lower drop-out rates (25) compared to those with a lower self-efficacy. Thus, self-efficacy may be a more useful marker when assessing health behaviour modification and subsequent change in cardio-metabolic disease risk from baseline to final study follow-up as part of the broader MODERN intervention trial. Interestingly, it has been suggested that self-efficacy may only be predictive for those who have a higher internal health locus of control (26) while individuals with an internal health locus of control have previously been associated with an unrealistic optimism.
concerning their own health risks (27). Our intervention follow-up may provide further indication of this.

The effectiveness of an intervention is extremely dependent on an individual’s willingness and adherence to prescribed behavioural or lifestyle modifications, which is typically poor (28). While a person’s perception of their own health risk can influence health behaviour change, barriers often present themselves. Whether an individual adopts a novel health behaviour or not can strongly depend on both their perception of, and their ability to do so. A person’s own health perception can sometimes be misleading (29). Although we showed an association between perceived and calculated CVD risk, we found an overwhelming number of participants were generally conservative with the risk estimates. Although it is not known why the majority of individuals perceived their own health to be worse than their calculated risk, the fact that most participants have made the conscious decision to participate in a lifestyle intervention may imply that they hold a negative perception of their risk. The impact of an individual’s health perception on intervention response, behaviour change and subsequent risk may be appropriately elucidated during our follow-up, particularly given poor health perception has been previously associated with both disease incidence and mortality (30-32).

Limitations

There are a number of limitations that require comment. As cohorts were self-selected from two regional communities, our overall findings should not be generalised without consideration of ethnic, socioeconomic and environmental factors pertaining to each community. The current cross-sectional data analyses pertained to the baseline characteristics of the MODERN study. Self-efficacy and health perceptions may be
useful tools for predicting behaviour change, and it is expected that the follow-up analyses of this cohort will provide further insight into the associations between self-efficacy, health perceptions, and intervention response.

Although a plethora of studies have revealed the association between self-efficacy, locus of control, and health behaviour change (5), and extensive research has revealed the impact of lifestyle interventions on behaviour modification, risk factor change and subsequent cardio-metabolic risk (33), it is not clear whether these interventions have a positive impact on health perceptions, self-efficacy or locus of control as putative mediators of behaviour change. Overall, with the exception of locus of control, CVD risk could not be predicted from beliefs about health, control or ability to execute health behaviours, or risk perceptions. Health perceptions and self-efficacy may be more informative predictors of interventional response as a means of reducing cardio-metabolic disease risk. Their role, if any, in the primary prevention and interventional efforts against cardio-metabolic diseases will be investigated as part of our longer-term follow up study.

Funding

The MODERN study is funded by a Project Grant (ID No. APP1069043) from the National Health and Medical Research Council of Australia who had no involvement in the design of the study and in writing the manuscript and played no role in the collection, analysis and interpretation of data derived from the study. MJC is supported by a Future Leader Fellowship (Award Reference 100,802) from the National Heart Foundation of Australia.

Conflicts of Interest
The authors declare that they have no conflicts of interest.

References


Sen, A. (2002). Health: perception versus observation: self reported morbidity has severe limitations and can be extremely misleading.


Table 1. Screened Population Risk, Beliefs, Perceptions and Behaviours

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Overall</th>
<th>Male</th>
<th>Female</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>853</td>
<td>333</td>
<td>520</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>56.35 ± 7.93</td>
<td>56.20 ± 9.05</td>
<td>56.45 ± 7.86</td>
<td>0.656</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.26 ± 5.84</td>
<td>28.97 ± 4.65</td>
<td>29.45 ± 6.49</td>
<td>0.246</td>
</tr>
<tr>
<td>Triglycerides (mmol)</td>
<td>1.70 ± 1.10</td>
<td>1.68 ± 1.02</td>
<td>1.72 ± 1.15</td>
<td>0.603</td>
</tr>
<tr>
<td>HDL-c (mmol)</td>
<td>1.65 ± 0.44</td>
<td>1.42 ± 0.38</td>
<td>1.80 ± 0.41</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HbA1c (mmol)</td>
<td>37.25 ± 6.04</td>
<td>37.42 ± 6.39</td>
<td>37.15 ± 5.81</td>
<td>0.524</td>
</tr>
<tr>
<td>Waist Circumference (cm)</td>
<td>98.01 ± 14.36</td>
<td>102.64 ±</td>
<td>95.05 ± 14.90</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic Blood Pressure (mmHg)</td>
<td>128.33 ±</td>
<td>131.43 ±</td>
<td>126.34 ± 16.04</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>15.70</td>
<td>14.66</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastolic Blood Pressure (mmHg)</td>
<td>77.31 ± 10.05</td>
<td>78.54 ±</td>
<td>76.53 ± 9.98</td>
<td>0.004</td>
</tr>
<tr>
<td></td>
<td>10.05</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-Efficacy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nutrition</td>
<td>3.93 ± 1.09</td>
<td>3.86 ± 1.12</td>
<td>3.97 ± 1.07</td>
<td>0.147</td>
</tr>
<tr>
<td>Physical Activity</td>
<td>3.71 ± 1.19</td>
<td>3.63 ± 1.25</td>
<td>3.76 ± 1.15</td>
<td>0.142</td>
</tr>
<tr>
<td>Smoking Cessation</td>
<td>4.20 ± 1.85</td>
<td>4.14 ± 1.91</td>
<td>4.24 ± 1.82</td>
<td>0.777</td>
</tr>
<tr>
<td>Alcohol Consumption</td>
<td>4.52 ± 1.32</td>
<td>4.22 ± 1.26</td>
<td>4.73 ± 1.32</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Locus of Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal</td>
<td>27.29 ± 4.13</td>
<td>27.35 ± 4.24</td>
<td>27.25 ± 4.06</td>
<td>0.711</td>
</tr>
<tr>
<td>Powerful Others</td>
<td>16.40 ± 5.15</td>
<td>17.89 ± 5.46</td>
<td>17.26 ± 5.77</td>
<td>0.113</td>
</tr>
<tr>
<td>Chance</td>
<td>17.50 ± 5.66</td>
<td>16.77 ± 4.98</td>
<td>16.17 ± 5.25</td>
<td>0.097</td>
</tr>
<tr>
<td>Cardiovascular Risk Perception</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>During Clinic Visit (1-3)</td>
<td>2.68 ± 0.93</td>
<td>2.61 ± 0.92</td>
<td>2.73 ± 0.94</td>
<td>0.086</td>
</tr>
<tr>
<td>Cardiovascular Disease Risk</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
ACR

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th>&lt;0.001</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.66±3.99</td>
<td>6.88±4.61</td>
<td>3.24±2.71</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Data are mean ± SD or n (%)

BP, Blood Pressure; BMI, body mass index; ACR, absolute cardiovascular risk; 1, Low; 2, Average; 3, High; HDL, high-density lipoprotein Cholesterol; HbA1c, glycated haemoglobin. Low Risk, ≤ 9%; Moderate Risk, 10-15%; High Risk ≥ 16%

The p-value pertains to the test of gender differences
Table 2. Self-Efficacy, Locus of Control and Risk Perception by CVD Risk Status

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Overall</th>
<th>Low Risk</th>
<th>Moderate</th>
<th>High Risk</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n = 853</td>
<td>754</td>
<td>79</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>56.35 ± 7.93</td>
<td>55.49 ± 7.81</td>
<td>62.54 ± 5.31</td>
<td>64.25 ± 5.82</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Men (%)</td>
<td>333 (39.0)</td>
<td>254 (33.7)</td>
<td>62 (78.5)</td>
<td>17 (85.0)</td>
<td></td>
</tr>
<tr>
<td>Behaviour specific Self-Efficacy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Activity</td>
<td>3.71 ± 1.19</td>
<td>3.71 ± 1.19</td>
<td>3.78 ± 1.25</td>
<td>3.55 ± 1.14</td>
<td>0.731</td>
</tr>
<tr>
<td>Nutrition</td>
<td>3.93 ± 1.09</td>
<td>3.92 ± 1.09</td>
<td>4.00 ± 1.13</td>
<td>3.88 ± 1.03</td>
<td>0.814</td>
</tr>
<tr>
<td>Smoking Cessation</td>
<td>4.20 ± 1.85</td>
<td>4.25 ± 1.88</td>
<td>3.89 ± 1.82</td>
<td>4.27 ± 1.64</td>
<td>0.768</td>
</tr>
<tr>
<td>Alcohol Consumption</td>
<td>4.52 ± 1.32</td>
<td>4.54 ± 1.31</td>
<td>4.38 ± 1.36</td>
<td>4.28 ± 1.43</td>
<td>0.534</td>
</tr>
<tr>
<td>Locus of Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Internal</td>
<td>27.29 ± 4.13</td>
<td>27.29 ± 4.07</td>
<td>27.49 ± 4.43</td>
<td>26.40 ± 5.42</td>
<td>0.576</td>
</tr>
<tr>
<td>Powerful Others</td>
<td>17.50 ± 5.66</td>
<td>17.32 ± 5.53</td>
<td>18.79 ± 6.44</td>
<td>19.50 ± 6.55</td>
<td>0.025</td>
</tr>
<tr>
<td>Chance</td>
<td>16.40 ± 5.15</td>
<td>16.23 ± 5.30</td>
<td>16.72 ± 5.30</td>
<td>21.70 ± 5.36</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CVD Risk Perception</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>During Clinic Visit (1-3)</td>
<td>1.78 ± 0.74</td>
<td>1.74 ± 0.74</td>
<td>2.03 ± 0.72</td>
<td>2.15 ± 0.59</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Risk Factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triglycerides (mmol/L)</td>
<td>1.70 ± 1.10</td>
<td>1.65 ± 1.10</td>
<td>2.04 ± 1.10</td>
<td>2.12 ± 1.13</td>
<td>0.003</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>1.65 ± 0.44</td>
<td>1.70 ± 0.43</td>
<td>1.31 ± 0.31</td>
<td>1.18 ± 0.20</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HbA1c (mmol)</td>
<td>37.25 ± 6.04</td>
<td>36.84 ± 5.50</td>
<td>39.90 ± 8.71</td>
<td>42.35 ± 8.06</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>98.01 ± 14.36</td>
<td>97.29 ± 14.49</td>
<td>103.39 ±</td>
<td>104.12 ±</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>12.07</td>
<td>12.21</td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>128.33 ±</td>
<td>126.04 ±</td>
<td>142.59 ±</td>
<td>158.38 ±</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>77.31 ±</td>
<td>76.42 ± 9.44</td>
<td>82.61 ± 8.14</td>
<td>90.28 ± 19.87</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>---------------------</td>
<td>----------</td>
<td>--------------</td>
<td>--------------</td>
<td>---------------</td>
<td>--------</td>
</tr>
<tr>
<td></td>
<td>10.048</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Data are mean ± SD or n (%)

BP, blood pressure; CVD, cardiovascular disease; 1, Low; 2, Average; 3, High; HDL, high-density lipoprotein Cholesterol; HbA1c, glycated haemoglobin.

Low Risk, ≤ 9%; Moderate Risk, 10-15%; High Risk ≥ 16%.

