Evidence-to-Practice Gap in the Translation of Dietary Intake Advice for the Prevention of Cardiovascular Disease

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Statement of Originality

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. I give consent to the final version of my thesis being made available worldwide when deposited in the University's Digital Repository, unless an embargo has been approved for a determined period, subject to the provisions of the Copyright Act 1968.

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Tracy L Schumacher

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Publications and presentations arising from this thesis

Published manuscripts in peer-reviewed journals:

1. Schumacher TL, Dewar DL, Lubans DR, Morgan PJ, Watson J, Guest M, *et al.* Dietary patterns of adolescent girls attending schools in low-income communities highlight low consumption of core foods. *Nutrition & Dietetics*. 2014;71(2):127-34.

2. Schumacher TL, Burrows TL, Cliff DP, Jones RA, Okely AD, Baur LA, *et al.* Dietary intake is related to multifactor cardiovascular risk score in obese boys. *Healthcare*. 2014;2(3):282-98.

3. Schumacher TL, Burrows TL, Thompson DI, Spratt NJ, Callister R, Collins CE. Feasibility of recruiting families into a heart disease prevention program based on dietary patterns. *Nutrients*. 2015;7(8):7042-57.

4. Schumacher TL, Burrows TL, Neubeck L, Redfern J, Callister R, Collins CE. How dietary evidence for the prevention and treatment of CVD is translated into practise in those with, or at high risk of CVD: A systematic review. *Public Health Nutrition.* FirstView:1-16 DOI:10.1017/S1368980016001543.

5. Schumacher TL, Burrows TL, Rollo ME, Wood LG, Callister R, Collins CE. Comparison of fatty acid intakes assessed by a cardiovascular-specific food frequency questionnaire with red blood cell membrane fatty acids in hyperlipidaemic Australian adults: A validation study. **Accepted for publication July 2016:** *European Journal of Clinical Nutrition*

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7. Schumacher TL, Burrows TL, Thompson DI, Callister R, Spratt NJ, Collins CE. Preventative dietary patterns within the context of a family cardiovascular disease risk reduction intervention. **Under review:** *Healthcare*

Conference abstracts: Published in conference proceedings or peer-reviewed journals

1. Schumacher TL, Burrows TL, Boggess M, Cliff D, Callister R, Collins CE. Evaluation of a cardiovascular risk tool for use in overweight Australian children. Heart Foundation National Conference, Adelaide, Australia, May 2013 (poster: 240)

2. Schumacher TL, Burrows TL, Spratt N, Callister R, Collins CE. Can families improve their diet quality when trying to reduce their CVD risk? Australian Cardiac Rehabilitation Association 24th Annual Scientific Meeting, Sydney, Australia, August 2014 (oral presentation: concurrent free papers)

3. Schumacher TL, Burrows TL, Spratt NJ, Thompson DI, Callister R, Collins CE. "I know what I'm supposed to eat but…": What families participating in a CVD risk reduction diet study think about eating the 'right' food for heart health. Australian Cardiac Rehabilitation Association 24th Annual Scientific Meeting, Sydney, Australia, August 2014 (oral presentation: concurrent free papers)

4. Schumacher, TL, Burrows TL, Callister R, Spratt NJ, Collins CE. Families at risk of CVD can improve diet quality through targeted dietary advice.38th Annual Scientific Meeting of the Nutrition Society of Australia, Hobart, Australia, 2014 (poster presentation)

5. Schumacher TL, Burrows TL, Thompson DI, Spratt NJ, Callister R, Collins CE. Can families improve their dietary intakes when trying to reduce their CVD risk? 2nd Australian Society for Medical Research Satellite Scientific Meeting. Newcastle, Australia, April 2015 (oral presentation)

6. Schumacher TL, Burrows TL, Callister R, Spratt NJ, Thompson DI, Collins CE. "I know what I am supposed to eat but…" What families thing about eating the "right" food for heart health. 2nd Australian Society for Medical Research Satellite Scientific Meeting. Newcastle, Australia, April 2015 (poster presentation)

7. Schumacher TL, Burrows TL, Spratt N, Callister R, Collins CE. Effectiveness of a dietary intervention to reduce cardiovascular risk factors in a hyperlipidaemic population. Australian Cardiac Rehabilitation Association 25th Annual Scientific Meeting, Melbourne, Australia, August 2015 (oral presentation: research prize session)

8. Schumacher TL, Burrows TL, Rollo ME, Callister R, Collins CE. Initiating and measuring appropriate dietary changes in cardiovascular populations. 23rd Annual conference for Cardiovascular Health and Rehabilitation Association of NSW and ACT inc. Sydney, Australia, October 2015 (oral presentation)

9. Collins CE, Schumacher TL, Burrows TL, Spratt NJ, Callister R. Effectiveness of a nutrition knowledge translation intervention in the dietary management of hyperlipidaemia. 4th Annual NHMRC symposium on Research Translation jointly with CIPHER. Sydney, Australia, October 2015 (poster presentation)

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1. Schumacher TL. The Food Heart Family study. Rotary Club. Newcastle, Australia, February 2013 (oral presentation)

2. Schumacher TL. Dietary changes following a CVD diagnosis. Three minute thesis Finalist. University of Newcastle, Australia, July 2013 (oral presentation)

3. Schumacher TL. The Food Heart Family study. Hunter New England Cardiac committee. Newcastle, Australia, September 2013 (oral presentation)

4. Schumacher TL. Nutrition and CVD. Hunter New England Cardiac committee. Newcastle, Australia, June 2015 (oral presentation)

5. Schumacher TL. What is the evidence for diet in the prevention and treatment of cardiovascular disease?...and how do we apply it? Hunter dietitians peer networking and CPD event. Newcastle, Australia, November 2015 (oral presentation)

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Glossary of common abbreviations

Abbreviation	Full form of abbreviation
$AUD	Australian dollar
95% CI	95% confidence interval
ABS	Australian Bureau of Statistics
ACAES	Australian Child and Adolescent Eating Survey
ACS	Acute coronary syndrome
AES	Australian Eating Survey
AGHE	Australian guide to healthy eating
AHA	American Heart Association
AHS	Australian health survey
AIHW	Australian Institute of Health and Welfare
AMI	Acute myocardial infarction
ARFS	Australian Recommended Food Score
BGL	Blood glucose level
BMI	Body mass index
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
CE	Cholesterol esters
CerVD	Cerebrovascular disease
CETP	Cholesterol ester transfer protein

Abbreviation	Full form of abbreviation
CHD	Coronary heart disease
CHEQ UP	Cardiovascular health eating questionnaire using propensities
CHO	Carbohydrate
cm	Centimetre
CONSORT	Consolidated Standards of Reporting Trials
CR services	Cardiac rehabilitation services
CRP	C-reactive protein
CVD	Cardiovascular disease
CVD-AES	Cardiovascular disease Australian Eating Survey
DALY	Disability-adjusted life year
DHA	docosahexaenoic acid
DPA	docosapentaenoic acid
EPA	eicosapentaenoic acid
FFQ	Food frequency questionnaire
FHF	Love your Food, Love your Heart, Love your Family
g	gram
GI	Glycaemic index
GL	Glycaemic load
HDL	High-density lipoprotein cholesterol
HMRI	Hunter Medical Research Institute
HOMA IR	Homeostasis assessment model insulin resistance

Abbreviation	Full form of abbreviation
HR	Hazard ratio
ICH	Ideal cardiovascular health
IDL-C	Intermediate density lipoprotein cholesterol
IHD	Ischaemic heart disease
kg	Kilogram
kJ	Kilojoule
KPI	Key performance indicator
LA	Linoleic acid
LCn3	Long chain omega 3 fats
LDL	Low-density lipoprotein cholesterol
MetS	Metabolic syndrome
MI	Myocardial infarction
mL	Millilitre
mmHg	Millimetre of mercury
N/A	Not applicable
NHANES	National Health and Nutrition Examination Survey
NHF	National Heart Foundation of Australia
PAD	Peripheral artery disease
PCI	Percutaneous coronary intervention (stent)
RBC	Red blood cell
RCT	Randomised controlled trial

Abbreviation	Full form of abbreviation
RR	Risk ratio
T2DM	Type 2 diabetes mellitus
TG	Triglycerides
VLDL	Very low-density lipoprotein cholesterol
WHO	World Health Organization
YLD	Years of healthy life lost to disability
YLL	Years of healthy life lost to premature mortality
SD	Standard deviation

Abstract

The term cardiovascular disease (CVD) covers a wide range of conditions that affect the heart and blood vessels, the most recognised of which are myocardial infarctions (heart attacks) and strokes. CVDs may be acute or chronic and approximately 21% of Australian adults were affected by CVD in 2011-2012. A key strategy in CVD prevention and treatment is addressing modifiable risk factors, including those conferred by lifestyle such as smoking status, physical activity levels and nutrition. Dietary patterns influence several biomedical CVD risk factors such as high blood pressure, serum cholesterols, high body mass and type II diabetes. However, modifying dietary patterns to align with National recommendations for preventing chronic diseases such as CVD involves many environmental, social and individual factors that impact on food choices. The translation of current best evidence for the prevention and treatment of CVD needs to account for a wide range of individual influences and be applicable to a wide range of population groups.

The aims of this thesis were to assess gaps in the process of translating best-practice nutrition and dietary evidence to populations who may benefit from interventions targeting prevention of CVD events. The process was assessed in four areas of the translation spectrum:

- Eating patterns that contribute to the evidence for prevention of CVD
- Strategies that may be used by clinicians and practitioners to translate current evidence
- Identification of populations at increased risk of CVD that may be amenable to changes in dietary patterns
- Quantification of translation of best-practice evidence by measuring long-term changes in dietary patterns efficacious for CVD prevention.

The literature review identified that unsaturated fats and unrefined carbohydrates are the macronutrients most likely to influence the underlying biomedical factors, which play a role in CVD development. Eating patterns with increased intakes of unrefined plant-based foods, such as the Mediterranean, Portfolio and DASH diets

contain the types of fats and carbohydrates that are most beneficial to CVD health. Characteristics that make these eating patterns unique, such as the high unsaturated to saturated fat intake of the Mediterranean diet, the use of functional and nutraceutical foods of the Portfolio diet (soy foods and products containing plant sterols), and the low sodium focus of the DASH diet also individually contribute to reducing dietary CVD risk.

A systematic review of the literature was used to identify dietary interventions that had translated dietary evidence to populations with or at high risk of CVD or health professionals who were most likely to treat CVD patients. The results of the review aimed to inform strategies undertaken in two dietary translation studies in populations at high risk of CVD. The strategies identified in the published studies were of limited value in informing the best approach as few studies provided sufficient details regarding how the translation was accomplished.

Identification of populations at increased risk of CVD, who may be amenable to changing their dietary pattern in terms of CVD risk, was investigated through two secondary data analyses and two intervention studies. Both secondary data analyses assessed diet-related CVD risk in young people (< 15 years old) with at least one risk factor for CVD previously identified. The first investigated associations between dietary intakes, anthropometric and serum lipid data in prepubertal children with overweight or obesity as the known CVD risk factor. Results from this analysis indicated an association between multiple risk factors for CVD and dietary intakes in boys, with no association found in girls. The other secondary data analysis investigated low socioeconomic status as a CVD risk factor in a population of adolescent girls. The dietary patterns of the girls were shown to be sub-optimal, with 47% of the girls' dietary intakes being derived from energy-dense, nutrient-poor food and few girls meeting dietary guideline recommendations.

The intervention studies translated current evidence-based dietary advice to two different populations at increased risk of CVD. The first study targeted families with a demonstrated family history of CVD. This allowed both adults and children who may share genetic risks and lifestyle behaviours within a home environment

to be targeted. This study identified that CVD risk in this particular population was not always accurately perceived. Also, many influences, including facilitators and barriers were demonstrated to impact on family and individual eating patterns. However, families were able to make a number of small dietary changes within their home environment.

The second intervention study tested the translation of evidence-based dietary advice on serum lipids in a population who self-identified as at increased risk of CVD through increased serum cholesterol. The dietary advice delivered in a single personalised counselling session achieved reductions of 0.51mmol/L in total cholesterol, 0.28 mmol/L in LDL cholesterol and 0.38 mmol/L in triglycerides over an average of 9.5 ± 2.5 weeks. This was most likely mediated through a 1006kJ per day reduction in energy from nutrient poor foods.

The final area of research in this thesis was the validation of a method of measuring the translation of dietary advice for the prevention of CVD by quantifying efficacious changes in the dietary patterns of individuals at increased risk of CVD. A food frequency questionnaire (FFQ) previously validated in a healthy population was modified to include additional food items specifically associated with heart health. Dietary estimates were provided using both the original and modified versions of the FFQ by 39 study participants who self-identified as having increased serum cholesterol levels. Better estimates of dietary fatty acid intakes were achieved using the modified FFQ. Populations who identify as having CVD health conditions may be regularly consuming foods or supplements regarded as beneficial to that condition but which have been inadequately assessed using standard dietary assessment measures.

This thesis identified that there are gaps in the current translation of dietary evidence for the prevention of CVD where improvements may be possible. Although dietary patterns are considered a modifiable risk factor, adapting intakes towards dietary recommendations for improving heart health remains challenging. However, dietary modifications can be an effective component of primary and secondary CVD prevention and effective strategies that translate evidence-based dietary advice for populations with or at risk of CVD are needed.

Chapter 1: Introduction

1.1 Overview

The focus of this thesis is to examine disparities between current evidence relating to nutrition and diet in the prevention and management of cardiovascular disease (CVD) and the subsequent translation into practice. This thesis begins with definitions and prevalence rates of common CVDs, then summarises the economic and social costs of these disease states. Diet is a key lifestyle influence on many of the risk factors that predict CVD development and contributes to the progression of the disease. Diet and nutrition-based risk factors are largely preventable, yet not targeted at the same level of treatment as medication and surgery, creating an evidence-to-practice gap in the prevention of CVD. This chapter concludes with a description of the research aims and hypotheses, the thesis structure and a summary of the chapters to follow.

1.2 Common cardiovascular diseases

The World Health Organization (WHO) describes CVDs as a group of disorders that affect the heart and blood vessels (1). These disorders are further categorised according to the part of the body affected and may be of acute or chronic duration (2). Common CVD disorders are coronary heart disease (CHD), ischaemic heart disease (IHD), peripheral artery disease (PAD) and cerebrovascular disease (CerVD), which are also umbrella terms for additional specific diagnoses (see Figure 1.1) (1).

CHD is defined as a disease that affects the blood vessels supplying the heart whilst IHD is the term used when blood supply to the heart is reduced. IHD may be either acute or chronic and also covers a range of diagnoses (2). Acute IHD conditions includes angina pectoris (stable and unstable angina) and acute myocardial infarctions (AMI). Chronic IHD conditions are defined as those where the duration between ischaemic episode and admission to care is longer than 28 days and includes conditions such as atherosclerotic heart disease, which is better known as

coronary artery disease (CAD) (2). Acute coronary syndromes (ACS) is a term often used prior to CHD and IHD diagnoses and refers to the spectrum of clinical symptoms that are consistent with AMI (3). CerVD is the term used where the vessels specifically supplying the brain are affected (4). Stroke is a common example of this condition where an occlusion occurs in a vessel and results in reduced blood supply to the brain, spinal cord or retinal cell (5).

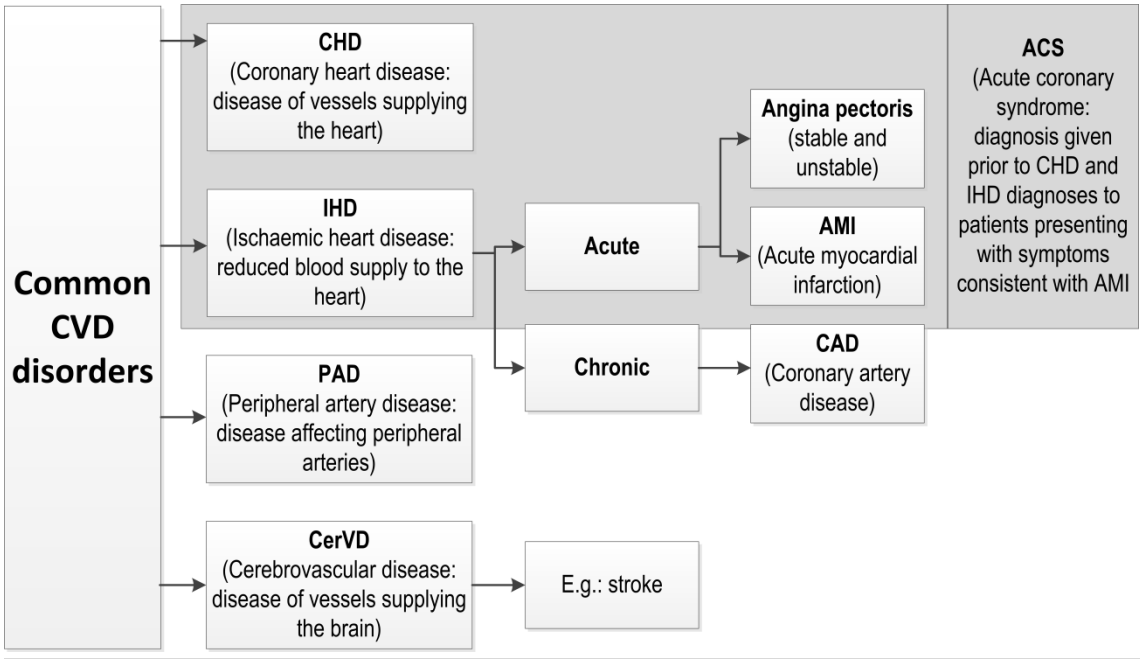

Figure 1.1: Common cardiovascular diseases

1.3 The economic and social costs of CVD

CVD has been identified as a National Health Priority Area in Australia since 1996 due to the high prevalence rates in the population and economic burden (6). The Australian Institute of Health and Welfare (AIHW) estimated that CVD was the underlying cause of 31% of deaths in 2011 (7). In the same year, the overall death rate from CHD was 1.8 times higher for males compared to females, and between 4-5 times higher for men in the 35-64 years age range compared to women (7). The prevalence rate of CVD was 20.9% of the Australian adult population in 2011-2012, with the condition self-reported as more prevalent in women (females: 22.1%, males 19.8%) and older populations (50.6% aged 65-74 years and 64.2% ≥75 years) (8).

The most recent release of Australian health expenditure by disease conditions shows that CVD accounted for $7,605 million or 12% of the total health care budget for the years 2008-2009 (9). Hospital admitted patient services accounted for 59% of this expenditure, with each hospital separation costing approximately $9406 (9). A subsequent 22% ($1,648 million) of CVD expenditure was for prescription pharmaceuticals to manage the condition. People presenting at hospitals with a first MI are a greater cost to treat, with an estimated cost per patient of $20,502 for the first 12 months following the event (2005 costing) (10).

A two year projection based on 2010-2011 prices was estimated to cost $AUD 7544 (standard deviation of $AUD 10,758) per person in direct healthcare expenditure for those aged 45 years or older with either established CVD or three or more CVD risk factors such as diabetes, hypercholesterolemia treated with medication, or hypertension despite medication (11). Of the 2856 subjects followed for two years, 37.2% had one hospitalisation in the intervening period and 6.4% had two hospitalisations (11). For the people experiencing ACS events, a cross-sectional study in 2012 showed that almost a third (27%) of those admitted to hospital with ACS symptoms had previously experienced a myocardial infarction (12).

Social costs of CVD are not included in economic models for health expenditure as they are not paid directly by the health system. Instead these are borne by the individuals with CVD and their families and carers and include costs relating to lost productivity, quality and length of life. Deloitte Access Economics estimated that in 2010 6,278 years of healthy life were lost from disability (YLDs) due to ACS events, with 4,109 YLDs owing to initial ACS events and 2,169 YLDs due to secondary ACS events (13). An estimated 10,021 people died in 2010 due to AMI (13). From this, years of healthy life lost (YLL) due to premature death from ACS were calculated as 103,975, with 48,610 YLL due to initial events and 55,365 YLL due to secondary events (13). As repeat events are more likely to end in mortality the greater burden of years lost falls on the secondary ACS events (13).

1.4 Risk factors for development of CVD

A risk factor is any factor that predisposes a person to a particular condition or increases the likelihood of an adverse event occurring. Risk factors, along with protective factors, may also be determinates of health (14). Age, sex, systolic blood pressure, total cholesterol and smoking status are commonly used in models, with other factors such as family history, ethnicity, and presence of other disease states or conditions often used (15-17).

The main risk factor for the development of CVD is age, which in conjunction with sex and genetic predisposition, is not able to be modified (18). However, other risk factors, such as those associated with lifestyle are largely modifiable (see Table 1.1). Lifestyle encompasses the areas of physical activity, smoking, alcohol consumption and diet and has strong influences on biomedical risk factors such as body mass, diabetes, increased blood pressure and serum cholesterol levels (19-21).

Table 1.1: Major risk factors for cardiovascular disease (14, 22-25)

CARDIOVASCULAR DISEASE RISK FACTORS		
Not modifiable	***Modifiable***	
	Biomedical risk factors	Behavioural risk factors
Age	High blood pressure	Physical inactivity
Sex	High cholesterol (total)	Smoking
Family history / genetics	Type II diabetes	Poor diet
Ethnicity	High body mass index	Risky alcohol consumption
Type I diabetes		

Multiple risk factors increase the risk of CVD and place a greater burden on the health care system (22). They are associated with reduced life expectancy, with those assessed at low risk estimated to live between 5.9 and 9.5 years longer than their multiple risk factor counterparts (26).

Data from the Australian Health Survey (AHS) (2011-2012) related to single risk factors that may be affected by poor diet showed 32% of adults had high blood pressure, 32.8% had abnormal total cholesterol results, 33.2% had abnormal LDL results, 23.1% had abnormal HDL results and 63.2% of people had dyslipidaemia,

with approximately 5% of adults also diagnosed with either type I or II diabetes (27, 28).

The AHS (2011-2012) also found the combined prevalence of overweight or obese adults to be 63.4%, with a higher prevalence in males (70.3%) compared to females (56.2%) (29). The prevalence of overweight and obesity in children was 17.7% and 7.6% respectively (29), with the proportion of girls (27.1%) who are overweight or obese was higher than that of boys (23.6%).

1.5 The role of nutrition

In 2010, dietary risks were ranked as the leading contributor to total burden of disease in Australia (11%), followed by high body mass index (9%) (27). Dietary risk factors also accounted for high disability-adjusted life year (DALY) burden on a global scale, with diets low in nuts and seeds attributed 40%, fruit 30% and oily fish 22%, and diets high in sodium attributed 17% and processed meats 13% (30).

The Australian recommendations for behavioural risk factors related to diet address fruit and vegetable intakes, with two pieces of fruit and five servings of vegetables per day recommended for adults to lower risk of chronic disease, and age-specific recommendations for children (31). The AHS (2011-2012) found that 54% of Australians met the age-specific guidelines for fruit, but only 6.8% met the guidelines for vegetables (29). The guideline for minimisation of lifetime risk of harm from alcohol is to limit intake to no more than two standard drinks on any day (32). The AHS (2011-2012) showed 19.5% of adult Australians exceeded this recommendation, although prevalence has decreased since 2007-2008 (20.9%).

Systematic analysis of dietary factors and their contribution to disease burden showed that protective dietary risk factors such as fruits, vegetables and wholegrains tended to be correlated with each other, as well as being negatively correlated with harmful risk factors (30). The same study concluded that relative risks calculated using dietary patterns and singular risk factors were similar (30). This highlights the importance of overall dietary patterns. A further challenge is then to quantify changes in dietary patterns and the impact this makes on the

health of an individual. A detailed justification of these issues can be found in Chapter 2 (section 1.2: The role of dietary patterns).

Making dietary changes to improve heart health can be difficult as complex environmental, social and individual influences impact on food choices (33). Individuals can also be at different stages of readiness to consider or make dietary changes, which impacts on type and extent of information provided, as well as techniques utilised in the delivery process (33). Therefore, effective dietary cardiovascular interventions must be able to account for large variations in individual circumstances and readiness.

1.6 Research aims and hypotheses

The overall aim of this thesis is to assess the evidence-to-practice gap in the translation of dietary advice for the prevention of CVD. In particular, to identify:

A. efficacious eating patterns in lowering risk factors for CVD
B. intervention strategies demonstrated to improve eating patterns of CVD populations
C. populations with or at increased risk of CVD that are amenable to change in dietary patterns, and
D. methods of quantifying dietary intakes shown to improve dietary CVD risk factors.

Secondary aims relating to individual chapters are summarised below in Table 1.2

Table 1.2: Research aims and hypotheses

RESEARCH TOPIC AREAS, QUESTIONS ADDRESSED AND RESEARCH HYPOTHESIS			
Aim	**Secondary aims for individual chapters**	**Research hypothesis**	**Chp**
A	To describe the role nutrition plays in the development of atherosclerosis	No hypothesis	2
	Identify how eating patterns that lower CVD risk can be quantified	No hypothesis	2
	Describe populations at increased risk of CVD	No hypothesis	2
B	To identify aspects of successful health service nutrition translation studies in CVD in terms of methodology, including implementation strategies, program design, resources, use of technology and message transmission channels	Successful health service nutrition translation studies in CVD will follow a clearly defined protocol, yet allow flexibility in the delivery	3

Table 1.2: Research aims and hypotheses

RESEARCH TOPIC AREAS, QUESTIONS ADDRESSED AND RESEARCH HYPOTHESIS			
Aim	**Secondary aims for individual chapters**	**Research hypothesis**	**Chp**
	Evaluate the methodological quality of these translation studies and the effectiveness of the nutrition-evidence translation on diet-related CVD risk factors	Methodology of dietary translation studies will be of low translation quality	3
	Develop a multifactor CVD risk score for children using paediatric reference ranges and assess its application in overweight and obese pre-pubertal children	Overweight and obese children will be at increased risk of developing CVD.	4
C	Examine the differences in dietary intakes of overweight and obese children in high and low category CVD risk scores	Children in high CVD risk categories will have significantly poorer nutrient intakes compared to those in low CVD risk categories	4
	Examine the strength of association between anthropometric and biomedical risk factors in overweight and obese boys and girls	Sub-optimal anthropometrics related to adiposity will have significant associations with biomedical risk factors	4
C	Determine the relative contributions of healthy core foods and energy-dense, nutrient-poor food groups to dietary intakes in adolescent girls attending schools in low-income communities in an Australian population	Girls from low socioeconomic positions would have low intakes of core foods and disproportionally high intakes of energy-dense, nutrient poor foods	5
	Investigate differences in dietary behaviours by weight status (BMI) in adolescent girls attending schools in low-income communities	Dietary behaviours in adolescent girls attending schools in low-income communities will vary based on weight status	5
	To test the feasibility of recruiting and retaining families at increased risk of CVD into a dietary intervention program targeting alignment of existing eating patterns with heart health recommendations	Families recruited into a dietary intervention study on the basis of one member having had an adverse CVD event, or being assessed at high risk of CVD, may be more receptive to changing their diet	6
B,C	To assess the dietary changes made by families undertaking a CVD prevention program	Families will make small, incremental changes to a number of food groups to align dietary intakes with heart health recommendations	6
	To test the acceptability of the dietary CVD prevention program	Families will find the overall program acceptable and highlight areas where improvements may be achieved	6
	To identify how risk of CVD is perceived by those at increased risk	Risk perception will correlate with actual risk in those identified at high risk of CVD	7
B,C	To investigate perceptions of the role healthy eating patterns in those at increased risk of CVD	Healthy eating patterns will be recognised as an important component in reducing CVD risk	7
	To investigate the extent of family influence on dietary patterns	Family will have considerable influence over dietary patterns	7

Table 1.2: Research aims and hypotheses

RESEARCH TOPIC AREAS, QUESTIONS ADDRESSED AND RESEARCH HYPOTHESIS			
Aim	**Secondary aims for individual chapters**	**Research hypothesis**	**Chp**
B,C	Determine the effectiveness of a brief dietetic intervention on diet-related CVD risk factors in hyperlipidaemic adults	A brief dietetic intervention using best-available dietary evidence will improve lipid profiles	8
D	To develop and validate a tool to quantify dietary intakes that is specific to populations with risk factors for CVD	Existing dietary intake tools designed for healthy populations will require modification to improve dietary intake estimates in populations at increased risk of CVD.	9

Abbreviations: Chp Chapter; CVD cardiovascular disease

1.7 Thesis structure and chapter description

An overview of the thesis structure is presented in Figure 1.2. This thesis begins with a review of the literature (Chapter 2). The focus of the literature review centres on dietary risk factors that contribute to the development of the disease and biomedical targets posited to prevent or delay the disease. The review identifies eating patterns protective against the development of CVD and measures used to quantify them. The literature review also identifies potential populations that may benefit from preventative CVD dietary patterns. Chapter 2 concludes with a summary of current guidelines for the treatment and prevention of CVD.

The objective of the systematic review in Chapter 3 was to identify how evidence from nutrition and dietetic research in populations with or at increased risk of CVD is translated into practice by clinicians. This is based on sections of the knowledge translation cycle and indicates the numerous stages to be addressed in order for evidence from research to progress to the end-stage user. In particular, the systematic review aimed to identify strategies that have successfully translated dietary knowledge for the purpose of prevention and treatment of CVD that can be replicated in a local population using current best evidence for dietary advice. This included identifying strategies to improve the dietetic practices of clinicians who work with CVD patients on a regular basis.

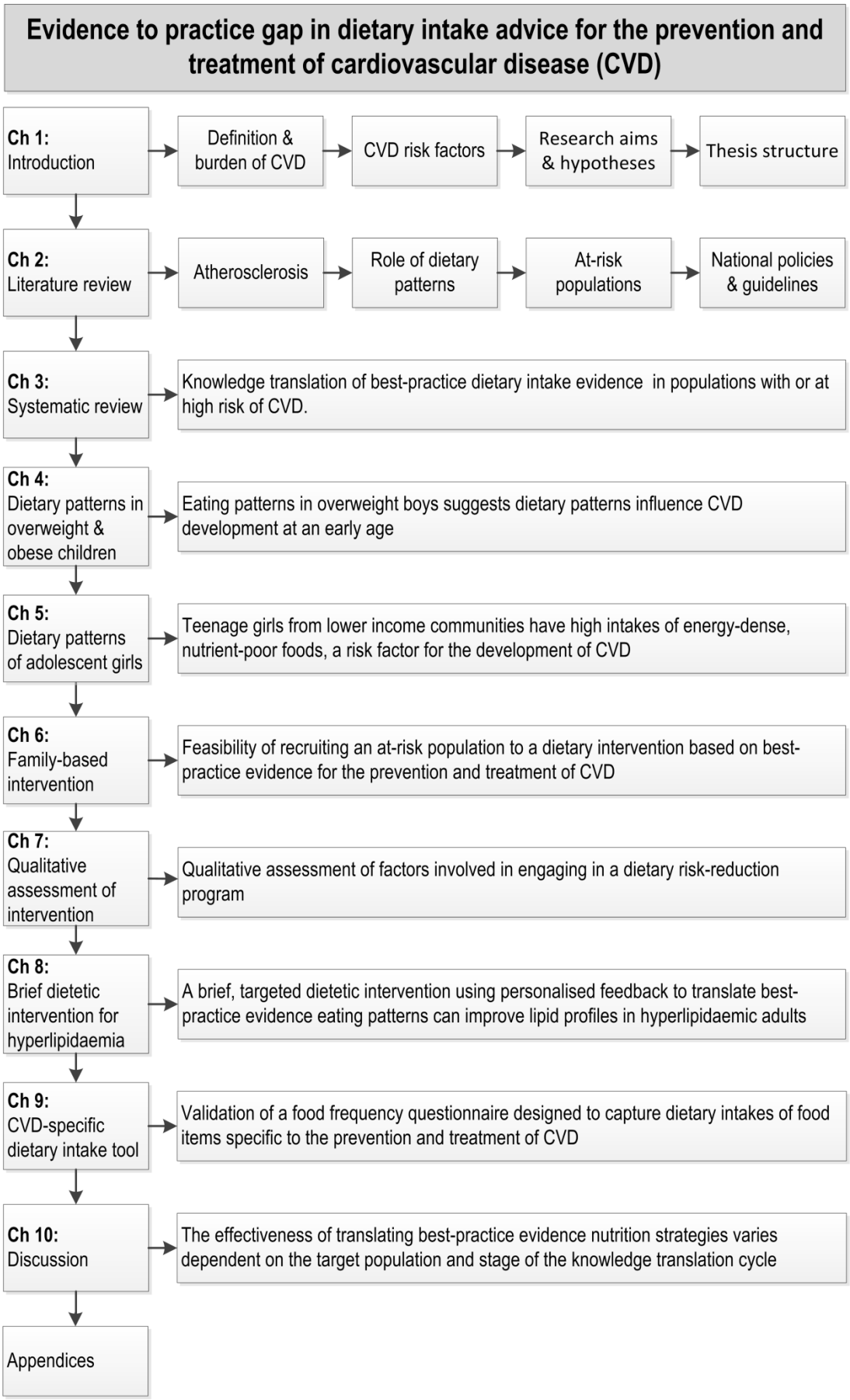

Figure 1.2: Thesis structure

Secondary data relating to dietary intakes of children and adolescents were obtained to establish the role of nutrition in the development of CVD during their formative years and described in Chapters 4 and 5. Firstly, dietary intake data from prepubertal boys and girls with a demonstrated prior risk factor for CVD were obtained. All shared the same anthropometric risk factor, though of differing magnitudes, as they were determined to be overweight or obese. An overall CVD risk score from paediatric reference ranges was developed to determine if an association between dietary intakes and the number and extent of CVD risk markers existed in children at a young age. The second data source was from females at a later stage of development (adolescence) with a different common risk factor for CVD, which was a low socio-economic background (15). The data were stratified by weight status to determine whether weight status as a risk factor would influence the overall diet quality being consumed.