The p-value pertains to the test of risk group differences
Figure 1.
Figure 2.

a. Internal

b. Powerful Others

c. Chance
Figure 1. Relationship between Locus of Control and Cardiovascular Disease Risk (a. Internal: Low (27.29, 95% CI [27.00, 27.58]); Moderate (27.49, 95% CI [26.49, 28.49]); High (26.40, 95% CI [23.86, 28.94]; b. Powerful Others: Low (17.32, 95% CI [16.92, 17.71]; Moderate (18.79, 95% CI [17.34, 20.25]; High (19.50, 95% CI [16.43, 22.57]; c. Chance: Low (16.23, 95% CI [15.87, 16.59]; Moderate (16.72, 95% CI [15.52, 17.91]; High (21.70, 95% CI [19.19, 24.21])

Figure 2. Relationship between Locus of Control and Diabetes Risk (a. Internal: Low (27.88, 95% CI [26.88, 28.88]); Intermediate (27.50, 95% CI [27.03, 27.98]; High (27.10, 95% CI [26.72, 27.48]; b. Powerful Others: Low (15.71, 95% CI [14.32, 17.10]; Intermediate (16.46, 95% CI [15.84, 17.09]; High (18.11, 95% CI [17.60, 18.62]; c. Chance: Low (16.51, 95% CI [15.32, 17.70]; Intermediate (15.83, 95% CI [15.22, 16.44]; High (16.75, 95% CI [16.29, 17.20])
Summary

Chapter 5 presented findings of screening analyses where we assessed behaviour specific self-efficacy, health locus of control and risk perceptions as modulators for developing CVD and/or T2DM. As anticipated, individuals at high risk for cardiometabolic disease showed an external health locus of control (higher powerful others and chance), and individuals at lower risk showed higher internal health locus of control scores, supporting that people who believe they are in control of their health are more likely to have better health outcomes.

Lack of a relationship between self-efficacy and cardiometabolic risk was not expected however it is likely explained by a high rating of self-efficacy for health behaviours across-the-board at baseline, with no disparities between low and high cardio-metabolic risk profiles.

It is unknown whether the MODERN intervention will positively impact upon self-efficacy and health locus of control to have (more) favourable changes on cardiometabolic risk.
Chapter 6

Self-efficacy, Health Locus of Control and Cardio-metabolic Disease Risk: Nurse-led Intervention
Chapter 6 presents the 12-month follow-up results to determine the effect of nurse-led lifestyle intervention on health behaviour change, behaviour-specific self-efficacy, health locus of control, cardio-metabolic risk.

This Chapter includes the following submitted manuscript:

Preface

Chapter 6 reports the mid-study intervention effect of behaviour-specific self-efficacy and health locus of control as potential determinants of health behaviour change.

Research has shown self-efficacy and health locus of control to be malleable, with interventions typically exuding a positive effect. Enhancing self-efficacy for a particular behaviour typically improves adherence, and internalising health locus of control likewise may lead to an individual taking more control of their own behaviour.

Chapter 6 divulges findings from the interim 12-month visits as part of the MODERN study. The submitted manuscript herein reports the effects of nurse-led intervention on behaviour specific self-efficacy, health locus of control, health behaviours and cardio-metabolic disease risk and seeks to further evaluate the potential influence of these psychological constructs on cardio-metabolic risk factors.
Determinants of cardiovascular risk factors and health behaviour change after 12 months in the ‘Management to Optimise Diabetes and mEtabolic syndrome risk Reduction via Nurse-led intervention (MODERN)’ randomised controlled trial

Andre L. Rodrigues \(^1, 2\) | andre.rodrigues@baker.edu.au
Ralf Schwarzer \(^3, 5\) | ralf.schwarzer@fu-berlin.de
Chantal Ski \(^4\) | c.ski@qub.ac.uk
Melinda J. Carrington \(^1, 2\) | melinda.carrington@baker.edu.au

\(^1\) Pre-Clinical Disease and Prevention Unit, Baker Heart and Diabetes Institute, Victoria, Australia
\(^2\) Dept. of Epidemiology and Preventive Medicine, Monash University, Victoria, Australia
\(^3\) Dept. of Psychology, Freie Universität Berlin, Berlin, Germany
\(^4\) School of Nursing and Midwifery, Queen’s University Belfast, Belfast, Northern Ireland, UK
\(^5\) Dept. of Clinical, Health, and Rehabilitation Psychology, SWPS University of Social Sciences and Humanities, Wroclaw, Poland

**Corresponding Author:**

Melinda Carrington, PO Box 6492, Melbourne, VIC 3004, Australia

*Telephone: +61 3 8532 1749; Fax: +61 3 8532 1100;*

*Email: melinda.carrington@baker.edu.au*
Enabling positive health behaviour modification is fundamental for the prevention of cardio-metabolic diseases. Self-efficacy and health locus of control are important factors for behaviour change and intervention response. Their ability to be modified and contribute to improved cardio-metabolic disease risk is not clear.

Two hundred and seventy-six adults aged 40-70 years with metabolic syndrome were enrolled into either nurse-led intervention or usual care as part of the MODERN randomised controlled trial. Twelve-month interventional effects on behaviour-specific self-efficacy, health locus of control and their association with health behaviours and cardio-metabolic disease risk were investigated.

Both groups showed improvements over 12 months in cardio-metabolic risk factors which were exacerbated within the intervention group compared to usual care. Intervention participants had a greater reduction in systolic blood pressure (-7.6 ± 14.6 v -1.7 ± 13.9, p < 0.001), waist circumference (-2.8 ± 5.4 v -1.5 ± 5.0, p = 0.046) and cardiovascular disease risk (-1.5 ± 2.4 v -0.6 ± 2.6, p = 0.002) compared to usual care. Both groups showed improvements in diet but not physical activity and reductions in behaviour specific self-efficacy. Powerful others health locus of control increased in the usual care group only. In adjusted analyses, sex, baseline level of risk and an internal health locus of control were consistently found to influence 12 month cardio-metabolic risk values. Our findings show that nurse-led health and lifestyle
modification can reduce cardio-metabolic disease risk and the potential for participants to initially overestimate their self-efficacy for key health behaviours.
INTRODUCTION

Cardiovascular disease (CVD) and type 2 diabetes (T2DM) are largely preventable cardio-metabolic diseases yet they remain a major global health concern with alarming mortality rates\(^1\). The first step in managing cardio-metabolic risk factors is lifestyle change through modification of key health behaviours; increasing physical activity, reducing sedentary behaviour, improving diet, reducing alcohol consumption and smoking cessation have been widely reported as effective\(^2\). Compared to metropolitan locations, there is an incremental need for risk factor management to address greater cardio-metabolic disease burden in regional, rural and remote areas\(^3\). Additionally, achievement of ideal risk factor levels to remain healthy can depend on the strength of one’s belief in being able to succeed in adopting positive behaviour changes (self-efficacy\(^4\)) as well as the perceived amount of control one has over their health (health locus of control\(^5\)). An individual with a high self-efficacy for a health behaviour, may be more likely to adopt the particular novel behaviour\(^4\). An internal locus of control has been associated with a reduced risk for common chronic diseases\(^5\) as these people are more adaptive and likely to engage in health-enhancing behaviours\(^6\). People with a high self-efficacy for a health behaviour are likely to have an internal health locus of control and may be better equipped to succeed in adopting the behaviour. These psychological constructs can influence positive health behaviours, however less is known in regard to their potential influence in reducing cardio-metabolic disease risk.

This research aimed to investigate the impact of a change in behaviour-specific self-efficacy and health locus of control via a nurse-led lifestyle intervention targeting cardio-metabolic disease risk reduction. We hypothesised that over a 12-month period, improvements in cardio-metabolic disease risk would be greater in the intervention group and be associated with a higher self-efficacy for key health behaviours and an
internal health locus of control. We tested this hypothesis by analysing mid-study results after 12 months within the Management to Optimise Diabetes and mEtabolic syndrome risk reduction via the Nurse-led intervention (MODERN) Study⁸.

METHODS

Setting and Study Design

MODERN is a 24-month, multi-centre, randomised controlled trial of a nurse-led intervention to improve cardio-metabolic health. The full protocol and design has been described previously⁸. Briefly, two regional clinics were established and individuals from the community were screened by registered nurses for eligibility into the study. Eligible participants with metabolic syndrome (MetS) were randomised to receive either the nurse-led lifestyle intervention or usual care (control group). MODERN is registered with the Australian New Zealand Clinical Trials Registry (ACTRN12616000229471) and the study protocol approved by the Australian Catholic University (ACU) Human Research Ethics committee (2014 244V). Written informed consent was provided by all participants.

Participants

Of 853 participants screened, 276 were randomised into the MODERN trial. Inclusion criteria were residents of Shepparton or Colac, aged between 40 and 70 years with any three or more measured risk factors for MetS (hypertension [≥130/85 mmHg], elevated triglycerides [≥ 1.7mmol/L], elevated glucose [glycated haemoglobin/HbA1c, ≥ 5.7% or 39mmol/L], reduced high-density lipoprotein cholesterol [HDL-C, <1.03 mmol/L for men, <1.29 mmol/L for women] and central obesity (elevated waist circumference [≥94 cm for men, ≥80 cm for women] and no previously diagnosed
CVD. Participants were excluded if they had two or less identifiable risk factors for MetS, had any diagnosed form of CVD or kidney disease.

**Procedures**

Participants were randomised on a 1:1 ratio to receive a nurse-led intervention of a multidisciplinary health behaviour modification program or usual care. The intervention was applied by nurses on an individual basis, acting independently of healthcare professionals and within the scope of their trained responsibilities. The intervention aimed to deliver coaching and support to individuals to change health behaviours and optimise risk factor management. Briefly, participants received (1) tailored risk factor management advice; (2) health education on CVD and T2DM; (3) motivational interviewing to facilitate risk factor improvement; and (4) routine follow-up visits commensurate with the level of risk.

Usual care consisted of a screening assessment with brief health education and advice. Usual care participants received management by their usual health care providers.

**Data Collection**

Baseline assessments were conducted and repeated at 12 months (mid) study follow-up. We collected demographic information (age, sex and education level) and lifestyle data including diet, alcohol intake, physical activity and smoking. Diet was assessed using the Dietary Questionnaire for Epidemiological Studies (DQES v2)\(^9\) and daily consumption data for total kilojoules, fat, carbohydrates, cholesterol, protein and sodium were analysed. The Australian Recommended Food Score and fruit and
vegetable consumption indexes were also computed. For physical activity, GTX3 accelerometers [ActiGraph, FL, USA] were used for 7-day continuous quantitative measurement and output variables of interest included energy expenditure, total steps and time spent undertaking moderate, moderate-to-vigorous or vigorous physical activity. Medical history, family history of CVD and diabetes and data on medications were also collected. Anthropometric and sitting blood pressure (BP) (with an automated BP monitor [Omron HEM-907, Omron Healthcare Co. Ltd, Kyoto, Japan]) were measured. Fasting capillary/fingerstick whole blood tests were taken of total (TC), LDL-C (low-density lipoprotein cholesterol and HDL-C, HbA1C and triglycerides with a point of care analyser (Afinion™ AS100, Alere, MA, USA). Cardiovascular 5-year risk was calculated via the Framingham Risk Equation\textsuperscript{10} based on sex, age, smoking history, diabetes status, systolic blood pressure (SBP) and TC:HDL ratio. T2DM 5-year risk was also calculated via the 10-item AUSDRISK prediction model\textsuperscript{11} based on sex, age, country of birth, history of high blood glucose, treatment for high BP, smoking history, family history of diabetes, vegetable and fruit consumption, waist circumference and physical activity levels. Risk was classified into; Low (<10% risk of events in the next 5 years), Moderate (10-15%) and High (>15%) for CVD and similarly, Low (scoring <6), Intermediate (6-11) or High (>11) for T2DM.