Chapter 6 is a description of the feasibility of undertaking a dietary intervention that targeted adults with or at increased risk of CVD and their offspring. The intervention dietary advice was informed by findings in the literature review with the intervention structure influenced by findings from the systematic review. In addition to testing feasibility, the acceptability of the intervention was evaluated and the types of dietary changes made by families assessed.

Chapter 7 is a qualitative study that describes the motivations of participants in Chapter 6 to enter into an intervention that aimed to improve their eating patterns and decrease their risk of CVD. This study is designed to improve our understanding of some of the processes that affect eating decisions for those at increased risk of CVD. These findings may be useful for determining which behaviour change strategies will be of most value in future studies.

Chapter 8 reports the results from the efficacy of a refined version of the previous intervention. In order to test the effectiveness of the dietary patterns and the methods by which the knowledge was translated, a new population was targeted. This population were those who identified as at increased risk of CVD through elevated levels of blood cholesterol (hyperlipidaemia). This study showed that it is possible to reduce risk conferred by eating patterns in a single, highly personalised

dietetic session that encompasses knowledge translation and behaviour change strategies.

Findings from the literature review indicated a need for a specific tool to measure eating patterns for those at increased risk of CVD. Data were collected on eating patterns in the first intervention trial and tested for accuracy in the second. Chapter 9 reports on a validation study of a food frequency questionnaire that collected data on dietary patterns in adults at increased risk of CVD due to hyperlipidaemia to inform dietetic treatment options following diagnosis. The primary outcome for validation was that of fatty acid intakes as this macronutrient has been shown to effect cholesterol components and triglycerides.

The final discussion chapter of the thesis reviews and integrates the findings from the series of studies. The current best evidence for dietetic advice is effective in reducing some CVD risk factors if translated using appropriate strategies. Interventions designed to adapt eating patterns to those shown to reduce risk factors for CVD may be of benefit in overweight or obese children, or adolescents from economically disadvantaged areas. Different strategies need to be found to target young people likely to be at increased risk of CVD, as intervening through family risk was found to be of limited success. A CVD-specific food frequency questionnaire can provide a reasonable estimate of dietary fatty acids intakes in populations at increased risk of CVD through hyperlipidaemia. However, validation of many other dietary factors, such as the quality of the diet pattern, quantities of energy-dense, nutrient-poor food choices and intakes of unrefined plant foods, particularly those high in soluble and insoluble fibre that also impact on CVD risk factors is also required.

References

1. World Health Organization. World Health Organization media centre fact sheet Nº317 Cardiovascular diseases (CVDs) Geneva, Switzerland: World Health Organization; 2015 [updated January 2015; cited 2015 2nd June]. Available from: http://www.who.int/mediacentre/factsheets/fs317/en/.

2. International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10 Version: 2015) [Internet]. World Health Organization. 2015 [cited 2nd June, 2015]. Available from: http://apps.who.int/classifications/icd10/browse/2015/en.

3. Kumar A, Cannon CP. Acute Coronary Syndromes: Diagnosis and Management, Part I. *Mayo Clin Proc*. 2009;84(10):917-38.

4. World Health Organization. Cardiovascular diseases Denmark: European World Health Organization; 2013 [cited 2013 26 March]. Available from: http://www.euro.who.int/en/what-we-do/health-topics/noncommunicable-diseases/cardiovascular-diseases/definition.

5. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJ, Culebras A, *et al.* An Updated Definition of Stroke for the 21st Century: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke*. 2013;44(7):2064-89.

6. National Health and Medical Research Council. Cardiovascular disease (National Health Priority Area) Canberra: National Health and Medical Research Council; 2014 [cited 2015 12 October]. Available from: http://www.nhmrc.gov.au/grants-funding/research-funding-statistics-and-data/cardiovascular-disease-nhpa.

7. Australian Institute of Health and Welfare. Cardiovascular disease, diabetes and chronic kidney disease-Australian facts: Mortality. Cardiovascular, diabetes and chronic kidney disease series no.1. Canberra: AIHW; 2014.

8. Australian Institute of Health and Welfare. Cardiovascular disease, diabetes and chronic kidney disease - Australian Fact: Prevalence and Incidence. Cardiovascular, diabetes and chonic kidney disease series no. 2. Canberra: Australian Institute of Health and Welfare; 2014.

9. Australian Institute of Health and Welfare. Health care expenditure of cardiovascular diseases 2008-09. Canberra: AIHW; 2014.

10. Ioannides-Demos LL, Makarounas-Kirchmann K, Ashton E, Stoelwinder J, McNeil JJ. Cost of Myocardial Infarction to the Australian Community A Prospective, Multicentre Survey. *Clin Drug Investig*. 2010;30(8):533-43.

11. Ademi Z, Liew D, Zomer E, Gorelik A, Hollingsworth B, Steg PG, *et al.* Outcomes and excess costs among patients with cardiovascular disease. *Heart Lung Circ*. 2013;22(9):724-30.

12. Chew DP, French J, Briffa TG, Hammett CJ, Ellis CJ, Ranasinghe I, *et al.* Acute coronary syndrome care across Australia and New Zealand: the SNAPSHOT ACS study. *Med J Aust*. 2013;199(3):185-91.

13. Deloitte Access Economics. ACS in Perspective: The importance of secondary prevention. Deloitte Access Economics Pty Ltd, 2011.

14. Australian Institute of Health and Welfare. Cardiovascular disease: Australian Facts 2011. Canberra: AIHW; 2011.

15. Perk J, De Backer G, Gohlke H, Graham I, Reiner Z, Verschuren M, *et al.* European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts). *Eur Heart J*. 2012;33(13):1635-701.

16. National Vascular Disease Prevention Alliance. Guidelines for the assessment of absolute cardiovascular disease risk. National Heart Foundation of Australia; 2009.

17. Kavousi M, Leening MG, Nanchen D, *et al.* Comparison of application of the ACC/AHA guidelines, Adult Treatment Panel III guidelines, and European Society of Cardiology guidelines for cardiovascular disease prevention in a European cohort. *JAMA*. 2014;311(14):1416-23.

18. Australian Bureau of Statistics. Australian demographic statistics Canberra: Australian Bureau of Statistics; 2011 [cited 2013 8 April]. Available from: http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/AB7C58CECD09B058CA257968000C6320/$File/31010_jun%202011.pdf.

19. Canfi A, Gepner Y, Schwarzfuchs D, Golan R, Shahar DR, Fraser D, *et al.* Effect of Changes in the Intake of Weight of Specific Food Groups on Successful Body Weight Loss during a Multi–Dietary Strategy Intervention Trial. *J Am Coll Nutr*. 2011;30(6):491-501.

20. Brunner E, Rees K, Ward K, Burke M, Thorogood M. Dietary advice for reducing cardiovascular risk. Cochrane DB Syst Rev [Internet]. 2007; (4). Available from: http://0-onlinelibrary.wiley.com.library.newcastle.edu.au/doi/10.1002/14651858.CD002128.pub3/abstract.

21. Brand-Miller J, McMillan-Price J, Steinbeck K, Caterson I. Carbohydrates--the good, the bad and the whole grain. *Asia Pac J Clin Nutr*. 2008;17 Suppl 1:16-9.

22. O'Brien K. Living dangerously: Australians with multiple risk factors for cardiovascular disease. *Bulletin* 2005;No.24 Cat. No. AUS 57.

23. Begg SJ, Vos T, Barker B, Stanley L, Lopez AD. Burden of disease and injury in Australia in the new millennium: Measuring health loss from diseases, injuries and risk factors. *Med J Aust*. 2008;188(1):36-40.

24. Lusis AJ. Atherosclerosis. *Nature*. 2000;407(6801):233-41.

25. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, *et al.* Defining and setting national goals for cardiovascular health promotion and disease

reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation*. 2010;121(4):586-613.

26. Stamler J, Stamler R, Neaton JD, *et al.* Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: Findings for 5 large cohorts of young adult and middle-aged men and women. *JAMA*. 1999;282(21):2012-8.

27. Australian Institute of Health and Welfare. Australia's health 2014. Canberra: AIHW; 2014.

28. Australian Bureau of Statistics. Australian Health Survey: Biomedical results for Chronic Diseases, 2011-12. Canberra: Commonwealth of Australia; 2013.

29. Australian Bureau of Statistics. 4364.0.55.001 - Australian Health Survey: First Results, 2011-2012. Canberra: Commonweath of Australia; 2012.

30. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, *et al.* A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012;380(9859):2224-60.

31. National Health and Medical Research Council. Australian Dietary Guidelines. In: National Health and Medical Research Council, editor. Canberra: Commonwealth of Australia; 2013.

32. National Health and Medical Research Council. Australian Guidelines to reduce health risks from drinking alcohol. Canberra: Commonwealth of Australia; 2009.

33. Rippe JM, Waite MA. Implementing Heart Healthy Dietary Guidelines: Moving From Ideal to Real. *Am J Lifestyle Med*. 2012;6(2):96-112.

Chapter 2: Literature review

The literature review aims to:

- Describe the process of atherosclerosis and how this contributes to cardiovascular diseases
- Examine the role dietary patterns play in the process of atherosclerotic development
- Identify dietary patterns that may assist in halting or reversing atherosclerosis
- Identify how eating patterns related to CVD health can be quantified in order to prove effectiveness
- Identify characteristics of people who would most benefit from dietary pattern modification
- Provide an overview of the current policies and recommendations in place for those with or at increased risk of CVD (see Figure 2.1)

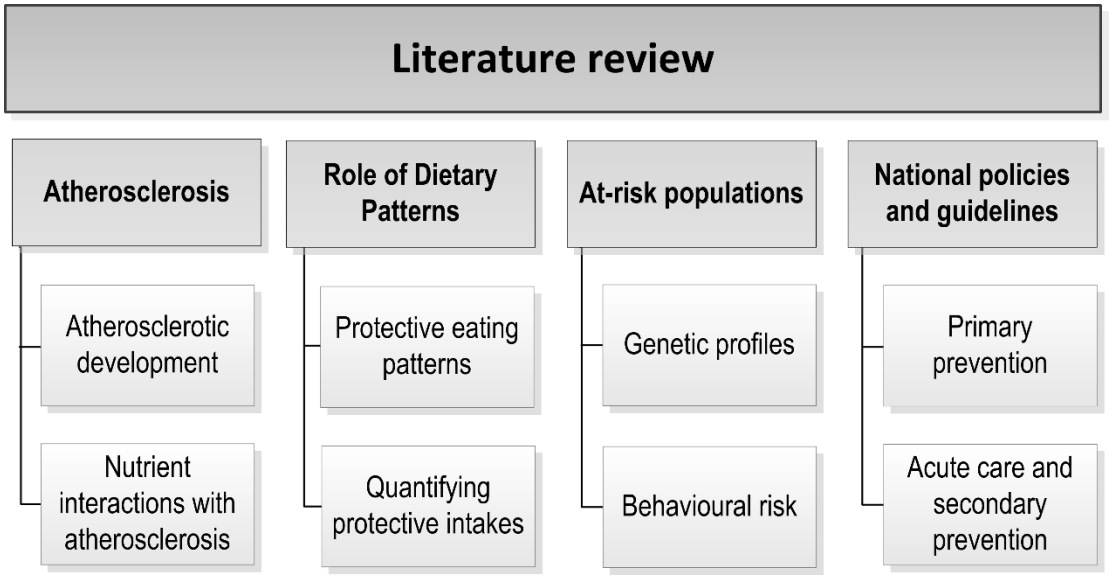

Figure 2.1: Structure of the literature review

2.1 Atherosclerosis and cardiovascular diseases

2.1.1 Development of atherosclerosis

2.1.1.1 *Atherosclerosis*

Atherosclerosis is the underlying disease state in most cardiovascular diseases (CVD). It is a progressive, chronic disease of the large arteries, with initial signs of the disease shown to occur in some children in their first decade of life (1, 2). A healthy artery consists of three distinct layers of cells: the intima or the inner most layer which contains the endothelium, the cells that are in direct contact with the artery blood flow; the media, the middle layer consisting of smooth muscle cells; and the adventitia, the outer layer of connective tissues and smooth muscle cells (2). Atherosclerosis is characterised by the accumulation of lipids and fibrous elements under the endothelium in the intima layer (2). The first stage of atherosclerosis is the appearance of lesions or fatty streaks in major blood vessels. This occurs at sites where flow of blood is disturbed or when there is an increased amount of stimuli present, such as excess lipids in the blood (1, 2). This causes the endothelium to become more permeable and allows the entry and retention of low-density lipoproteins into the intima, which subsequently become oxidised. In turn, the endothelium expresses adhesion molecules which capture monocytes that mature into macrophages once inside the artery wall (1). The macrophages then absorb the oxidised lipoproteins and form foam cells. Over time, the

foam cells die and become part of the core of the lesion or fatty streak. Subsequent to this, lesions can either become permanently or temporarily stable or progress into the next stage. This next stage involves the recruitment of smooth muscle cells, which migrate from the media to the intima layer (1). Once in the intima layer, the smooth muscle cells proliferate and produce a matrix containing substances such as collagen and elastin and this forms a cap over the plaque produced by contents of foam cells that have died by apoptosis (1). Plaques vulnerable to rupture have generally been shown to have thin plaque caps which have not been adequately maintained or have a high macrophage content (1, 2). Once ruptured, the contents of the plaque come into direct contact with the blood flow which triggers coagulation, forming a thrombus. This thrombus can then cause a blockage in a subsequent blood vessel. Thrombi to heart vessels are usually termed heart attacks, while thrombi lodging in vessels supplying the brain are termed strokes.

The relationship between blood cholesterol and atherosclerotic CVD is strong and has been demonstrated from meta-analysis of prospective cohort studies of approximately 900,000 participants (3). Every 1mmol/L reduction in usual serum total cholesterol was associated with a decrease in the hazard ratio for IHD mortality of 0.44 for those aged 40-49, 0.58 for those aged 50-59 years, 0.72 for those 60-69 years, 0.82 for those 70-79 years and 0.85 for those aged 80-89 years (3).

2.1.1.2 *Serum lipids*

Total cholesterol is a combination of specific lipoprotein families, namely the chylomicrons, very low density lipoproteins (VLDL), intermediate density lipoproteins (IDL), low density lipoproteins and high density lipoproteins (HDL) (4-7). Lipoproteins are particles that transport a blend of proteins and lipids through plasma and vary in size, density, composition and function. Apolipoproteins are proteins on the surface of the lipid particles and stabilise the core and assist with regulation of lipid levels (8). Chylomicrons are the principle carriers of triglycerides (TG) in the non-fasting state, with VLDL taking the same role in the fasting state (9). The primary purpose of LDL is to provide cells with cholesterol esters (10). The esters are used to modulate the fluidity of the cell membrane, as a substrate for steroid hormones and vitamin D, and as a

precursor for bile acids (11). The role of HDL is the clearance and transport of tissue cholesterols to the liver for reprocessing (10).

TGs are absorbed from the small intestine into chylomicrons which are transported into the lymphatic system and thereby into the wider circulatory system, acquiring apolipoproteins on the way (8). The apolipoproteins allow the chylomicrons to bind to adipose tissue and muscle cells where the TGs are hydrolysed (8). Under normal conditions, the remaining cholesterol-rich particle (chylomicron remnant) is then transported to the liver for processing (8). Chylomicron remnants that remain in circulation have been demonstrated experimentally to contribute to the progression of atherosclerosis through a number of pathways involving inflammation (8). VLDL-Cs containing TGs are produced in the liver via a complex regulatory system (8). Once excreted from the liver to the plasma, cholesterol esters are added. VLDL-C TGs are hydrolysed, causing the VLDL-C to shrink into smaller VLDL-C and then into IDL-C and subsequently into LDL-C (8). The cholesterol ester transport protein (CETP) is responsible for transferring cholesterol esters (CE) and TGs between different classes of lipoproteins (12).

ApoB is the major apolipoprotein of LDL and it exists in two forms (13). ApoB100 is expressed on lipoproteins only from the liver and therefore only present on VLDL, IDL and LDL (13). ApoB48, an abbreviated version of ApoB100, is expressed on particles from the intestine and thus found on chylomicrons (13). Both ApoB types can bind to sites in the intima and this retention and subsequent modification of lipoproteins by oxidation in the intima results in the development of foam cells (13, 14).

HDL is protective of the development of CVD because of its role in reverse cholesterol transport and importantly, its relationship with CVD is independent of other risk factors (15, 16). It is also hypothesised to inhibit the expression of adhesion molecules on endothelial cells, inhibit the oxidation of LDL and promote the efflux of cholesterol from foam cells (10).

TGs are not directly atherogenic, but are a biomarker of the disease state because of their relationship with atherogenic particles and inflammation states (17). They are associated with the development of metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) as a marker of insulin resistance (18). The complete role of TGs in the

development of atherosclerosis is still unclear, but recognised to have intricate biological relationships with lipoproteins, particularly HDL-C, where a strong inverse association is found (8). In healthy subjects, VLDL concentration determines the amount of CE transferred between HDL-C and LDL-C and the TG-rich VLDL and chylomicrons via the CETP (12). In hypertriglyceridaemia, CETP is the rate limiting factor and removes CE from HDL and LDL and transfers it to VLDL and chylomicrons. At the same time, it removes TG from the VLDL and chylomicrons and transfers it to HDL and LDL. The altered content of HDL and LDL causes them to become functionally aberrant, disrupting their cholesterol delivery and removal systems (19). Hydrolysis of the TG-rich VLDL and chylomicrons in the presence of high plasma TG can result in the production of remnant lipoprotein particles (RLP) (8, 20), which are posited to be atherogenic (21). Non-fasting levels of TGs have been shown to be strongly correlated with these remnant lipoprotein particles and a better predictor of incidence of CVD events compared to fasting samples (8, 20).

Much is still unknown in this area as increased levels of RLP have been shown to reduce rates of mortality following an AMI event (6). A recent theory proposes that low-levels of inflammation in CVD may be due to elevated RLPs, rather than to excess levels of LDL (22). BMI has also been shown to correlate with an increase in the lipoproteins that are more likely to be atherogenic, even in individuals with lipid levels in acceptable ranges (23).

2.1.1.3 *Hypertension*

Hypertension is a different disease state to atherosclerosis (24, 25) but causes similar pathological changes in blood vessels, mainly in the small arteries and arterioles. Atherosclerosis development is accelerated by hypertension, due to the increased pressures and forces that cause vessel walls to remodel, leading to reduced lumen diameter, increased thickness of the media, and disrupted endothelial function (24, 26). In particular, increased blood pressure in combination with a low serum ratio of HDL to total cholesterol has been shown to be a strong predictor of mortality from specific atherosclerotic diseases such as IHD (3). In an analysis of disability-adjusted life years due to IHD, the disease burden attributed to hypertension and increased total cholesterol were 53% and 29% respectively (27).

2.1.2Nutrient interactions with atherosclerotic development

2.1.2.1*Macronutrients*

The macronutrients of proteins, fats and carbohydrates are major contributors to intakes of dietary energy. The current understanding of the role these nutrients play in the development of atherosclerosis and CVD is described below.

Fats (saturated, monounsaturated and polyunsaturated)

Dietary fat intakes and their respective sub-classes of saturated, monounsaturated and polyunsaturated fatty acids are contentious in the prevention and treatment of cardiovascular disease (28-31). Dietary intakes of fats are difficult to measure by biomarkers because many types of fat can be synthesised within the body and therefore confound associations with dietary consumption (32).

The role of saturated fats in the development of CVD is controversial (28-30). A recent meta-analysis of prospective cohort studies investigating classes of fatty acid consumed in the diet in relation to cardiovascular-related mortality found little relationship between saturated fat intake and either CVD (0.97 95%CI: 0.84, 1.12) or CHD mortality (1.15, 95%CI: 0.97, 1.36) (33). Of particular importance in this meta-analysis was the low confidence in the certainty of the estimates. These were determined to be low, due to low precision and major inconsistencies and therefore methodological limitations applied to these findings. Other methodological considerations in reviews investigating the role of saturated fat in CVD have also contributed to confusion related to dietary intakes and the saturated fat debate (28).

The most recent Cochrane Review investigating dietary intakes of saturated fat and CVD reported a 17% reduction in cardiovascular events in those who had reduced their intakes of saturated fat intakes for at least two years, compared to those consuming a usual diet (34). As to which macronutrient was best to replace saturated fat with, no benefit was shown by replacing saturated fat with either carbohydrate or protein (34). However, substituting saturated fat with polyunsaturated or monounsaturated fats may provide better protection against the developing CVD. More data are required to inform the statement related to substitution with monounsaturated fats, as limited studies were available at the time of that review. However, this review did not include one of

the most recent large CVD prevention studies, the Prevención con Dieta Mediterránea (PREDIMED) study, as it was still ongoing at the time (35). It is possible that inclusion of this study would provide substantially stronger evidence for the inclusion of monounsaturated fats, as this study recommended intakes of 50g olive oil each day for personal use and provided a litre each week for family use in order to test the efficacy of a Mediterranean diet on the reduction in major cardiovascular events.

Similar to this, incidence of CHD in relation to saturated fat intakes was analysed in over 84,500 women from the Nurse's Health Study (1980-2010) and almost 43,000 men from the Health Professionals Follow-up Study (1986-2010) (36). Results of this analysis demonstrated that the replacement of 5% of energy from saturated fat, with polyunsaturated and monounsaturated fats indicated a hazard ratio (HR) for incidence of CHD: 0.75 (95%CI: 0.67, 0.84) and HR: 0.85 (95%CI: 0.74, 0.97) respectively (36).

Modification of fat types in diets used for secondary prevention in populations with established CVD showed similarly low impact on cardiovascular mortality (31). Meta-analysis of 12 randomised controlled trials including 6744 participants with established CVD showed no effect for reducing fat intakes or modifying fat types on mortality (RR 0.93, 95%CI: 0.66, 1.31 and RR 0.96, 95% CI: 0.65, 1.42, respectively) or on combined CVD events (RR 0.93, 95% CI:0.65, 1.34 and RR 0.85 (95% CI: 0.63, 1.15, respectively).

Saturated fats have been linked to increases in serum LDL through: reductions in the number of LDL receptors on cell membranes, thereby impacting on the rate of serum LDL clearance; influencing gene expression for proteins regulating cholesterol synthesis and metabolism; and acting as coactivators that promote hepatic secretion of VLDL (37, 38). The length of the saturated fatty acid chain of fats supplied via the diet has also been linked to elevated LDL cholesterol levels, with stearic acid (C18:0) shown to raise LDL cholesterol more than lauric (C12:0) or myristic acid (C14:0)(38). Conversely, saturated fats have also been demonstrated to increase fractions of HDL cholesterols (39, 40).

The impact of consumption of dietary saturated fats in comparison to monounsaturated fats on serum cholesterols was demonstrated recently in habitual diets using common foods predominantly composed of saturated (butter) or monounsaturated fatty acids (olive oil) (41). In a five week cross-over trial in 47 healthy men and women, moderate

amounts of butter (approximately 4.5% of daily energy intake) were compared to equivalent amounts of olive oil. Total cholesterol increased from 5.22 ±0.90 mmol/L to 5.50±0.98 mmol/L ($p<0.005$) on the butter diet, with no change in the olive oil diet. Although increases in both LDL and HDL contributed to the rise in total cholesterol, the ratio of total:HDL cholesterol also slightly increased from 3.24±0.89 to 3.29±0.92 ($p<0.01$), showing that the rises were proportional.

Differences in the metabolic effects of saturated versus polyunsaturated fats was demonstrated in 61 abdominally obese subjects fed isocaloric diets for 10 weeks (42). Diets were either high in saturated fat (butter), accounting for approximately 20% of total energy, or linoleic acid from sunflower seeds, oil and margarine (C18:2), accounting for approximately 15% of total energy (42). Whilst both diets increased body weight slightly (0.8kg saturated fat diet, 0.4kg LA diet), total plasma cholesterol slightly decreased on the LA diet (0.2±0.4 mmol/L), with no change seen in the saturated fat diet. However, other metabolic effects were seen in this particular population. Liver fat decreased with the LA diet (-0.5%, 95% CI: -2.3, 0.2) and increased on the saturated fat diet (0.7%, 95%CI: -0.2, 2.1) ($p<0.03$ difference between groups), although no difference was demonstrated in markers of inflammation and oxidised stress (42). A meta-analysis of prospective cohort studies investigating the dietary effects of LA showed that highest intakes of LA compared to lowest had a 15% reduction in CHD events (43). Differences in saturated fat intakes based on increments of 5% of LA from total energy was associated with 9% lower risk of CHD events and 13% lower risk of CHD deaths (43). A review of the evidence surrounding omega 6 polyunsaturated fats by the AHA concluded that higher intakes appear to be safe, and possibly beneficial when consumed as part of a low saturated fat and low cholesterol diet (44).

The long chain omega 3 (LCn3) fatty acids are a class of polyunsaturated fatty acids with particular properties due to the placement of the double bond in the carbon chain. LCn3s include eicosapentaenoic (EPA), docosahexaenoic (DHA) and docosapentaenoic (DPA) acids which have been demonstrated to have beneficial CVD effects through reductions in arrhythmias, platelet aggregation, and serum triglycerides (8, 45). For example, daily consumption of approximately 4g of marine-based LCn3 has been shown to decrease TG between 9-51% and is therefore recommended for those with hypertriglyceridaemia (46, 47). A more recently identified potential action of LCn3 is

counteracting post-prandial rises in serum lipids following consumption of saturated fats, although this may have sex differences in effect size (48). Meta-analysis of studies testing LCn3 supplementation and cardiovascular events showed a risk reduction of 0.91 (95%CI: 0.85, 0.98) for cardiac death rates with a mean LCn3 dose of 1.51g per day, although no association was found with all-cause mortality (49).

Trans fats are a type of unsaturated fatty acid, and is the opposing state of the *cis* configuration of carbon double bonds, which is most commonly found in unsaturated fats. Trans fats occur naturally in low levels in ruminant animal products, such as dairy foods and meats from cows and sheep. However, *trans* fats can also be found in commercially made food products from partially hydrogenating oils. *Trans* fats are of particular interest to CVD risk as they have been demonstrated to increase serum LDL cholesterol whilst decreasing HDL cholesterol (50). Meta-analysis of trials demonstrated that *trans* fatty acids increased the ratio of serum LDL to HDL by 0.055 (95%CI: 0.044, 0.066) for each % increase in energy from industrial *trans* fatty acid sources (50). Serum LDL was demonstrated to increase by 0.048mmol/L and serum HDL to decrease by 0.01mmol/L (95%CI: 0.007, 0.013) for each % increase of energy from *trans* fatty acids (50).

The WHO recommends dietary intakes of *trans* fatty acids to be less than 1% of total energy intake (51). Average Australian intakes have been currently estimated to be approximately 0.6% of total energy for those aged two years and over (52). Highest concentrations of *trans* fatty acids in the Australian food supply were reported in croissants (1.02g /100g) and prepared pastry items (0.96g/100g) (53).

Carbohydrates

The spectrum of carbohydrate classes, ranging from insoluble and soluble fibres, resistant and refined starches, and simple sugar complexes have differing effects on risk factors for CVD (8, 36, 54-62).

Meta-analysis of studies examining the role of fibre in CVD and CHD showed a dose responsive curve, whereby higher intakes of dietary fibre were associated with lower risk of CVD and CHD (54). Overall, a 9% lower risk was demonstrated for both CVD and CHD for every additional 7g of fibre consumed per day (54). Differences were seen based on the types of fibre. For every 4g increase in soluble fibre, a non-significant risk

estimate for CVD was reported as 0.88 (0.75-1.03) and CHD as 0.89 (0.78-1.02). The risk estimates based on a 7g per day increase in insoluble fibre were reported as 0.82 (0.70 – 0.96) for CVD and 0.82 (0.68 – 0.99) for CHD (54). More recent analysis from two large prospective cohort studies showed that replacing 5% of energy from saturated fat with equivalent amounts of carbohydrates from wholegrains gave a reported HR of 0.91 (95%CI: 0.85, 0.98) (36).

The mechanisms by which dietary fibres reduce CVD and CHD risk factors follow different pathways (55). Insoluble fibres such as brans are thought to work through displacement of other foods, thereby increasing satiety and slowing gastric emptying, with no direct link to serum lipids or hypertension (55, 63). Soluble fibres, which include psyllium husks and β-glucan fibres reduce serum cholesterol via their ability to form viscous gels (55). The soluble fibres bind bile acids in the intestine which contain high levels of cholesterol. The bound bile is excreted, requiring cholesterol to be drawn from serum for the production of new bile acids (56).

Starches and sugars differ from fibre in their impact on CVD risk factors. Glycaemic index (GI) and glycaemic load (GL) relate to the rate at which available CHO is digested and the subsequent requirement for plasma insulin to aid CHO metabolism (57). CVD risk factors such as hyperglycaemia, which may be caused by CHO excessive to rates of metabolism, is thought to increase oxidative stress and affect endothelial function, although a number of pathways exist by which high GI diets may influence CVD development (62, 64). Meta-analysis of the association between GI and GL with CHD events and mortality showed a relationship in women, but not men. (57). Comparing women in the highest versus lowest quintiles of GL and GI reported the relative risks of CHD events or mortality in follow up periods between 6-25 years was 1.55 (95% CI: 1.18, 2.03) and 1.26 (95% CI: 1.12, 1.41) respectively (57).

Diets containing higher amounts of added sugar are associated with increased CVD mortality (58). A prospective study of approximately 10,000 Americans showed greater hazard ratios by quintile of added sugar intake after adjusting for other CVD risk factors such as ages, sex, ethnicity, education and smoking status of 1.07 (95%CI: 1.02, 1.12), 1.18 (95%CI: 1.06, 1.31), 1.38 (95%CI: 1.11, 1.70) and 2.03 (95%CI: 1.26, 3.27) (quintiles two to five respectively) compared to the lowest quintile (58). Meta-analysis

of the relationship between sugar intakes and serum lipids shows increases in plasma TGs, total cholesterol, LDL and HDL particles (60). Triglycerides were shown to have a mean difference of 0.11 mmol/L (0.07, 0.15) between higher sugar intakes and lower sugar intakes, a mean difference of 0.16mmol/L in total cholesterol, 0.12 mmol/L (0.05, 0.19) difference in LDL cholesterol, 0.02 mmol/L (0.00, 0.03) increase in HDL (60).

Research has shown that in comparison to isocaloric amounts of low fat milk for a six month period, a sucrose-sweetened beverage increased visceral fat by 31% (95%CI: 10, 53) and liver fat by 143% (95%CI: 50, 236) (65). Whilst LDL particle size has been shown to be affected by increased levels of monosaccharides such as fructose and sucrose, the cellular mechanism is still unclear; however it is thought that diets containing sugars enhance fat absorption and stimulates chylomicron secretion (66). Also, higher glucose has been shown to increase the size of particles carrying TGs, rather than increase the number of particles (59).

Protein

Protein as a macronutrient has little evidence of a causal link to the development of atherosclerosis. A European prospective cohort study of approximately 43,000 women found a slight increased incidence of CVD events in those with relatively high protein diets, whereas a cohort study of approximately 83,000 women from the United States found no association with protein intake (67, 68). These results may be influenced by the relative contributions of protein and carbohydrate (CHO) as suggested by a study in a healthy Greek population, where protein only became predictive of mortality when combined with a low carbohydrate diet (69).

A meta-analysis of high and low protein diets, with a relative 10% difference in contribution to total energy between each, found no difference in total and LDL cholesterol between diet types (70). However, a statistically significant though slight increase in HDL was reported for those following the high protein diets (0.04mmol/L, 95%CI: 0.01, 0.08), as well as a decrease in TGs (-0.24mmol/L, 95%CI: -0.37, -0.11) (70). A possible explanation for the difference in TGs may be due to the types of proteins consumed (71). Proteins of differing quality such as whey, casein, gluten and cod proteins have been demonstrated to affect the rate of gastric emptying, thereby

affecting chylomicron production, rather than a direct causal link to plasma TGs (72, 73).