Participants completed the Health Behaviour-Specific Self-Efficacy scale\textsuperscript{12} for four key health behaviours: nutrition, physical activity, alcohol consumption and smoking cessation. A composite score for each behavioural domain was ascertained by averaging the responses to all items. Values ranged from 1 to 6; the higher the score, the higher the perceived self-efficacy for the concerned behaviour. The 18-item Wallston Multidimensional Health Locus of Control scale\textsuperscript{13} was classified by three
domains (internal, chance and powerful others) each consisting of six items. For each item, participants indicated the extent to which they agreed or disagreed, from 1 (strongly disagree) to 6 (strongly agree). Scoring was calculated using the cumulative scores of 18 items, with scores ranging from -3 to +3 per item.

**Statistical Analyses**

Data from both clinics were pooled and summarised. For self-efficacy, analyses were carried out separately for each health behaviour (i.e. nutrition, physical activity and alcohol consumption) from which a mean score was derived. Similarly, for locus of control, item analysis was carried out separately for each classification and scores summed. For CVD and AUSDRISK categorical variables, moderate and high CVD risk categories, and low and intermediate diabetes risk categories were combined due to few individuals classified as high risk and low risk respectively.

Descriptive statistics were used to summarise the demographic, clinical and health profile of the sample at baseline and 12 months follow-up. For discrete variables, frequencies, percentages and group differences were assessed via χ² tests with 95% confidence intervals. For continuous variables, means with standard deviations were reported and paired sample t-tests were used to assess within group change over time, and between group comparisons were made using analysis of variance (ANOVA). Multiple backwards stepwise regression analysis was used to identify determinants of 12 month cardio-metabolic risk status using BP, waist circumference, total cholesterol, HDL-c, triglycerides and CVD and T2DM risks scores. As independent variables we entered age, sex, education level (secondary school/ TAFE/trade or degree/diploma/graduate certificate), group (intervention or usual care), change from baseline to 12 months in physical activity (energy expenditure, steps, moderate,
vigorous and moderate- to-vigorous activity), diet (fat, carbohydrates, protein, sodium, cholesterol, fruit and vegetable consumption index and the overall Australian Recommended Food Score), behaviour specific self-efficacy (nutrition and physical activity) and health locus of control (internal, chance and powerful others domains). Each model contained the baseline value of the dependent variable to account for change over time. Cut off levels for entry of variables was $p=0.05$ and for removal $p=0.10$. Statistical significance was interpreted as two-sided at the level $\alpha=0.05$. Data were analysed using SPSS Statistics 21.0 (SPSS Inc., Chicago, Illinois, USA).
RESULTS

Of 276 individuals randomised to the MODERN study, 136 entered into the nurse-led intervention and 140 received usual care. At baseline, 35% and 42% of participants in the intervention and usual care groups, respectively, were male (38% overall). Mean age was 57.4 ± 7.7 years, with no significant group or gender differences. A similar number of participants randomised to usual care and the nurse-led intervention were at moderate-to-high risk of CVD (22% v 24%, p = 0.910). Individuals randomised to usual care had a larger waist circumference (105.8cm ± 12.3 cm v 102.6cm ± 13.0 cm, p = 0.035) at baseline compared to those randomised to the intervention group. There were no significant group differences for BP, TC, HDL-C, triglycerides, HbA1c and overall CVD or T2DM risk measures at baseline. Table 1 represents baseline and 12-month characteristics, by treatment group and between groups.

12-month changes in cardio-metabolic risk and health behaviours

At 12 months, 248 (90%) participants completed follow-up assessments (122 usual care, 126 intervention). Figure 1 shows decreases in all variables in both groups from baseline to 12 months. Within the intervention group, there were significant differences over time for all variables except smoking self-efficacy and health locus of control parameters (Table 1). For the usual care group, only waist circumference, HbA1c, CVD and T2DM risk were significantly reduced in addition to alcohol self-efficacy and powerful others locus of control. Between groups, individuals in the nurse-led intervention group had a significantly greater reduction in SBP, waist circumference and CVD risk over 12 months compared to usual care (Table 1). There were no other statistically significant differences between groups for any other risk factor.
Figure 1. Change in Risk Factors from baseline to 12 months.
Health Behaviour Change

There were no statistically significant changes from baseline to 12 months concerning smoking status or alcohol consumption for either group. Within the intervention group, dietary data revealed a reduction in daily kilojoules (-1028 kJ ± 295kJ, \( p = 0.001 \)), fat (-11.0g ± 3.2g, \( p = 0.001 \)), carbohydrate (-23.3g ± 6.7g, \( p = 0.001 \)), cholesterol (-27.1mg ± 13.2mg, \( p = 0.042 \)), sodium (-340mg ± 117mg, \( p = 0.004 \)) and protein (-9.0g ± 4.4g, \( p = 0.042 \)) intake. There was also an increase (representing better nutrient adequacy of usual dietary intake) in the overall Australian Recommended Food Score (1.7 ± 0.6, \( p = 0.005 \)) and Vegetable Consumption Index (2.9 ± 1.3, \( p = 0.032 \)) after 12 months. For the usual care group, participants consumed less kilojoules (-610.0 kJ ± 254.4kJ, \( p = 0.018 \)), carbohydrates (-18.6g ± 6.3g, \( p = 0.004 \)) and sodium (-185.4mg ± 92.0mg, \( p = 0.046 \)) per day at 12 months compared to baseline. There were no statistically significant differences between groups in dietary data.

For physical activity, participants within the intervention group reduced their average daily energy expenditure (311 kJ ± 241kJ v 275 kJ ± 176kJ, \( p = 0.002 \)) and decreased their average daily steps (7161 ± 2709 v 6233 ± 2236, \( p < 0.001 \)) at 12 months compared to baseline. Similar reductions in average daily steps at 12 months were also seen within the usual care group (6938 ± 2466 v 6167 ± 2200, \( p < 0.001 \)). There were no differences in changes in moderate, vigorous or moderate-to-vigorous activity levels within or between groups.

Behaviour Specific Self-Efficacy and Health Locus of Control
As shown in Table 1, there was a significant reduction in nutrition, physical activity and alcohol self-efficacies within the intervention group. In the usual care group only alcohol self-efficacy was significantly lower after 12 months.

There were no statistically significant differences between treatment groups for any behaviour specific self-efficacies. Powerful others locus of control significantly increased in the control group from baseline to 12 months yet there were no other significant changes in health locus of control after 12 months.

**Predictors of Change**

Table 2 outlines the multiple linear regression models and independent correlates of various cardio-metabolic risk factors at 12 months. After adjustment for potential confounders, sex, baseline levels of risk and an internal health locus of control were consistently found to predict 12 month cardio-metabolic risk values. Overall, the variables analysed predicted between 40% and 88% of the variance in risk factors when including baseline variables. The predicted SBP, absolute CVD risk and T2DM risk scores were lower for females whereas diastolic blood pressure (DBP), TC, HDL-c and triglyceride were higher for females, compared to males. Higher baseline risk values increased 12 month risk levels, on average. Greater improvements (from baseline to 12 months) in internal health locus of control were expected to increase BP and ACVD but decrease HDL-c and T2DM risk scores. Compared to usual care participants, intervention group participants were expected to have a lower waist circumference, SBP and overall ACVD risk score after 12 months of participation, but there were no group effects for any other risk factor. Few other physical activity and dietary data were predictive of waist circumference and lipid variables. Greater fruit consumption was expected to decreased waist circumference yet greater increases in
vigorously, larger increases in dietary cholesterol was expected to increase blood TC levels whereas greater reductions in dietary fat was expected to reduce HDL-c. Greater increases in the Australian Recommended Food Score were expected to increase T2DM risk. Improvements in nutrition self-efficacy were expected to decrease triglycerides and increase CVD risk scores.

**DISCUSSION**

This research was an investigation into the interim effects of a nurse-led lifestyle intervention to reduce cardio-metabolic risk and the likely determinants of health behaviour change. It was hypothesised that over a 12-month period, improvements in cardio-metabolic disease risk would be greater in the intervention group and associated with a higher self-efficacy for key health behaviours and an internal health locus of control. We found that concerning self-efficacy and health locus of control, our hypotheses were not met, however modifications in health behaviours were found with significant benefits for cardio-metabolic health.

Our most notable findings were improvements in key cardio-metabolic risk factors which were exacerbated within the intervention group compared to usual care. Most notably, SBP and waist circumference decreased by 8 mmHg and 3 cm respectively, with concomitant decreases in overall CVD risk also observed. Amidst modest changes in physical activity, more substantial dietary changes, indicated by less total energy (kJ), carbohydrates and sodium consumed for all participants and less fat, cholesterol and protein consumed for intervention participants, appeared to be more influential on cardio-metabolic risk factors at 12 months. Self-efficacy for key health behaviours were reduced at 12 months in intervention participants while there were no
notable changes in health locus of control, aside from an increase in powerful others locus of control within the usual care group. In general, better cardio-metabolic risk levels at 12 months were anticipated for females and individuals with lower baseline risk levels, with some benefit of being randomised to the nurse-led intervention – as shown in adjusted analyses by lower expected waist circumference, SBP and overall CVD risk compared to the usual care group. There were some anomalous findings in identifying: 1) improvements in internal health locus of control and higher BP, CVD risk and lower HDL-c; 2) enhanced nutrition self-efficacy and higher CVD risk and 3) increased (vigorous) physical activity and higher waist circumference.

Improving key risk factors is the basis for the observed reductions in overall cardio-metabolic disease risk. Waist circumference\(^{14-15}\), BP\(^{16-17}\), lipids\(^{18}\) and glucose\(^{19}\) levels are all vital health determinants and are hence routinely targeted as part of effective lifestyle interventions aimed at reducing cardio-metabolic disease risk\(^{20}\). In the historical Finnish Diabetes Prevention Study, there was a 58% reduction in the incidence of diabetes with lifestyle intervention\(^{21}\). Further, in a systematic review and meta-analysis of interventions for reducing diabetes and CVD risk in people with MetS, lifestyle, most notably the combination of diet and exercise, had the largest likelihood of being the best intervention\(^{22}\). It follows that nurse-led interventions that embed health and lifestyle modification are beneficial in managing cardio-metabolic disease risk and preventing recurrent hospitalisation in primary and secondary care contexts\(^{23-27}\). As part of our nurse-led intervention, individuals were provided coaching with education about cardio-metabolic diseases, care planning and routine follow-up in order to help achieve health behaviour change for improved risk factor management. Most obviously, our nurse-led intervention elicited some positive dietary behaviour changes, although it may have been more difficult to change physical
activity habits of at risk individuals, as noted by others that have not been as successful in impacting behaviour change\textsuperscript{28} or attenuating risk\textsuperscript{29-31}. Maintaining positive health behaviour changes beyond the short-term represents a challenge\textsuperscript{20} and seasonal affects may have impacted on adherence to recommended physical activity\textsuperscript{32}.