2.1.2.2 *Micronutrients*

Micronutrients include those that are required in trace amounts for good health, and include vitamins and minerals. Two minerals in particular, sodium and potassium, are associated with atherosclerosis and CVD health, due to their relationship with hypertension. Although many mechanisms are involved in the development of hypertension, as discussed in section 2.1.1.3, sodium is consistently identified as a major contributor (74, 75). Excessive dietary sodium intakes increase blood pressure through two mechanisms in particular. The first mechanism is that excessive sodium intakes elevate plasma volume (extracellular fluid volume) and water retention by the kidneys (76). The second is that excessive dietary sodium results in increased levels of sodium in the cerebrospinal fluid which then triggers sympathetic nerve activity for vasoconstriction (77). Whereas sodium is a major contributor to the development of hypertension, higher intakes of potassium have been shown to reduce blood pressure through vasodilation and reducing sodium retention (78).

2.1.2.3 *Functional foods and nutraceuticals*

A functional food is defined as one that has a beneficial effect beyond their nutritional properties when consumed regularly, whereas a nutraceutical is a dietary supplement that is a concentrated form of a bioactive agent from a food (79). Soy-based foods and phytosterols are two examples of these with reported benefits to cardiovascular health (11, 79-82)

Phytosterols are the plant equivalent of mammal cholesterol analogues (11). Phytosterols are generally added to margarines, low-fat milks and soft cheeses, with intakes of approximately 2g per day shown to reduce serum cholesterol by 9-14% (11, 81). Phytosterols have three mechanisms by which they affect serum cholesterol. Firstly, phytosterols resist absorption by the body. Whilst both cholesterol and phytosterols are absorbed into the intestine from the lumen by the same transporter, the phytosterols are largely returned to the lumen and cholesterol is preferentially esterified allowing packaging into chylomicrons. Secondly, phytosterols inhibit

cholesterol absorption as they competitively displace cholesterol for uptake in the cholesterol transporter between the lumen and intestine. Thirdly, β-sitosterol, a common phytosterol, has been demonstrated to inhibit gene expression of the rate-limiting enzyme (HMG-CoA reductase) in cholesterol synthesis (11).

Meta-analysis of soy-based foods have been reported to reduce serum cholesterol by approximately 4% or 0.17mmol/L (95% CI: -0.25, -0.10) (82) in doses ranging from 25-133g/day. Three mechanisms that may be responsible for this effect include displacement of dietary animal protein, and increases in both phytoestrogens and lunasin, a component of soy protein (11, 82, 83).

Substitution of soy products for animal sources of protein reduces dietary sources of cholesterol and saturated fat (82). Displacement of animal proteins with soy proteins was estimated to achieve a 0.15mmol/L reduction in serum cholesterol at a daily dose of 13g if mean intakes of total and saturated fat were the equivalent to that found in NHANES III (33% and 11% respectively) (82).

Phytoestrogens are plant compounds that have weak affinity for binding to oestrogen receptors in mammals. These compounds have been suggested to both inhibit cholesterol synthesis and increase expression of LDL receptors (11). The most commonly consumed phytoestrogens in the human diet are soy isoflavones (11). However, investigations of the effect of soy isoflavones on serum lipids have had mixed results, including no demonstrated dose-dependency, indicating these may have limited contribution to cholesterol-lowering effects of soy (11). Thus it may be more likely that it is the soy protein itself which may be efficacious (83).

The third of the potential mechanisms through which soy-based foods may lower serum cholesterol relates to lunasin. Lunasin is a soy protein peptide which is thought to affect gene expression in both the enzyme HMG-CoA reductase and LDL receptors thereby reducing cholesterol synthesis, increasing LDL clearance from serum (83).

2.2 The role of dietary patterns

2.2.1 Efficacious dietary patterns for the prevention and treatment of CVD

Very few of the nutrients discussed above can be viewed in isolation as they are rarely consumed in a pure form, and more usually consumed as single or mixed food or beverage item. For this reason, epidemiology has shifted from nutrient-based research towards that of dietary patterns (84). Dietary patterns are more able to describe the overall diet, including common food combinations and variety of foods consumed. Dietary patterns allow for a complex mix of nutrients which vary in strength of evidence for the prevention or halting of progression of CVD and are able to incorporate synergistic effects of foods when consumed in combinations over a period of time (85, 86).

The quality of diet relates to the variety and comparative nutrient density and energy value of foods regularly consumed (86, 87). Interventions aimed to improve diet quality have indicated that intima-media thickness of the carotid artery, a sign of atherosclerosis, could be decreased, or lower inflammation (88-91). The most recent meta-analysis of dietary patterns and CVD from five observational studies has shown that those with highest quality diets had a RR:0.69 (95%CI: 0.60, 0.78), with RR 0.83 (95%CI: 0.75, 0.92) for CHD from 11 observational studies (92).

Three dietary patterns are described below, each with different purported mechanisms for the reduction of CVD risk factors, yet contain similarities as all have a predominantly plant-based focus.

2.2.1.1 *Mediterranean diet*

The Mediterranean diet is generally characterised by a high monounsaturated to saturated fat ratio, high intakes of legumes, fruits, vegetables, wholegrains and cereals with low processing, increased intakes of fish and nuts, low intakes of red meats and moderate amounts of dairy and alcohol in comparison to a western diet (35, 93, 94).

The Mediterranean diet has been researched for many years, with the benefits for prevention of CVD best demonstrated to date in the Prevención con Dieta Mediterránea (PREDIMED) study (35). This study investigated the mortality rates in a large adult cohort following two variations of the Mediterranean diet and compared to a control low-fat diet. One Mediterranean diet was supplemented with olive oil and the other supplemented with nuts (walnuts, almonds and hazelnuts). After following over 7,000 subjects for approximately five years, and adjusting for CVD risk factors, hazard ratios of 0.70 (95% CI: 0.54. 0.92) and 0.72 (0.54, 0.96) were observed for the Mediterranean diets with olive oil and nuts respectively, compared to the control group (35).

The Mediterranean diet has also been demonstrated to show positive effects in children (95). The IDEFICS study, investigated the effect of a Mediterranean diet in over 16,000 children aged 2-9 years in eight European countries. Results found that children consuming more of the food types that characterise the Mediterranean diet were less likely to be overweight or obese (OR: 0.85, 95%CI: 0.77, 0.94) (95). Another study investigated the effectiveness of the Mediterranean diet in prepubertal children with hypercholesterolaemia (96). After 12 months of following a Mediterranean diet, 36 hypercholesterolaemia children reduced their LDL from 4.7 to 4.2mmol/L and increased HDL from 1.4 to 1.6mmol/L (96). A reduction in mean intima-media thickness (mean of 12 carotid segment measures) was also shown (0.37mm to 0.32mm) (96).

The high unsaturated to saturated fat ratio is one of the key features of the Mediterranean diet as it is able to affect cholesterol metabolism, whilst the ratio is also purported to be responsible for a 10-15% reduction in TGs (8). The Mediterranean diet is predominantly a plant-based diet, and therefore high in fibre and antioxidants (96). It also contains moderate intakes of fish, which have been demonstrated to improve cardiovascular outcomes (97, 98). A meta-analysis of fish intakes and CHD mortality using 17 cohort studies reported that increments of 15g of fish consumption decreased risk of CHD mortality by 6% (RR:0.94, 95%CI: 0.90, 0.98) (98). Another meta-analysis investigating the relationship between fish consumption and CerVD reported RR:0.94 (95%CI: 0.90, 0.98) when comparing two to four servings per week of fish with one or less per week (97).

2.2.1.2 *Portfolio diet*

The Portfolio diet is a more prescriptive type of diet in comparison to the Mediterranean . It is a combination of four classes of functional foods (soluble fibre, plant sterols, soy protein and nuts) demonstrated to improve lipid profiles (see Table 2.1) (99-102). The cumulative effect of this combination of functional foods and nutraceuticals was theorised to be capable of inducing a 22.5% reduction in LDL cholesterol, and the different mechanisms by which these foods act posited to be additive (102, 103). LDL reductions from following this type of diet have been shown to vary from 35.0±3.1% in four weeks where foods were provided and menus prescribed to 0.62 mmol/L (95%CI: 0.78, 0.49) (approximately 13%) in a 6-month period where participants were counselled on the diet twice (99, 104). Reductions in systolic (1.5-4.3mmHg) and diastolic (1.5-2.3mmHg) blood pressure were also reported, although the diastolic reduction was attributed to weight loss (99, 105).

Table 2.1: Functional foods and recommended intakes of the Portfolio diet (99, 100, 104)

FUNCTIONAL FOODS	RECOMMENDED INTAKE
Plant sterols	0.94 - 1.2g / 1000kCal
Soluble fibres	8.3 – 10.3g / 1000kCal
Soy protein	16.2 – 22.5g / 1000kCal
Almonds	16.6 – 22.5g / 1000kCal
Peas/beans/lentils	High intakes encouraged

Compliance with the recommended intakes of the functional food items was shown to vary (101). Highest compliance was reported for almond recommendations (78.8%), with 67% compliance for sterol recommendations, 55% with soluble fibres and 51% compliance with soy protein (101). Participants found following a complete vegetarian diet challenging with only seven of 55 remaining vegetarian over a 12 month period (101).

Satiety induced by these foods may contribute to decreasing excessive energy intake, which is associated with obesity and adverse lipid profiles (103). The mechanisms by which the functional foods work are dissimilar, yet all reduce LDL cholesterol. High intakes of soluble fibre from plant matter bind bile acids (56), whilst the insoluble fibres present with the soluble fibres increase satiety (55). The diet is high in soy protein which is posited to effect the cholesterol cycle and displace animal proteins (11, 82, 83).

The plant sterols inhibit cholesterol reabsorption from the digestive tract and nuts are more complex as they contain a combination of nutrients, but are thought to suppress appetite and have been proposed to increase fat oxidation (106).

2.2.1.3 *DASH diet*

The Dietary Approaches to Stop Hypertension (DASH) diet aims to reduce systolic and diastolic blood pressure in populations with or without known hypertension (107). It is a predominantly plant-based diet with sodium restriction (107). Meta-analysis of prospective observational studies in healthy populations with no known CVD or CVD risk factors showed that following a DASH-style diet reduced overall CVD risk (RR: 0.80, 95% CI: 0.74, 0.86) for combined heart failure, CHD, stroke and CVD (108).

The diet is characterised by encouraging intakes of fruits and vegetables, aiming for 4.5 cups per day, two servings of 100g (preferably oily) fish per week, approximately 30g of fibre from wholegrains, sodium intakes restricted to 1500mg per day, and sugar sweetened beverages restricted to 1880kJ or approximately 1 litre per week (85). Nuts, legumes and seeds are encouraged (four or more servings per week), foods such as red and processed meats are discouraged, with a guideline of between none and two servings per week and a saturated fat intake of less than 7% of total energy intake recommended (85, 107). In contrast to the Mediterranean diet, it is relatively lower in fats and higher in protein and carbohydrate (109, 110).

The mechanism by which the DASH diet is purported to work is through a reduction of sodium intakes, an increase in dietary potassium, but may also be synergistic with increases in fruit and vegetable intakes (111-114).

Although the Portfolio and DASH diets are more prescriptive than the Mediterranean diet, there are a number of similarities between the diet types. All are based on low-processed plant foods, and are high in nuts, plant protein and fibre. Commonalities and differences for this diet types can be seen below in Figure 2.2.

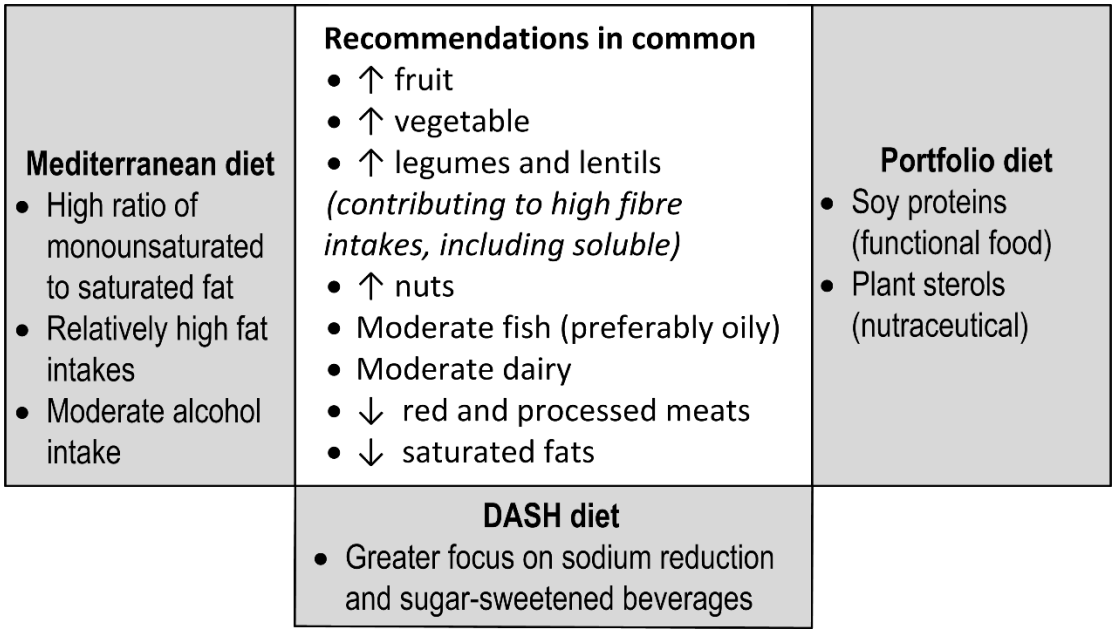

Figure 2.2: Commonalities and differences of eating patterns efficacious in reducing dietary CVD factors

2.2.2 Measuring dietary patterns in populations at risk or with cardiovascular disease

Common methods used to measure dietary intakes include 24 hour dietary recalls, diet histories, food diaries, weighed food records and food frequency questionnaires (FFQ). Evolving dietary intake methodologies incorporate the use of emerging technologies or hybrids of previous methods (115, 116). All methodologies have advantages and disadvantages, with methods generally chosen for their ability to measure prospectively or retrospectively the desired primary outcomes with reasonable accuracy, considering also validity, reproducibility, cost, burden to researchers and participants and skill levels required for administration and analysis (117). These methodologies are also subject to either systematic or random bias (118). Methodologies that collect short-term dietary intakes, such as the 24 hour recall and food diaries are subject to variation in day-to-day intakes that may or may not reflect usual intake (118). Those that use finite food lists, such as FFQs, have systematic bias built in and increased cognitive difficulty, due to respondents being required to estimate regular intake of food items over longer periods of time (117, 118). Whilst it is relatively easy to measure a single nutrient with reasonable accuracy, the effect of dietary patterns on CVD risk factors are more challenging due to the number of factors involved. The number of repeated dietary measures in longitudinal studies may also be important in assessing

associations with CVD events (119). One such large scale study, the Doetinchem Cohort Study, found that eating patterns changed slightly over time, and accounting for these changes increased the strength of association between diet and adverse CVD events (119). The hazard ratio for all composite CVD events within a 10 year time frame using only baseline data was 0.77 (95%CI: 0.53,1.11), which decreased to 0.65 (95%CI: 0.43, 0.97) when also using data from baseline, five and ten years (119).

For the most part, FFQs have been used in large cohort studies investigating the progression of CVD, although diet histories have also been used (35, 119-123). FFQs can be self-administered to large cohorts, are relatively inexpensive to analyse, may be validated for the outcome of interest and can be used to examine associations between diet and health, although they are subject to systematic bias (117, 118). In contrast, multiple-pass 24 hour recalls have been used for large population studies, such as those used for National Health and Nutrition Examination Survey (NHANES) and the AHS (52, 58). Whilst the 24 hour recall obtains more detail in regards to intakes on the previous day, it is essentially a short term measure, is potentially more expensive and requires more skill to administer and analyse than a FFQ (117).

As FFQs contain finite food lists that are linked to nutrient composition databases, FFQs should ideally be tested for validation and representativeness of the desired population (124). Food lists are constructed from food items that a significant proportion of the chosen population consume on a regular basis and contribute significant nutrients to their health (124). However, populations who identify with a health condition may regularly consume foods specific to their condition, especially supplements (125, 126). A food list designed for a non-specific population may not adequately capture all nutrients of interest in this situation. A number of FFQs have been validated in various Australian populations, such as those that are healthy, only women, aged populations, or children and adolescents (127-134). Two FFQs specifically related to CVD health were identified: one relating to intakes of EPA and DHA, and another relating to dietary habits (135, 136). Both are designed as screening tools, and as a result, neither FFQ is capable of providing a comprehensive assessment of nutrient intakes and dietary patterns of this population.

Diet quality indices or scores are measures relating to the quality of overall diets and tend to be based on national dietary guidelines (137). As the diet quality indices relate to eating patterns, rather than groups of individual nutrient groups, a diet quality score allows for food combinations and interactions of nutrients. Most variants of published diet quality scores internationally in adults are based around the following major scores (87, 137-139):

1. Healthy Eating Index (HEI) where scoring is proportional to the extent to which national dietary guidelines are met
2. Alternative Healthy Eating Index (AHEI) is scored to be consistent with dietary patterns and eating patterns associated with lower chronic disease risk
3. Diet Quality Index (DQI), measures the quality of diet that may reflect the risk of diet-related disease
4. Healthy Diet Indicator (HDI), based on dietary recommendations for preventing chronic disease from the World Health Organization (WHO)
5. Mediterranean Diet Score (MDS), based on adherence to traditional Mediterranean diets (high monounsaturated fat to saturated fat ratio, high legume, fruit, vegetable and cereal consumption, moderate ethanol, low meat and dairy consumption).

Diet quality scores are easily adapted to measuring compliance of dietary recommendations if the purpose is clearly defined and constructed in accordance with that purpose. A review of 20 diet quality scores in adults in 2007 concluded that higher diet quality scores have an association with decreased morbidity and mortality in general, but should be interpreted with care and limitations considered (138).

Meta-analysis of 15 cohort studies with diet quality scores and mortality or incidence of CVD reported a RR: 0.78 (95%CI: 0.75, 0.81) for the highest diet quality category as assessed by the HEI, AHEI and DASH diet score in comparison to the lowest (86). One of the more recent scores developed for CVD use is the American Heart Association (AHA) Diet and Lifestyle Score (140). This was developed to measure compliance with the AHA Dietary and Lifestyle Recommendations. To date, no validated FFQ or related score has been developed that measures compliance to Australian guidelines for the primary and secondary prevention of CVD.

2.3 Risk factors for cardiovascular disease

In 1948, the first participants were recruited to the Framingham Heart Study (141). This landmark study was the first to identify risk factors for CVD events (142). Since that time, the Framingham risk assessment method has been refined, with variations now used to assess CVD risk within many national guidelines (143-146). In Australia, a brief CVD assessment for risk of CVD events within the next five years considers age, sex, total to HDL ratio, systolic blood pressure, ethnicity, smoking status and diabetes as a co-morbidity (144). A comprehensive assessment of CVD risk also includes waist circumference and BMI, physical activity levels, alcohol intakes, family and social history as well as related conditions such as reduced kidney function, familial hypercholesterolaemia and evidence of atrial fibrillation (143).

2.3.1 Influence of genetic profiles

Genetic predisposition for the development of CVD may take a number of forms, for example by causing variations in plasma lipid concentrations (147, 148). A number of ethnicities such as South Asian, African American, African Caribbean, native American Indian, Mexican American have demonstrated predispositions to metabolic syndrome, hypertension or dyslipidaemia, especially when removed from a traditional diet (148-150).

Genetic variation may explain approximately 12% of variation in LDL, HDL and total cholesterol, as well as 10% of variation in TGs (148). Genetic variants associated with incidence of CVD (death, MI, angina, intermittent claudication) assessed in the Framingham cohort showed a HR 1.05 (95% CI: 1.01, 1.09) for each genetic risk allele (13 in total), independent of all other CVD risk factors for a 10 year risk period (151). Whilst genetic screening is becoming more prevalent, screening for a family history of premature CVD within a clinical setting remains a viable option, with a hazard ratio of 1.59 (95% CI: 1.14, 2.22) within 10-20 years for males with a family history of premature CVD events, after adjusting for other factors (152).

2.3.2 Lifestyle / behavioural risk

In 2010, the AHA published a definition of ideal cardiovascular health (ICH) (85). The definition for adults included seven different factors: non-smoking status, with a BMI of less than 25kg/m^2, minimum of 75 minutes of physical activity per week, a healthy diet score, total cholesterol less than 5.2mmol/L, blood pressure less than 120/80 mmHg, and a fasting plasma glucose of 5.6mmol/L, with appropriate ranges given for children (85). These measures of ideal cardiovascular health were applied to data from a cohort of children and young adults aged between 12 and 24 years that were followed for 21 years (153). The ICH measures were compared with physical signs of atherosclerosis, such as thickness of the carotid intima-media and coronary artery calcification. Results showed that a one standard deviation increase in ICH reduced risk of coronary artery calcification by 0.66 (95%CI: 0.53, 0.83) and high risk carotid intima-media thickness by 0.71 (95%CI: 0.59, 0.86). Further to this, those who improved their ICH over time had similarly low risk rates of 0.71 (95%CI: 0.56, 0.89) and 0.75 (95%CI: 0.63, 0.88) for coronary artery calcification and high risk carotid intima-media thickness, showing that by improving ICH, CVD health could be regained (153).

However, data from the 2011-2012 Australian Health Survey showed that ICH risk factors and behaviours may not be well identified or acted on in this population. Around 32% of Australian adults had high levels of total cholesterol (≥5.5mmol/L), yet only 10% identified high cholesterol as a health condition (154). Of those who were categorised as having unmedicated high blood pressure (≥140/90mmHg) in the AHS 2011-2012, almost half (48%) were not aware they had the condition (155, 156). Risk factors were also found in combinations as shown by the high rate of abnormal LDL readings (51.7%) in obese 18-44 year olds who currently smoked daily, compared to 15.8% in those of a healthy BMI and non-smoking status of the same age group (154).

Whilst smoking rates continue to decline in Australia, in 2011-2012 men were more likely to smoke (18.2%) than women (14.4%) (156). In regards to physical activity, approximately 67% of Australians had sedentary or low exercise levels, rising to 83% in adults aged ≥75 years (156). A healthy diet according to Australian recommendations would include two serves of fruit and five serves vegetables per day (157). Yet 48.3% of Australian adults were found to eat two piece of fruit each day and only 8.3% met the

vegetables recommendation (156). In addition, 63.4% of Australian adults and 25.3% of children were overweight or obese in 2012 (156).

Those with recognised risk factors for premature CVD, such as strong family history or requiring medication to manage CVD-related conditions, may also be demonstrating adverse CVD behaviours (158, 159). For example, smoking is an important risk factor for CVD, yet an Australian study reported that those with a strong family history of premature CVD smoked 0.82 more pack years than those with an average family history (20 cigarettes per day for a year is equal to one pack year) (158). The same study also reported that those with a strong family history were less likely to identify a history as an ex-smoker as a risk factor (OR 0.32, 95%CI: 0.12, 0.90) (158). Data from the National Health and Nutrition Examination Survey showed that in 1999-2000, those that used statins had lower BMI's, total energy and fat intakes ($p=0.02$, $p<0.01$ and $p<0.01$ respectively). For the years between 1999 to 2010, those that used statins slowly increased BMI (+1.3 kg/m^2), total energy (+803kJ/day) and fat intakes (+10.3g/day), whilst those not using statins were relatively stable for the same risk factors (+0.5kg/m^2; +171kJ/day; -1.9g/day respectively). This change in trend in important risk factors indicates that dietary advice may be being inadequately addressed in statin users or people are relying on the protective effects of the medication (159).

2.3.3 Families: both genetic and lifestyle behaviours

Risk factors may cluster in families because of inherited genetic risks and learned behaviours from other family members (160-162). CVD risk within families has been described in high quality research studies from the Bogalusa study in the late 1990's (163). The study detailed the progression of coronary artery disease (CAD) in children with a parental history of CAD through to adulthood (163). Results showed that children who had parents with CAD had higher BMIs than those whose parents did not have a CAD diagnosis (1.22, $p=<0.01$). Children with a family history also had higher prevalence of abnormal LDL (12.4 compared to 7.4 respectively, $p=0.01$) and total cholesterol levels (8.4 compared to 4.8 respectively, $p=0.03$). Higher prevalence rates of children with two or more risk factors were found in families with a parental history of CAD (9.1 to 14, $p=0.01$) (163).

More recently, other large cohort studies have reported childhood effects of negative and positive parent behaviours (160, 161). Examples of associations reported include those between parental smoking and a child's increased fasting blood glucose levels or BMIs, and parental energy intakes and physical activity levels with a relationship to a child's weight status (160, 161). Another smaller study has been shown to have correlations between specific paternal food intakes and those of their offspring, particularly for fruit, chips and biscuits (164). All of these studies infer that CVD risk factors occur within families making them potential avenues for prevention and treatment interventions including risk reduction.

Concordance in CVD risk factors linked to behaviours have also been reported between spouses (165). A meta-analysis reported smoking as having highest concordance (OR: 3.25, 95%CI: 2.94, 3.59), with obesity, hypertension and diabetes also being significantly related between spouses (OR: 1.44, 95%CI: 1.16, 1.78; OR: 1.21, 95%CI: 1.16, 1.26 and; OR: 1.16, 95%CI: 1.03, 1.31 respectively) (165).

These correlations may in part be explained by the transmission of eating behaviours between family members (166). Restraint and inhibitory eating patterns have been described to be influenced by parents, as a child's first home food environment is established by parents. Behaviours that are modelled by adults may be learned and replicated by their offspring, although family influences are more likely to be replaced by those of peer groups as the children age.

Core components for Australian cardiovascular disease secondary prevention and cardiac rehabilitation include the presence of family members in education and counselling sessions as a key data to collect (167). Reasons for the importance of family inclusion are threefold: family inclusion is culturally appropriate for many ethnic groups; families are likely to share similar risk factors; family engagement increases rates of participation in cardiac rehabilitation (167).

2.4 National policies and guidelines for cardiovascular disease prevention and treatment

Biological factors and identified risk factors are used to inform risk identification and prevention strategies as well as current guidelines and clinical management for CVD.

The Australian Institute of Health and Welfare classifies management of CVD care into three phases (168):

1. Primary prevention
2. Acute care
3. Secondary prevention

2.4.1 Primary prevention

Primary prevention may be either at general population or individual level. It is aimed to reduce prevalence of risk factors and address behaviour change related to lifestyle risk (169). The lifestyle risk factors addressed in Australia for the general population that form part of primary prevention of chronic disease include diet, physical activity, body mass and smoking.

The Australian Guide to Healthy Eating and the Australian Dietary Guidelines are the Australian dietary recommendations for the prevention of chronic disease (157, 170). Many countries have similar recommendations that are tailored for the specific requirements of their populations, and dietary recommendations are also available from the WHO (171).

Primary prevention is also addressed at the individual level. Guidelines for the assessment of CVD risk for use by Australian general practitioners (GPs), Aboriginal health workers and other health professionals in primary care recommend that individuals are stratified by risk of events (143). The stratification of risk is based on the Framingham equation and has three qualitative levels of risk with corresponding probability of a CVD event within five years: low (<10%), medium (10-15%) and high (>15%) (143).

Guidelines for the management of CVD risk aim to manage CVD risk factors through lifestyle changes and pharmacological therapy where indicated (172). The recommended level of care, treatments and advice provided to an individual relate to the risk stratification level. Those at a low level of risk are to be provided with lifestyle advice, although persistent hypertension is recommended to be treated, and reviewed in two years. Those at moderate risk are to be provided with lifestyle advice and support and may be treated for both hypertension and lipids and reviewed in 6-12

months. Those at high risk are to be provided with frequent and sustained lifestyle advice, support and follow-up, whilst treating hypertension and lipids and reviewing according to context (172). A summary of the lifestyle advice recommended for provision can be seen in Table 2.2, with comparisons given for other relevant international guidelines.

Table 2.2: Current Australian lifestyle recommendations for prevention of cardiovascular disease, with international guidelines for comparison.

	Guidelines for the management of absolute CVD risk (Australia) (172)*	AHA/ACC Lifestyle Management Guidelines (United States) (173)	Joint British Societies consensus recommendations (United Kingdom) (174)	European guidelines on CVD prevention in clinical practice (European) (146)
Smoking	Cessation recommended	-	Cessation recommended	Avoid passive exposure, cessation recommended
PA	At least 30 min moderate intensity on most or preferably every day of the week	3-4 sessions / week lasting an average of 40 minutes of moderate to vigorous intensity	30-40 min x 2-3 / week	2.5-5hr / week moderate intensity or 1-2.5 hr / week vigorous Multiple bouts ≥ 10 min 4-5 x week
Weight	Weight loss should be recommended for people who are overweight or obese. BMI <25kg/m^2 is desirable	-	-	Weight reduction recommended in overweight and obese people
WC	A waist circumference of ≥94cm in men and ≥80cm in women is suggestive of central obesity	-	-	≥94cm (M) and ≥ 80cm (F): no further weight gain ≥102cm (M) and ≥88cm (F): weight reduction recommended
DIETARY RECOMMENDATIONS				
Fat	Limit foods containing saturated and trans fats (172).	Aim for 5-6% of total energy from saturated fat. Reduce trans fats. I Dietary patterns includes intake of low-fat dairy products, non-tropical vegetable oils and nuts	Saturated <10% total fat intake Replace saturated fat with polyunsaturated wherever possible	Saturated < 10% total energy intake Replace with polyunsaturated fats Trans fats: as little as possible, <1% total energy from natural sources
Fish	Included as part of a wide variety of nutritious foods (157)	Dietary patterns include intakes of fish	2 servings / week, preferably oily	2 servings / week, one of which to be oily
Fruit and vegetable	Consume a diet rich in fruit and vegetables (172); Included as part of a wide variety of nutritious	Dietary patterns includes emphasis on intake of vegetables and fruit; includes legumes	5 portions / day	200g fruit / day (2-3 serves) 200g vegetables / day (2-3 servings)

Table 2.2: Current Australian lifestyle recommendations for prevention of cardiovascular disease, with international guidelines for comparison.

	Guidelines for the management of absolute CVD risk (Australia) (172)*	AHA/ACC Lifestyle Management Guidelines (United States) (173)	Joint British Societies consensus recommendations (United Kingdom) (174)	European guidelines on CVD prevention in clinical practice (European) (146)
	foods; Plenty of vegetables, including different types and colours, and legumes /beans; Fruit (157)			
Fibre / wholegrains	Included as part of a wide variety of nutritious foods; Grain (cereal) foods, mostly wholegrain and/or high cereal fibre varieties (157)	Dietary patterns includes emphasis on intake of wholegrains	Wholegrains: consider regular consumption	30-45g /day fibre from wholegrain products, fruits and vegetables
Salt	Limit salt <6g per day or approximately 2300mg sodium (172)	<240mg sodium/day	< 6g / day	< 5g /day
Alcohol	Limit alcohol intake to ≤2 standard drinks per day (172).	-	< 21 units / week (M) < 14 units / week (F)	20g / day (M) 10g / day (F)
Foods to avoid	Limit intake of foods high in saturated fats; foods and drinks containing added salt; foods and drinks containing added sugars such as confectionary and SSB. (157)	Limit intakes of sweets, SSB and red meats	Processed meats & commercially produced foods, refined CHO, SSB, energy-dense, nutrient-poor foods	-

* Australian guidelines for the management of absolute CVD risk refer to lifestyle advice for alcohol and nutrition to the Australian Guidelines to reduce health risks from drinking alcohol (175) and The Australian Dietary Guidelines (157), respectively for additional detail. Guidelines are referenced accordingly.

Abbreviations:

AHA American Heart Association; ACC American College of Cardiology; BGL Blood glucose level; BMI Body mass index; CHO Carbohydrate; (F) Females; HDL High density lipoprotein; LDL Low density lipoprotein; (M) Males; min Minute; SSB Sugar sweetened beverages;

2.4.2 Acute care and secondary prevention

Acute care is treatment on an individual basis following a CVD event and is managed primarily by paramedics, emergency departments and specialised units following a CVD event (168). Most of acute care treatment is performed within hospitals and is aimed at improving survival rates and minimising damage from these events.

Those presenting at a medical care facility with symptoms that are consistent with AMI are to be treated according to the guidelines for ACS (176, 177). These guidelines focus on appropriate management of the acute phase, and also provide recommendations for the long-term management of the adverse event. For example, whilst an appropriate medication regime should be initiated on discharge from the facility, so too should patients have been advised on appropriate lifestyle changes, including smoking cessation, nutrition, alcohol, physical activity and weight management when relevant. Patients should also be actively advised to attend cardiac rehabilitation services (176, 177).

Following discharge from the acute phase, guidelines for secondary prevention of adverse CVD events are appropriate to be followed at an individual level (178). The guidelines incorporate recommendations for lifestyle and behavioural risk factors with pharmacological and biomedical risk factor management, state the importance of initiating and sustaining behaviour changes and include psychosocial assessment (178). The secondary prevention guidelines regarding diet and nutrition are prescriptive as the AGHE and also provides detail on other lifestyle risk factors (170). In addition to the secondary prevention guidelines, Australian core components for cardiac rehabilitation services were developed to guide effective service delivery (167). The core components recommend measuring nutritional status, including alcohol and caffeine intakes and the number of referrals to dietitians (167). Key performance indicators (KPIs) from the same guidelines recommend using the percentage of patients who received education on the AGHE, the percentage that made dietary changes and the percentage that were referred to dietitians (167).

Adherence to lifestyle advice plays a key role in reducing the risk of repeat events

(179). However, provision of advice in regards to the long-term management of conditions following an ACS has been shown to be less than adequate (180). In 2012, a large cross-sectional study investigating levels of care in patients presenting with ACS in Australia showed that 71% of people were discharged with an appropriate medication plan and 46% were referred to cardiac rehabilitation services (180). In regards to lifestyle risk factors, 69% of smokers received smoking cessation advice, 43% of patients received advice for physical activity and approximately a third (36%) received dietary modification advice whilst in hospital (180). The extent and detail of the advice provided is unclear, only that it was documented to occur.

Although 46% of ACS patients received a referral to cardiac rehabilitation services, the NHFA reported that only one in three eligible people actually attend (181). In regards to patient education for diet and nutrition, a recent systematic review of cardiac rehabilitation classes showed that measures for diet tend to focus on singular components rather than overall diet quality (182). Additionally, receipt of dietary advice may be poorly retained, as 55% of people eligible to attend outpatient cardiac rehabilitation reported remembering being told to follow a modified fat diet and those that did not attend were even less likely to recall being advised to do so (odds ratio: 0.34, 95% confidence interval 0.28-0.46) (183).

The guidelines for secondary prevention state that where indicated, the patient should be referred to allied health professions (178). Therefore, the decision to refer a patient to a dietitian as the allied health dietary expert is at the discretion of the treating physician. The clinical reasoning process for this decision is complex (184). Factors reported to lower referrals to dietitians include whether the doctor believes the patient has attended a cardiac rehabilitation program, or that medication in combination with surgical procedures will be as effective as including lifestyle modification, or that the patient would be unwilling to attend dietetic services (184).

A compilation of international data has shown that following an ACS event, adherence to medication is substantially higher than adherence to dietary and physical activity advice (179). Six months after the event adherence to medication was shown to vary between 78% for β-blockers to 95% for anti-platelet

medications (179). In contrast to this, 28% of ACS patients did not make either dietary or physical activity changes, with 42% making a change to either diet or exercise, and 30% making changes to both (179). It must be noted that the extent and intensity of the dietary changes were not measured, only whether compliance with recommendations was positively assessed. Less than desirable prescriptions for and adherence to advice indicates that the current model of care may not be effective for a significant proportion of people experiencing ACS events. This is demonstrated by the high rates of ACS hospitalisations of those who have already experienced a MI or undergone invasive surgery to prevent an event (185). For example, the Australian and New Zealand SNAPSHOT ACS study in 2012 showed that of the cohort admitted to hospital in a two week period presenting with ACS, 27% had already experienced a prior MI, 20% had a prior percutaneous coronary intervention (PCI), and 11% had already undergone a coronary artery bypass graft (CABG) (185).

2.5 Summary of the literature review

- Atherosclerosis is a disease state that may be affected by nutrient intakes and dietary patterns
- Those with and without established CVD are less likely to experience adverse CVD events if following a high quality diet
- Lifestyle modification is an important adjunct to other medical therapies such as medication and interventional procedures as it can assist with reversal of the condition or prevent further deterioration. Appropriate diet patterns can impact on multiple risk factors of CVD, namely overweight and obesity, hypertension, diabetes and some causes of inflammation
- A high quality diet for the prevention and treatment of CVD is one that includes relatively high amounts of unrefined plant matter, with moderate amounts fish and low fat dairy foods and a high unsaturated to saturated ratio of fatty acids
- Dietary intakes are generally measured by FFQ in large scale CVD cohort studies as they have relatively low participant and researcher burden at

relatively low expense. No CVD-specific FFQ appropriate for the comprehensive assessment of dietary patterns currently exists in Australia

- Whilst genetic profiles play a significant role in the development of CVD in some populations, lifestyles and behaviours also contribute significant risk
- Families with a history of premature CVD are a population at increased risk through a combination of both genetic and lifestyle risk factors
- Primary prevention policies exist at both population and individual level in Australia. Population guidelines exist to prevent all chronic lifestyle diseases, whilst those identified at increased risk through screening tools based on risk factors present are to be treated according to individual-level prevention guidelines
- Those experiencing adverse CVD events are to be treated according to acute guidelines and be provided a long-term care plan following discharge from hospital which includes appropriate medication, referral to cardiac rehabilitation services and advice for lifestyle modification where necessary
- A high proportion of people presenting with ACS symptoms have already experienced an adverse CVD event such as a MI, or have previously undergone a preventative invasive procedure. This indicates that the current model of care may be inappropriate for a significant proportion of people

This review of the literature provided the rationale for the series of studies conducted for this thesis.

References

1. Libby P, Ridker PM, Hansson GK. Progress and challenges in translating the biology of atherosclerosis. *Nature*. 2011;473(7347):317-25.

2. Lusis AJ. Atherosclerosis. *Nature*. 2000;407(6801):233-41.

3. Lewington S, Whitlock G, Clarke R, Sherliker P, Emberson J, Hasley J, et al. Blood cholesterol and vascular mortality by age, sex, and blood pressure: ci meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. *Lancet*. 2007;370(9602):1829-39.

4. Sullivan DR, Watts GF, Nicholls SJ, Barter P, Grenfell R, Chow CK, et al. Clinical guidelines on hyperlipidaemia: recent developments, future challenges and the need for an Australian review. *Heart Lung Circ*. 2015;24(5):495-502.

5. Beaumont JL, Carlson LA, Cooper GR, Fejfar Z, Fredrickson DS, Strasser T. Classification of hyperlipidaemias and hyperlipoproteinaemias. *Bull World Health Organ*. 1970;43(6):891-915.

6. Martin SS, Faridi KF, Joshi PH, Blaha MJ, Kulkarni KR, Khokhar AA, et al. Remnant Lipoprotein Cholesterol and Mortality After Acute Myocardial Infarction: Further Evidence for a Hypercholesterolemia Paradox From the TRIUMPH Registry. *Clin Cardiol*. 2015:n/a-n/a.

7. Brown MS, Goldstein JL. A receptor-mediated pathway for cholesterol homeostasis. *Science*. 1986;232(4746):34-47.

8. Miller M, Stone NJ, Ballantyne C, Bittner V, Criqui MH, Ginsberg HN, et al. Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation*. 2011;123(20):2292-333.

9. Varbo A, Benn M, Tybjaerg-Hansen A, Jorgensen AB, Frikke-Schmidt R, Nordestgaard BG. Remnant cholesterol as a causal risk factor for ischemic heart disease. *J Am Coll Cardiol*. 2013;61(4):427-36.

10. Barter P. The role of HDL-cholesterol in preventing atherosclerotic disease. *Eur Heart J Suppl*. 2005;7(F):F4-F8.

11. Chen ZY, Jiao R, Ma KY. Cholesterol-lowering nutraceuticals and functional foods. *J Agric Food Chem*. 2008;56(19):8761-73.

12. Mann CJ, Yen FT, Grant AM, Bihain BE. Mechanism of plasma cholesteryl ester transfer in hypertriglyceridemia. *J Clin Invest*. 1991;88(6):2059-66.

13. Fogelstrand P, Boren J. Retention of atherogenic lipoproteins in the artery wall and its role in atherogenesis. *Nutr Metab Cardiovasc Dis*. 2012;22(1):1-7.

14. Maiolino G, Rossitto G, Caielli P, Bisogni V, Rossi GP, Calo LA. The role of oxidized low-density lipoproteins in atherosclerosis: the myths and the facts. *Mediators Inflamm*. 2013;2013:714653.

15. Graham I, Cooney MT, Bradley D, Dudina A, Reiner Z. Dyslipidemias in the prevention of cardiovascular disease: risks and causality. *Curr Cardiol Rep*. 2012;14(6):709-20.

16. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. *Am J Med*. 1977;62(5):707-14.

17. Talayero BG, Sacks FM. The role of triglycerides in atherosclerosis. *Curr Cardiol Rep*. 2011;13(6):544-52.

18. Astrup A, Dyerberg J, Elwood P, Hermansen K, Hu FB, Jakobsen MU, et al. The role of reducing intakes of saturated fat in the prevention of cardiovascular disease: where does the evidence stand in 2010? *Am J Clin Nutr*. 2011;93(4):684-8.

19. Skeggs JW, Morton RE. LDL and HDL enriched in triglyceride promote abnormal cholesterol transport. *J Lipid Res*. 2002;43(8):1264-74.

20. Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. *JAMA*. 2007;298(3):299-308.

21. Aguiar C, Alegria E, Bonadonna RC, Catapano AL, Cosentino F, Elisaf M, et al. A review of the evidence on reducing macrovascular risk in patients with atherogenic dyslipidaemia: A report from an expert consensus meeting on the role of fenofibrate-statin combination therapy. *Atheroscler Suppl*. 2015;19:1-12.

22. Varbo A, Benn M, Nordestgaard BG. Remnant cholesterol as a cause of ischemic heart disease: evidence, definition, measurement, atherogenicity, high risk patients, and present and future treatment. *Pharmacol Ther*. 2014;141(3):358-67.

23. Guardiola M, Solà R, Vallvé JC, Girona PJ, Godàs G, Heras M, et al. Body mass index correlates with atherogenic lipoprotein profile even in nonobese, normoglycemic, and normolipidemic healthy men. *J Clin Lipidol*. 2015.

24. Hollander W. Role of hypertension in atherosclerosis and cardiovascular disease. *Am J Cardiol*. 1976;38(6):786-800.

25. International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10 Version: 2015) [Internet]. World Health Organization. 2015 [cited 2nd June, 2015]. Available from: http://apps.who.int/classifications/icd10/browse/2015/en.

26. Unger T. The role of the renin-angiotensin system in the development of cardiovascular disease. *Am J Cardiol*. 2002;89(2A):3A-9A.

27. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012;380(9859):2224-60.

28. Chowdhury R, Warnakula S, Kunutsor S, Crowe F, Ward HA, Johnson L, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. *Ann Intern Med*. 2014;160(6):398-406.

29. Dawczynski C, Kleber ME, Marz W, Jahreis G, Lorkowski S. Association of dietary, circulating, and supplement fatty acids with coronary risk. *Ann Intern Med*. 2014;161(6):453-4.

30. Willett WC, Stampfer MJ, Sacks FM. Association of dietary, circulating, and supplement fatty acids with coronary risk. *Ann Intern Med*. 2014;161(6):453.

31. Schwingshackl L, Hoffmann G. Dietary fatty acids in the secondary prevention of coronary heart disease: a systematic review, meta-analysis and meta-regression. *BMJ Open*. 2014;4(4):e004487.

32. Arab L. Biomarkers of fat and fatty acid intake. *J Nutr*. 2003;133 Suppl 3(3):925S-32S.

33. de Souza RJ, Mente A, Maroleanu A, Cozma AI, Ha V, Kishibe T, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. *BMJ*. 2015;351:3978.

34. Hooper L, Martin N, Abdelhamid A, Davey Smith G. Reduction in saturated fat intake for cardiovascular disease. *Cochrane Database Syst Rev*. 2015;6:CD011737.

35. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013;368(14):1279-90.

36. Li Y, Hruby A, Bernstein AM, Ley SH, Wang DD, Chiuve SE, et al. Saturated Fats Compared With Unsaturated Fats and Sources of Carbohydrates in Relation to Risk of Coronary Heart Disease: A Prospective Cohort Study. *J Am Coll Cardiol*. 2015;66(14):1538-48.

37. Mustad VA, Etherton TD, Cooper AD, Mastro AM, Pearson TA, Jonnalagadda SS, et al. Reducing saturated fat intake is associated with increased levels of LDL receptors on mononuclear cells in healthy men and women. *J Lipid Res*. 1997;38(3):459-68.

38. Calder PC. Functional Roles of Fatty Acids and Their Effects on Human Health. *JPEN J Parenter Enteral Nutr*. 2015;39(1 Suppl):18S-32S.

39. Berglund L, Oliver EH, Fontanez N, Holleran S, Matthews K, Roheim PS, et al. HDL-subpopulation patterns in response to reductions in dietary total and saturated fat intakes in healthy subjects. *Am J Clin Nutr*. 1999;70(6):992-1000.

40. Turner JD, Le NA, Brown WV. Effect of changing dietary fat saturation on low-density lipoprotein metabolism in man. *Am J Physiol*. 1981;241(1):E57-63.

41. Engel S, Tholstrup T. Butter increased total and LDL cholesterol compared with olive oil but resulted in higher HDL cholesterol compared with a habitual diet. *Am J Clin Nutr*. 2015;102(2):309-15.

42. Bjermo H, Iggman D, Kullberg J, Dahlman I, Johansson L, Persson L, et al. Effects of n-6 PUFAs compared with SFAs on liver fat, lipoproteins, and inflammation in abdominal obesity: a randomized controlled trial. *Am J Clin Nutr*. 2012;95(5):1003-12.

43. Farvid MS, Ding M, Pan A, Sun Q, Chiuve SE, Steffen LM, et al. Dietary linoleic acid and risk of coronary heart disease: a systematic review and meta-analysis of prospective cohort studies. *Circulation*. 2014;130(18):1568-78.

44. Harris WS, Mozaffarian D, Rimm E, Kris-Etherton P, Rudel LL, Appel LJ, et al. Omega-6 fatty acids and risk for cardiovascular disease: a science advisory from the American Heart Association Nutrition Subcommittee of the Council on Nutrition, Physical Activity, and Metabolism; Council on Cardiovascular Nursing; and Council on Epidemiology and Prevention. *Circulation*. 2009;119(6):902-7.

45. Saravanan P, Davidson NC, Schmidt EB, Calder PC. Cardiovascular effects of marine omega-3 fatty acids. *Lancet*. 2010;376(9740):540-50.

46. Harris WS. n-3 fatty acids and serum lipoproteins: human studies. *Am J Clin Nutr*. 1997;65(5 Suppl):1645S-54S.

47. Leslie MA, Cohen DJ, Liddle DM, Robinson LE, Ma DW. A review of the effect of omega-3 polyunsaturated fatty acids on blood triacylglycerol levels in normolipidemic and borderline hyperlipidemic individuals. *Lipids Health Dis*. 2015;14:53.

48. Dias CB, Phang M, Wood LG, Garg ML. Postprandial lipid responses do not differ following consumption of butter or vegetable oil when consumed with omega-3 polyunsaturated fatty acids. *Lipids*. 2015;50(4):339-47.

49. Rizos EC, Ntzani EE, Bika E, Kostapanos MS, Elisaf MS. Association between omega-3 fatty acid supplementation and risk of major cardiovascular disease events: a systematic review and meta-analysis. *JAMA*. 2012;308(10):1024-33.

50. Brouwer IA, Wanders AJ, Katan MB. Effect of Animal and Industrial Trans Fatty Acids on HDL and LDL Cholesterol Levels in Humans – A Quantitative Review. *PLoS ONE*. 2010;5(3):e9434.

51. Nishida C, Uauy R. WHO Scientific Update on health consequences of trans fatty acids: introduction. *Eur J Clin Nutr*. 2009;63:S1-S4.

52. Australian Bureau of Statistics. 4364.0.55.007 - Australian Health Survey: Nutrition First Results - Food and Nutrients, 2011-12. Canberra: Commonwealth of Australia; 2014.

53. Food Standards Australia New Zealand. Supporting Document 1: 2013 Survey of the tran fatty acid content of Australian and New Zealand foods. In: Zealand FSAN, editor.: Food Standards Australia New Zealand; 2013.

54. Threapleton DE, Greenwood DC, Evans CE, Cleghorn CL, Nykjaer C, Woodhead C, et al. Dietary fibre intake and risk of cardiovascular disease: systematic review and meta-analysis. *BMJ*. 2013;347:f6879.

55. Erkkila AT, Lichtenstein AH. Fiber and cardiovascular disease risk: how strong is the evidence? *J Cardiovasc Nurs*. 2006;21(1):3-8.

56. Ellegard L, Andersson H. Oat bran rapidly increases bile acid excretion and bile acid synthesis: an ileostomy study. *Eur J Clin Nutr*. 2007;61(8):938-45.

57. Mirrahimi A, de Souza RJ, Chiavaroli L, Sievenpiper JL, Beyene J, Hanley AJ, et al. Associations of glycemic index and load with coronary heart disease events: a systematic review and meta-analysis of prospective cohorts. *J Am Heart Assoc*. 2012;1(5):e000752.

58. Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Intern Med*. 2014;174(4):516-24.

59. Morgantini C, Xiao C, Dash S, Lewis GF. Dietary carbohydrates and intestinal lipoprotein production. *Curr Opin Clin Nutr Metab Care*. 2014;17(4):355-9.

60. Te Morenga LA, Howatson AJ, Jones RM, Mann J. Dietary sugars and cardiometabolic risk: systematic review and meta-analyses of randomized controlled trials of the effects on blood pressure and lipids. *Am J Clin Nutr*. 2014;100(1):65-79.

61. Nichenametla SN, Weidauer LA, Wey HE, Beare TM, Specker BL, Dey M. Resistant starch type 4-enriched diet lowered blood cholesterols and improved body composition in a double blind controlled cross-over intervention. *Mol Nutr Food Res*. 2014;58(6):1365-9.

62. Mathews MJ, Liebenberg L, Mathews EH. How do high glycemic load diets influence coronary heart disease? *Nutr Metab (Lond)*. 2015;12(6):6.

63. Smith CE, Tucker KL. Health benefits of cereal fibre: a review of clinical trials. *Nutr Res Rev*. 2011;24(1):118-31.

64. Ludwig DS. The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *JAMA*. 2002;287(18):2414-23.

65. Maersk M, Belza A, Stodkilde-Jorgensen H, Ringgaard S, Chabanova E, Thomsen H, et al. Sucrose-sweetened beverages increase fat storage in the liver, muscle, and visceral fat depot: a 6-mo randomized intervention study. *Am J Clin Nutr*. 2012;95(2):283-9.

66. Stanhope KL, Schwarz JM, Havel PJ. Adverse metabolic effects of dietary fructose: results from the recent epidemiological, clinical, and mechanistic studies. *Curr Opin Lipidol*. 2013;24(3):198-206.

67. Lagiou P, Sandin S, Lof M, Trichopoulos D, Adami HO, Weiderpass E. Low carbohydrate-high protein diet and incidence of cardiovascular diseases in Swedish women: prospective cohort study. *BMJ*. 2012;344:e4026.

68. Halton TL, Willett WC, Liu S, Manson JE, Albert CM, Rexrode K, et al. Low-carbohydrate-diet score and the risk of coronary heart disease in women. *N Engl J Med*. 2006;355(19):1991-2002.

69. Trichopoulou A, Psaltopoulou T, Orfanos P, Hsieh CC, Trichopoulos D. Low-carbohydrate-high-protein diet and long-term survival in a general population cohort. *Eur J Clin Nutr*. 2007;61(5):575-81.

70. Santesso N, Akl EA, Bianchi M, Mente A, Mustafa R, Heels-Ansdell D, et al. Effects of higher- versus lower-protein diets on health outcomes: a systematic review and meta-analysis. *Eur J Clin Nutr*. 2012;66(7):780-8.

71. Bohl M, Bjornshave A, Rasmussen KV, Schioldan AG, Amer B, Larsen MK, et al. Dairy proteins, dairy lipids, and postprandial lipemia in persons with abdominal obesity (DairyHealth): a 12-wk, randomized, parallel-controlled, double-blinded, diet intervention study. *Am J Clin Nutr*. 2015;101(4):870-8.

72. Stanstrup J, Schou SS, Holmer-Jensen J, Hermansen K, Dragsted LO. Whey protein delays gastric emptying and suppresses plasma fatty acids and their metabolites compared to casein, gluten, and fish protein. *J Proteome Res*. 2014;13(5):2396-408.

73. Holmer-Jensen J, Mortensen LS, Astrup A, de Vrese M, Holst JJ, Thomsen C, et al. Acute differential effects of dietary protein quality on postprandial lipemia in obese non-diabetic subjects. *Nutr Res*. 2013;33(1):34-40.

74. Whelton PK, Appel LJ, Sacco RL, Anderson CA, Antman EM, Campbell N, et al. Sodium, blood pressure, and cardiovascular disease: further evidence supporting the American Heart Association sodium reduction recommendations. *Circulation*. 2012;126(24):2880-9.

75. Unger T. Decade in review--hypertension: the past decade in hypertension--facts, hopes, and hypes. *Nat Rev Cardiol*. 2014;11(11):633-5.

76. Freis ED. Salt, volume and the prevention of hypertension. *Circulation*. 1976;53(4):589-95.

77. Blaustein MP, Leenen FH, Chen L, Golovina VA, Hamlyn JM, Pallone TL, et al. How NaCl raises blood pressure: a new paradigm for the pathogenesis of salt-dependent hypertension. *Am J Physiol Heart Circ Physiol*. 2012;302(5):H1031-49.

78. Castro H, Raij L. Potassium in hypertension and cardiovascular disease. *Semin Nephrol*. 2013;33(3):277-89.

79. Espin JC, Garcia-Conesa MT, Tomas-Barberan FA. Nutraceuticals: facts and fiction. *Phytochemistry*. 2007;68(22-24):2986-3008.

80. Chen ZY, Ma KY, Liang YT, Peng C, Zuo YY. Role and classification of cholesterol-lowering functional foods. *J Funct Foods*. 2011;3(2):61-9.

81. Gylling H, Plat J, Turley S, Ginsberg HN, Ellegard L, Jessup W, et al. Plant sterols and plant stanols in the management of dyslipidaemia and prevention of cardiovascular disease. *Atherosclerosis*. 2014;232(2):346-60.

82. Jenkins DJ, Mirrahimi A, Srichaikul K, Berryman CE, Wang L, Carleton A, et al. Soy protein reduces serum cholesterol by both intrinsic and food displacement mechanisms. *J Nutr*. 2010;140(12):2302S-11S.

83. Lule VK, Garg S, Pophaly SD, Hitesh, Tomar SK. "Potential health benefits of lunasin: a multifaceted soy-derived bioactive peptide". *J Food Sci*. 2015;80(3):R485-94.

84. Cespedes EM, Hu FB. Dietary patterns: from nutritional epidemiologic analysis to national guidelines. *Am J Clin Nutr*. 2015;101(5):899-900.

85. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. *Circulation*. 2010;121(4):586-613.

86. Schwingshackl L, Hoffmann G. Diet quality as assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension score, and health outcomes: a systematic review and meta-analysis of cohort studies. *J Acad Nutr Diet*. 2015;115(5):780-800 e5.

87. Wirt A, Collins CE. Diet quality--what is it and does it matter? *Public Health Nutr*. 2009;12(12):2473-92.

88. Sala-Vila A, Romero-Mamani ES, Gilabert R, Nunez I, de la Torre R, Corella D, et al. Changes in ultrasound-assessed carotid intima-media thickness and plaque with a Mediterranean diet: a substudy of the PREDIMED trial. *Arterioscler Thromb Vasc Biol*. 2014;34(2):439-45.

89. Petersen KS, Clifton PM, Blanch N, Keogh JB. Effect of improving dietary quality on carotid intima media thickness in subjects with type 1 and type 2 diabetes: a 12-mo randomized controlled trial. *Am J Clin Nutr*. 2015;102(4):771-9.

90. Gardener H, Wright CB, Cabral D, Scarmeas N, Gu Y, Cheung K, et al. Mediterranean diet and carotid atherosclerosis in the Northern Manhattan Study. *Atherosclerosis*. 2014;234(2):303-10.

91. Dias JA, Wirfalt E, Drake I, Gullberg B, Hedblad B, Persson M, et al. A high quality diet is associated with reduced systemic inflammation in middle-aged individuals. *Atherosclerosis*. 2015;238(1):38-44.

92. Rodriguez-Monforte M, Flores-Mateo G, Sanchez E. Dietary patterns and CVD: a systematic review and meta-analysis of observational studies. *Br J Nutr*. 2015;114(9):1341-59.

93. Rees K, Hartley L, Flowers N, Clarke A, Hooper L, Thorogood M, et al. 'Mediterranean' dietary pattern for the primary prevention of cardiovascular disease. *Cochrane Database Syst Rev*. 2013;8:CD009825.

94. Trichopoulou A, Martinez-Gonzalez MA, Tong TY, Forouhi NG, Khandelwal S, Prabhakaran D, et al. Definitions and potential health benefits of the Mediterranean diet: views from experts around the world. *BMC Med*. 2014;12(1):112.

95. Tognon G, Hebestreit A, Lanfer A, Moreno LA, Pala V, Siani A, et al. Mediterranean diet, overweight and body composition in children from eight European countries: cross-sectional and prospective results from the IDEFICS study. *Nutr Metab Cardiovasc Dis*. 2014;24(2):205-13.

96. Giannini C, Diesse L, D'Adamo E, Chiavaroli V, de Giorgis T, Di Iorio C, et al. Influence of the Mediterranean diet on carotid intima-media thickness in hypercholesterolaemic children: a 12-month intervention study. *Nutr Metab Cardiovasc Dis*. 2014;24(1):75-82.

97. Chowdhury R, Stevens S, Gorman D, Pan A, Warnakula S, Chowdhury S, et al. Association between fish consumption, long chain omega 3 fatty acids, and risk of cerebrovascular disease: systematic review and meta-analysis. *BMJ*. 2012;345:e6698.

98. Zheng J, Huang T, Yu Y, Hu X, Yang B, Li D. Fish consumption and CHD mortality: an updated meta-analysis of seventeen cohort studies. *Public Health Nutr*. 2012;15(4):725-37.

99. Jenkins DJ, Jones PJ, Lamarche B, Kendall CW, Faulkner D, Cermakova L, et al. Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. *JAMA*. 2011;306(8):831-9.

100. Jenkins DJ, Chiavaroli L, Wong JM, Kendall C, Lewis GF, Vidgen E, et al. Adding monounsaturated fatty acids to a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *CMAJ*. 2010;182(18):1961-7.

101. Jenkins DJ, Kendall CW, Faulkner DA, Nguyen T, Kemp T, Marchie A, et al. Assessment of the longer-term effects of a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *Am J Clin Nutr*. 2006;83(3):582-91.

102. Jenkins DJ, Srichaikul K, Mirrahimi A, Chiavaroli L, Kendall CW. Functional foods to increase the efficacy of diet in lowering serum cholesterol. *Can J Cardiol*. 2011;27(4):397-400.

103. Jenkins DJ, Kendall CW, Faulkner D, Vidgen E, Trautwein EA, Parker TL, et al. A dietary portfolio approach to cholesterol reduction: combined effects of plant sterols, vegetable proteins, and viscous fibers in hypercholesterolemia. *Metabolism*. 2002;51(12):1596-604.

104. Jenkins DJ, Kendall CW, Marchie A, Faulkner D, Vidgen E, Lapsley KG, et al. The effect of combining plant sterols, soy protein, viscous fibers, and almonds in treating hypercholesterolemia. *Metabolism*. 2003;52(11):1478-83.

105. Jenkins DJ, Kendall CW, Faulkner DA, Kemp T, Marchie A, Nguyen TH, et al. Long-term effects of a plant-based dietary portfolio of cholesterol-lowering foods on blood pressure. *Eur J Clin Nutr*. 2008;62(6):781-8.

106. Tan SY, Dhillon J, Mattes RD. A review of the effects of nuts on appetite, food intake, metabolism, and body weight. *Am J Clin Nutr*. 2014;100 Suppl 1(Supplement 1):412S-22S.

107. Fung TT, Chiuve SE, McCullough ML, Rexrode KM, Logroscino G, Hu FB. Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. *Arch Intern Med*. 2008;168(7):713-20.

108. Salehi-Abargouei A, Maghsoudi Z, Shirani F, Azadbakht L. Effects of Dietary Approaches to Stop Hypertension (DASH)-style diet on fatal or nonfatal cardiovascular diseases--incidence: a systematic review and meta-analysis on observational prospective studies. *Nutrition*. 2013;29(4):611-8.

109. Harnden KE, Frayn KN, Hodson L. Dietary Approaches to Stop Hypertension (DASH) diet: applicability and acceptability to a UK population. *J Hum Nutr Diet*. 2010;23(1):3-10.

110. Appel LJ, Champagne CM, Harsha DW, Cooper LS, Obarzanek E, Elmer PJ, et al. Effects of comprehensive lifestyle modification on blood pressure control: main results of the PREMIER clinical trial. *JAMA*. 2003;289(16):2083-93.

111. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. *N Engl J Med*. 2001;344(1):3-10.

112. Huang CL, Kuo E. Mechanisms of Disease: WNK-ing at the mechanism of salt-sensitive hypertension. *Nat Clin Pract Nephr*. 2007;3(11):623-30.

113. Lin PH, Allen JD, Li YJ, Yu M, Lien LF, Svetkey LP. Blood Pressure-Lowering Mechanisms of the DASH Dietary Pattern. *J Nutr Metab*. 2012;2012:472396.

114. Miura K, Torii S. Diet, Nutrients, and the Prevention of Hypertension. *Curr Nutr Rep*. 2012;1(2):87-92.

115. Illner AK, Harttig U, Tognon G, Palli D, Salvini S, Bower E, et al. Feasibility of innovative dietary assessment in epidemiological studies using the approach of combining different assessment instruments. *Public Health Nutr*. 2011;14(6):1055-63.

116. Illner AK, Freisling H, Boeing H, Huybrechts I, Crispim SP, Slimani N. Review and evaluation of innovative technologies for measuring diet in nutritional epidemiology. *Int J Epidemiol*. 2012;41(4):1187-203.

117. National Cancer Institute. Dietary Assessment Primer Naational Institutes of Health. Available from: http://dietassessmentprimer.cancer.gov/.

118. Kirkpatrick SI, Reedy J, Butler EN, Dodd KW, Subar AF, Thompson FE, et al. Dietary Assessment in Food Environment Research: A Systematic Review. *Am J Prev Med*. 2014;46(1):94-102.

119. Hoevenaar-Blom MP, Spijkerman AM, Boshuizen HC, Boer JM, Kromhout D, Verschuren WM. Effect of using repeated measurements of a Mediterranean style diet on the strength of the association with cardiovascular disease during 12 years: the Doetinchem Cohort Study. *Eur J Nutr*. 2014;53(5):1209-15.

120. O'Neil CE, Deshmukh-Taskar P, Mendoza JA, Nicklas TA, Liu Y, Relyea G, et al. Dietary, Lifestyle, and Health Correlates of Overweight and Obesity in Adults 19 to 39 Years of Age: The Bogalusa Heart Study. *American Journal of Lifestyle Medicine*. 2011;6(4):347-58.

121. Ma J, Fox CS, Jacques PF, Speliotes EK, Hoffmann U, Smith CE, et al. Sugar-sweetened beverage, diet soda, and fatty liver disease in the Framingham Heart Study cohorts. *J Hepatol*. 2015;63(2):462-9.

122. Sauvageot N, Alkerwi A, Albert A, Guillaume M. Use of food frequency questionnaire to assess relationships between dietary habits and cardiovascular risk factors in NESCAV study: validation with biomarkers. *Nutr J*. 2013;12(1):143.

123. Duffey KJ, Steffen LM, Van Horn L, Jacobs DR, Jr., Popkin BM. Dietary patterns matter: diet beverages and cardiometabolic risks in the longitudinal Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Am J Clin Nutr*. 2012;95(4):909-15.

124. Cade J, Thompson R, Burley V, Warm D. Development, validation and utilisation of food-frequency questionnaires - a review. *Public Health Nutr*. 2002;5(4):567-87.

125. Lyle BJ, Mares-Perlman JA, Klein BE, Klein R, Greger JL. Supplement users differ from nonusers in demographic, lifestyle, dietary and health characteristics. *J Nutr*. 1998;128(12):2355-62.

126. Dickinson A, MacKay D. Health habits and other characteristics of dietary supplement users: a review. *Nutr J*. 2014;13(1):14.

127. Ireland P, Jolley D, Giles G, O'Dea K, Powles J, Rutishauser I, et al. Development of the Melbourne FFQ: a food frequency questionnaire for use in an Australian prospective study involving an ethnically diverse cohort. *Asia Pac J Clin Nutr*. 1994;3(1):19-31.

128. Swierk M, Williams PG, Wilcox J, Russell KG, Meyer BJ. Validation of an Australian electronic food frequency questionnaire to measure polyunsaturated fatty acid intake. *Nutrition*. 2011;27(6):641-6.

129. Collins CE, Boggess MM, Watson JF, Guest M, Duncanson K, Pezdirc K, et al. Reproducibility and comparative validity of a food frequency questionnaire for Australian adults. *Clin Nutr*. 2014;33(5):906-14.

130. Lassale C, Guilbert C, Keogh J, Syrette J, Lange K, Cox DN. Estimating food intakes in Australia: validation of the Commonwealth Scientific and Industrial Research Organisation (CSIRO) food frequency questionnaire against weighed dietary intakes. *J Hum Nutr Diet*. 2009;22(6):559-66.