Self-efficacy for key health behaviours (nutrition, physical activity and alcohol) were all unexpectedly reduced after 12 months. Although self-efficacy has been shown to be malleable and often enhanced through behaviour interventions\textsuperscript{33}, it may not always be the case\textsuperscript{34}. Our findings are commensurate with a review of interventions that aimed to modify self-efficacy whereby 11 out of 25 studies were associated with a reduction over time\textsuperscript{35}. One explanation for this may be a self-reporting overestimation at baseline of one’s self-efficacy for certain health behaviours. Initially, participants may not have had the previous experience required to accurately postulate their self-efficacy for certain health behaviours and subsequently over-estimated their capacity. After 12 months of engaging in a lifestyle intervention however, participants’ re-evaluation of the difficulties or barriers associated with changing or adopting certain health behaviours may have led to a more well-informed assessment of their capabilities. McAuley et al. supported this hypothesis in finding that older adults seemed to overestimate physical activity self-efficacy prior to commencing a physical activity intervention program, but afterwards, participants’ self-efficacy for physical activity reduced in just 3 weeks\textsuperscript{36}. It has been suggested that inactive older adults may not have enough relevant experience to accurately assess their self-efficacy for certain health behaviours\textsuperscript{37}. Self-efficacy may hence be considered strongest at the beginning of an intervention when the behavior is novel, however with time and waning of nurse
contact\textsuperscript{38}, barriers have the ability to exacerbate the perceived difficulty associated with engaging in the behavior, thereby reducing self-efficacy\textsuperscript{39}.

Likewise for health locus of control which has also been shown to be modifiable\textsuperscript{40-44}, our results showed minimal interventional effects. Powerful others health locus of control may have increased in the usual care group due to regular contact with their health professional outside of the intervention. After adjusting for potential confounders, some counterproductive findings suggested that greater increases in internal health locus of control was indicative of higher SBP and DBP and CVD risk scores (but lower HDL-c and T2DM risk scores) at 12 months. Despite that an internal health locus of control has been associated with reduced chronic disease risk\textsuperscript{5}, health behaviours are not always explicitly associated with either internal or external locus of control\textsuperscript{4}; an individual with a strong internal locus of control may be generally more likely to engage in health behaviours but a person with a low internal locus of control or high powerful others locus of control may be more likely to comply with behaviour modifications prescribed by a medical practitioner. The effects of this at the completion of the MODERN trial will be assessed.

Albeit nurse intervention was not able to boost self-efficacy for key health behaviours or internal health locus of control, our other observed influences on 12 month cardiovascular risk reconfirm the gender disparities of an overall better cardio-metabolic risk profile for women than men and higher baseline values, that require larger and a faster rate of change, as important independent risk factors expected to increase cardio-metabolic risk. The clinical value of the nurse-led intervention and improvements in diet and physical activity for prevention of chronic disease remains
obvious. Taken together, the effect of nurse-led lifestyle intervention on positive health behaviour change should have notable longer term individual and public health benefit.

Limitations

Favourable changes in risk factors and health behaviours observed in the control group may have arisen as a result of being involved in research via the well-known Hawthorne effect\textsuperscript{45}. The intervention was designed as a nurse-led, multi-faceted, tailored, lifestyle intervention with the principal aim of targeting risk factor modification to reduce cardio-metabolic disease risk, and was not intended to specifically modify health locus of control or self-efficacy. An alternate health professional, such as a psychologist, might achieve this more effectively. This was a self-selected sample from two regional populations such that overall findings may not be generalised. Furthermore, this manuscript reports mid-study findings pertaining to a 24-month randomised control trial, as such, it is expected that the 24-month follow-up analyses of this cohort will provide further insight into the associations between self-efficacy, locus of control, interventional response and subsequent cardio-metabolic disease risk.

Conclusions

Our findings predicate the potential for a nurse-led intervention to reduce cardio-metabolic disease risk through risk factor modification and changing (predominantly dietary) health behaviours. Nurse management of higher risk individuals with multiple risk factors does not appear to rely on modification of health beliefs (i.e. self-efficacy or health locus of control) which may have been overestimated at the beginning of the
trial, but then normalised thereafter. Extended follow-up to 24 months will confirm the benefit of nurse-led clinics in preventing cardio-metabolic disease and the longer term interventional effects on self-efficacy, health locus of control and their relationship with cardio-metabolic risk.

**Funding**

The MODERN study is funded by a Project Grant (ID No. APP1069043) from the National Health and Medical Research Council of Australia who had no involvement in the design of the study and in writing the manuscript and played no role in the collection, analysis and interpretation of data derived from the study. MJC is supported by a Future Leader Fellowship (Award Reference 100,802) from the National Heart Foundation of Australia.

**Conflicts of Interest**

The authors declare that they have no conflicts of interest.

**References**

4. Tong, B., Cardiovascular medicines and primary health care: a regional


Carrington, Melinda J., and Paul Zimmet. "Nurse health and lifestyle modification versus standard care in 40 to 70 year old regional adults: study protocol of the Management to Optimise Diabetes and mEtabolic syndrome Risk reduction via Nurse-led intervention (MODERN) randomized controlled trial." BMC health services research 17.1 (2017): 813


"Development of the multidimensional health locus of control (MHLC) scales."  


Seo, Dong-Chul, Siyoung Choe, and Mohammad R. Torabi. "Is waist circumference $\geq 102/88$ cm better than body mass index $\geq 30$ to predict hypertension and diabetes development regardless of gender, age group, and race/ethnicity? Meta-analysis." *Preventive medicine* 97 (2017): 100-108.


Wu, Yu-Tzu, et al. "Weather, day length and physical activity in older adults:


40 Brousseau, Kenneth R., and Mark A. Mallinger. "Internal-external locus of


Table 1. Baseline and 12-month results for Usual Care and Intervention groups

<table>
<thead>
<tr>
<th>Demographics</th>
<th>n</th>
<th>Baseline</th>
<th>12 Month</th>
<th>p</th>
<th>n</th>
<th>Baseline</th>
<th>12 Month</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>122</td>
<td>57.53 ± 7.04</td>
<td>58.67 ± 6.93</td>
<td>&lt;0.001***</td>
<td>126</td>
<td>58.07 ± 7.93</td>
<td>59.09 ± 7.94</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>Biomedical</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>122</td>
<td>135.64 ± 13.12</td>
<td>133.96 ± 13.85</td>
<td>0.183</td>
<td>126</td>
<td>137.15 ± 17.00</td>
<td>129.51 ± 13.70</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>122</td>
<td>82.11 ± 9.15</td>
<td>81.26 ± 11.05</td>
<td>0.276</td>
<td>126</td>
<td>81.08 ± 9.19</td>
<td>78.51 ± 9.14</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>122</td>
<td>105.85 ± 12.45</td>
<td>104.34 ± 11.93</td>
<td>0.001**</td>
<td>126</td>
<td>102.01 ± 12.75</td>
<td>99.17 ± 13.23</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>122</td>
<td>5.57 ± 1.10</td>
<td>5.47 ± 1.10</td>
<td>0.123</td>
<td>126</td>
<td>5.68 ± 1.10</td>
<td>5.47 ± 1.02</td>
<td>0.018*</td>
</tr>
<tr>
<td>HDL-c (mmol)</td>
<td>122</td>
<td>1.38 ± 0.38</td>
<td>1.36 ± 0.36</td>
<td>0.386</td>
<td>126</td>
<td>1.38 ± 0.39</td>
<td>1.37 ± 0.36</td>
<td>0.526</td>
</tr>
<tr>
<td>HbA1c (mmol)</td>
<td>122</td>
<td>5.85 ± 0.57</td>
<td>5.66 ± 0.71</td>
<td>&lt;0.001***</td>
<td>126</td>
<td>5.76 ± 0.46</td>
<td>5.59 ± 0.56</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>122</td>
<td>1.68 ± 0.80</td>
<td>1.62 ± 0.78</td>
<td>0.288</td>
<td>126</td>
<td>1.86 ± 1.01</td>
<td>1.68 ± 0.94</td>
<td>0.003**</td>
</tr>
<tr>
<td>Risk</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVD Risk</td>
<td>122</td>
<td>6.77 ± 4.90</td>
<td>6.26 ± 4.59</td>
<td>0.030*</td>
<td>125</td>
<td>6.62 ± 4.85</td>
<td>5.14 ± 3.59</td>
<td>&lt;0.001***</td>
</tr>
<tr>
<td>T2DM Risk</td>
<td>112</td>
<td>17.33 ± 5.13</td>
<td>14.60 ± 5.27</td>
<td>&lt;0.001***</td>
<td>117</td>
<td>16.94 ± 4.88</td>
<td>14.55 ± 5.26</td>
<td>&lt;0.001***</td>
</tr>
</tbody>
</table>
### Self-Efficacy

<table>
<thead>
<tr>
<th></th>
<th>Mean ± SD</th>
<th>T-Stat</th>
<th>P-value</th>
<th>Mean ± SD</th>
<th>T-Stat</th>
<th>P-value</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutrition</td>
<td>4.04 ± 0.94</td>
<td>3.91 ± 0.93</td>
<td>0.114</td>
<td>114</td>
<td>4.07 ± 1.06</td>
<td>3.72 ± 1.02</td>
<td><strong>0.001</strong></td>
</tr>
<tr>
<td>Physical Activity</td>
<td>3.78 ± 1.17</td>
<td>3.63 ± 1.13</td>
<td>0.215</td>
<td>113</td>
<td>3.85 ± 1.10</td>
<td>3.56 ± 1.04</td>
<td><em>0.014</em></td>
</tr>
<tr>
<td>Alcohol</td>
<td>4.72 ± 1.30</td>
<td>4.25 ± 1.45</td>
<td>&lt;0.001***</td>
<td>91</td>
<td>4.64 ± 1.26</td>
<td>4.32 ± 1.53</td>
<td><em>0.026</em></td>
</tr>
<tr>
<td>Smoking</td>
<td>4.48 ± 1.66</td>
<td>4.08 ± 1.45</td>
<td>0.113</td>
<td>14</td>
<td>3.95 ± 1.89</td>
<td>4.07 ± 1.74</td>
<td>0.828</td>
</tr>
</tbody>
</table>

### Locus of Control

<table>
<thead>
<tr>
<th></th>
<th>Mean ± SD</th>
<th>T-Stat</th>
<th>P-value</th>
<th>Mean ± SD</th>
<th>T-Stat</th>
<th>P-value</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internal</td>
<td>27.18 ± 4.38</td>
<td>26.52 ± 4.27</td>
<td>0.125</td>
<td>126</td>
<td>26.89 ± 4.43</td>
<td>27.08 ± 4.64</td>
<td>0.663</td>
</tr>
<tr>
<td>Chance</td>
<td>16.98 ± 4.88</td>
<td>16.70 ± 5.27</td>
<td>0.514</td>
<td>126</td>
<td>16.00 ± 5.55</td>
<td>16.70 ± 5.84</td>
<td>0.185</td>
</tr>
<tr>
<td>Powerful Others</td>
<td>17.67 ± 5.72</td>
<td>18.48 ± 5.73</td>
<td>0.036*</td>
<td>126</td>
<td>18.06 ± 5.90</td>
<td>18.35 ± 5.93</td>
<td>0.510</td>
</tr>
</tbody>
</table>
Table 2. Multiple linear regression models of significant predictors of cardiometabolic risk factors at 12 months