131. Lai JS, Attia J, McEvoy M, Hure AJ. Biochemical Validation of the Older Australian's Food Frequency Questionnaire Using Carotenoids and Vitamin E. *Nutrients*. 2014;6(11):4906-17.

132. Tan LM, Charlton KE, Tan SY, Ma G, Batterham M. Validity and reproducibility of an iodine-specific food frequency questionnaire to estimate dietary iodine intake in older Australians. *Nutr Diet*. 2013;70(1):71-8.

133. Ambrosini GL, de Klerk NH, O'Sullivan TA, Beilin LJ, Oddy WH. The reliability of a food frequency questionnaire for use among adolescents. *Eur J Clin Nutr*. 2009;63(10):1251-9.

134. Burrows T, Berthon B, Garg ML, Collins CE. A comparative validation of a child food frequency questionnaire using red blood cell membrane fatty acids. *Eur J Clin Nutr*. 2012;66(7):825-9.

135. Dickinson KM, Delaney CL, Allan R, Spark I, Miller MD. Validation of a Brief Dietary Assessment Tool for Estimating Dietary EPA and DHA Intake in Australian Adults at Risk of Cardiovascular Disease. *J Am Coll Nutr*. 2015;34(4):333-9.

136. McKellar S, Horsley P, Chambers R, Pullen M, Vandersee P, Clarke C, et al. Development of the Diet Habits Questionnaire for use in cardiac rehabilitation. *Aust J Prim Health*. 2008;14(3):43-7.

137. Fransen HP, Ocke MC. Indices of diet quality. *Curr Opin Clin Nutr Metab Care*. 2008;11(5):559-65.

138. Waijers PM, Feskens EJ, Ocke MC. A critical review of predefined diet quality scores. *Br J Nutr*. 2007;97(2):219-31.

139. McCullough ML, Willett WC. Evaluating adherence to recommended diets in adults: the Alternate Healthy Eating Index. *Public Health Nutr*. 2006;9(1A):152-7.

140. Bhupathiraju SN, Lichtenstein AH, Dawson-Hughes B, Tucker KL. Adherence index based on the AHA 2006 diet and lifestyle recommendations is associated with select cardiovascular disease risk factors in older Puerto Ricans. *J Nutr*. 2011;141(3):460-9.

141. Framingham Heart Study. History of the Framingham Heart Study 2015. Available from: https://www.framinghamheartstudy.org/about-fhs/history.php.

142. Dawber TR, Kannel WB, Revotskie N, Stokes J, Kagan A, Gordon T. Some Factors Associated with the Development of Coronary Heart Disease—Six Years' Follow-Up Experience in the Framingham Study. *Am J Public Health Nations Health*. 1959;49(10):1349-56.

143. National Vascular Disease Prevention Alliance. Guidelines for the assessment of absolute cardiovascular disease risk. National Heart Foundation of Australia; 2009.

144. National Vascular Disease Prevention Alliance. Australian Cardiovascular Risk Charts. PDF: Heart Foundation of Australia; 2009.

145. Anderson TJ, Gregoire J, Hegele RA, Couture P, Mancini GB, McPherson R, et al. 2012 update of the Canadian Cardiovascular Society guidelines for the diagnosis and treatment of dyslipidemia for the prevention of cardiovascular disease in the adult. *Can J Cardiol*. 2013;29(2):151-67.

146. Perk J, De Backer G, Gohlke H, Graham I, Reiner Z, Verschuren M, et al. European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts). *Eur Heart J*. 2012;33(13):1635-701.

147. Kelishadi R, Poursafa P. A review on the genetic, environmental, and lifestyle aspects of the early-life origins of cardiovascular disease. *Curr Probl Pediatr Adolesc Health Care*. 2014;44(3):54-72.

148. Teslovich TM, Musunuru K, Smith AV, Edmondson AC, Stylianou IM, Koseki M, et al. Biological, clinical and population relevance of 95 loci for blood lipids. *Nature*. 2010;466(7307):707-13.

149. Forouhi NG, Sattar N. CVD risk factors and ethnicity--a homogeneous relationship? *Atheroscler Suppl*. 2006;7(1):11-9.

150. Gasevic D, Ross ES, Lear SA. Ethnic Differences in Cardiovascular Disease Risk Factors: A Systematic Review of North American Evidence. *Can J Cardiol*. 2015;31(9):1169-79.

151. Thanassoulis G, Peloso GM, Pencina MJ, Hoffmann U, Fox CS, Cupples LA, et al. A genetic risk score is associated with incident cardiovascular disease and coronary artery calcium: the Framingham Heart Study. *Circ Cardiovasc Genet*. 2012;5(1):113-21.

152. Bachmann JM, Willis BL, Ayers CR, Khera A, Berry JD. Association between family history and coronary heart disease death across long-term follow-up in men: the Cooper Center Longitudinal Study. *Circulation*. 2012;125(25):3092-8.

153. Laitinen TT, Pahkala K, Magnussen CG, Oikonen M, Viikari JS, Sabin MA, et al. Lifetime measures of ideal cardiovascular health and their association with subclinical atherosclerosis: The Cardiovascular Risk in Young Finns Study. *Int J Cardiol*. 2015;185:186-91.

154. Australian Bureau of Statistics. Australian Health Survey: Biomedical results for Chronic Diseases, 2011-12. Canberra: Commonwealth of Australia; 2013.

155. Australian Institute of Health and Welfare. Australia's health 2014. Canberra: AIHW; 2014.

156. Australian Bureau of Statistics. 4364.0.55.001 - Australian Health Survey: First Results, 2011-2012. Canberra: Commonweath of Australia; 2012.

157. National Health and Medical Research Council. Australian Dietary Guidelines. In: National Health and Medical Research Council, editor. Canberra: Commonwealth of Australia; 2013.

158. Sud R, Roy B, Emerson J, Hennessy A. Associations between family history of cardiovascular disease, knowledge of cardiovascular disease risk factors and health behaviours. *Aust J Prim Health*. 2013;19(2):119-23.

159. Sugiyama T, Tsugawa Y, Tseng CH, Kobayashi Y, Shapiro MF. Different time trends of caloric and fat intake between statin users and nonusers among US adults: gluttony in the time of statins? *JAMA Intern Med*. 2014;174(7):1038-45.

160. Khanolkar AR, Byberg L, Koupil I. Parental influences on cardiovascular risk factors in Swedish children aged 5-14 years. *Eur J Public Health*. 2012;22(6):840-7.

161. Schwandt P, Haas GM, Liepold E. Lifestyle and cardiovascular risk factors in 2001 child-parent pairs: the PEP Family Heart Study. *Atherosclerosis*. 2010;213(2):642-8.

162. Austin MA, McKnight B, Edwards KL, Bradley CM, McNeely MJ, Psaty BM, et al. Cardiovascular disease mortality in familial forms of hypertriglyceridemia: A 20-year prospective study. *Circulation*. 2000;101(24):2777-82.

163. Bao W, Srinivasan SR, Valdez R, Greenlund KJ, Wattigney WA, Berenson GS. Longitudinal changes in cardiovascular risk from childhood to young adulthood in offspring of parents with coronary artery disease: the Bogalusa Heart Study. *JAMA*. 1997;278(21):1749-54.

164. Hall L, Collins CE, Morgan PJ, Burrows TL, Lubans DR, Callister R. Children's intake of fruit and selected energy-dense nutrient-poor foods is associated with fathers' intake. *J Am Diet Assoc*. 2011;111(7):1039-44.

165. Di Castelnuovo A, Quacquaruccio G, Donati MB, de Gaetano G, Iacoviello L. Spousal concordance for major coronary risk factors: a systematic review and meta-analysis. *Am J Epidemiol*. 2009;169(1):1-8.

166. Kral TV, Rauh EM. Eating behaviors of children in the context of their family environment. *Physiol Behav*. 2010;100(5):567-73.

167. Woodruffe S, Neubeck L, Clark RA, Gray K, Ferry C, Finan J, et al. Australian Cardiovascular Health and Rehabilitation Association (ACRA) core components of cardiovascular disease secondary prevention and cardiac rehabilitation 2014. *Heart Lung Circ*. 2015;24(5):430-41.

168. Australian Institute of Health and Welfare. Cardiovascular disease: Australian Facts 2011. Canberra: AIHW; 2011.

169. Lawson JS, Bauman AE. Public health Australia: an introduction. Second ed. Roseville: McGraw Hill, Australia; 1998.

170. Australian Government, NHMRC. Australian Guide to Healthy Eating: NHMRC; 2013 [cited 2014 3 November, 2014]. Available from: http://www.eatforhealth.gov.au/guidelines/australian-guide-healthy-eating.

171. World Health Organization. Healthy diet. Fact sheet No394: World Health Organization; 2015 [cited 2015 10 November]. Available from: http://www.who.int/mediacentre/factsheets/fs394/en/.

172. National Vascular Disease Prevention Alliance, Lalor E, Boyden A, Cadilhac D, Colagiur S, Doust J, et al. Guidelines for the management of absolute cardiovascular disease risk. 2012 Report: 0987283014.

173. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Miller NH, Hubbard VS, et al. 2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2013;129(25_suppl_2):S76-S99.

174. J.B.S. Board. Joint British Societies' consensus recommendations for the prevention of cardiovascular disease (JBS3). *Heart*. 2014;100 Suppl 2(Suppl 2):ii1-ii67.

175. National Health and Medical Research Council. Australian Guidelines to reduce health risks from drinking alcohol. Canberra: Commonwealth of Australia; 2009.

176. Aroney C, Aylward P, Kelly A, Chew D, Clune E. National Heart Foundation of Australia Cardiac Society of Australia and New Zealand Guidelines for the management of acute coronary syndromes 2006. *Med J Aust*. 2006;184(8):S1-+.

177. Aroney CN, Aylward P, Chew DP, Huang N, Kelly AM, White H, et al. 2007 addendum to the National Heart Foundation of Australia/Cardiac Society of Australia and New Zealand Guidelines for the management of acute coronary syndromes 2006. *Med J Aust*. 2008;188(5):302-3.

178. National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand. Reducing risk in heart disease: an expert guide to clinical practice for secondary prevention of coronary heart disease. Melbourne: National Heart Foundation of Australia; 2012. p. 1-19.

179. Chow CK, Jolly S, Rao-Melacini P, Fox KA, Anand SS, Yusuf S. Association of diet, exercise, and smoking modification with risk of early cardiovascular events after acute coronary syndromes. *Circulation*. 2010;121(6):750-8.

180. Redfern J, Hyun K, Chew DP, Astley C, Chow C, Aliprandi-Costa B, et al. Prescription of secondary prevention medications, lifestyle advice, and referral to rehabilitation among acute coronary syndrome inpatients: results from a large prospective audit in Australia and New Zealand. *Heart*. 2014;100(16):1281-8.

181. National Heart Foundation of Australia. Secondary prevention of cardiovascular diease. 2010.

182. Ghisi GL, Abdallah F, Grace SL, Thomas S, Oh P. A systematic review of patient education in cardiac patients: do they increase knowledge and promote health behavior change? *Patient Educ Couns*. 2014;95(2):160-74.

183. Johnson NA, Inder KJ, Ewald BD, James EL, Bowe SJ. Association between Participation in Outpatient Cardiac Rehabilitation and Self-Reported Receipt of Lifestyle Advice from a Healthcare Provider: Results of a Population-Based Cross-Sectional Survey. *Rehabil Res Pract*. 2010;2010:541741.

184. Pomeroy SE, Cant RP. General practitioners' decision to refer patients to dietitians: insight into the clinical reasoning process. *Aust J Prim Health*. 2010;16(2):147-53.

185. Chew DP, French J, Briffa TG, Hammett CJ, Ellis CJ, Ranasinghe I, et al. Acute coronary syndrome care across Australia and New Zealand: the SNAPSHOT ACS study. *Med J Aust*. 2013;199(3):185-91.

Chapter 3: How dietary evidence for the prevention and treatment of CVD is translated into practice in those with or at high risk of CVD: A systematic review

This chapter was submitted in 2015.

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3.1 Overview

This chapter is a systematic review of the literature to identify interventions in populations with or at high risk of CVD where dietary evidence was successfully translated. The translational strategies identified as effective were incorporated into the intervention studies reported in Chapters 6 and 8. Additional files referred to in text were offered as available to be published electronically. These documents are presented in the appendices and cross-referenced according.

3.2 Abstract

Objective: Cardiovascular disease (CVD) is a leading cause of mortality and morbidity, and nutrition is an important lifestyle factor. The aim of this systematic review was to synthesise the literature relating to knowledge translation of dietary evidence for the prevention and treatment of CVD into practice in populations with or at high risk of CVD.

Design: A systematic search of six electronic databases (CINAHL, Cochrane, EMBASE, Medline, PsycINFO and Scopus) was performed. Studies were included if a nutrition or dietary knowledge translation was demonstrated to occur with a relevant separate measureable outcome. Quality was assessed using a tool adapted from two quality checklists.

Subjects: Population with or at high risk of CVD or clinicians likely to treat this population.

Results: A total of 4420 titles and abstracts were screened for inclusion, with 354 full texts retrieved to assess inclusion. Forty-three articles were included in the review, relating to 35 separate studies. No studies specifically stated their aim to be KT. Thirty-one studies were in patient or high-risk populations and four targeted health professionals. Few studies stated a theory on which the intervention was based (n=10) and provision of instruction was the most common behaviour change strategy used (n=26).

Conclusions: Knowledge translation in nutrition and dietary studies has been inferred, not stated, with few details provided regarding how dietary knowledge is

translated to the end user. This presents challenges for implementation by clinicians and policy and decision makers. Consequently a need exists to improve the quality of publications in this area.

Protocol Registration: PROSPERO 2014:CRD42014007404

3.3 Introduction

Cardiovascular disease (CVD) is the leading cause of non-communicable deaths worldwide (1). The direct and indirect costs associated with CVD are high, with CVD-related healthcare costs accounting for 12% of the total Australian heath care budget in 2008-09 (AUD $7,605 million) and an estimated lost income of AUD$1.1 billion due to exit from the labour force (2009) (2, 3). As the population in Australia ages, so too does the economic burden of chronic CVD conditions (4). Nutrition is recognised as an important contributor to the prevention of primary and secondary CVD events (5-8). Dietary intakes affect the biochemical pathways contributing to hypercholesterolaemia, hypertension, hyperglycaemia, insulin resistance and inflammation which contribute to the development and progression of CVD (9). High quality diets, such as those containing greater amounts and variety of fruits and vegetables and lower amounts of energy-dense, nutrient-poor foods, are associated with a lower risk of subsequent CVD related morbidity and mortality in those with pre-existing CVD risk factors (10). However, the Prospective Urban Rural Epidemiology study found that of 7519 individuals from 17 countries who had experienced a self-reported CVD event, only 39% were considered to have a healthy diet in the 4-5 years following the event (11). This indicates that appropriate nutrition knowledge for the prevention and treatment of CVD failed to be incorporated into long-term behaviour change for these individuals. It is likely that this was due to a range of reasons.

Knowledge translation (KT) describes the process that encompasses the stages from development and synthesis of the evidence-based knowledge through to the translation of this knowledge by health care providers to consumers into subsequent health behaviours, with the end goal of improved individual health. The Canadian Institute of Health Research defines this process as “a dynamic and iterative process that includes synthesis, dissemination, exchange and ethically-

sound application of knowledge" (12). It is also described as the knowledge-to-action cycle where a knowledge creation process precedes an action cycle, in which the created knowledge is utilised by a range of decision makers and stakeholders, ranging from patients to healthcare policy makers (13, 14). It is the action cycle that is the focus of this review, where end-users implement and utilise the evidence-based knowledge (15). As this is a behaviour change process, it is suggested that implementation of KT should be based on a theoretical framework (16, 17).

Scott et al. (2012) (18) in a previous review of KT in the allied health fields of nutrition and dietetics, occupational therapy, pharmacy, physiotherapy and speech-pathology found that research publications reported mixed results from studies seeking to translate knowledge into practice. Studies of KT strategies were reported generally to be of poor methodological quality, and no particular type of KT strategy was shown to be more effective than others. Education only as a KT strategy was commonly employed, with consistent non-significant results. A literature review of strategies used to achieve lifestyle changes following CVD events found that education was commonly used to support adherence to heart-healthy dietary recommendations, with staff highly trained to provide the intervention (19). However, that review did not focus on diet exclusively, nor how nutrition knowledge was translated. It is the need for this nutrition KT that was specifically highlighted in the European Guidelines on CVD prevention in clinical practice (2012): "*The challenge for coming years is to translate nutritional guidelines into diets that are attractive to people and to find ways in which to make people change their (long-standing) dietary habits"* (7).

The objective of this systematic review was to identify how the best available current evidence on diet for the prevention and treatment of CVD is translated into practice in those with or at high risk of CVD. The primary aim was to identify aspects of successful health service nutrition translation studies in CVD, in terms of the methodology, including theoretical framework, implementation strategies, program design, resources, use of technology, and message transmission channels. A secondary aim was to evaluate the methodological quality of these translation

studies and the effectiveness of nutrition evidence translation on diet-related CVD risk factors.

3.4 Methods

The conduct and reporting of this systematic review adhered to the guidelines stated in the Preferred Reporting Items for Systematic Reviews and Meta-analysis (PRISMA) Statement (20). The systematic review protocol was registered with PROSPERO (CRD 42014007404):

http://www.crd.york.ac.uk/PROSPERO/display_record.asp?ID=CRD42014007404

3.4.1 Eligibility criteria

3.4.1.1 *Participants*

Adults (classed as 18 years or older) with one or more CVD diagnoses were included. Relevant CVD diagnoses included angina (stable or unstable), coronary artery disease (CAD), coronary heart disease (CHD), myocardial infarction (MI), acute myocardial infarction (AMI) or interventions such as coronary artery by-pass graft (CABG) or percutaneous transluminal coronary angioplasty (PTCA). In cases where adults and minors were included together, results for adults had to be reported separately. Due to the nature of some CVDs where living skills may be diminished, interventions targeting the carers of people with a CVD diagnosis were also included. As KT studies can also relate to service providers, health professionals directly treating those with a CVD diagnosis were included, as well as interventions targeting heath care systems or policies directly related to the treatment of patients with diagnosed CVD conditions. This review also intended to provide KT strategies for nutrition in the treatment and prevention of CVD, thus those defined as high risk, 16% and above by the five year absolute cardiovascular disease risk assessment (21), were also included. If an intervention population was a combination of those at moderate and high risk of CVD, either a minimum of 50% of the population had to be at high risk, or results reported separately.

3.4.1.2 *Study inclusion criteria*

Studies were included with either experimental or quasi-experimental designs, with comparators, control groups, wait-list control groups or pre-post designs. Studies were limited to English language in the years from 1985 to 2013, as there is no evidence to show that a lack of studies in languages other than English will bias the results (22), and the timespan reflects recent acknowledgement of the need for translation strategies (see additional file 1: Search strategy and results for the systematic review (Appendix 2)). To be relevant for inclusion, studies needed to assess and report diet or nutrition KT as a separate measureable outcome.

The KT needed to be applicable to one or more of the following areas: evidence synthesis, dissemination, exchange, application or ethically sound application of knowledge. No set length of follow-up was determined as this would be dependent on the intervention delivered. As the focus of the review was on diet and nutrition, the KT had to relate to whole food, not nutrient supplements only. For example, KT regarding fish intakes would be considered eligible, whereas a focus on omega-3 fatty acid supplementation would not. Studies including supplementation within their intervention were not excluded if whole foods were reported separately.

The KT could be either at the personal, community or health care level. Both pre- and post-intervention outcome measures had to be reported in the results to evaluate effectiveness.

3.4.1.3 *Outcomes*

The primary outcomes were dependent upon the stage of the KT spectrum at which the intervention was determined to occur. Outcomes for KT at patient, care-giver, health professional, health system and public health levels as follows:

1. Patient level: Cardiovascular risk markers e.g. serum lipids, blood pressure, arterial stiffness, anthropometrics
2. Care giver level: Cardiovascular risk markers e.g. serum lipids, blood pressure, arterial stiffness, anthropometrics
3. Health professional level: Changes in practice

4. Health system level: Changes in cardiovascular prevention or treatment policies, guidelines, recommendations or best practice
5. Public health or community level:

    a. Improvement in cardiovascular risk rates, as measured by biochemical risk markers
    b. Hospitalisations, morbidity or mortality due to cardiovascular disease
    c. Health expenditure per capita

Secondary outcomes appropriate to the stage of KT were:

1. Patient level: Knowledge or behaviour change related to dietary intake
6. Care giver level: Knowledge or behaviour change related to dietary intake
7. Health professional level: None
8. Health system level: None
9. Public health or community level: None

3.4.2 Search strategy and selection of studies (information sources)

A search strategy was developed and implemented with the search conducted in the databases of CINAHL, Cochrane, EMBASE, Medline, PsycINFO and Scopus. Key terms for KT were sourced from Armstrong *et al.* (2011) (23) and Scott *et al.* (2011, 2012) (18, 24) and cardiovascular terms were identified from two Cochrane systematic reviews by Hooper *et al.* (25) and Hartley *et al.* (26). Nutrition terms encompassed nutrients and nutrition, eating, foods, diets and terms specific to CVD such as dietary fats. Reference lists from included and related studies, relevant conference abstracts, and thesis were searched for additional citations. Protocol publications or references to gain further information on methods related to included studies were sourced and included if relevant. Multiple publications from the same intervention were combined and all relevant outcomes reported.

3.4.3 Process of study selection

Two reviewers independently assessed records based on title and abstract for eligibility and full text retrieval. Full text articles were assessed independently by two reviewers. Disagreements were discussed and until consensus was reached, with a third reviewer consulted when consensus was not reached.

3.4.4 Data extraction

Data were extracted using a spreadsheet, initially piloted for consistency and to ensure all required data were obtained. One reviewer extracted all the data and a second reviewer checked the extracted data for accuracy and consistency. Disagreements were discussed until consensus was reached. Data items included for extraction related to details about the population, intervention, use of control groups, study outcomes related to nutrition or diet. Details regarding the KT strategy used were also extracted, such as the framework, theory or principle on which the translation strategy was based, the behaviours targeted, and change techniques used, as defined by Abraham and Michie (27).

3.4.5 Assessment of study quality

Risk of bias in individual studies was assessed by two reviewers independently using a tool adapted from the American Dietetic Association Evidence Analysis Manual quality criteria checklist (28) and relevant items from the checklist of Workgroup for Intervention Development and Evaluation Research (WIDER) recommendations (29). These items included a detailed intervention description, clarification of the assumed change process and access to intervention manuals (29). The combined tool (Additional file 2: Quality checklist (Appendix 3)) was required to assess both the quality of the nutrition study and also of the behaviour change intervention. Each item was coded as Yes (clearly indicated and present), No (missing or not appropriate), Unclear (if indicated, but insufficient information provided) or Not Applicable (N/A) (see Additional file 2: Quality checklist (Appendix 3) for questions to which N/A could be applied). Each item was considered individually. Relevance questions 1-4 (see Additional file 2: Quality checklist) and validity questions B,C, F and G were weighted for importance

according to the American Dietetic Association Evidence Analysis Manual (28), and quality for the items from WIDER recommendations (29) were reported separately. Quality items as per the WIDER recommendations were given as questions K, L and M. Quality was designated as positive if both questions K and L were yes, neutral if either K and L were positive and M was positive, and negative for one or zero yes in total for all WIDER questions. K and L were designated as the more important translation items as clinicians would have access to their own resources as appropriate (question M).

3.4.6 Data analysis / synthesis

Meta-analysis of data were not expected to be possible due to the heterogeneity in both stage of KT in which interventions could occur and in the expected reporting of outcomes. Data were synthesised into the study characteristics, KT characteristics, intervention content and study quality by one reviewer with data synthesis checked for accuracy and consistency by a second reviewer. Data were further stratified by target population: those with a CVD diagnosis / diagnoses, populations assessed at high risk of CVD, interventions targeting heart failure patients, and those targeting health professionals. Data were stratified using these methods as:

- those with a CVD diagnosis would be expected to have different perceptions of CVD risk or may be at a different stage of change compared to those assessed at high risk of CVD
- those with heart failure would be expected to receive dietary advice primarily addressing sodium and fluid restrictions
- interventions targeting health professionals would be expected to use a substantially different theoretical framework from those perceiving risk to self.

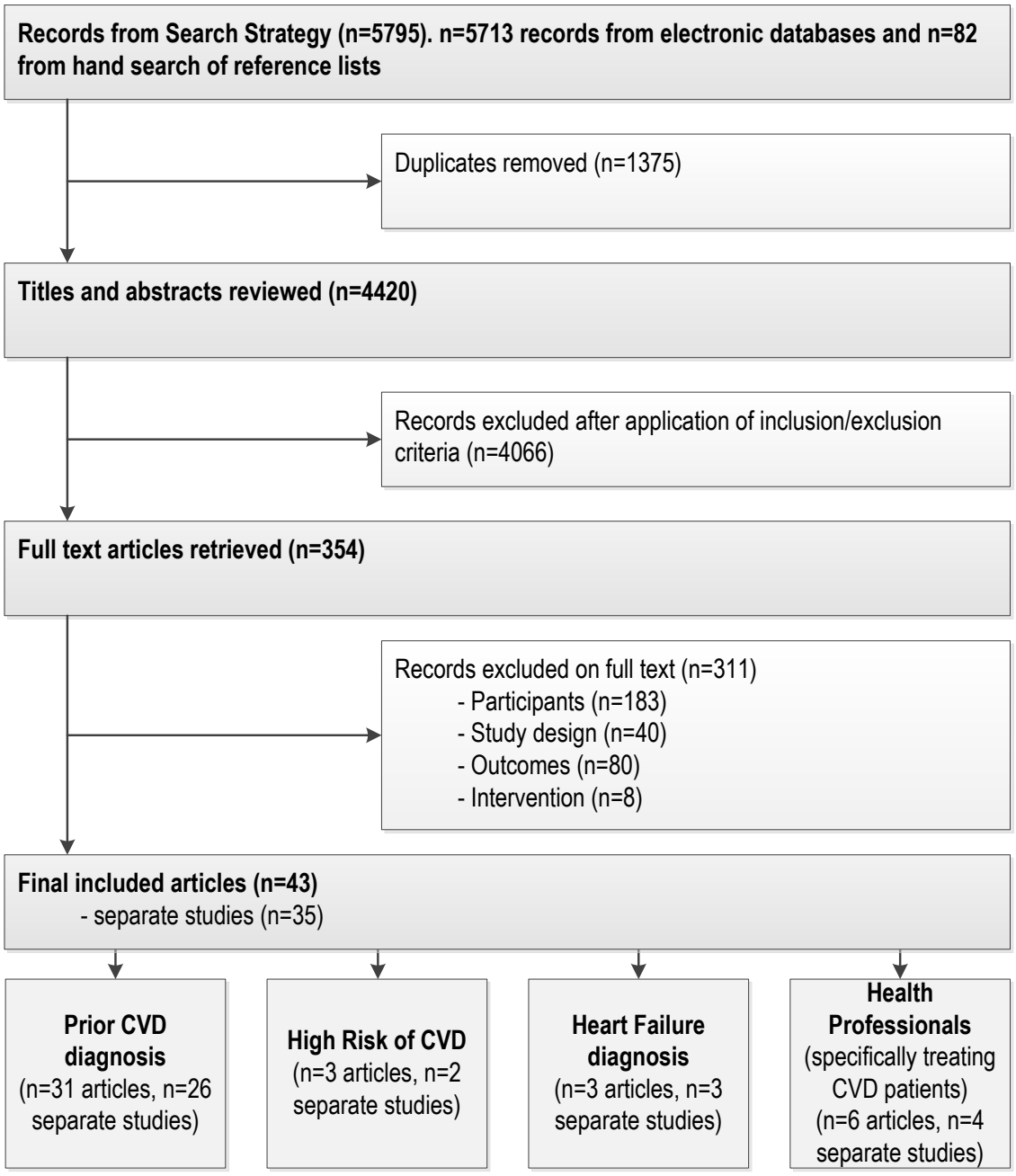

Figure 3.1: Flow chart showing results of search strategy

3.5 Results

The search strategy identified 4420 titles after duplicates were removed (see Flow chart showing results of search strategy Figure 3.1), with 354 full texts retrieved and screened for inclusion/exclusion, and 43 texts included that described 35 separate studies (30-72). Primary reasons for exclusion were populations not

specifically related to cardiovascular disease and outcomes not related to nutrition KT.

3.5.1 Risk of bias within studies

The quality of the studies was assessed using the composite tool described above and is provided in Additional file 2: Search strategy for the systematic review (Appendix 2). Three major areas of quality were assessed as relevance, internal validity and strength of KT reporting. The results of the quality assessments are reported in Additional file 3: Methodological quality scores and risk bias assessment in nutrition knowledge translation studies for cardiovascular disease (Appendix 4). No studies were excluded based on quality, as all studies were determined to have limitations in at least one major area related to study quality. Five studies were scored negatively for relevance, due to lack of feasibility of study replication with a limited budget, with the remaining 29 studies assessed as positive in terms of relevance. Seven studies achieved a positive rating for internal validity, with 20 scored as neutral and seven scored as negative. There was limited reporting in terms of details related to the translation of nutrition and dietary knowledge as only three studies were scored positive, three scored as neutral and 28 scored negative. No studies scored positive responses in all three areas, although four studies scored two positive and one neutral response (32, 45, 49, 66). Three scored neutral for validity (32, 45, 49) and one for KT strategy (66). Three studies scored negatively or neutral in all three areas (44, 46, 54). In particular, these three studies all scored negatively for relevance as they were judged as not being feasible for a clinician to replicate the context of current cardiovascular clinics due to high resourcing costs, although they may have been feasible during the time at which the studies were performed. Excluding relevance, these studies were not significantly different from many of the other studies included in the review in either the way KT was reported or in the level of details provided to determine validity.

Disagreements between evaluators of manuscript quality were found in five of the 35 reviewed manuscripts, in individual questions contributing to the overall

categories of validity and knowledge translation strategy. However, these did not affect the overall category grading given for these manuscripts.

3.5.2 Study characteristics

Although all included studies were evaluated as translating nutrition-based knowledge as part of their intervention strategy, no studies specifically stated nutrition or dietary KT to be their primary or secondary aim. Therefore the internal validity of the KT strategy cannot be established as it cannot be proven that it was directly responsible for the primary and secondary outcomes. However, as KT was a fundamental part of the intervention strategy, the effectiveness of the translation has been be inferred through the measures used.

In total, 37 publications from 31 separate studies targeted patients (30-66), whereas four studies (six publications) targeted health professionals (67-72) (see Table 3.1). Of those targeting patients, 18 measured the primary outcome of cardiovascular risk markers (33-36, 39, 40, 42-45, 50, 52-54, 57, 59-62, 64-66). Eight of these had diet or nutrition as the only risk factor being targeted (33, 34, 39, 40, 42, 44, 50, 52, 64, 65), whereas 10 had intervention strategies for multiple risk factors (35, 36, 43, 45, 53, 54, 57, 59-62, 66). All four studies targeting health professionals addressed the primary outcome of changes in practice (67-72), and three had diet as their sole focus (67-71), with the remaining study targeting multiple risk factors (72). In total, 17 included studies focused only on diet (30, 31, 33, 34, 38-40, 42, 44, 47-50, 52, 55, 63-65, 67-71), with the remaining 18 extending the focus to other risk factors, such as smoking, physical activity or adherence to medication.

All participants in the patient-focused studies were on medications for CVD, with only two studies accounting for the effect of medication by requiring participants to maintain constant dosage (44) or initially stratifying based on dosage of relevant medications, although some dosages for this particular study were changed throughout the study (65). Therefore, all patient studies are reported as measures for the secondary outcome of knowledge or behaviour change relating to dietary intake for consistency. Primary outcome results for the two studies

accounting for the effects of medication are included with secondary outcomes in Table 3.2.

There were 26 studies in adults with prior CVD diagnoses (angina: stable or unstable angina, CAD, CHD, MI, AMI, CABG or PTCA) (30-60). Two studies included populations identified as at high risk of CVD (61-63), three studies were in those with a heart failure diagnosis (64-66) and four interventions targeted health professionals treating patients with CVD (67-72).