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unstandardised</th>
<th>( \beta )</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Waist circumference (R(^2) = 0.882)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>constant</td>
<td>4.027</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Intervention vs Usual Care</td>
<td>-2.139</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>Baseline waist circumference</td>
<td>0.950</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Change in fruit consumption index</td>
<td>-0.105</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>Change in vigorous physical activity</td>
<td>4.774</td>
<td>0.033</td>
<td></td>
</tr>
<tr>
<td><strong>Systolic blood pressure (R(^2) = 0.397)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>constant</td>
<td>67.282</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Intervention vs Usual Care</td>
<td>-4.968</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td>Female vs. Male</td>
<td>-3.884</td>
<td>0.044</td>
<td></td>
</tr>
<tr>
<td>Baseline systolic blood pressure</td>
<td>0.533</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Change in internal health locus of control</td>
<td>0.512</td>
<td>0.001</td>
<td></td>
</tr>
<tr>
<td><strong>Diastolic blood pressure (R(^2) = 0.525)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>constant</td>
<td>22.581</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Female vs. Male</td>
<td>1.152</td>
<td>0.014</td>
<td></td>
</tr>
<tr>
<td>Baseline diastolic blood pressure</td>
<td>0.749</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Change in Internal health locus of control</td>
<td>0.120</td>
<td>0.017</td>
<td></td>
</tr>
<tr>
<td><strong>Total cholesterol (R(^2) = 0.515)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>constant</td>
<td>1.390</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Female vs. Male</td>
<td>0.347</td>
<td>0.007</td>
<td></td>
</tr>
<tr>
<td>Baseline total cholesterol</td>
<td>0.606</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Change in cholesterol consumption</td>
<td>0.002</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td><strong>HDL-c (R(^2) = 0.747)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>constant</td>
<td>0.171</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Female vs. Male</td>
<td>0.075</td>
<td>0.024</td>
<td></td>
</tr>
<tr>
<td>Baseline HDL-c</td>
<td>0.771</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Fat consumption</td>
<td>-0.001</td>
<td>0.016</td>
<td></td>
</tr>
<tr>
<td>Change in internal health locus of control</td>
<td>-0.006</td>
<td>0.034</td>
<td></td>
</tr>
<tr>
<td><strong>Triglycerides (R(^2) = 0.642)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>constant</td>
<td>0.792</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Coefficient</td>
<td>p-value</td>
<td></td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------</td>
<td>---------</td>
<td></td>
</tr>
<tr>
<td>Female vs. Male</td>
<td>0.218</td>
<td>0.006</td>
<td></td>
</tr>
<tr>
<td>Baseline triglycerides</td>
<td>0.705</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Change nutrition self-efficacy</td>
<td>-0.148</td>
<td>0.001</td>
<td></td>
</tr>
</tbody>
</table>

**CVD Risk Score** (\(R^2 = 0.809\))

<table>
<thead>
<tr>
<th></th>
<th>Coefficient</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>3.567</td>
<td></td>
</tr>
<tr>
<td>Intervention vs. Usual care</td>
<td>-0.979</td>
<td>0.004</td>
</tr>
<tr>
<td>Female vs. Male</td>
<td>-1.344</td>
<td>0.001</td>
</tr>
<tr>
<td>Baseline CVD risk score</td>
<td>0.708</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Change in internal health locus of control</td>
<td>0.096</td>
<td>0.005</td>
</tr>
<tr>
<td>Change in nutrition self-efficacy</td>
<td>0.282</td>
<td>0.042</td>
</tr>
</tbody>
</table>

**T2DM Risk Score** (\(R^2 = 0.461\))

<table>
<thead>
<tr>
<th></th>
<th>Coefficient</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>constant</td>
<td>5.813</td>
<td></td>
</tr>
<tr>
<td>Female vs. Male</td>
<td>-1.759</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Baseline T2DM risk score</td>
<td>0.648</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Change in internal health locus of control</td>
<td>-0.164</td>
<td>0.012</td>
</tr>
<tr>
<td>Change in Australian Recommended Food Score</td>
<td>0.106</td>
<td>0.042</td>
</tr>
</tbody>
</table>
Summary

Chapter 6 presented the 12-month follow-up analyses to determine interventional effects on behaviour-specific self-efficacy, health locus of control and their association with health behaviours and cardio-metabolic disease risk. There were overall reductions in risk factors which were more advantageous for the intervention group than usual care, evidently due to the structured and more intensive intervention over 12 months. These beneficial findings appeared to be driven by changes in diet rather than physical activity or health beliefs whereupon self-efficacy and health locus of control showed minimal variation after 12 months. Unexpectedly, behaviour specific self-efficacy showed decreases likely due to the potential ‘optimistic’ overestimation of self-efficacy made at baseline by participants; a reassessment of the challenges related to changing a particular behaviour may have taken place during the course of the intervention, which resulted in a more accurate evaluation of self-efficacy at 12 months. The potential for nurse intervention to enhance both self-efficacy and health locus of control was therefore not apparent raising the possibility that disease risk cannot be associated with any particular health belief type. Instead, the key determinants of cardiometabolic risk after 12 months were changes in biomedical risk factors and diet.

Extended follow-up to 24 months will determine the influence of the intervention on self-efficacy, locus of control and their relationships with overall cardiometabolic disease risk.
Chapter 7
General Discussion and Conclusion
Chapter 7 discusses the overall contents of this thesis work and provides a general discussion of the major findings and implications. The limitations of this research are also divulged, and potential future research directions are provided.
7.1. Thesis Summary

The aim of this thesis was to investigate behaviour-specific self-efficacy and health locus of control as potential determinants of cardio-metabolic disease risk and interventional response within the wider MODERN nurse-led randomised controlled trial of at-risk regional dwelling adults. This thesis provided a review of the effective characteristics of cardio-metabolic lifestyle interventions and highlighted the exacerbated cardio-metabolic risk levels existing in regional communities; thus presenting them as ideal candidates for a lifestyle modification program. Associations between behaviour-specific self-efficacy, health locus of control, behaviour change and cardio-metabolic disease risk factors were investigated at baseline and their ability to be enhanced after a 12-month lifestyle intervention was assessed in individuals prone to the development of CVD and/or T2DM by virtue of their MetS status.

Given the strong burden of CVD/T2DM on non-metropolitan populations (Chapter 1) and the potential influence of self-efficacy and health locus of control on health behaviour change (Chapter 2), this research focussed on regional populations who are routinely identified as being at heightened risk of poorer health outcomes. To better understand this disparity and the characteristics of an effective intervention targeting regional, rural or remote populations, a systematic review and meta-analysis of non-urban primary prevention interventions was undertaken (Chapter 3). The findings highlighted that short-term, community based programs delivered in a pre/post framework were the most effective in improving cardio-metabolic disease risk. The results aided in the design of a lifestyle modification program to reduce
CVD and/or T2DM risk in a non-urban setting. Chapter 4 presented the methodology of the RCT of a multi-disciplinary, nurse-led, health and lifestyle modification trial (MODERN). Screening results were also reported showing high levels of risk factors common in this regional sample. There was a high prevalence of overweight/obesity affecting around 80% of participants, dyslipidemia impacting over 80% of individuals’ total and LDL-C levels and dysglycemia affecting one third of adults. In addition, health behaviours such as high risk alcohol consumption and insufficient physical activity were reported by around one third of participants. Baseline (Chapter 5) associations between behaviour-specific self-efficacy, health locus of control, health perceptions and cardio-metabolic disease risk were also assessed in the screening cohort of 853 participants. Briefly, findings showed that participants rated their self-efficacy for health behaviours (nutrition, physical activity, alcohol consumption and smoking cessation) quite highly, with no disparities between individuals with low and high cardio-metabolic risk profiles. Contrastingly, individuals at high risk of cardio-metabolic disease risk had an external health locus of control and scored higher in powerful others and chance health locus of control domains than their lower risk counterparts.

In Chapter 6 the effects of the nurse-led intervention on behaviour-specific self-efficacy, health locus of control and health behaviours were described and associations with cardio-metabolic risk were evaluated over 12 months in 276 randomised participants. The intervention was effective in reducing cardio-metabolic disease risk factors significantly greater within the intervention group than usual care, although reductions were observed in both groups. Reductions in behaviour-specific self-efficacy for key health behaviours were observed over 12 months. The key determinants of
cardiometabolic risk after 12 months were changes in biomedical risk factors and diet. Chapter 7 discusses the overall contents of the thesis and provides a general discussion on the major findings and implications. The limitations of this research are also considered and potential future research directions are provided.

7.2. Research Findings

To our knowledge, Chapter 3 represented the first publication to systematically review the effectiveness of primary prevention interventions aiming to improve cardio-metabolic risk in non-urban populations. Our findings indicated that more pronounced changes in behaviour and risk occurred in short-term interventions (≤ 12 months), highlighting the difficulties associated with health behaviour change maintenance over longer periods of time. Frequent contact with participants and regular follow-up were key to an effective intervention, aiding in a participant’s adherence to novel behaviour changes which often declines over time or as contact is reduced [123]. It identified that pre/post study designs were marginally more effective but this may have been due to overestimation/confounding as well as any potential Hawthorne effect occurring within randomised controlled trials. Community-based interventions were also particularly effective at reducing cardio-metabolic disease risk, likely due to utilisation of community constructs to deliver their intervention. The overall capacity for non-urban primary prevention programs to attenuate risk factors and cardio-metabolic disease risk via improved risk factor control was evident, however, it was concluded that the key individual determinants of cardio-metabolic risk and intervention response required further evaluation.
Therefore, as part of the MODERN study, we sought to evaluate an individual’s health perceptions, behaviour-specific self-efficacy and health locus of control as determinants of behaviour change and cardio-metabolic disease risk. MODERN presents a pragmatic, multi-centre, appropriately powered, 24-month, RCT of an innovative nurse-led intervention aiming to develop a cost-effective regional health care program to optimise the reduction of risk factors in individuals with MetS with or without T2DM. Chapter 4 of this thesis presented the methodology and rationale of MODERN, together with the screening results which revealed high levels of cardio-metabolic risk factors, demonstrating participants’ eligibility for a lifestyle modification program. Baseline and 12-month follow-up data contributed to the findings in this research, with the 24-month follow-up of the MODERN study extending beyond the scope of this thesis.

Given the scarcity of research focussing on key psychological determinants of behaviour change and their associations with chronic disease risk, baseline analyses of the screened cohort (Chapter 5) focussed on identifying associations between behaviour-specific self-efficacy (nutrition, physical activity, alcohol consumption and smoking), multidimensional health locus of control, risk perception and cardio-metabolic disease risk. It was hypothesised that there would be an inverse correlation between self-efficacy for key health behaviours, health locus of control and cardio-metabolic disease risk. We found that individuals at high risk of CVD and T2DM had a greater external (chance and powerful others) health locus of control compared to those at low risk who had a greater internal health locus of control. Our findings are commensurate with previous research findings indicating there may be some benefit and more favourable health outcomes in having an internal health
locus of control [100-101]. However what has yet to be shown is whether health beliefs (locus of control and self-efficacy) can be modified by trained nurses, coinciding with changes to risk factor levels.

The final research manuscript, Chapter 6, investigated the effectiveness of a nurse-led lifestyle intervention on cardio-metabolic disease risk, behaviour-specific self-efficacy, health locus of control and health behaviours compared to usual care after 12 months follow-up. As a consequence of the intervention, it was anticipated that there would be an increase in behaviour-specific self-efficacy and shift towards an internal locus of control reflecting greater participant engagement in making positive health behaviour changes, with concomitant reductions in cardio-metabolic risk.