Table 3.1: Study characteristics

Author , year	Country	Setting	n	Diagnosis	Length of follow-up	Population (age, sex)	Risk factors targeted	Study design	Control group
CARDIOVASCULAR DISEASE DIAGNOSES									
Aish, 1996 (30) Aish, 1996 (31)	Canada	Community general hospitals	104	MI	7 weeks	34-83 years 60% M	Diet	RCT	Yes (concurrent)
Allen 1996 (32)	United States	Teaching hospital	138	CABG	12 months	Age not stated 100% F	Smoking, Exercise, Diet	RCT	Yes (concurrent)
Arntzenius,1985 (33) Kromhout,1986 (34)	Netherlands	University hospital	53	Angina	24 months	<60 years unclear	Diet	Pre-, post-test	No
Billings, 2000 (35) Koertge, 2003 (36)	United States	Hospital	440	CAD	12 months	58±10 years 79% M	Diet, Exercise	Pre-, post-test	No
Campbell,1998 (37)	Scotland	General practices	1343	CHD	12 months	Age unclear Sex unclear	Smoking, Medication, Diet	RCT	Yes (concurrent)
Dalgard, 2001 (38)	Denmark	University hospital	36	MI, CABG, PTCA	12 months	<70 years 86% M	Diet	RCT	Yes (concurrent)
de Lorgeril, 1994 (39) de Lorgeril, 1999 (40)	France	Multi-clinic	605	MI	5 years	<70 years 90% M	Diet	RCT	Yes (concurrent)
Evon, 2004 (41)	United States	Multi-outpatient cardiac rehab	80	MI, CABG, PTCA, Angina	12 weeks	Age not stated 100% M	Exercise, Diet, Medication	Case series	No
Frost, 2004 (42)	United Kingdom	Hospital	55	MI, Angina, CAD	12 weeks	30-70 years 89% M	Diet	RCT	Yes (concurrent)
Giannuzzi, 2008 (43)	Italy	Cardiac rehabilitation centre	3241	MI	3 years	57.9±9.2 years 86% M	Smoking, Exercise, Medication, Diet	RCT	Yes (concurrent)

Table 3.1: Study characteristics

Author , year	Country	Setting	n	Diagnosis	Length of follow-up	Population (age, sex)	Risk factors targeted	Study design	Control group
Gleason, 2002 (44)	United States	Home	35	CHD	8 weeks	40-79 years 60%M	Diet	Pre-, post-test	No
Goodwin, 2012 (45)	United States	Community	16	MI, Angina	unclear	18-75 years 69%F	Smoking, Exercise, Diet	Pre-, post-test	No
Hofman-Bang, 1999 (46)	Sweden	Residential stay	93	PTCA	24 months	<65 years 84% M	Smoking, Exercise, Diet	RCT	Yes (concurrent)
Jackson, 2005a (47) Jackson, 2005b (48)	United Kingdom	CHD clinic in primary care	120	CHD	3 months	65±9 years 59% M	Diet	RCT	Yes (concurrent)
Luszczynska, 2007 (49)	Unclear	Unclear	114	MI	8 months	39-67 years 64% M	Diet	RCT	Yes (concurrent)
Masley, 2001 (50)	United States	Multi-speciality clinics	97	CAD	12 month	Mean: 65 years 30% F	Diet	RCT	Yes (concurrent)
Mildestvedt, 2007 (51)	Norway	Cardiac rehabilitation centre	176	CAD	24 months	56.0±9.3 78% M	Smoking, Exercise, Diet	RCT	Yes (concurrent)
Shenberger, 1992 (52)	United States	Unclear	59	CABG	2 months	38-73 years 100% M	Diet	Pre-, post-test	No
Singh, 2002 (53)	India	Unclear	1000	Angina, MI	2 years	28-75 years 90% M	Diet, Exercise	RCT	Yes (concurrent)
Sundin, 2003 (54)	Sweden	Residential stay + community	132	PTCA, CABG, AMI	12 months	58.8 years 100% M	Smoking, Exercise, Diet	RCT	Yes (concurrent)

Table 3.1: Study characteristics

Author , year	Country	Setting	n	Diagnosis	Length of follow-up	Population (age, sex)	Risk factors targeted	Study design	Control group
Timlin, 2002 (55)	United States	Cardiac out-patient rehabilitation	104	MI, PTCA, CABG, Angina	3 months	35-85 years 81% M	Diet	Pre-, post-test	Yes (concurrent)
Toobert, 1998 (56)	Not stated	Residential stay + community	28	CHD	Unclear	57-63 years 100% F	Diet, Exercise, Smoking	RCT	Yes (concurrent)
Vale, 2003 (57)	Australia	Telephone service	792	CABG, PTCA, AMI, Angina	6 months	58.5±10.6 years 77% M	Smoking, Exercise, Medication, Diet	RCT	Yes (concurrent)
van Elderen-van Kemenade, 1994 (58)	Netherlands	Hospital + telephone service	60	AMI	12 months	33-69 years 82% M	Exercise, Smoking, Medication, Diet	Pre-, post-test	Yes (historical)
Vestfold Heartcare Study, 2003 (59)	Norway	Rehabilitation centre in hospital	197	CABG, Angina, AMI, PTCA	24 months	55±8 years 82% M	Diet, Smoking, Exercise, Medication	RCT	Yes (concurrent)
Wallner, 1999 (60)	Austria	University cardiology & metabolism clinic	60	CAD, Angina, PTCA	18 months	< 70 years 78% M	Smoking, Exercise, Diet	RCT	Yes (concurrent)
HIGH RISK OF CVD									
Gorder, 1986 (61) Van Horn, 1997 (62)	United States	Community-based	6428	High risk	6 years	35-57 years 100% M	Smoking, Diet, Medication	RCT	Yes (concurrent)
Siero, 2000 (63)	Netherlands	Community-based	262?	High risk	4 years	30-70 years 55% F	Diet	Pre-, post	Yes (concurrent)

Table 3.1: Study characteristics

Author , year	Country	Setting	n	Diagnosis	Length of follow-up	Population (age, sex)	Risk factors targeted	Study design	Control group
HEART FAILURE									
Donner Alves, 2012 (64)	Brazil	Outpatients	46	Heart failure, NYHA class I-III	6 months	58±10 years 70% M	Diet	RCT	Yes (concurrent)
Philipson, 2010 (65)	Sweden	Unclear. Community?	30	Heart failure, NYHA class II-IV	12 week	74±8 years 73% M	Diet	RCT	Yes (concurrent)
Powell, 2010 (66)	United States	Outpatients + telephone service	902	Heart failure, NYHA class II-III	2-3 years	63.6±13.5 47% F	Medication, Diet, Exercise	RCT	Yes (concurrent)
HEALTH PROFESSIONALS									
Banz, 2004 (67)	United States	Community	172	CVD prevention & treatment	11 weeks	Dietitians	Diet	RCT	Yes (concurrent)
Carson, 2002 (68)	United States	University	196	CVD patients	25 days	4th year medical students	Diet	Pre-, post-test	Yes (historical)
Perry & McLaren, 2000, 2003a, 2003b (69-71)	United Kingdom	Hospital	400	Stroke patients	Hospital stay only	Doctors, nurses, therapists	Diet	Pre-, post-test	No
Van der Weijden, 1998 (72)	Netherlands	General practice	32	Hypercholesterolaemia patients	Unclear	General practitioners	Medication, Diet, Smoking	RCT	Yes (concurrent)

Abbreviations: MI myocardial infarction; M male; RCT Randomised Controlled Trial; CABG coronary artery bypass graft; F female; Angina Stable or Unstable; CAD coronary artery disease; CHD coronary heart disease; PTCA percutaneous transluminal coronary angioplasty; AMI acute myocardial infarction; NYHA New York Heart Association; CVD cardiovascular disease

Studies were predominately RCTs (n=24, 69%), with the remainder using uncontrolled pre-post interventions (n=6), pre-post interventions with a control group (n=4; two concurrent and two historical control groups) and one case series. Sample sizes ranged between 16 and 6428 participants. Studies targeting participants with a prior CVD diagnosis primarily included MI (n=10), angina (n=9) or CABG (n=8). Interventions targeting populations with or at risk of CVD were followed up between seven weeks and six years, with most following patients for ≤3 months (n=8) or up to 12 or 24 months (n=6, n=5, respectively). Those targeting health professionals were medium or short term (11 weeks, n=1 and 25 days, n=1 respectively) or unclear (n=2). Population ages ranged from 18 to 85 years and were primarily male, with four studies exclusively including males, and ranging from 53-90% male in 22 studies. Only two studies focused exclusively on females, with two other studies including more women than men (55% and 96% female). Studies were primarily implemented in hospital settings (general, teaching and university) (n=9), outpatients or cardiac rehabilitation centres (n=7), clinics or general practices (n=5). Three studies used a residential stay as part of their intervention. Five studies (30-34, 52, 58) were published prior to the release of the first CONSORT statement in 1996 (73) and another 10 were published within five years of the release date (37-40, 46, 50, 56, 60-63, 72).

Table 3.2 summarises the KT characteristics of the included studies, including the nutrition-focused KT outcomes and associated results. The heterogeneity between KT outcomes and measures precluded any combining of results. Validated dietary intake assessment measurement tools or methods were used for the KT outcomes in 22 of the studies with the remaining 24 studies using measured dietary outcomes using methods that were either unclear or not validated. Twenty-two studies found statistically significant results for outcomes related to nutrition and/or dietary KT, with 11 being non-significant, mixed significance for outcomes or significance not stated. One study (38) found the usual care group (comprehensive counselling) had greater improvements than the brief counselling intervention being tested.

The theoretical framework for the intervention was not stated (n=18) or was unclear (n=7) in the majority of studies. Ten studies specifically stated the

theoretical framework, with no one theory being predominantly used (see Table 2). The most commonly used behaviour change strategies, as defined by Abraham and Michie (27) were provision of instruction (e.g. “no day without fruit”) (n=26), followed by provision of feedback on performance (e.g. from analysis of dietary intake) (n=12), the prompting of intention formation (e.g. new resolutions about health habits) (n=11), and the provision of the behaviour-health link (e.g. information on the influence of diet on blood cholesterol levels) (n=8). Five studies reported provision of instruction as the only behaviour change strategy used, with 25 studies employing three or more identifiable behaviour change strategies. It must be noted that these behaviour change strategies may not have been used exclusively for diet when other risk factors such as physical activity or smoking cessation were also the target of the intervention.

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
CARDIOVASCULAR DISEASE DIAGNOSIS					
Aish, 1996 (30) Aish, 1996 (31)	Nutrients & food habits reflecting adherence to guidelines for primary prevention of cardiac disease (Health & Welfare, Canada 1990)	Dietary intake: 3 day food record [UC]. Nutritional self-care: FHQ [Y]	Orem's self-care deficit theory	Prompt intention formation, Provide feedback on performance, Provide general encouragement	Significant differences between Ctrl. & Int. at week 7 for PE total fat (26.4±5.6, 32.38±6.3 respectively, p<0.01) & sat. fat (8.8±2.9,11.1±3.6 respectively, p<0.01). Int. significantly healthier food habits (2.2±0.5, 2.3±0.4, p<0.05)
Allen (1996) (32)	Adherence to low fat diet	Modified block FFQ [UC]	Social Cognitive Theory	Provide instruction, Prompt intention formation, Prompt practice, Prompt specific goal setting, Provide feedback on performance	PE total fat ↓from 38% to 35% (Int. grp > Ctrl, p=0.008). PE sat. fat ↓1% (Int. grp > ctrl p=0.02). No ↓ total energy.
Arntzenius,1985 (33) Kromhout,1986 (34)	Adherence to vegetarian diet : P:S ratio of 2:1 & cholesterol <100mg/day	WFR [Y]	Not stated	Provide instruction	↑in P:S ratio from 0.91±0.62 to 2.54±0.47 (p<0.001), cholesterol↓ (mg/1000 kCal) 88.6±23.5 to 29.5±11.5, p<0.001)
Billings, 2000 (35) Koertge, 2003 (36)	Dietary adherence: PE fat ≤10% total energy, whole foods, plant-based diet	3 day food diary [UC]	Unclear	Provide instruction, Model or demonstrate behaviour, Plan social support or social change, Stress management	Men: ↓PE total fat 12.8±7.8 to 6.3±2.6, women: ↓ PE total fat 16.9±8.5 to 7.6±4.1 (p=0.00, value over time)
Campbell,1998 (37)	Low fat diet (DINE score <30)	DINE score [Y]	Not stated	Prompt specific goal setting, Provide feedback on performance	Proportion of Int. achieving DINE score <30 ↑ from 49.0% to 56.5% (95%CI:2.4-12.6%). Ctrl: no change 48.6% (baseline) & 48.6% (12 month).

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Dalgard, 2001 (38)	Comparison of total & saturated fat intakes from brief (BDC) (Plate Model) & comprehensive dietary counselling (CDC) (NCEP Step 1) groups.	7 day WFR [Y]	Not stated	Provide instruction, Provide feedback on performance, Provide information about behaviour-health link, Unclear	↓4.8% PE total fat in CDC group ($p<0.005$) from baseline (32.7±6.2), BDC NS; ↓ 2.7% PE sat. fat in CDC ($p<0.005$), BDC NS; ↑3.9%PE CHO in CDC, BDC NS.
de Lorgeril, 1994 (39) de Lorgeril, 1999 (40)	Adoption of a Mediterranean-type diet (Int.) compared to Western prudent diet (Ctrl.)	24 hr recall [UC], FFQ [UC], plasma lipid analysis [Y]	Not stated	Provide instruction	After mean 48 months years (Int. compared to Ctrl, g/day ± SEM, $p\leq0.01$): ↑ bread (167±6,145±7), ↑ fruit (251±12, 203±12), ↑ margarine (19.0±1.0, 5.1±0.6); ↓butter & cream (2.8±0.6, 16.6±1.6), ↓meat (40.8±5.0, 60.4±5.5), & ↓delicatessen meats 6.4±1.5, 13.4±2.4).
Evon, 2004 (41)	Whether amount of total fat and cholesterol consumed is mediated by dietary self-efficacy	Quick check for Diet Progress [UC], Cardiac Diet Self-efficacy Instrument (questionnaire) [Y]	Unclear	Provide instruction, Provide general encouragement, Unclear	Significant correlations between early-mid self-efficacy & PE fat intake (-.31) & mid-late self-efficacy & fat intake (-.29) ($p<0.05$).

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Frost, 2004 (42)	20% reduction in glycaemic index (GI) of diet	4 x (unclear) day food record [Y]	Not stated	Provide instruction, Prompt review of behavioural goals ,Provide general encouragement, Unclear	13% reduction achieved between baseline to 12 weeks Int. group (mean ±SEM): GI units: Int.↓ 81±2 to 71±1, p<0.05. Ctrl: 82±2 to 81±1 (NS). GI load: Int. ↓ 195±9 to 164±11. Ctrl. ↓176±10 to152±9. NS difference in GI load between groups
Giannuzzi, 2008 (43)	Adherence to healthy Mediterranean-like diet	Mediterranean diet score [N]	Not stated	Provide instruction, Plan social support or social change, Stress management ,Unclear,	Baseline: 26.1% of patients had dietary score ≤19/24. At 3 years, the 56.1% of Ctrl (usual care) & 64.4% of Int. had score ≤19/24 (p<0.001 time x treatment)
Gleason, 2002 (44)	Dietary compliance with NCEP Step 2 by consuming meals provided	1[O] outcome: serum cholesterol 2[O] outcome: 3 day food record [UC]	Not stated	Provide instruction, Prompt barrier identification, Provide general encouragement, Provide feedback on performance, Prompt specific goal setting	1O: ↓0.17±0.08mmol/L total cholesterol, (p<0.05), ↓0.19±0.09mmol/L LDL cholesterol (p<0.05) 2O: 93% of energy consumed was part of prescribed menu

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Goodwin, 2012 (45)	Adherence to heart-healthy lifestyle: decreased caloric, fat and sodium intake	ASA-24 (NCI) [Y] & weight [Y]	Acceptance-based behaviour therapy	Provide instruction, Prompt barrier identification, Prompt intention formation, Prompt review of behavioural goals, Prompt self-monitoring of behaviour, Provide opportunities for social comparison, Stress management, Teach to use prompts or cues, Time management	↓ 523kCal/day (ES 1.03), ↓32g fat/day (ES 1.15), ↓1509mg sodium/day (ES 1.63). BMI ↓ 0.74 (ES -0.05)
Hofman-Bang, 1999 (46)	Knowledge of healthy-heart diet & actual dietary behaviours aligning with Swedish official guidelines: <30% fat, <10% saturated fat, 15% protein, 50% CHO	Questionnaire [N] & diet index [N]	Not stated	Prompt practice	Significant improvement in knowledge (p=0.002)& self-reported dietary habits (p=0.01) (unclear extent of change).
Jackson, 2005a (47) Jackson, 2005b (48)	Increase of 2 portion of F&V per day	24 hr recall with focus on F&V intakes [UC]	Theory of Planned Behaviour	Ctrl: Provide instruction Int. A: Provide instruction, Prompt intention formation Int. B: Provide instruction, Prompt intention formation, Prompt specific goal setting	No difference between any groups. All significantly ↑ F&V portions from baseline to 3 months. Ctrl.: 2.6±1.5 to 4.1±2.2. Int. A: 3.1±1.9 to 4.6±2.0. Int. B: 3.0±1.7 to 4.2±2.6.

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Luszczynska, 2007 (49)	Reduction in total and saturated fat intake	Rapid Food Screener [Y]	Theory of Planned Behaviour	Provide instruction, Prompt intention formation, Prompt specific goal setting, Provide feedback on performance, Provide general encouragement	Pre-intervention to 6 months total fat (g/day): Int ↓78.7±14.2 to 69.7±10.3, Ctrl 74.1±14.0 to 74.9±13.6. Sat. fat (g/day): Int.↓ 22.9±6.0 to 19.7±4.6, Ctrl 22.3±4.8 to 22.5±5.2.
Masley, 2001 (50)	Mean fruit & vegetable intakes based on Mediterranean-type diet, anti-oxidant rich, ≤20% energy from fat, focus on fat quality.	WHI FFQ [UC]; + questionnaire for legumes & fat intakes [N]	Not stated	Provide instruction, Model or demonstrate behaviour, Plan social support or social change, Prompt self-monitoring of behaviour, Unclear	Int: ↑F&V intake 3.1 to 4.9 ½ cup serves/day. Control↓3.3 to 2.9 serves/day (p=0.002 change difference). No significant difference in change in fat intakes.
Mildestvedt , 2007 (51)	Mediterranean-type diet, low saturated fat, ↑fish, F&V intake	Three questions with 5-level responses [N]	Self-determination theory	Motivational interviewing, Prompt barrier identification, Prompt intention formation, Use follow-up prompts	No difference between Int. & cardiac rehab (Ctrl) baseline to 2 years. Low saturated fat (unit unclear): Int 3.5±0.8 to 3.8±0.6 / Ctrl 3.3±0.9 to 3.8±0.8. Fish dinners/week: Int 2.3±0.7 to 2.4±0.7 Ctrl 2.2±0.6 to 2.2±0.5. F&V units/day: Int 3.1±1.3 to 3.7±1.2 Ctrl 3.2±1.4 to 3.7±1.2
Shenberger, 1992 (52)	Total energy (kCal), dietary cholesterol & PE from total & sat. fats, based on NCEP Step 1	24 hr recall [Y]	Not stated	Provide instruction, Unclear	↓kCal from 1754±74 to 1502± 64($p<0.05$), PE fat ↓33.4±1.3 to 25.2±1.4 ($p<0.05$), PE sat. fat ↓11.1±0.6 to 7.0±0.4 ($p<0.05$), cholesterol ↓122±6.1 to 90±6.3 ($p<0.05$)

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Singh, 2002 (53)	Indo-Mediterranean diet Ctrl: NCEP Step 1 Int: NCEP Step 1 with ↑wholegrains, legumes, F&V, nuts, mustard/ soybean oil	1 week food record [UC]	Not stated	Provide instruction, Unclear	Significant differences (p<0.001) between Int & Ctrl at 2 years: ↑total fibre (23g/day), ↑soluble fibre (12g/day),↑ fruit/vegetables/nuts combined (334g/day), ↑wholegrains (127g/day), ↑soy/mustard oil (18g/day)
Sundin, 2003 (54)	Changes in diet knowledge index, diet habit index & diet habits based on standard low-fat diet according to Swedish official guidelines	Diet knowledge index [N] Diet habits: diary assessment [N]	Type A behaviour?	Provide instruction, Model or demonstrate behaviour, Prompt practice, Prompt review of behavioural goals, Prompt self-monitoring of behaviour, Provide feedback on performance, Provide information about behaviour-health link, Stress management	From baseline to 1 year: dietary habits: 11.4 to 13.7, diet knowledge 4.8 to 7.0.
Timlin, 2002 (55)	Changes in fat, sat. fat, cholesterol, CHO and restaurant eating based on AHA Step 2 dietary advice recommendations	Diet Habit Survey [Y], Cardiac Diet Self-Efficacy instrument [Y]	Social Cognitive Theory, Trans-theoretical Model	Prompt intention formation, Provide general encouragement, Unclear,	No significant difference in improvement between Ctrl (standard care) & Int group (baseline to 3 months) except restaurant score: Ctrl 16.4±4.4 to 17.5±3.7, Int 16.8±3.9 to 18.6±3.1 (p=0.01)

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Toobert, 1998 (56)	Adherence to Reversal diet (Ornish): high fibre, PE total fat ≤10, PE CHO 70-75, PE protein 15-20, 5mg /day cholesterol	Kristal Food Habits Questionnaire [Y], 4 day food record [UC], screeners for dietary fats [Y] & fibre [Y]	Social Cognitive Theory?	Provide instruction, Model or demonstrate behaviour, Plan social support or social change, Provide information about behaviour-health link, Provide opportunities for social comparison, Stress management	Baseline to 12 months: PE fat 27.0±10.6 to 13.1±7.0 (p<0.01), CHO not reported. Non-significant difference reported for fibre. Animal protein (g/day) 43.7±18.9g to 21.8±8.9 (p<0.02). Cholesterol (mg/day) 173.9±115 to 34.2±31.8 (p<0.01)
Vale, 2003 (57)	Changes in total fat, saturated fat, cholesterol & fibre intakes	FFQ [Y]	Unclear. Model based on 5 stage continuous quality improvement cycle	Prompt intention formation, Prompt self-monitoring of behaviour, Prompt specific goal setting, Provide feedback on performance, Provide information about behaviour-health link, Unclear	Significant differences between Int & Ctrl (usual care) for total fat (↓15.3g,↓10.5g respectively, p=0.4), saturated fat (↓8.0g ,↓4.9g respectively, p=0.002), cholesterol (↓36mg, 20mg respectively, p=0.04) & fibre (↑0.5g,↓0.7g respectively, p=0.02).
van Elderen-van Kemenade, 1994 (58)	Healthy eating: moderation of salt, fat, cholesterol and sugar intake	General questionnaire for Heart Patients [UC]	Unclear	Provide instruction, Provide information about behaviour-health link, Prompt intention formation, Prompt review of behavioural goals, Unclear	Significant differences in healthy eating habits at 12 months (p<0.05). 12.1±4.0 to 15.5±4.1. Units unclear.

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Vestfold Heartcare Study, 2003 (59)	Dietary intake of fat, fibre, cholesterol & sugar based on low fat diet and Mediterranean regimen	Comprehensive FFQ Y	Not stated	Provide instruction, Plan social support or social change, Provide information about behaviour-health link, Stress management	Significant differences in g/day at 2 years between Int & Ctrl (usual care) for sat. fat (↓12.7±11.8, ↓3.6±14.3), mono. fat (↓9.1±8.9, ↓5.5±11.6), sugar (↓17.3±31.2, 6.2±37.7) & cholesterol (mg/day) (↓79.4±94, 31.6±114.4) respectively
Wallner, 1999 (60)	Adherence to diet "close to" actual AHA step 2, designed according to Reversal diet (Ornish)	7 day WFR N	Not stated	Provide instruction	Significant ↓ in Int for PE total fat (9±6%) & ↑CHO (8±6%).
HIGH RISK OF CVD					
Gorder, 1986 (61) Van Horn, 1997 (62)	Fat-modified food patterns (basic and progressive nutrient and food pattern targets) Dietary changes made and adherence to advice	24 hour recall, UC, 3 day record evaluation, subjective assessments N	Not stated	Provide instruction, Provide information about behaviour-health link, Provide information on consequences, Unclear	Major food group changes from year 1 sustained throughout trial. ↓energy from foods recommended to avoid. Significant change difference between groups: Int.↓ total fat 4.2%, ↓Sat. fat. 3.5%, ↓Mono. fat 2.3%, ↑Poly. fat 1.9%. Higher levels of dietary adherence as determined by FRR show correlations with ↓ in serum cholesterol. 58.7% of participant adherence rated subjectively as excellent / good in initial 20 months.

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Siero, 2000 (63)	Mediterranean diet adapted to Dutch situation: ↑ bread, green and root vegetables, fish, ↓beef, lamb, pork (replace with poultry), no day without fruit, sufficient dairy, oil & margarine instead of butter & cream	Food frequency questionnaire Y	Prochaska stage of change	Provide instruction, Model or demonstrate behaviour, Prompt intention formation, Prompt specific goal setting, Provide feedback on performance, Provide opportunities for social comparison, Set graded tasks,	Intervention groups A & B↑ fish ~15,16g/day (respectively, p<0.05)) & fruit & vegetable ~70, 100g/day (respectively, p<0.05), compared to control group at week 16 (usual care: Dutch national nutrition guidelines)
HEART FAILURE					
Donner Alves, 2012 (64)	Guidelines regarding salt restriction & diet quality. Nutritional knowledge	24 hour recall Y, knowledge scale Y	Not stated	Prompt review of behavioural goals, Provide information about behaviour-health link, Unclear	↑nutritional knowledge Int. v Ctrl (p=0.007, extent unclear). Diet quality not defined. Salt intake (from foods, but not including added salt) ↓(p=0.017, extent unclear)
Philipson, 2010 (65)	Reduction in sodium & fluid	1^{O} outcome: urine samples using PABA for sodium & urea UC, 2^{O} outcome: FFQ UC,	Not stated	Provide instruction, Provide information on consequences, Unclear	1^{O} outcome: No sig. diff between groups, but Int. sig. ↓urine sodium (p=0.04) & ↓urine volume (0.04). Int. sig. ↓fluid intake (1.6±0.4 to 1.2±0.5 L/day) compared to Ctrl (1.9±0.6 to 1.7±0.7 L/day) (p<0.05) 2^{O} outcome: not reported

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Powell, 2010 (66)	Sodium restriction	CALS FFQ UC	Social Cognitive Theory?	Provide instruction, Plan social support or social change, Prompt barrier identification, Prompt self-monitoring of behaviour, Stress management	Baseline median sodium intake of 3338mg/day. 28% Int. participants and 18% Ctrl participants ↓intake to 2400mg/day (p=0.01 for time effect)
HEALTH PROFESSIONALS					
Banz, 2004 (67)	Knowledge, consumption & recommendation of soy foods for CVD prevention & treatment	Survey N	Transtheoretical Model	Provide instruction, Model or demonstrate the behaviour	↑38% in knowledge of soy food benefits, ↑37% recommending soy foods to clients, no change in personal consumption. No change in ctrl.
Carson, 2002 (68)	Cardiovascular nutrition therapy Knowledge, attitude and self-efficacy regarding cardiovascular nutrition	Questionnaire (21 knowledge items, 22 attitude and 9 self-efficacy) Y	Social learning	Provide instruction, Model or demonstrate behaviour, Provide feedback on performance, Prompt practice, Provide opportunities for social comparison, Set graded tasks	Knowledge score ↑from 10.3±2.5 to 14.4±2.5 (p<0.001), mean performance <70%; self-efficacy ↑ from 26.2±35.7 to 35.7±5.4 (p<0.001); attitude ↑from 90.0±8.6 to 92.4±9.9 (p<0.001)

Table 3.2: Knowledge translation characteristics

Author , year (country)	Knowledge translation aims	Knowledge translation measure*	Intervention theory	Behaviour change strategies employed *	Results
Perry & McLaren, 2000, 2003a, 2003b (69-71)	Screening and assessment of dysphagia a) Screening for nutritional risk and nutritional status b) initiation of nutrition support c) Patient outcomes e.g. time spend without nutrition	a) Screening for nutritional risk and nutritional status b) initiation of nutrition support c) Patient outcomes e.g. time spend without nutrition Y	Not stated	Provide instruction, Prompt barrier identification, Prompt intention formation, Provide feedback on performance, Prompt practice	a) 17% increase in screening for nutritional risk using validated tool within 24 hrs ($p<0.01$) b) increase in decision making instituted from 35% to 81% for patients nil oral by day 5 ($p<0.01$) c) Mean reduction in time spent without nutrition from 10.2 days to 4.7 days ($p<0.001$)
Van der Weijden, 1998 (72)	Low-fat diet or referral to dietitian (cholesterol guidelines) Improved knowledge and attitudes toward cholesterol guidelines	Questionnaire of guideline topics N	Not stated	Provide instruction, Model or demonstrate behaviour, Provide feedback on performance	No significant differences in agreement with guideline topics between control & intervention groups; high rates of provision of dietary advice or leaflets (46% of consultations), low support through diet therapy (3%)

Abbreviations: FHQ Food Habits Questionnaire; Ctrl. Control group; PE percentage energy; sat. fat saturated fat; Int. Intervention group; ↓ decreased; grp group; P:S polyunsaturated:saturated fat ratio; WFR weighed food record; ↑ increased; 95% CI 95% confidence interval; NCEP National cholesterol education program; CHO carbohydrate; NS non-significant; SEM standard error of the mean; NCI National Cancer Institute; ES effect size; F&V fruit and vegetables; mono. fat monounsaturated fat; AHA American Heart Association; poly. fat polyunsaturated fat; FRR Food record rating; 1^{O} Primary outcome; PABA para-aminobenzoic acid; 2^{O} Secondary outcome;

Validated measure Y Yes; N No; UC Unclear

* "Not stated" was given if no information on the topic could be found and "Unclear" if the description given was too vague to determine which (if any) behaviour change strategy was used

Many studies reported that the dietary advice provided to participants targeted the reduction in total fat intakes (n=12) or asked participants to adopt a Mediterranean or Mediterranean-like eating pattern (n=7, see Table 3.2: knowledge translation outcome). The range of dietary advice provided extended from asking participants to adhere to the particular country's guidelines for the prevention and treatment of CVD, such as the National Cholesterol Education Program Step 2 Diet, through to general healthy eating. This advice was provided by a range of health professionals (n=9), dietitians (n=6), nurses (n=5), nutritionists (n=2), physicians (n=2), psychologists (n=2), or local experts (n=1, local opinion leader). The nutrition advice provider was unclear in seven studies. All interventions were delivered interpersonally, in either individual (n=16) or group-based sessions (n=15), or a combination of both (n=2) with one study unclear. The degree of intervention standardisation was either unclear or not stated in 25 studies; five studies undertook rigorous measures to ensure standardisation and four had varying levels. Further details on intervention content, resources and intensity are summarised in Additional file 4: Intervention content (Appendix 5).

3.6 Discussion

This review aimed to determine how nutrition-related evidence for the prevention and treatment of CVD was effectively translated into practice. The results indicate that KT is inferred, not stated in this area, and is being under-reported in terms of reproduction for clinicians, and policy and decision-makers. No studies were identified with the primary aim to translate dietetic knowledge to impact on objective CVD risk markers, which indicates a need for KT strategies in this area to be purposefully conducted and evaluated. The evidence base confirms the relationship between dietary change and improved outcomes of populations living with CVD in clinical interventions, but the KT studies are lacking. Overall, methods describing strategies to initiate and maintain nutrition behaviour changes were of limited value. In addition to this, the measures used to assess the dietary outcomes were varied, with the sensitivity of the tests to determine the extent of change in the outcome of interest unclear in many cases.

Only 10 of the 34 studies clearly identified a theoretical framework that addressed processes involved in the changing of behaviours. This is in contrast to the fact that all studies required participants to change behaviour if the intervention was to be successfully implemented. The results of larger-scale successful dietary intervention studies such as the GOSPEL study (n=3241) (43) and Lyon Diet Heart Study (n=605) (39, 40) found that dietary behaviour changes were initiated and persisted in the longer term. However, the detail as to how this was achieved was not described and no basis given for why the chosen strategies were appropriate for the context in which they were applied. Therefore replication using the same KT strategy in a given population is not possible and the external validity is unclear. This issue is not limited to nutrition KT strategies in CVD, and has been identified more generally in primary care and general medicine (74). Also, the need for standardised end-points for key performance indicators of cardiovascular outcomes in Australia has also been indicated (75). This extends to dietary intake because of the role nutrition plays in the prevention and treatment of CVD. From this review, it is evident that standardisation of approaches would be of benefit here as well.

In terms of study quality, all interventions focused on risk outcomes important to the prevention and treatment of CVD, although five scored negatively for relevance, as they are unlikely to be replicated in the current financial climate and funding models due to their intensive resourcing (44, 46, 54, 56, 59). Inconsistencies in the published details required to confirm validity may also be due to their older publication dates, as 15 were published either before or within five years of the first CONSORT guidelines. Of these 15 studies, only two scored a positive for validity (39, 40, 56). No studies scored positive for all three areas of quality. This may be a publication limitation, as intervention methods were either limited or focused on describing the measures used. Glasziou *et al*. showed that authors of publications of non-pharmacological interventions, selected for high validity and relevance, were able to provide information supplemental to that published upon request, to provide a more complete description of interventions to aid replication by clinicians (74).