There were overall reductions in risk factors which were more advantageous for the intervention group than usual care These beneficial findings appeared to be driven by changes in diet rather than physical activity or health beliefs. Unexpectedly, behaviour specific self-efficacy showed decreases likely due to the potential ‘optimistic’ overestimation of self-efficacy made at baseline by participants; a more accurate evaluation of self-efficacy may have occurred at 12 months. Although nurses were not explicitly targeting the modification of self-efficacy or health locus of control, the potential for nurse intervention to enhance both self-efficacy and health locus of control was not apparent, raising the possibility that disease risk may not be explicitly associated with any particular health belief direction.

7.3. Implications of Research Findings

The data of this thesis indicate that nurse-led interventions can be effective in reducing cardio-metabolic disease risk via risk factor management. Although
nurses are ideally placed to deliver multifactorial lifestyle interventions targeting behavioural risk factors, they may not be as adept for eliciting change in behaviour specific self-efficacy and/or health locus of control. Modifying these psychological constructs may extend beyond a nurse’s scope of training. It might be useful for interventions aiming to modify these constructs to include training in health behaviour change principles or to incorporate clinically-trained psychologists in the delivery of the intervention.

Although the cardio-metabolic disease risk improvements achieved by this nurse-led intervention are not attributable to either health locus of control or self-efficacy in this instance, they remain key principles and widely accepted determinants of health behaviour change. Furthermore, other causes, such as health professional contacts and medications must be considered as contributing factors for improved risk management, which were not assessed as part of this thesis. Extended 24-month follow-up will provide more wide-ranging insight into the influences of risk behaviours and cardio-metabolic health in a higher risk regional cohort.

7.4. Limitations

The overall MODERN study was designed as a nurse-led, multi-faceted, tailored, lifestyle intervention with the principal aim of targeting risk factor modification to reduce cardio-metabolic disease risk, and was not designed or powered to specifically target or modify health locus of control or behaviour-specific self-efficacy. Behavioural changes and risk reductions observed within the usual care group may have been a result of being involved in research (the well-known Hawthorne effect [124]), reducing group differences making (significant) change in the intervention arm more difficult.
to achieve. This however would be accounted for in power calculations of the main 24 month follow-up MODERN trial. Selection bias may limit the generalisability of findings; as participants were self-selected from two regional communities, overall findings should consider the environmental, socioeconomic and ethnic factors specific to each community which may differ from wider population data. Participants may have also been considered “worried well” and seeking reassurance about their health, whereas other participants may have volunteered seeking more comprehensive management of their known elevated risk. Regardless, all participants randomised into the longer-term MODERN RCT were identified with MetS and as such presented as interested and ideal candidates for lifestyle and risk factor modification intervention. Potential desirability bias or exaggeration of certain self-reported health behaviour data also cannot be excluded.

7.5. Future Research

The most paramount short-term objective for continued research is the completion of the MODERN study and the analysis and interpretation of the 24-month follow-up data. The longer-term influence of nurse-led intervention on health locus of control and behaviour-specific self-efficacy may be further explained, and the latter’s influence on changes in health behaviours and cardio-metabolic disease risk further elucidated. Future analyses might focus on more refined mechanisms of change and examine the potential moderator role of self-efficacy that could reveal differential effects for subgroups of the study sample.
An evident extension of the current research should involve the undertaking of a dedicated intervention implementing behaviour-specific self-efficacy and health locus of control modifications, administered by allied health professions, other than nurses, such as clinical psychologists. This could also be investigated within a metropolitan context. Contextually, findings from a metropolitan population would have wider spanning implications for clinical cardio-metabolic risk factor management and primary prevention strategies.

7.6. Conclusions

Residents of regional, rural and remote communities present as ideal candidates for targeted risk factor management and lifestyle intervention. Although interventions are typically effective, understanding the underlying determinants of reducing an individual’s risk via prescribed lifestyle or health behaviour modifications is difficult. This research program investigated health perceptions, namely behaviour-specific self-efficacy and multidimensional health locus of control, as possible determinants of interventional response (behaviour changes), and as potential predictors of cardio-metabolic disease risk.

Our findings predicate the potential for a nurse-led intervention to be effective in reducing overall cardio-metabolic disease risk via risk-factor modification. Health locus of control and behaviour-specific self-efficacy did not present as significant determinants of cardio-metabolic disease risk, although the later may have been overestimated at the beginning of the trial, but then normalised thereafter. Extended follow-up to 24 months will confirm
the benefit of nurse-led clinics in preventing cardio-metabolic disease and the longer term interventional effects on behaviour-specific self-efficacy, health locus of control and their relationship with cardio-metabolic disease risk.
This **bibliography** contains all references pertaining to the main body of the thesis work. For published papers or submitted manuscripts, references remain separate, as part of the publication or manuscript.


68. Artinian, Nancy T., et al. "Interventions to promote physical activity and dietary lifestyle changes for cardiovascular risk factor reduction in


99. Cobb-Clark, Deborah A., Sonja C. Kassenboehmer, and Stefanie Schurer. "Healthy habits: The connection between diet, exercise, and


116. Williams, Stefanie L., and David P. French. "What are the most effective intervention techniques for changing physical activity self-efficacy and physical activity behaviour—and are they the same?." *Health education research* 26.2 (2011): 308-322.


Nurse health and lifestyle modification versus standard care in 40 to 70 year old regional adults: study protocol of the Management to Optimise Diabetes and mEtabolic syndrome Risk reduction via Nurse-led intervention (MODERN) randomized controlled trial

Melinda J. Carrington and Paul Zimmet

Abstract

Background: Metabolic syndrome (MetS), the clustering of multiple leading risk factors, predisposes individuals to increased risk for developing type 2 diabetes and/or cardiovascular disease (CVD). Cardio-metabolic disease risk increases with greater remoteness where specialist services are scarce. Nurse-led interventions are effective for the management of chronic disease. The aim of this clinical trial is to determine whether a nurse-implemented health and lifestyle modification program is more beneficial than standard care to reduce cardio-metabolic abnormalities and future risk of CVD and diabetes in individuals with MetS.

Methods: MODERN is a multi-centre, open, parallel group randomized controlled trial in regional Victoria, Australia. Participants were self-selected and individuals aged 40 to 70 years with MetS who had no evidence of CVD or other chronic disease were recruited. Those attending a screening visit with any 3 or more risk factors of central obesity, dyslipidemia (high triglycerides or low high density lipoprotein cholesterol) elevated blood pressure and dysglycemia were randomized to either nurse-led health and lifestyle modification (intervention) or standard care (control). The intervention included risk factor management, health education, care planning and scheduled follow-up commensurate with level of risk. The primary cardio-metabolic end-point was achievement of risk factor thresholds to eliminate MetS or minimal clinically meaningful changes for at least 3 risk factors that characterise MetS over 2 year follow-up. Pre-specified secondary endpoints to evaluate between group variations in cardio-metabolic risk, general health and lifestyle behaviours and new onset CVD and type 2 diabetes will be evaluated. Key outcomes will be measured at baseline, 12 and 24 months via questionnaires, physical examinations, pathology and other diagnostic tests. Health economic analyses will be undertaken to establish the cost-effectiveness of the intervention.

(Continued on next page)
Discussion: The MODERN trial will provide evidence for the potential benefit of independent nurse-run clinics in the community and their cost-effectiveness in adults with MetS. Findings will enable more nurse-led clinics to be adopted outside of major cities and encompassing other chronic diseases as a key primary preventative initiative.

Trial registration: MODERN is registered with the Australian New Zealand Clinical Trials Registry (ACTRN12616000229471) on 19 February 2016 (retrospectively registered).

Secondary identifiers: MODERN is an investigator-initiated trial funded by the National Health and Medical Research Council of Australia from 2014 to 2017 via a Project Grant (ID No. APP1069043) and was approved by the Australian Catholic University Human Research Ethics Committee (Project No: 2014 244 V) and the Department of Health Human Research Ethics Committee (Project No:38/2014) for the release of Medicare claims information.

Keywords: Metabolic syndrome, Cardio-metabolic risk factors, Cardiovascular disease, Type 2 diabetes mellitus, Nurse-led intervention, Health and lifestyle modification, Clinical trial

Background
Metabolic syndrome (MetS), the constellation of central obesity, dyslipidemia, elevated blood pressure (BP) and dysglycemia [1], encapsulates some of the leading non-communicable disease risk factors in the world [2]. For three of these risk factors, 9% of the Australian adult population are overweight or obese, have dyslipidemia and elevated BP in combination, with 66% of the population having three or more of any behavioural or biomedical risk factor (at the same time) for developing cardiovascular disease (CVD), diabetes and chronic kidney disease [3]. The proportion of adults with antecedent risk for cardio-metabolic disease increases with increasing remoteness [3]; this geographical pattern of inequality is reflected by higher rates of CVD and diabetes prevalence [4], deaths [5] and hospitalizations [6].

Notwithstanding the definition used to identify subjects with MetS, having multiple risk factors is detrimental to increased disease risk in a dose-response gradient with more components of the MetS [7, 8]. CVD morbidity and mortality outcomes are between 2 to 2.4-fold higher [9], and stronger for type 2 diabetes (T2DM) risk of between 3 to 5-fold greater [8] in individuals with MetS. CVD is the most common clinical sequelae in those with T2DM and MetS itself promotes the development of both of these conditions due to the association between components and atherosclerosis and dysglycemia. Fortunately, in people with MetS, a meta-analysis has demonstrated the benefits of lifestyle and pharmacological interventions for reversal of MetS [10] and decreased likelihood of developing CVD [11] and T2DM [12]. Robust evidence exists for the clinical usefulness of nurse-led interventions for improving cardio-metabolic risk factors in adults with chronic conditions [13–15], fostered by written protocols embedding clinical practice guidelines and a structured framework to enable the titration and intensification of therapy and retraction of the frequency of management when deemed appropriate [16]. However, no randomized controlled trial with standard care as the comparator over the longer term to evaluate health benefit and cost-effectiveness of community nurse-led clinics has been established to date in higher risk, non-urban settings.

Therefore, our aim was to assess the effectiveness of a nurse-led clinic program that modulated health and lifestyle factors to reduce cardio-metabolic abnormalities and future risk of CVD and T2DM in individuals with MetS. It was hypothesised that there would be more adults with MetS in the nurse-facilitated health and lifestyle modification group compared to standard care who achieve the primary endpoint of meeting target risk factor thresholds or minimum changes that would be considered clinically significant for any 3 or more risk factors that characterise the MetS over 2 year follow-up.

Methods/Design
Study design
MODERN is a multi-centre, open, parallel group randomized controlled trial evaluating the effect of nurse-facilitated health and lifestyle modification (intervention group) vs. standard care (control group) on a primary cardio-metabolic end-point (achievement of risk factor levels to eliminate MetS or minimal clinically meaningful change in risk factor levels for MetS) in at risk regional residents aged between 40 and 70 years. The study flow diagram over 2 year follow up and extended 5 year longer-term follow-up is shown in Fig. 1. The final study protocol (Version 2; 5 August 2014) was approved by the Australian Catholic University Human Research Ethics Committee (Project No: 2014 244 V). Study nurses obtained written informed consent to participate and additional consent for the release of Medicare claims information (medical services and prescriptions), as approved by the Department of Health Human Research Ethics Committee (Project No: 38/2014). Subjects could discontinue their participation upon request, in which case personal information collected up to
the time of withdrawal would be retained for analyses, unless the participant stated otherwise.

**Study population and recruitment strategy**
Participants were self-selected from two regional locations in Shepparton, northern Victoria, and Colac, south-west Victoria (estimated resident population 40–70 years at 30 June 2015 was 23,437 and 13,055, respectively). Census data were used to ensure that a representative sample of the age and sex distribution of each regional community between 40 and 70 years were recruited. The sampling strategy involved large scale promotion of the study to key local workplaces, community groups (e.g. Rotary) and Local Members of Parliament, community engagement at local markets and stalls, direct and indirect postal invitations, radio and newspaper advertising and social media (e.g. Facebook). Individuals responded to participate and a brief screening questionnaire administered via telephone or face-to-face was initially used to determine age and history of CVD or other chronic disease. Eligible participants made an appointment to undergo screening to identify MetS from 26 September 2014 and recruitment was completed on 1 April 2016. The study protocol conformed to SPIRIT guidelines and a full CONSORT diagram will be finalised upon trial completion.

**Selection criteria**
Participants residing in, or in surrounding regions of Shepparton or Colac and aged between 40 and 70 years with any three or more risk factors for MetS [1] as shown in Table 1 at the time of screening were eligible. The equivalent HbA1c level for increased risk of diabetes was used as a substitute for fasting plasma glucose [17]. Participants were included if they were capable of attending scheduled study clinic visits and able to provide informed consent. Participants outside the eligible age
range with two or less identifiable risk factors, any diagnosed form of CVD, chronic kidney disease or other forms of chronic disease that resulted in the belief that participation would not be appropriate, were excluded. Participants were omitted from participating if they had neurological/cognitive impairment and/or were unable to provide written informed consent.

Randomisation
Participants who satisfied the selection criteria were randomized into one of two trial arms according to a 1:1 allocation ratio using a pre-determined computer generated sequentially numbered randomisation schedule centrally performed via SPSS Statistics 22.0 (SPSS Inc., IL, USA) and transferred to the receptionist at each study clinic to assign group distribution. Group allocation was concealed from nurses to eliminate selection bias during recruitment. Block randomisation (per regional study clinic) occurred with block sizes of 20 and stratified according to MetS without T2DM or with a diagnosis of T2DM. Due to the nature of the intervention, blinding subjects and nurses was not possible.

Primary endpoint
The primary endpoint was the between group difference in achievement of the target risk factor thresholds (Table 1) or minimum changes shown in Table 2 that would be considered clinically significant for any 3 or more of the risk factors that characterise the MetS. These clinically significant minimum changes represent > 0.5 standard deviation change from baseline, calculated utilising pilot data from adults with MetS [18].

Secondary endpoints
Secondary endpoints included between group changes at 24 months in meeting the individual components of the primary endpoint (i.e. target thresholds and clinically significant changes); resolution of MetS status; incidence of T2DM, as indicated by initiation of glucose-lowering medication following confirmation of a diagnosis of T2DM or a HbA1C level ≥ 6.5% [17]; incidence of CVD, defined by fatal events, a diagnosis of CVD or non-fatal CVD-related events requiring hospitalization; within and between group variations in health behaviours, general health and cardio-metabolic specific medical interventions and hospitalizations to establish the cost-effectiveness of the MODERN intervention.

Procedures
A self-report questionnaire posted to participants in the week prior to their scheduled visit collected information regarding: socio-demographic factors including marital and work status, primary language spoken, income, education and ethnicity; health behaviours including smoking, diet and alcohol via the Dietary Questionnaire for Epidemiological Studies (DQES v2) [19], sleep and physical activity and sedentary behaviour via the International Physical Activity Questionnaire (IPAQ) [20] and; general health including history of CVD, associated conditions or other serious conditions, family history of CVD or diabetes, prescribed medications and adherence via the Medication Adherence Questionnaire (MAQ) [21], signs and symptoms of angina via the Rose Angina questionnaire [22], health related quality of life via the Assessment of Quality of Life – 8 Dimension (AQoL-8D) [23], perceived risk and health belief evaluation via item specific self-efficacy [24] and locus of control [25].

An on-line, discrete choice experiment during the week prior to their scheduled visit at baseline and at study end evaluated participant preferences for the attributes of a health and lifestyle management program they consider of greatest importance affecting their choice to participate in such programs.

The screening assessments were undertaken at a dedicated clinic or on-site at workplaces (for few organisations with large numbers of workers) by

---

**Table 1** Criteria for metabolic syndrome classification [1]

<table>
<thead>
<tr>
<th>Risk factor for MetS</th>
<th>Cut-point criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated WC</td>
<td>Men: ≥ 94 cm</td>
</tr>
<tr>
<td></td>
<td>Women: ≥ 80 cm</td>
</tr>
<tr>
<td>Elevated triglycerides</td>
<td>≥ 1.7 mmol/L</td>
</tr>
<tr>
<td>Reduced HDL-C</td>
<td>Men: &lt; 1.03 mmol/L</td>
</tr>
<tr>
<td></td>
<td>Women: &lt; 1.29 mmol/L</td>
</tr>
<tr>
<td>Elevated BP</td>
<td>≥ 130/85 mmHg</td>
</tr>
<tr>
<td>Elevated HbA1c</td>
<td>≥ 5.7% (39 mmol/L)</td>
</tr>
</tbody>
</table>

**Table 2** Criteria for minimum change in components of metabolic syndrome to assess the primary endpoint

<table>
<thead>
<tr>
<th>Risk factor for MetS</th>
<th>Clinically significant minimum change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated WC</td>
<td>Reduce by ≥ 5 cm</td>
</tr>
<tr>
<td></td>
<td>Reduce by ≥ 6 cm</td>
</tr>
<tr>
<td>Elevated triglycerides</td>
<td>Reduce by ≥ 0.6 mmol/L</td>
</tr>
<tr>
<td></td>
<td>Increase by ≥ 0.15 mmol/L</td>
</tr>
<tr>
<td>Reduced HDL-C</td>
<td>Reduce by ≥ 0.5 mmol/L</td>
</tr>
<tr>
<td></td>
<td>Increase by ≥ 0.18 mmol/L</td>
</tr>
<tr>
<td>Elevated BP</td>
<td>Reduce by ≥ 7/3 mmHg</td>
</tr>
<tr>
<td></td>
<td>Reduce by ≥ 8/4 mmHg</td>
</tr>
<tr>
<td>Elevated HbA1c</td>
<td>Reduce by ≥ 0.4%</td>
</tr>
<tr>
<td></td>
<td>Reduce by ≥ 0.5%</td>
</tr>
</tbody>
</table>

MetS, metabolic syndrome; WC, waist circumference; HDL-C, high-density lipoprotein cholesterol; BP, blood pressure; HbA1c, glycated haemoglobin.
registered nurses according to a standardised protocol at baseline, prior to randomisation. The same registered nurse who conducted the screening assessment remained responsible for the follow-up assessments of each participant until study completion; due to standardising the management of participants and frequency of follow-up, a different nurse that managed participants from only one study group was not required to alleviate bias.

Waist and hip circumference were measured using a Figure™ Tape Measure (Novel Products, IL, USA) in accord with the World Health Organization (WHO) STEPwise approach to surveillance (STEPS) procedure [26] in the horizontal plane whilst standing; the level mid-way between the lowest rib and iliac crest at the end of a gentle expiration was taken for waist circumference and the level at the maximum extension of the buttocks defined hip circumference. Body mass index (BMI, kg/m²) was determined from height, measured using a portable stadiometer (seca®, Hamburg, Germany) and weight, using digital weighing scales (A&D Medical, SA, Aus), after the removal of shoes and heavy garments. Total cholesterol (TC), high- (HDL-C) and low-density lipoprotein cholesterol, triglycerides and HbA1C in capillary/fingerstick whole blood were analysed by the reflectance photometry technique using a calibrated Afinion™ AS100 analyser (Alere, MA, USA). Sitting blood pressure (BP) was measured in the brachial artery with a suitable cuff size using a calibrated Omron HEM-907 automated monitor (Omron Healthcare Co. Ltd., Kyoto, Japan) after 5 min of rest; the average of two measurements separated by a one-minute interval were analysed provided BP did not vary by ≥10/≥5 mmHg, in which case another reading was taken and the closest two readings were analysed. Calculation of CVD risk via the Framingham Risk Equation [27], and T2DM risk via the AUSDRISK tool [28], was estimated from applicable health and lifestyle risk factors and results from the thorough screening assessment to predict risk of a cardiovascular event [29] and developing T2DM [28], respectively, over the next 5 years.

If MetS was indicated and subjects agreed to be randomized into a study group, additional information was collected including: electrocardiography (ECG) via a 12-lead Universal ECG™ in accordance with standard electrode placement and recorded using Office Medic™ Software (QRS Diagnostic, MN, USA); biochemistry assessment from venous blood of creatinine to estimate glomerular filtration rate (eGFR) using the Modification of Diet in Renal Disease study equation, and high-sensitive C-reactive protein (hs-CRP) as an inflammatory marker of CVD risk; actigraphy for 7-day continuous quantitative measurement of sleep, physical activity and sedentary behaviour using GTX3 accelerometers (ActiGraph, FL, USA) and; arterial stiffness from ankle brachial (ABI) and cardio ankle vascular pressure indexes (CAVI) using the VaSera™ VS-1500 N vascular device (Fukuda Denshi Co Ltd., Tokyo, Japan).

Qualitative evaluation of intervention fidelity [30] to assess 1) adherence to key aspects of the protocol being implemented, and 2) competence in delivering the program in terms of communication, technical abilities and skills in responding to participants was established by video recordings of nurse/participant clinic visit interactions that were later evaluated by two trained observers, with feedback of results to improve nurse’s performance. For selection of a random set of observations to provide a representative sample for measuring intervention fidelity, nurses were advised (3–5 days prior) by the study co-ordinator that all participants scheduled to attend clinic visits in the following week will be asked to provide written informed consent to video recording their clinic encounter. Fidelity monitoring continued until the target number of 2 observations from each of the two trial arms (4 in total) per nurse at baseline and mid (12 month) study visits was completed.