Of the three studies scoring positive quality for KT, only Luszczynska *et al*. (49) targeted diet as single risk factor. Luszczynska *et al*. used implementation intentions training in patients as an adjunct to Phase 2 cardiac rehabilitation. The intervention itself was brief (10-20 minutes), yet results showed significant changes persisted up to six months. The authors also provided a sufficiently detailed structure of the intervention that could be replicated within a cardiac rehabilitation setting by other health care professionals. Allen (32) and Goodwin *et al.* (45) both targeted multiple risk factors, with Allen using Social Cognitive Theory (SCT) to improve self-efficacy and Goodwin *et al.* using Acceptance-Based Behaviour Therapy (ABBT) to facilitate participant changes to healthier behaviours. Both studies provide details that are less prescriptive than those found in Luszczynska *et al*. and therefore more challenging for clinicians to replicate. Allen targeted self-efficacy by the development of specific strategies to attain goals (see Additional file 4: Intervention content (Appendix 5)) and reported positive results after one year, but the dietary measure used may not have been of sufficient sensitivity to detect the change in outcome reported of total and saturated fat. Goodwin *et al.* had a small sample size (n=16) and it is therefore more difficult to determine whether the intervention can be applied to a more diverse population sample, such as those found in current clinical CVD prevention and treatment settings, and attain similar results.

Approximately half of the studies here focused on diet alone, with the remainder targeting other CVD risk factors as well. The KT results are inconclusive here in regards to which is the more successful approach. It has been suggested that it may be easier to translate efficacious dietary patterns, such as the Mediterranean-style diet, instead of focusing on single nutrients, and thereby contribute to better CVD outcomes (76). The American Heart Association Scientific Statement (2010) also considered the advantages of focusing on single lifestyle factors compared to multiple factors, such as physical activity, smoking and dietary modification on CVD biochemical risk factors, with a similar inconclusive result (77). However, European guidelines for prevention of CVD in clinical practice recommend multimodal behavioural interventions for individuals at very high risk (7).

These constraints may be the result of a difference in focus between explanatory and pragmatic designs in relation to KT. Bhattacharyya *et al.* highlighted the differences in intervention design between controlled trials that investigate the efficacy of a treatment and pragmatic studies that aim to assess the effectiveness of the treatment in the context of clinical practice (78). In particular, the focus of the outcomes from these two very different types of studies necessarily varies greatly. Explanatory designs use process measures for outcomes whereas pragmatic designs use outcomes relevant to healthcare stakeholders, such as the patients, the health services and funding bodies.

Very few studies were found where nutrition evidence specifically for the prevention and treatment of CVD was passed between researchers and clinicians. There is a gap in the literature about how researchers are passing on their findings of what works to clinicians. Whilst this is most likely taking place in settings such as conferences, seminars and workshops, the translation strategies are not being described, evaluated or appearing in publications. One such example of publication was the study by Banz *et al.* (67) but the study quality was poor which may be due to the short report format.

A number of further shortcomings were identified within the review that reduced the usefulness for KT replication. For example, it was identified that many of the measures and power calculations used may have been of insufficient quality to detect the extent of change in dietary patterns due to the KT strategy. In particular, the dietary measures used cast doubt on the significance of the results. Whilst many studies declared their dietary measures to be validated, it was unclear from the methods whether the instrument used in the study was validated for the outcome for which it was used. Food records and 24 hour dietary recalls are regularly used for obtaining data on usual dietary intakes, but adequate standardised protocols for data collection need to be described in the methods to ensure the data were collected in sufficient detail to be considered valid. Many of the participants were also on medication for their condition, which makes the extent of change in biochemical risk markers attributable to diet unclear. This is typified in the Masley *et al.* study in which a healthcare fund-driven medication campaign occurred prior to randomisation, accounting for a decrease in LDL

cholesterol from 3.7 mmol/L to 3.1 mmol/L (50). The use of biochemical risk markers is limited unless medication and other factors are controlled for, such as in the case of Gleason *et al.* and Philipson *et al.*, where medication was held consistent or results stratified by medication use, respectively (44, 65). In addition to this, the type of dietary advice people were given, such as replacing dietary fat with carbohydrates of unclear nutritional quality is likely to have confounded long-term CVD outcomes (79).

Studies published before the recommendations from the CONSORT statement took effect (73) were of lower methodological quality. This limitation may have been overcome if the inclusion year were changed to a more recent date, such as to approximately 2001. The rationale for this date includes the release of the first updated CONSORT statement (80) with a five year lag after the primary statement to allow reporting to become more consistent. As expected, meta-analysis of data were not possible due to heterogeneity in nutrition KTs and outcome measures.

3.6.1 Recommendations for future research and practice

There is a clear need for both efficacy and effectiveness KT trials in the area of dietary prevention and treatment of CVD. Studies need to provide a sound basis for choosing particular theoretical frameworks, and behaviour changes strategies should be adequately detailed to allow for replication. In addition, study outcomes should use valid measures that are appropriate for the KT and behaviour change strategies, and describe links to a clinically useful outcome. Further information is required as to how clinically useful research findings are effectively translated to clinicians and then patients. Health professionals are the conduits to translating best-evidence to at-risk persons, but little evidence currently exists to demonstrate efficacy or effectiveness of the translation link between CVD nutrition research and health professionals. A summary of recommended inclusions for KT publications is given in Table 3.3: Recommended checklist for nutrition and dietary translation studies.

Table 3.3: Recommended checklist for nutrition and dietary translation studies

Item	Recommended items for inclusion in nutrition and dietary translation studies
These recommendations are specific to nutrition and diet translation studies and to be used in conjunction with the WIDER recommendations checklist as per Albrecht et al (2013) [29].	
A	Provide sound basis for why the theoretical framework chosen was appropriate for the stated population
B	Provide sufficient detail on behaviour change strategies used to allow for replication or adaption by a qualified diet or nutrition specialist in a comparable situation or setting
C	Nutrient or dietary pattern outcomes must be measured with a validated tool or methodology of sufficient sensitivity to detect the expected changes in a sample size to be adequately powered
D	If a behaviour change is the primary outcome, it is recommended that a clear link is established between the behaviour and a clinically useful outcome
E	Confounders such as medication are common in dietetic interventions. It is recommended to account for such confounders in study design so as to provide a sound basis for why the dietary or nutrition changes instigated were responsible for or assisted in the clinical outcome
F	Clearly designate the purpose of the study design as explanatory or pragmatic

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References

1. World Health Organization. Global status report on noncommunicable diseases 2014. Geneva, Switzerland: World Health Organization, 2014.

2. Schofield D, Shrestha R, Percival R, Passey M, Callander E, Kelly S. The personal and national costs of CVD: impacts on income, taxes, government support payments and GDP due to lost labour force participation. *Int J Cardiol*. 2013;166(1):68-71.

3. Australian Institute of Health and Welfare. Health care expenditure of cardiovascular diseases 2008-09. Canberra: AIHW; 2014.

4. Ha NT, Hendrie D, Moorin R. Impact of population ageing on the costs of hospitalisations for cardiovascular disease: a population-based data linkage study. *BMC Health Serv Res*. 2014;14(1):554.

5. Anderson TJ, Gregoire J, Hegele RA, Couture P, Mancini GB, McPherson R, *et al.* 2012 update of the Canadian Cardiovascular Society guidelines for the diagnosis and treatment of dyslipidemia for the prevention of cardiovascular disease in the adult. *Can J Cardiol*. 2013;29(2):151-67.

6. Mosca L, Benjamin EJ, Berra K, Bezanson JL, Dolor RJ, Lloyd-Jones DM, *et al.* Effectiveness-based guidelines for the prevention of cardiovascular disease in women--2011 update: a guideline from the american heart association. *Circulation*. 2011;123(11):1243-62.

7. Perk J, De Backer G, Gohlke H, Graham I, Reiner Z, Verschuren M, *et al.* European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts). *Eur Heart J*. 2012;33(13):1635-701.

8. J.B.S. Board. Joint British Societies' consensus recommendations for the prevention of cardiovascular disease (JBS3). *Heart*. 2014;100 Suppl 2(Suppl 2):ii1-ii67.

9. Mathews MJ, Liebenberg L, Mathews EH. How do high glycemic load diets influence coronary heart disease? *Nutr Metab (Lond)*. 2015;12(6):6.

10. Dehghan M, Mente A, Teo KK, Gao P, Sleight P, Dagenais G, *et al.* Relationship between healthy diet and risk of cardiovascular disease among patients on drug therapies for secondary prevention: a prospective cohort study of 31 546 high-risk individuals from 40 countries. *Circulation*. 2012;126(23):2705-12.

11. Teo K, Lear S, Islam S, Mony P, Dehghan M, Li W, *et al.* Prevalence of a healthy lifestyle among individuals with cardiovascular disease in high-, middle- and low-income countries: The Prospective Urban Rural Epidemiology (PURE) study. *JAMA*. 2013;309(15):1613-21.

12. Tetroe J. Knowledge translation at the Canadian Institutes of Health Research: A primer. National Center for the Dissemination of Disability Research,, 2007 2007. Report No.: Contract No.: Technical brief No. 18.

13. Graham ID, Logan J, Harrison MB, Straus SE, Tetroe J, Caswell W, *et al.* Lost in knowledge translation: time for a map? *J Contin Educ Health Prof*. 2006;26(1):13-24.

14. Straus SE, Tetroe JM, Graham ID. Knowledge translation is the use of knowledge in health care decision making. *J Clin Epidemiol*. 2011;64(1):6-10.

15. Straus SE, Tetroe J, Graham I. Defining knowledge translation. *CMAJ*. 2009;181(3-4):165-8.

16. Eccles M, Grimshaw J, Walker A, Johnston M, Pitts N. Changing the behavior of healthcare professionals: the use of theory in promoting the uptake of research findings. *J Clin Epidemiol*. 2005;58(2):107-12.

17. Graham ID, Tetroe J. Some theoretical underpinnings of knowledge translation. *Acad Emerg Med*. 2007;14(11):936-41.

18. Scott SD, Albrecht L, O'Leary K, Ball GD, Hartling L, Hofmeyer A, *et al.* Systematic review of knowledge translation strategies in the allied health professions. *Implement Sci*. 2012;7:70.

19. Cobb SL, Brown DJ, Davis LL. Effective interventions for lifestyle change after myocardial infarction or coronary artery revascularization. *J Am Acad Nurse Pract*. 2006;18(1):31-9.

20. Moher D, Liberati A, Tetzlaff J, Altman DG, Group P. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *J Clin Epidemiol*. 2009;62(10):1006-12.

21. National Vascular Disease Prevention Alliance. Guidelines for the assessment of absolute cardiovascular disease risk. National Heart Foundation of Australia; 2009.

22. Morrison A, Polisena J, Husereau D, Moulton K, Clark M, Fiander M, *et al.* The effect of English-language restriction on systematic review-based meta-analyses: a systematic review of empirical studies. *Int J Technol Assess Health Care*. 2012;28(2):138-44.

23. Armstrong R, Waters E, Dobbins M, Lavis JN, Petticrew M, Christensen R. Knowledge translation strategies for facilitating evidence-informed public health decision making among managers and policy-makers. *The Cochrane Library*. 2011.

24. Scott SD, Albrecht L, O'Leary K, Ball GD, Dryden DM, Hartling L, *et al.* A protocol for a systematic review of knowledge translation strategies in the allied health professions. *Implement Sci*. 2011;6:58.

25. Hooper L, Bartlett C, Davey SG, Ebrahim S. Advice to reduce dietary salt for prevention of cardiovascular disease. *Cochrane Database Syst Rev*. 2004(1):CD003656.

26. Hartley L, Flowers N, Lee MS, Ernst E, Rees K. Tai chi for primary prevention of cardiovascular disease. *Cochrane Database Syst Rev*. 2014;4:CD010366.

27. Abraham C, Michie S. A taxonomy of behavior change techniques used in interventions. *Health Psychol*. 2008;27(3):379-87.

28. American Dietetic Association. ADA Evidence Analysis Manual. American Dietetic Association, Chicago; 2005.

29. Albrecht L, Archibald M, Arseneau D, Scott SD. Development of a checklist to assess the quality of reporting of knowledge translation interventions using the Workgroup for Intervention Development and Evaluation Research (WIDER) recommendations. *Implement Sci*. 2013;8:52.

30. Aish A. A comparison of female and male cardiac patients' response to nursing care promoting nutritional self-care. *Can J Cardiovasc Nurs*. 1996;7(3):4-13.

31. Aish AE, Isenberg M. Effects of Orem-based nursing intervention on nutritional self-care of myocardial infarction patients. *Int J Nurs Stud*. 1996;33(3):259-70.

32. Allen JK. Coronary risk factor modification in women after coronary artery bypass surgery. *Nurs Res*. 1996;45(5):260-5.

33. Arntzenius AC, Kromhout D, Barth JD, Reiber JH, Bruschke AV, Buis B, *et al.* Diet, lipoproteins, and the progression of coronary atherosclerosis. The Leiden Intervention Trial. *N Engl J Med*. 1985;312(13):805-11.

34. Kromhout D, Arntzenius AC, van der Velde EA. Diet and coronary heart disease: the Leiden Intervention Trial. *Bibl Nutr Dieta*. 1986(37):119-20.

35. Billings JH. Maintenance of behavior changes in cardiorespiratory risk reduction: a clinical perspective from the Ornish Program for reversing coronary heart disease. *Health Psychol*. 2000;19(1 Suppl):70-5.

36. Koertge J, Weidner G, Elliott-Eller M, Scherwitz L, Merritt-Worden TA, Marlin R, *et al.* Improvement in medical risk factors and quality of life in women and men with coronary artery disease in the Multicenter Lifestyle Demonstration Project. *Am J Cardiol*. 2003;91(11):1316-22.

37. Campbell NC, Ritchie LD, Thain J, Deans HG, Rawles JM, Squair JL. Secondary prevention in coronary heart disease: a randomised trial of nurse led clinics in primary care. *Heart*. 1998;80(5):447-52.

38. Dalgard C, Thuroe A, Haastrup B, Haghfelt T, Stender S. Saturated fat intake is reduced in patients with ischemic heart disease 1 year after comprehensive counseling but not after brief counseling. *J Am Diet Assoc*. 2001;101(12):1420-9.

39. de Lorgeril M, Renaud S, Mamelle N, Salen P, Martin JL, Monjaud I, *et al.* Mediterranean alpha-linolenic acid-rich diet in secondary prevention of coronary heart disease. *Lancet*. 1994;343(8911):1454-9.

40. de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation*. 1999;99(6):779-85.

41. Evon DM, Burns JW. Process and outcome in cardiac rehabilitation: an examination of cross-lagged effects. *J Consult Clin Psychol*. 2004;72(4):605-16.

42. Frost GS, Brynes AE, Bovill-Taylor C, Dornhorst A. A prospective randomised trial to determine the efficacy of a low glycaemic index diet given in addition to healthy eating and weight loss advice in patients with coronary heart disease. *Eur J Clin Nutr*. 2004;58(1):121-7.

43. Giannuzzi P, Temporelli PL, Marchioli R, Maggioni AP, Balestroni G, Ceci V, *et al.* Global secondary prevention strategies to limit event recurrence after myocardial infarction: results of the GOSPEL study, a multicenter, randomized controlled trial from the Italian Cardiac Rehabilitation Network. *Arch Intern Med*. 2008;168(20):2194-204.

44. Gleason JA, Bourdet KL, Koehn K, Holay SY, Schaefer EJ. Cardiovascular risk reduction and dietary compliance with a home-delivered diet and lifestyle modification program. *J Am Diet Assoc*. 2002;102(10):1445-51.

45. Goodwin CL, Forman EM, Herbert JD, Butryn ML, Ledley GS. A pilot study examining the initial effectiveness of a brief acceptance-based behavior therapy for modifying diet and physical activity among cardiac patients. *Behav Modif*. 2012;36(2):199-217.

46. Hofman-Bang C, Lisspers J, Nordlander R, Nygren A, Sundin O, Ohman A, *et al.* Two-year results of a controlled study of residential rehabilitation for patients treated with percutaneous transluminal coronary angioplasty. A randomized study of a multifactorial programme. *Eur Heart J*. 1999;20(20):1465-74.

47. Jackson C, Lawton R, Jenkinson J, Conner M. Increasing daily fruit and vegetable consumption: what changes do cardiac patients make? *J Hum Nutr Diet*. 2005;18(3):195-204.

48. Jackson C, Lawton R, Knapp P, Raynor DK, Conner M, Lowe C, *et al.* Beyond intention: do specific plans increase health behaviours in patients in primary care? A study of fruit and vegetable consumption. *Soc Sci Med*. 2005;60(10):2383-91.

49. Luszczynska A, Scholz U, Sutton S. Planning to change diet: a controlled trial of an implementation intentions training intervention to reduce saturated fat intake among patients after myocardial infarction. *J Psychosom Res*. 2007;63(5):491-7.

50. Masley S, Phillips S, Copeland JR. Group office visits change dietary habits of patients with coronary artery disease-the dietary intervention and evaluation trial (D.I.E.T.). *J Fam Pract*. 2001;50(3):235-9.

51. Mildestvedt T, Meland E, Eide GE. No difference in lifestyle changes by adding individual counselling to group-based rehabilitation RCT among coronary heart disease patients. *Scand J Public Health*. 2007;35(6):591-8.

52. Shenberger DM, Helgren RJ, Peters JR, Quiter E, Johnston EA, Hunninghake DB. Intense dietary counseling lowers LDL cholesterol in the recruitment phase of a clinical trial of men who had coronary artery bypass grafts. *J Am Diet Assoc*. 1992;92(4):441-5.

53. Singh RB, Dubnov G, Niaz MA, Ghosh S, Singh R, Rastogi SS, *et al.* Effect of an Indo-Mediterranean diet on progression of coronary artery disease in high risk patients (Indo-Mediterranean Diet Heart Study): a randomised single-blind trial. *Lancet*. 2002;360(9344):1455-61.

54. Sundin O, Lisspers J, Hofman-Bang C, Nygren A, Ryden L, Ohman A. Comparing multifactorial lifestyle interventions and stress management in coronary risk reduction. *Int J Behav Med*. 2003;10(3):191-204.

55. Timlin MT, Shores KV, Reicks M. Behavior change outcomes in an outpatient cardiac rehabilitation program. *J Am Diet Assoc*. 2002;102(5):664-71.

56. Toobert DJ, Glasgow RE, Nettekoven LA, Brown JE. Behavioral and psychosocial effects of intensive lifestyle management for women with coronary heart disease. *Patient Educ Couns*. 1998;35(3):177-88.

57. Vale MJ, Jelinek MV, Best JD, Dart AM, Grigg LE, Hare DL, *et al.* Coaching patients On Achieving Cardiovascular Health (COACH): a multicenter randomized trial in patients with coronary heart disease. *Arch Intern Med*. 2003;163(22):2775-83.

58. van Elderen-van Kemenade T, Maes S, van den Broek Y. Effects of a health education programme with telephone follow-up during cardiac rehabilitation. *Br J Clin Psychol*. 1994;33 (Pt 3)(Pt 3):367-78.

59. Vestfold Heartcare Study G. Influence on lifestyle measures and five-year coronary risk by a comprehensive lifestyle intervention programme in patients with coronary heart disease. *Eur J Cardiovasc Prev Rehabil*. 2003;10(6):429-37.

60. Wallner S, Watzinger N, Lindschinger M, Smolle KH, Toplak H, Eber B, *et al.* Effects of intensified lifestyle modification on the need for further revascularization after coronary angioplasty. *Eur J Clin Invest*. 1999;29(5):372-9.

61. Gorder DD, Dolecek TA, Coleman GG, Tillotson JL, Brown HB, Lenz-Litzow K, *et al.* Dietary intake in the Multiple Risk Factor Intervention Trial (MRFIT): nutrient and food group changes over 6 years. *J Am Diet Assoc*. 1986;86(6):744-51.

62. Van Horn L, Dolecek TA, Grandits GA, Skweres L. Adherence to dietary recommendations in the special intervention group in the Multiple Risk Factor Intervention Trial. *Am J Clin Nutr*. 1997;65(1 Suppl):289S-304S.

63. Siero FW, Broer J, Bemelmans WJ, Meyboom-de Jong BM. Impact of group nutrition education and surplus value of Prochaska-based stage-matched

information on health-related cognitions and on Mediterranean nutrition behavior. *Health Educ Res*. 2000;15(5):635-47.

64. Donner Alves F, Correa Souza G, Brunetto S, Schweigert Perry ID, Biolo A. Nutritional orientation, knowledge and quality of diet in heart failure: randomized clinical trial. *Nutr Hosp*. 2012;27(2):441-8.

65. Philipson H, Ekman I, Swedberg K, Schaufelberger M. A pilot study of salt and water restriction in patients with chronic heart failure. *Scand Cardiovasc J*. 2010;44(4):209-14.

66. Powell LH, Calvin JE, Jr., Richardson D, Janssen I, Mendes de Leon CF, Flynn KJ, *et al.* Self-management counseling in patients with heart failure: the heart failure adherence and retention randomized behavioral trial. *JAMA*. 2010;304(12):1331-8.

67. Banz MF, Most PV, Banz WJ. A workshop designed to educate dietetics professionals about the cardiovascular benefits of soyfoods. *J Nutr Educ Behav*. 2004;36(2):103-4.

68. Carson JA, Gillham MB, Kirk LM, Reddy ST, Battles JB. Enhancing self-efficacy and patient care with cardiovascular nutrition education. *Am J Prev Med*. 2002;23(4):296-302.

69. Perry L, McLaren SM. An evaluation of implementation of evidence-based guidelines for dysphagia screening and assessment following acute stroke: Phase 2 of an evidence-based practice project. *Journal of Clinical Excellence*. 2000;2(3):147-56.

70. Perry L, McLaren S. Implementing evidence-based guidelines for nutrition support in acute stroke. *Evid Based Nurs*. 2003;6(3):68-71.

71. Perry L, McLaren S. Nutritional support in acute stroke: the impact of evidence-based guidelines. *Clin Nutr*. 2003;22(3):283-93.

72. Van Der Weijden T, Grol RPTM, Schouten BJ, Knottnerus JA. Barriers to working according to cholesterol guidelines: A randomized controlled trial on implementation of national guidelines in 20 general practices. *Eur J Public Health*. 1998;8(2):113-8.

73. Begg C, Cho M, Eastwood S, Horton R, Moher D, Olkin I, *et al.* Improving the quality of reporting of randomized controlled trials. The CONSORT statement. *JAMA*. 1996;276(8):637-9.

74. Glasziou P, Meats E, Heneghan C, Shepperd S. What is missing from descriptions of treatment in trials and reviews? *BMJ*. 2008;336(7659):1472.

75. Clark AM, Redfern J, Thompson DR, Briffa T. More data, better data or improved evidence translation: what will improve cardiovascular outcomes? *Int J Cardiol*. 2012;155(3):347-9.

76. Anand SS, Hawkes C, de Souza RJ*, et al.* Food Consumption and its Impact on Cardiovascular Disease: Importance of Solutions Focused on the Globalized Food

System: A Report From the Workshop Convened by the World Heart Federation. *J Am Coll Cardiol* 2015;66(14) 1590-614.

77. Artinian NT, Fletcher GF, Mozaffarian D, Kris-Etherton P, Van Horn L, Lichtenstein AH, *et al.* Interventions to promote physical activity and dietary lifestyle changes for cardiovascular risk factor reduction in adults: a scientific statement from the American Heart Association. *Circulation*. 2010;122(4):406-41.

78. Bhattacharyya OK, Estey EA, Zwarenstein M. Methodologies to evaluate the effectiveness of knowledge translation interventions: a primer for researchers and health care managers. *J Clin Epidemiol*. 2011;64(1):32-40.

79. Fan J, Song Y, Wang Y, Hui R, Zhang W. Dietary glycemic index, glycemic load, and risk of coronary heart disease, stroke, and stroke mortality: a systematic review with meta-analysis. *PLoS ONE*. 2012;7(12):e52182.

80. Moher D, Schulz KF, Altman DG. The CONSORT statement: revised recommendations for improving the quality of reports of parallel-group randomised trials. *Lancet*. 2001;357(9263):1191-4.

Chapter 4: Dietary intake is related to multifactor cardiovascular risk score in obese boys

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4.1 Overview

This chapter is the first of the secondary data analyses used to assess diet-related CVD risk in young people where one risk factor is already present. The CVD risk factor in this chapter is overweight and obesity and the population is prepubertal children. Data for this analysis was sourced from a randomized controlled trial performed in 2005-2008.

4.2 Abstract

Cardiovascular disease (CVD) originates in childhood and early identification of risk factors provides an early intervention opportunity. The aim was to identify children at higher risk using a CVD risk score, developed from factors known to cluster in childhood. Risk was scored as very high (≥97.5th centile), high (≥95th), moderate (≥90th) or threshold (<90th) using normal pediatric reference ranges for 10 common biomedical risk factors. These were summed in a multifactor CVD risk score and applied to a sample of 285 observations from 136 overweight Australian children (41% male, aged 7–12 years). Strength of associations between CVD risk score and individual biomedical and dietary variables were assessed using univariate logistic regression. High waist circumference (Odds Ratio: 5.48 [95% CI: 2.60–11.55]), body mass index (OR: 3.22 [1.98–5.26]), serum insulin (OR: 3.37 [2.56–4.42]) and triglycerides (OR: 3.02 [2.22–4.12]) were all significantly related to CVD risk score. High intakes of total fat (OR: 4.44 [1.19–16.60]), sugar (OR: 2.82 [1.54–5.15]) and carbohydrate (OR 1.75 [1.11–2.77]) were significantly related to CVD risk score in boys only. This multifactor CVD risk score could be a useful tool for researchers to identify elevated risk in children. Further research is warranted to examine sex-specific dietary factors related to CVD risk in children.

4.3 Introduction

The World Health Organization (WHO) reported in 2008 that 17.3 million deaths worldwide were due to cardiovascular disease (CVD) (1). A major contributor to CVD is atherosclerosis which is a dynamic process that can begin in childhood and develop or regress, depending on the presence or absence of a range of risk factors,

including obesity, inflammation, hyperglycemia, hypertension and hyperlipidemia (2).

The International Obesity Taskforce estimates that approximately 40–50 million school aged children are obese (3). Obese children are at high risk of becoming obese adults, especially if their parents are also obese (4). Abdominal obesity in children is associated with low grade inflammation (5), a significant contributor to the development of atherosclerosis (2). Both body mass index (BMI) and waist circumference (WC) correlate with intra-abdominal fat in primary school aged children (6) and are used as clinical measures to identify CVD risk (7). Obese children are also at increased risk of hypertension and dyslipidemia as they age (8).

A number of risk factors cluster within both adults and children (9, 10). Those most commonly identified in overweight and obese children are elevated fasting serum insulin and glucose (5, 11, 12), high blood pressure (5, 11, 13, 14), raised triglycerides (5, 11, 12, 14), total (5, 14) and LDL-cholesterol (11, 12, 14); and low HDL-cholesterol (11-14). There are a number of accepted standard reference ranges used to quantify these risk factors in adults (15, 16), with age- and sex-specific reference values recently developed for children (17, 18) using population data from France (18). The Framingham method for assessing 30 year risk in adults includes age, sex, systolic blood pressure (SBP), anti-hypertensive medications, smoking, presence of diabetes, total and HDL cholesterol (19). Although scoring methods for determining CVD risk in children exist (20), a straightforward method of assessing and describing a gradation of risk in overweight children by comparing individual clinical data to population-derived percentile bands could assist in estimating future risk of CVD. Therefore the aims of this study were to; (1) develop a multifactor CVD risk score using these reference ranges for pediatric researchers; (2) assess its application in overweight and obese pre-pubertal children; (3) examine the difference in dietary intake between high *versus* low category CVD risk scores; and (4) to examine the strength of associations between individual anthropometric and biomedical risk factors in both boys and girls.

4.4 Experimental Section

Data for the current study were obtained for secondary analysis from a primary study: the Hunter Illawarra Kids Challenge Using Parent Support (HIKCUPS) randomized controlled trial. Overweight and obese children aged 5 to10 years were recruited between 2005–2006 and followed up for a period of 24 months from baseline. The children were from predominantly middle-class families with English-speaking backgrounds. The original purpose of this study was to determine the efficacy of three different weight loss interventions in overweight children. Detailed methods for the study and outcomes are described elsewhere (21-25). Data were collected at up to four time points (baseline, 6-, 12-, and 24-month follow-up). The current analyses included those children with complete blood, dietary and anthropometric data sets at individual time points for children aged seven years or older at the time of the first measurement, reflecting the minimum age of the reference values used (18). This included 285 observations on 136 individuals, comprised of 112 baselines measures with 79, 57 and 37 measures respectively from the remaining time points. This provides data from children at a wider range of ages and with changing weight status. Written informed consent was obtained from the parents, the trial was registered with the U.S. National Center for Clinical Trials (00107692) and approval was obtained from the human research ethics committees of the University of Newcastle and University of Wollongong.

4.4.1 Anthropometry and serum sampling

Participants were weighed in light clothing to the nearest 0.1 kg using Tanita HD646 scales (Tanita Corporation, IL, USA). Height was measured to the nearest 0.1 cm using PE87 portable stadiometers (Mentone Educational Center, Victoria, Australia). Waist circumference (WC) was measured at the midpoint between the lower costal border and the iliac crest (26). Blood pressure was measured using an automated monitor (Critikon, Tampa, FL, USA) under standardized conditions. Blood samples were collected for glucose, insulin and blood lipid concentrations from the children after an overnight fast by trained phlebotomists and analyzed at

a single accredited pathology service (National Association of Testing Authorities, Newcastle, Australia).

4.4.2 HIKCUPS CVD risk score

BMI, WC, systolic blood pressure (SBP), diastolic blood pressure (DBP), serum HDL, LDL and total cholesterol, serum triglycerides, serum fasting glucose and serum fasting insulin were used to develop a multifactor CVD risk score based on the premise that elevated childhood values for these individual measures have been shown to predict later CVD in adulthood (27-29). Values greater than or equal to the top tenth percentile of normal reference ranges were used to identify those at the greatest risk and to identify risk score cut-points. This is in a manner comparable to those used in the International Diabetes Federation (IDF) definition of pediatric metabolic syndrome (30) and the National Heart, Lung and Blood Institute (31) for identification of elevated plasma lipids, with a scoring similar to that used by Bergmann *et al.* (32) to identify individuals at higher odds of CVD risk factors. BMI reference values were from the US Centers for Disease Control and Prevention age and sex specific growth charts (33) and those for all other measures were developed from a healthy population by Mellerio *et al.* (18), although in that study WC was measured at the umbilicus. The 90th, 95th and 97.5th percentile values for each measure for children aged 7–11 years are provided in Table 4.1.

Table 4.1: Percentiles of clinical and biochemical cardiovascular disease (CVD) risk score factors by age and sex.

Age	7 Years			8 Years			9 Years			10 Years			11 Years		
Percentile	**90**	**95**	**97.5**	**90**	**95**	**97.5**	**90**	**95**	**97.5**	**90**	**95**	**97.5**	**90**	**95**	**97.5**
BOYS															
BMI *	17.7	18.8	19.5	18.3	19.7	20.9	20.4	22.3	23.1	20.1	22	23.1	21.7	23	25
WC	61.4	63.6	65.5	63.7	66	68.1	66	68.5	70.8	68.4	71.1	73.6	70.9	73.7	76.4
Systolic BP	114	118	122	116	120	124	118	123	127	121	125	129	124	128	132
Diastolic BP	70	73	75	71	74	76	71	74	77	72	75	78	73	76	79
F. glucose	4.87	5	5.11	4.96	5.09	5.21	5.04	5.17	5.3	5.11	5.24	5.37	5.16	5.3	5.42
Insulin	5.99	7.05	8.09	7.16	8.45	9.73	8.47	10	11.6	9.88	11.8	13.6	11.3	13.5	15.7
Total chol	5.5	5.84	6.15	5.52	5.85	6.15	5.53	5.85	6.14	5.51	5.83	6.11	5.47	5.78	6.06
LDL	3.79	4.07	4.33	3.75	4.03	4.29	3.7	3.99	4.24	3.66	3.94	4.2	3.62	3.9	4.15
HDL	1.94	2.07	2.19	1.95	2.08	2.2	1.95	2.08	2.2	1.94	2.07	2.18	1.91	2.03	2.15
Triglycerides	0.72	0.81	0.91	0.8	0.91	1.03	0.87	1.01	1.15	0.94	1.1	1.27	1.01	1.19	1.38
GIRLS															
BMI	17.8	19.2	20	18.9	19.9	21.6	20.1	22.9	24.3	21	23.2	23.8	22.4	24.7	27
WC	61.2	63.6	66	63.9	66.6	69.2	66.5	69.4	72.2	68.9	72	75	71.1	74.4	77.6
Systolic BP	112	116	120	115	118	122	117	121	124	119	123	127	121	125	129
Diastolic BP	70	73	75	71	74	76	71	74	77	72	75	78	73	76	79
F. glucose	4.81	4.97	5.11	4.88	5.04	5.18	4.96	5.11	5.24	5.03	5.17	5.3	5.08	5.22	5.34
Insulin	7.59	9.18	10.8	8.55	10.2	12	9.67	11.5	13.4	11.1	13.1	15.2	13	15.3	17.7
Total chol.	5.77	6.15	6.5	5.67	6.05	6.39	5.58	5.95	6.29	5.51	5.87	6.2	5.45	5.8	6.13
LDL	4.1	4.47	4.82	3.98	4.34	4.69	3.88	4.23	4.56	3.78	4.13	4.46	3.71	4.05	4.37

Table 4.1: Percentiles of clinical and biochemical cardiovascular disease (CVD) risk score factors by age and sex.