**Intervention group – Health and lifestyle modification**

The nurse-facilitated intervention required that two nurses at each site actively deliver a health and lifestyle intervention and remain responsible for assessing participant progress in achieving their health goals until study completion. Nurses applied the intervention on an individual basis and acted within the scope of their nursing responsibilities and independent of the participant’s health care professionals. The aim of the intervention was to deliver coaching to support individuals to achieve health behaviour change for improved risk factor management. Intervention participants were permitted to seek any relevant comitant care. Key components were:

1. Cardio-metabolic risk factor management - to achieve ideal goal levels based on recommended guidelines and with consideration for the circumstances of unique individuals. For weight management, waist circumference, BMI measurements and responses to the DQES food frequency questionnaire were all evaluated followed by tailored advice on healthy eating in accord with Australian Dietary Guidelines [31] to strive for a healthy weight range of BMI 18.5–24.9 kg/m² or waist circumference < 80 cm (women) or < 94 cm (men). For physical activity, responses to the IPAQ were assessed and according to Australian Government Department of Health guidelines [32], subjects were advised to aim towards accumulating at least 30 min of moderate intensity physical activity on all or most days of the week and if capable, vigorous activity for 30 min or more for 3
or 4 days a week, and to limit sedentary behaviour. For dyslipidaemia (defined as not meeting any one target lipid level) and dysglycemia, results from fasting point of care profiling were reviewed to achieve optimal levels for reduced risk of coronary artery disease [33] of < 4.5 mmol/L for TC, < 2.5 mmol/L for LDL-C, > 1.0 mmol/L for HDL-C, and < 2.0 mmol/L for triglycerides, and for reduced risk of diabetes [17] of < 5.7% (39 mmol/mol) for HbA1C. For BP, average clinic readings were classified in accord with National Heart Foundation guidelines [34] with the aim to achieve a BP of < 140/90 mmHg. Smokers were assessed on their readiness to cease smoking and provided appropriate counselling to encourage them to attempt to quit. People who chose to drink alcohol were advised to limit their intake to no more than 2 standard drinks on any day to reduce the lifetime risk of alcohol-related harm in accord with Australian Guidelines for Alcohol Consumption [35]. For overall CVD and T2DM health, absolute CVD and T2DM calculated risk levels were categorised in accord with recommended guidelines [28, 29] with the goal to reduce risk of CVD and T2DM. Diet and lifestyle modification was initially indicated for all subjects with dyslipidemia, dysglycemia and elevated BP, regardless of CVD and T2DM risk, except in the case of extreme results whereby participants were referred to their nominated primary health practitioner for consideration for drug therapy or additional clinical care. The results of the baseline screening assessment were assessed in accord with the Green, Amber, Red Delineation of Risk and Need (GARDIAN) management system to determine the frequency of health care intervention over study follow up [16]. Figure 2 shows that eligible intervention participants were designated a traffic light colour RED (high) or AMBER (intermediate) [noting the unavailability of GREEN (low) at baseline since eligibility required the presence of risk factors] to denote their overall degree of risk and corresponding GARDIAN-guided level of nurse intervention. GARDIAN designated RED subjects as those with high absolute CVD risk scores ≥16%, T2DM or identification of diabetes by HbA1C ≥ 6.5%, markedly elevated levels of individual risk factors or who were smokers. GARDIAN designated AMBER subjects were those with moderate absolute CVD risk scores 10–15% or with risk factors above recommended ranges. GARDIAN designated GREEN subjects (at 12 month review) were those identified by low absolute CVD risk scores < 10%, HbA1C < 5.7% or risk factor levels within recommended limits.

Fig. 2 GARDIAN-guided classification of risk and follow-up plan over 24 months
2. Health education – to put risk factor management in context, participants received education on CVD and T2DM including causes, risks, symptoms, treatment and management; interpretation of individualised test results in contrast to target goal levels and benefits of changing behaviour(s); understanding how to read food labels; managing portion serving sizes and comprehension and adherence to prescribed medication. All participants received a brief report containing their risk assessment results as well as a Health Passport to document before and after photographs, record appointment times and to maintain a “health identity” profile to track risk factor measurements over time and change to their GARDIAN (traffic light) risk colour code. Written education information and risk factor targets to aim for to remain healthy was consolidated within the Health Passport.

3. Care planning – to help reduce identified risk factors, nurses trained in motivational interviewing [36] applied the 5As model (assess, advise, agree, assist, arrange) [37] of behavioural counselling and prompted participants to develop self-care and management plans to stimulate behaviour change. With nurse support, participants’ motivations to change, including self-nominated goals/priority areas and obstacles or catalysts to achieve them were identified. Attainable targets to achieve individualised health goals were agreed and suitable community lifestyle programs or health professional referral for improved care were recommended, with formal re-evaluation of care plans mid-study.

4. Scheduled follow-up - to assist subjects to adhere to the care plan and reinforce positive behaviour change, specified follow-up visits and supplemental telephone coaching was implemented in accord with GARDIAN status. The frequency of contact with participants was aligned with their level of risk. As shown in Fig. 2, GARDIAN designated RED subjects at baseline received a telephone call at 1 month and clinic visits at 3, 6 and 12 months. GARDIAN designated AMBER subjects received a telephone call at 1 month and clinic visits at 6 and 12 months. After 12 months, screening assessments were repeated and GARDIAN classification was reassessed; revised RED-coded subjects received a telephone call at 13 months and clinic visits at 15, 18 and 24 months. Updated AMBER coded subjects received a telephone call at 13 months and clinic visits at 18 and 24 months. Newly designated GREEN subjects received a telephone call at 18 months and a clinic visit at 24 months.

Control group - standard care
Participants allocated to the standard care arm of MODERN received screening assessments but diluted risk factor management which was limited to receiving a brief risk assessment report and Health Passport with less intensive CVD and T2DM education and dietary and lifestyle advice. Ideal risk factor targets were identical yet care plans were not developed. GARDIAN status was not used to direct management and follow-up contacts at set times; aside from baseline, 12 month (mid), and 24 month (end) study visits, all management and follow-up was at the discretion of their usual health care providers. As per intervention group participants, control group participants were permitted to seek any relevant concomitant care.

Follow-up visit format
For all participants randomized into a study group, self-report questionnaires posted 1 week prior to their scheduled visit collected updated information regarding health behaviours and general health at 12 and 24 month visits. Screening assessments, biochemistry, actigraphy and arterial stiffness tests were also repeated at 12 and 24 month visits for all participants. For intervention participants only, just screening assessments were conducted at all other (GARDIAN directed) clinic visits to help assess participant’s progress in achieving their health goals. Extended surveillance to 5 years post randomization of cardio-metabolic health and associated health care activity to determine the longer-term impact of the MODERN intervention is planned.

Study power
A 15% difference (20% intervention, 5% control) in the number of subjects who achieved target risk factor thresholds or clinically significant changes in any 3 MetS risk factors was derived from sub-group analyses (of individuals identified with MetS) from a pilot intervention study [18] and a randomized controlled trial of a similar lifestyle intervention delivered by health professionals versus standard care [12]. With two-sided α = 0.05, an estimated minimum of 125 participants per study arm would provide 95% power to detect a 0.15 difference in the primary endpoint after 2 year follow-up. Accounting for potential loss-to-follow up of 15%, overall the trial would require approximately 150 participants randomized in each group.

Data management, confidentiality and dissemination
Information collected under unique study identification will be returned to an independent data management unit for double data entry, range checks for data values, data queries to be resolved by data lock and coding according to standard operating procedures. Storage of
paper documents will be filed in a locked compactus controlled by proxy access to authorised personnel. Electronic files will be saved in a secure server storage system that is accessible by authorised personnel in accord with information technology security processes. Data sharing and use by organisations involved in the study is guaranteed by clinical trial research agreements between collaborating institutions. Upon study completion, participants will receive a summary of group results and study findings will be presented in publications and presentations.

Statistical analyses
All analyses will be performed according to a statistical analysis plan and on an intention-to-treat basis. Tests will be two-sided at the nominal level $\alpha = 0.05$. Data from each clinic will be pooled and summarised. Descriptive statistics will summarise the socio-demographic, clinical and overall health profile of the sample at baseline and 24 months follow-up; discrete variables will be assessed by frequencies and percentages and group differences tested using $\chi^2$ tests with calculation of odds ratios and 95% CI, whereas continuous variables will be assessed using mean (SD) or median (IQR) and group differences compared using independent $t$-tests or Mann-Whitney tests for variables with skewed distributions. For dichotomous variables, multiple logistic regression using 24 month results as dependent variables will evaluate the independent correlates of achieving the primary and secondary endpoints, after adjusting for baseline results and socio-demographic characteristics. For continuous variables, mixed-model ANOVA will be performed to assess within (pre-post) and between-group (control-intervention) differences over time and multiple linear regression used to evaluate important predictors of response.

Discussion
This study will be the first Australian-based trial to establish the evidence for the potential benefit of independent nurse-run clinics in the community and their cost-effectiveness in adults with common cardio-metabolic abnormalities. Findings will determine whether a nurse-implemented health and lifestyle modification program is more effective at improving MetS components and other major risk factors for the development of CVD and T2DM compared to standard care in adults who are in jeopardy the most by living away from urban areas where socioeconomic disadvantage is higher and specialist health care increasingly scarce [38, 39]. Importantly, our longer term study over 2-years with re-evaluation at 5 years of changes in health outcomes and health care utilisation will assess the enduring effect of nurse education and self-care and management coaching on CVD and T2DM prevention. Pending positive results and cost-effectiveness, we envisage more nurse-led clinics to be adopted outside of major cities and encompassing other chronic diseases as a key primary preventative initiative.

The novelty of the MODERN study will establish the feasibility of nurses to safely and effectively screen for the presence of MetS and deliver cardio-metabolic management based on a written protocol that integrates a systematic approach to individual assessment of risk and tailored management [16]. Nurse-led models of care provide the opportunity to maximise the use of finite resources and reserve health professional contacts for higher risk individuals. The response to the MODERN trial results however should not discount the role of general practitioners and specialists in managing chronic disease. In practical terms, MODERN will provide a model that improves the earlier detection and quality of preventative care through clear therapeutic targets for lifestyle change and risk factors, techniques to instigate behaviour change, schedules of follow-up visits and guidance to seek additional GP (or other health professional) input. With MetS driving the twin global epidemics of CVD and T2DM and the extent of the underlying burden imposed by MetS, the model being tested represents a pragmatic utilisation of the potential for nurses to manage cardio-metabolic disease and prevent or delay the development of CVD and/or T2DM.

The detailed health economic evaluation component of the MODERN trial has the distinct advantage to enhance the translation of research findings into sustainable improvements in routine clinical practice and patient outcomes. Supporting health economic analyses are lacking in the Australian context and evidence relating to the cost-effectiveness of nurse-led clinics remains an obstacle to overcome before next generation models of care can improve the quality of preventative health. At present, it is incontrovertible that components of the MetS cause risk for CVD and T2DM and the MetS is an ideal target for risk reduction programs, carried out by nurses with the competence and skill set that uphold cardio-metabolic disease prevention. The results of the MODERN trial will add to the evidence-base for the success or failure of a nurse-led health service delivery of care for combatting the likelihood of CVD and T2DM in individuals with the MetS.

Acknowledgements
Appreciation to Associate Professor John Furler for contributing to the funding success of the MODERN study.

Funding
The MODERN study is funded by a Project Grant (ID No. APP1069043) from the National Health and Medical Research Council of Australia who had no involvement in the design of the study and in writing the manuscript and will play no role in the collection, analysis and interpretation of data to be derived from the study. MJC is supported by a Future Leader Fellowship (Award Reference 100,802) from the National Heart Foundation of Australia.
Availability of data and materials
Not applicable.

Authors’ contributions
Both authors devised the study plan, including the integration of ideas and methodologies and are the recipients of project research funding. MJC was a major contributor in writing the manuscript. Both authors read and approved the final manuscript.

Ethics approval and consent to participate
The study protocol was approved by the Australian Catholic University Human Research Ethics Committee (Project No: 2014 244 V) and the Department of Health Human Research Ethics Committee (Project No:38/2014) for the release of Medicare claims information. All participants gave written informed consent. Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

Publisher’s Note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Author details
1 Pre-Clinical Disease and Prevention Unit, Baker Heart and Diabetes Institute, PO Box 6492, Melbourne, Victoria 3004, Australia. 2 Centre for Primary Care and Prevention, MacKillop Institute for Health Research, Australian Catholic University, Melbourne, Victoria, Australia. 3 Department of Diabetes, Faculty of Medicine, Nursing and Health Sciences, Monash University, Melbourne, Victoria, Australia.

Received: 20 September 2017 Accepted: 27 November 2017

References