Age	7 Years			8 Years			9 Years			10 Years			11 Years		
HDL	1.78	1.9	2.02	1.82	1.94	2.06	1.82	1.95	2.07	1.82	1.94	2.06	1.8	1.93	2.04
Triglycerides	0.84	0.95	1.07	0.92	1.06	1.19	1.00	1.15	1.29	1.07	1.23	1.38	1.13	1.3	1.47
Percentile	**10**	**5**	**2.5**	**10**	**5**	**2.5**	**10**	**5**	**2.5**	**10**	**5**	**2.5**	**10**	**5**	**2.5**
BOYS															
HDL	1.23	1.15	1.09	1.23	1.16	1.09	1.23	1.16	1.09	1.23	1.15	1.09	1.21	1.13	1.07
GIRLS															
HDL	1.11	1.03	0.98	1.13	1.05	1	1.13	1.06	1	1.13	1.06	1	1.12	1.05	0.99

* BMI is 97th percentile not 97.5th percentile. BMI values are taken from CDC reference ranges (33) and waist circumference, blood pressure, fasting glucose, insulin, triglycerides, LDL, HDL and total cholesterol are those developed by Mellerio (18). BMI—Body Mass Index; BP—blood pressure; F. glucose—fasting glucose; HDL—high density lipoprotein cholesterol; LDL—low density lipoprotein cholesterol; Total chol.—total cholesterol; WC—waist circumference.

Values were designated as threshold, moderate, high and very high risk, with each category contributing a certain number of points to the CVD score. Threshold risk was defined for values below the 90th centile and allocated zero points. Moderate risk, where the individual is currently at increased risk, was between the 90th and 95th centile (scoring one point), high risk was assessed as between the 95th and 97.5th centile (scoring two points) and values ≥97.5th centile were categorized as very high risk (scoring three points). Given that low levels of HDL-cholesterol are a risk factor but higher levels have a protective effect, both positive and negative scores were awarded for HDL with low levels scored as 3, 2 and 1 for the ≤2.5th, 2.5–5th and 5–10th centiles respectively, and protective levels were scored -1, -2, -3, for 90–95th, 95–97.5th and ≥97.5th centiles respectively. The scores from all variables were summed to provide a multifactor CVD index with a range of −3 to 30 where negative numbers or zero would be deemed as low risk of future CVD. A score above eight indicates that risk factors such as raised blood pressure, abnormal lipids or impaired glucose metabolism, are also present in addition to obesity, as obese children would score highly on BMI and WC, which combine to a maximum score of six.

4.4.3 Dietary Intake

Dietary intake was assessed by parent report as usual intake frequency over the six months prior to each assessment using the previously validated Australian Child and Adolescent Eating Survey, a 135 item semi-quantitative food frequency questionnaire (FFQ) (34-36). More extensive dietary outcomes have been reported previously (24, 37). Nutrient intakes were computed using FoodWorks version 4.00.1158, 2005 (Xyris Software, Queensland, Australia) and the Australian AusNut 1999 nutrient database (All Foods, Revision 17) and AusFoods (Brands, Revision 5).

4.4.4 Statistical Analysis

Counts and percentages were used to describe the distribution of the children's biomedical data within each CVD risk score category. Medians and interquartile range (IQR) (1st–3rd quartile) were used to describe the dietary data relative to

low and high CVD risk scores. The CVD risk score was dichotomized as follows: a score of 0 if CVD risk score <9; a score of 1 if CVD risk score ≥9. The value of nine was chosen as it identifies those with more than one risk factor for CVD in addition to high risk WC and BMI, similar to the IDF definition of pediatric metabolic syndrome (30). Medians of dietary data were compared across the two CVD risk score groups using the Wilcoxon rank-sum test. Multivariate logistic regression models, with standard errors clustered by child, to estimate the strength of the association of individual CVD risk scores for the factor and each dietary measure with the multifactor CVD score while controlling for total energy intake. The dietary measures were sugar (g), total energy (kJ), protein (g), carbohydrate (g), sodium (mg), fat (g), saturated fat (g), mono- and polyunsaturated fat (g). For each explanatory variable, two models were fit, observations of boys only and observations of girls only. Odds ratios, 95% confidence intervals and *p*-values from each model were reported. All data manipulation and statistical analyses were performed in Stata version 12 MP (38). Significance was determined at the 5% level.

4.5 Results and Discussion

Data were obtained from 56 boys and 80 girls, resulting in 285 sets of observations (121 on boys and 164 on girls), with 51 individuals (37.5%) having a single observation, with 36 (26.5%), 34 (25%) and 15 (11%) having two, three and four observations respectively. Table 4.2 reports the baseline data of these children.

Table 4.3 reports the count and percentage of observations for boys and girls in each multifactor CVD risk score category for each biomedical measure. Eighty five percent of observations of boys and 75% of observations of girls were at or above the 95th centile (intermediate to high risk) for BMI and WC as expected in this cohort. In addition, 40% of observations of boys and almost 35% of observations of girls were at or above high risk for serum triglycerides and insulin, and over 15% of all observations had low levels of HDL cholesterol. In contrast, five of the other risk factors (systolic and diastolic blood pressure, fasting glucose, total and LDL cholesterol) had 95% or more of observations in the low risk category.

Table 4.2: Characteristics of the children at baseline.

Variables	Boys (*n* = 44) *	Girls (*n* = 44) *	Total (*n* = 112) *
Age (years)	8.89 ± 0.8	8.53 ± 0.9	8.7 ± 0.84
BMI (kg/m^2)	25.48 ± 3.5	25.04 ± 4.0	25.20 ± 3.8
WC (cm)	80.50 ± 9.1	76.68 ± 9.4	78.18 ± 9.4
Systolic BP (mmHg)	100.65 ± 9.3	99.08 ± 8.6	99.65 ± 8.9
Diastolic BP (mmHg)	57.32 ± 6.0	55.92 ± 5.5	56.47 ± 5.7
Fasting glucose (mmol/L)	4.22 ± 0.4	4.19 ± 0.5	4.2 ± 0.5
Insulin (mIU/L)	11.63 ± 7.0	12.05 ± 8.2	11.88 ± 7.7
Total Cholesterol (mmol/L)	4.25 ± 0.6	4.37 ± 0.7	4.32 ± 0.7
LDL (mmol/L)	2.44 ± 0.6	2.61 ± 0.6	2.54 ± 0.6
HDL (mmol/L)	1.28 ± 0.3	1.25 ± 0.3	1.27 ± 0.3
TG (mmol/L)	1.15 ± 0.6	1.11 ± 0.6	1.12 ± 0.6

* Baseline measures include only those with full data available for this time point. Data given as mean ± standard deviation

Table 4.4 reports median and IQR of dietary measures by low or high CVD risk score separately for boys and girls. For boys, but not girls, the median of all dietary measures was significantly higher in the high CVD risk score group (with the exception of saturated fat). For girls, there were no significant differences between the medians of high and low CVD risk score groups in any dietary measures.

The results of logistic regression modeling of CVD risk score are reported in Table 4.5. For girls, all factors other than systolic and diastolic blood pressure were significantly associated with the multifactor CVD risk score. For boys, the multifactor CVD risk score was associated with all factors other than blood pressure, fasting glucose, total and LDL cholesterol. The greatest odds ratios for both boys and girls were for waist circumference (14.36 and 4.59, respectively), with BMI, insulin, triglycerides and HDL also being significant. The odds ratios and 95% confidence intervals are displayed in Figure 4.1.

Table 4.3: Count and percent of observations by CVD risk score factor level and sex.

CVD risk factor	Threshold Risk (<90th)	Moderate Risk (90th–95th)	High Risk (95th–97.5th)	Very High Risk (≥97.5th)
BOYS				
BMI	6 (5.0%)	13 (10.7%)	13 (10.7%)	89 (73.6%)
Waist circumference	5 (4.1%)	8 (6.6%)	9 (7.4%)	99 (81.8%)
Systolic BP	117 (96.7%)	4 (3.3%)	0 (0.0%)	0 (0.0%)
Diastolic BP	120 (99.2%)	0 (0.0%)	0 (0.0%)	1 (0.8%)
Fasting glucose	119 (98.3%)	1 (0.8%)	0 (0.0%)	1 (0.8%)
Insulin	54 (44.6%)	17 (14.0%)	13 (10.7%)	37 (30.6%)
Total cholesterol	120 (99.2%)	1 (0.8%)	0 (0.0%)	0 (0.0%)
LDL	117 (96.7%)	3 (2.5%)	0 (0.0%)	1 (0.8%)
Triglycerides	59 (48.8%)	14 (11.6%)	9 (7.4%)	39 (32.2%)
	10th–90th	**5th–10th**	**2.5th–5th**	**≤2.5th**
HDL	70 (57.9%)	24 (19.8%)	5 (4.1%)	19 (15.7%)
		90–95th	**95th–97.5th**	**≥97.5th**
HDL (protective)		1 (0.8%)	2 (1.7%)	0 (0.0%)
GIRLS				
BMI	9 (5.5%)	30 (18.3%)	31 (18.9%)	94 (57.3%)
Waist circumference	20 (12.2%)	14 (8.5%)	22 (13.4%)	108 (65.9%)
Systolic BP	161 (98.2%)	2 (1.2%)	0 (0.0%)	1 (0.6%)
Diastolic BP	163 (99.4%)	0 (0.0%)	1 (0.6%)	0 (0.0%)
Fasting glucose	158 (96.3%)	2 (1.2%)	0 (0.0%)	4 (2.4%)
Insulin	82 (50.0%)	24 (14.6%)	7 (4.3%)	51 (31.1%)
Total cholesterol	159 (97.0%)	1 (0.6%)	1 (0.6%)	3 (1.8%)
LDL	155 (94.5%)	4 (2.4%)	1 (0.6%)	4 (2.4%)
Triglycerides	86 (52.4%)	19 (11.6%)	9 (5.5%)	50 (30.5%)
	10th–90th	**5th–10th**	**2.5th–5th**	**≤2.5th**
HDL	113 (68.9%)	17 (10.4%)	7 (4.3%)	23 (14.0%)
		90th–95th	**95th–97.5th**	**≥97.5th**
HDL (protective)		4 (2.4%)	0 (0.0%)	0 (0.0%)

285 observations on 136 individuals; 121 observations on 56 boys, 164 observations on 80 girls; BMI—Body Mass Index; BP—blood pressure; HDL—high density lipoprotein cholesterol; LDL—low density lipoprotein cholesterol; WC—waist circumference.

Table 4.4: Median and IQR of dietary measures by CVD risk score category (< 9 and ≤ 9) and sex.

	Low risk (CVD Score < 9)		High risk (CVD Score ≥ 9)			
DIET MEASURE	**Boys**	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**
	n = 60 (49.9%)	n = 89 (54.3%)	n = 61 (50.1%)	n = 75 (45.7%)		
	← **Median (IQR)** →				**p-value**	
Sugars (×100 g)	1.65 (1.3–2.0)	1.64 (1.3–2.2)	2.10 (1.6–2.7)	1.67 (1.4–2.1)	<0.001	0.629
Energy (×1000 kJ)	9.87 (8.5–11.0)	9.82 (7.5–11.6)	11.45 (9.2–13.8)	9.72 (8.0–11.5)	0.004	0.936
Protein (×10 g)	9.30 (7.8–10.6)	9.06 (8.0–11.3)	10.68 (8.1–13.3)	8.98 (7.1–11.5)	0.018	0.493
Carbohydrate (×100 g)	3.24 (2.6–3.7)	2.97 (2.5–3.7)	3.63 (3.0–4.5)	3.04 (2.7–3.6)	0.004	0.936
Total Fat (×100 g)	0.74 (0.6–0.9)	0.74 (0.5–0.9)	0.84 (0.6–1.1)	0.71 (0.5–0.9)	0.047	0.941
Saturated fat (×10 g)	3.24 (2.5–3.9)	3.18 (2.2–4.1)	3.49 (2.8–4.8)	3.00 (2.3–3.8)	0.099	0.803
Monounsaturated fat (×10 g)	2.59 (2.2–3.1)	2.61 (1.9–3.2)	2.84 (2.3–3.7)	2.60 (1.9–3.4)	0.048	0.986
Polyunsaturated fat (×1 g)	8.44 (7.3–11.0)	8.71 (6.6–11.4)	10.23 (8.6–12.3)	8.43 (6.9–11.2)	0.015	0.829
Sodium (×1000 mg)	2.03 (1.6–2.3)	1.92 (1.6–2.5)	2.24 (1.8–2.7)	1.98 (1.5–2.5)	0.017	0.901

Table 4.5: Odds ratios, 95% confidence intervals (CI) and p-values from multivariate logistic regression models of CVD risk score on individual multifactor CVD risk score factors and total energy (e indicates total energy was significant in the model at the 5% level).

	Boys (*n* = 121)			Girls (*n* = 164)		
CVD RISK SCORE FACTOR	**Odds Ratio**	**95% CI**	***p*-Value**	**Odds Ratio**	**95% CI**	***p*-Value**
BMI (kg/m2)	6.20 e	2.33, 16.46	<0.001	2.59	1.50, 4.48	0.001
Waist circ. (cm)	14.01 e	2.50, 78.49	0.003	4.59	2.14, 9.83	<0.001
Systolic BP (mmHg)	0.27 e	0.02, 2.99	0.286	2.23	0.63, 7.86	0.213
Diastolic BP (mmHg)	1.00	1.00, 1.00	1.000	1.00	1.00, 1.00	1.000
Fasting glucose (mmol/L)	1.50 e	0.59, 3.81	0.395	3.23	1.25, 8.30	0.015
Insulin (mIU/L)	3.25 e	2.25, 4.71	<0.001	3.43	2.31, 5.10	<0.001
Total cholesterol (mmol/L)	2.85 e	0.61, 13.40	0.185	3.01	1.16, 7.79	0.023
LDL (mmol/L)	1.00	1.00, 1.00	1.000	2.90	1.15, 7.32	0.024
HDL (mmol/L)	2.22 e	1.30, 3.77	<0.001	2.39	1.65, 3.47	<0.001
Triglycerides (mmol/L)	3.31	2.24, 4.90	<0.001	2.84	1.85, 4.35	<0.001

The response variable is dichotomous, where 0 means CVD risk score <9 and 1 means CVD risk score ≥9 for boys and girls separately. Each explanatory variable is used to estimate the strength of its association with the multifactor CVD risk score. For example, boys increasing their waist circumference from the 90th to the 95th centile, or from the 95th to above 97.5th, are 14 times more likely to have other risk factors for CVD such as lipid or blood pressure abnormalities, in addition to a high BMI and WC.

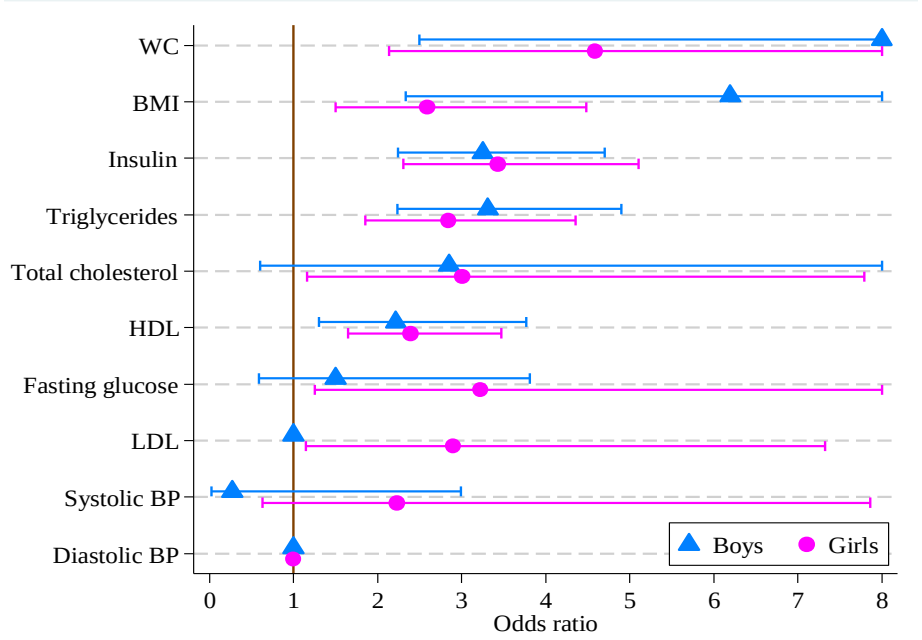

Figure 4.1: Odds ratios, with 95% confidence intervals, for boys and girls from multivariate logistic regression models of CVD risk score on individual CVD risk score factors (boys, *n* = 121; girls, *n* = 164).

Significant associations were found in boys between the HIKCUPS CVD risk score and all dietary measures except polyunsaturated fat (Table 4.6). No significant associations were demonstrated using similar models for girls, or in models using observations from both sexes, with or without the interaction between sex and dietary measures. These odds ratios and 95% confidence intervals are displayed in Figure 4.2.

Table 4.6: Odds ratios, 95% confidence intervals (CI) and *p*-values from multivariate logistic regression models of CVD risk score on individual diet measures and total energy (e indicates total energy was significant in the model at the 5% level).

	Boys (*n* = 121)			Girls (n=164)		
DIET MEASURE	**Odds Ratio**	**95% CI**	***p*-Value**	**Odds Ratio**	**95% CI**	***p*-Value**
Sugars/100 (g)	2.82	1.54, 5.15	0.001	0.92	0.59, 1.42	0.696
Energy/1000 (kJ)	1.22	1.05, 1.41	0.010	0.96	0.87, 1.07	0.479
Protein/10 (g)	1.21	1.04, 1.39	0.011	0.95	0.85, 1.07	0.435
Carbohydrate/100 (g)	1.75	1.11, 2.77	0.016	0.88	0.66, 1.18	0.400
Total Fat/100 (g)	4.44	1.19, 16.60	0.027	0.76	0.23, 2.46	0.646
Saturated fat/10 (g)	1.34	1.03, 1.74	0.027	0.95	0.73, 1.23	0.690
Monounsaturated fat/10 (g)	0.73 e	0.36, 1.46	0.366	0.93	0.68, 1.29	0.678
Polyunsaturated fat (g)	0.76 e	0.13, 4.34	0.761	0.73	0.30, 1.80	0.493
Sodium/1000 (mg)	2.05	1.01, 4.15	0.047	0.90	0.55, 1.49	0.694

The response variable is dichotomous, where 0 means CVD risk score <9 and 1 means CVD risk score ≥9 for boys and girls separately. Each explanatory variable is used to estimate the strength of its association with the multifactor CVD risk score. For sugars, each increase of 100 g triples the odds of the CVD risk score being equal to or above 9.

The current study describes the development of a multifactor CVD risk score that reflects the clustering of CVD risk score factors in pre-pubertal overweight and obese children. The main factors contributing to higher CVD risk score in this pediatric group were high WC and BMI, elevated serum insulin and triglycerides, and low HDL concentrations. Elevated blood pressure was rare. Dietary measures, including high intakes of total and saturated fat, carbohydrate and sugars were significantly related to CVD risk score in boys only with no associations between a range of dietary measures and multifactor CVD risk score in girls.

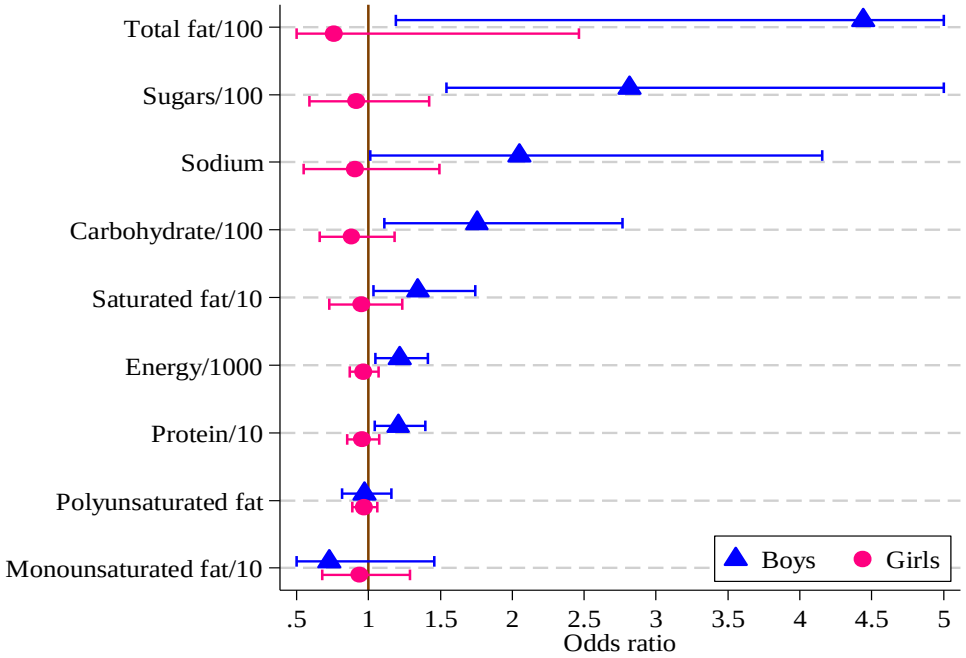

Figure 4.2: Odds ratios, with 95% confidence intervals, for boys and girls from multivariate logistic regression models of CVD risk score on diet measure (boys, *n* = 121; girls, *n* = 164)

The risk factors identified in the current study are not entirely consistent with other studies in overweight and obese children. Although 48% of girls and 50% of boys had triglyceride concentrations above the 90th centile in the current study, with similar results for fasting insulin levels, most had values for blood pressure, fasting glucose, total and LDL cholesterol below the 90th percentile. An Australian study of 1430 eight year olds (39) (boys and girls 15% and 20% overweight or obese, respectively) found significant differences between normal and overweight/obese participants for triglycerides, HDL, systolic and diastolic pressure. Data from the Bogalusa study (40) showed that overweight children (aged 5–10 years) had a higher prevalence of elevated insulin and triglyceride levels, as well as systolic blood pressure, total and LDL cholesterol, compared to those of normal weight (defined as >95th centile for race, age and sex, >130 mg/dL, >95th centile according to National High Blood Pressure Education Program methods, >200 mg/dL and >130 mg/dL respectively). In a sample of European overweight and obese children (13) aged 12 ± 3 years (31% pre-pubertal), elevated blood pressure was the most prevalent CVD risk score factor at 35% (assessed by height >95th centile of European reference ranges). Potential influences on the reference ranges include disparities in centile or population

reference values based on age, sex, race and height; differences in assay techniques; and possibly diurnal differences in serum risk factor concentrations.

The combination of low levels of HDL with elevated serum insulin and triglyceride concentrations increases CVD risk score and was found in approximately one third of children in the current study. Insulin resistance increases triglyceride production, which in turn facilitates development of small dense LDL particles that are more susceptible to oxidation (41), further increasing CVD risk. Data compiled from four major child cardiovascular studies (Bogalusa Heart, Muscatine, Young Finns and Childhood Determinants of Adult Health) (8, 42) were able to predict subclinical atherosclerosis in children aged nine years or older by identifying those with elevated total cholesterol, triglycerides, blood pressure and BMI. In addition, Lawlor *et al.* (43) and Nyugen *et al.* (28) argue that positive and rapid changes in BMI over time further increase the risk of CVD and metabolic syndrome and need to be considered when assessing risk.

Studies examining sex-based associations between diet and CVD risk in children, particularly amongst those overweight or obese, are limited and findings inconsistent making comparisons across studies difficult. A study of a large European pediatric cohort (44) found that boys aged 6 to 9 years who consumed nuts and seeds or had high intakes of chocolate and nut-based spreads had a lower CVD risk whereas girls had a higher risk in association with high intakes of manufactured juices and lower risk with chocolate and nut-based spreads. Ambrosini *et al.* (45) found 14 year old girls, but not boys, who had diets high in fat, refined sugars and sodium had greater clustering of risk factors for metabolic syndrome. In a Mexican population of a similar age (46), positive correlations were found between white bread and fasting insulin concentrations; between fasting glucose and sugar sweetened beverages and between added fats and serum triglycerides. However, no differences were reported by sex or weight status.

Sex-based responses to diet may influence CVD risk secondary to differences in hormone profiles, lipid metabolism or lifestyle behaviors, as suggested previously by Ambrosini *et al.* (45). Differences in maturation and hormonal status of children may have influenced the results and account for some of the sex differences in this

study. Reinehr and Toschke (47) found that CVD risk factors vary by stage of puberty. In the current study, pubertal status was assessed at baseline only, with follow-up time points up to two years included. Therefore it is not known whether any participants entered puberty during this time. Consequently the results need to be interpreted with some caution (47) and further investigation is warranted. Reinehr (48) suggests that future research focus on identifying which high-risk adolescents respond to specific treatment approaches, with the current study supporting a focus on dietary interventions particularly for boys shown to be at elevated CVD risk prior to puberty.

Clustering of CVD risk factors is common (5, 13, 40, 49). The strength of the CVD risk score developed in the current study is that it combines graded scores based on ten CVD risk variables (30, 50). Deriving a combined CVD risk score from measures of adiposity, blood lipids, carbohydrate metabolism and blood pressure referenced to normal ranges has been used in previous studies (5, 17, 51). Kelly *et al.* (52) found that clustered scores were better predictors of metabolic syndrome, and that it overcame issues associated with arbitrary cut-points for some criteria (53). The benefit of such a tool is that it is easier than calculating z-scores or quintiles, as is commonly used currently (52, 54). Using both BMI and WC allows more accurate identification of adiposity and abdominal obesity, rather than those with greater lean muscle mass.

When attempting to create a risk assessment tool, there are limitations that need to be acknowledged. The current tool would be strengthened by applying it to a larger and more varied cohort in a much longer study that followed children into adulthood to obtain objective CVD outcomes. Whilst this study used the 90th centile as the cut-point across all risk factors to ensure consistency across reference values, it is acknowledged that this may reduce sensitivity in identifying all those at increased risk secondary to high BMI. Some studies (28, 43) have suggested that change in body weight may be important and this is not included in the current study. Mattsson (29) suggests that true CVD risk assessment needs to include family history of CVD and metabolic syndrome to account for genetic contributions, which were not assessed as part of the HIKCUPS study. Weight status of parents and family lifestyle behaviors could also be included in future risk

assessment (4, 39). In practice, BP, BMI, WC and family history are more likely to be measured due to their low cost, whereas blood tests to assess plasma lipids are less regularly performed (55). The use of normal reference ranges developed on other pediatric populations also has inherent difficulties. Although reported to be consistent with other Caucasian children for WC, blood pressure, serum lipids and glucose metabolism (18), the reference ranges used here were developed in French children. Variations exist when obtaining WC measures as an indicator of central adiposity in children (umbilicus, narrowest point, mid-point). This can be difficult to measure in overweight and obese pediatric participants and variations are known to exist within the measurements (56) compared to measures such as height and weight. In the current study WC measures and reference values were obtained by different methods and may have influenced the results. The use of blood pressure centiles based on height could also yield different risk scores, by allowing higher blood pressure ranges in taller children (57). In the current study the CVD risk score for each factor was weighted as equally important but this may an oversimplification and hence results should be interpreted with caution. Future studies should investigate this further, and include additional factors associated with insulin resistance (53).

4.6 Conclusions

The assessment of CVD risk based on cut-point using greater than or equal to the top tenth percentile of published normal reference ranges of variables associated with CVD risk in overweight children has demonstrated that those with a WC or BMI at or above the 90th centile were more likely to also have other CVD risk factors present. Overweight boys with high dietary intakes of fat and carbohydrate in particular had significantly more CVD risk factors elevated above the 90th centile. Future work is needed in larger cohorts to examine the relationship of sex-specific CVD risk factors in association with dietary factors and whether this could provide opportunities for development and testing of early prevention programs targeting these factors.

References

1. World Health Organization. Global Atlas on Cardiovascular Disease Prevention and Control. Geneva: World Health Organization, 2011.

2. Francis AA, Pierce GN. An integrated approach for the mechanisms responsible for atherosclerotic plaque regression. *Experimental and clinical cardiology*. 2011;16(3):77-86.

3. International Obesity Taskforce. Obesity the Global Epidemic London, United Kingdom: International Obesity Taskforce; [cited 2013 12 September]. Available from: http://www.iaso.org/iotf/obesity/obesitytheglobalepidemic/.

4. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med*. 1997;337(13):869-73.

5. Galcheva SV, Iotova VM, Yotov YT, Bernasconi S, Street ME. Circulating proinflammatory peptides related to abdominal adiposity and cardiometabolic risk factors in healthy prepubertal children. *Eur J Endocrinol*. 2011;164(4):553-8.

6. von Schnurbein J, Klenk J, Galm C, Berg S, Gottmann P, Steinacker JM, *et al.* Reference values and early determinants of intra-abdominal fat mass in primary school children. *Horm Res Paediatr*. 2011;75(6):412-22.

7. Reinehr T, Wunsch R. Relationships between cardiovascular risk profile, ultrasonographic measurement of intra-abdominal adipose tissue, and waist circumference in obese children. *Clin Nutr*. 2010;29(1):24-30.

8. Juonala M, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, *et al.* Childhood adiposity, adult adiposity, and cardiovascular risk factors. *N Engl J Med*. 2011;365(20):1876-85.

9. Huang TTK, Ball GDC, Franks PW. Metabolic syndrome in youth: current issues and challenges. *App Physiol Nutr Metab*. 2007;32(1):13-22.

10. Raitakari OT, Porkka KV, Viikari JS, Ronnemaa T, Akerblom HK. Clustering of risk factors for coronary heart disease in children and adolescents. The Cardiovascular Risk in Young Finns Study. *Acta Paediatr*. 1994;83(9):935-40.

11. Reinehr T, de Sousa G, Toschke AM, Andler W. Long-term follow-up of cardiovascular disease risk factors in children after an obesity intervention. *Am J Clin Nutr*. 2006;84(3):490-6.

12. Freedman DS, Serdula MK, Srinivasan SR, Berenson GS. Relation of circumferences and skinfold thicknesses to lipid and insulin concentrations in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr*. 1999;69(2):308-17.

13. I'Allemand D, Wiegand S, Reinehr T, Muller J, Wabitsch M, Widhalm K, *et al.* Cardiovascular risk in 26,008 European overweight children as established by a multicenter database. *Obesity (Silver Spring)*. 2008;16(7):1672-9.

14. Flechtner-Mors M, Thamm M, Wiegand S, Reinehr T, Schwab KO, Kiess W, *et al.* Comorbidities related to BMI category in children and adolescents: German/Austrian/Swiss Obesity Register APV compared to the German KiGGS Study. *Horm Res Paediatr*. 2012;77(1):19-26.

15. Mosca L, Benjamin EJ, Berra K, Bezanson JL, Dolor RJ, Lloyd-Jones DM, *et al.* Effectiveness-based guidelines for the prevention of cardiovascular disease in women--2011 update: a guideline from the american heart association. *Circulation*. 2011;123(11):1243-62.

16. National Cholesterol Education Program Expert Panel. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106(25):3143-421.

17. Cook S, Auinger P, Huang TT. Growth curves for cardio-metabolic risk factors in children and adolescents. *J Pediatr*. 2009;155(3):S6 e15-26.

18. Mellerio H, Alberti C, Druet C, Capelier F, Mercat I, Josserand E, *et al.* Novel modeling of reference values of cardiovascular risk factors in children aged 7 to 20 years. *Pediatrics*. 2012;129(4):e1020-9.

19. Framingham Heart Study. Cardiovascular Disease (30 year risk) Framingham, MA: National Heart Lung and Blood Institute, Boston University; 2013 [updated 11 September, 2013; cited 2013 17 September]. Available from: http://www.framinghamheartstudy.org/risk/cardiovascular30.html.

20. Reed KE, Warburton DE, McKay HA. Determining cardiovascular disease risk in elementary school children: developing a healthy heart score. *J Sports Sci Med*. 2007;6(1):142-8.

21. Okely AD, Collins CE, Morgan PJ, Jones RA, Warren JM, Cliff DP, *et al.* Multi-site randomized controlled trial of a child-centered physical activity program, a parent-centered dietary-modification program, or both in overweight children: the HIKCUPS study. *J Pediatr*. 2010;157(3):388-94, 94 e1.

22. Jones RA, Okely AD, Collins CE, Morgan PJ, Steele JR, Warren JM, *et al.* The HIKCUPS trial: a multi-site randomized controlled trial of a combined physical activity skill-development and dietary modification program in overweight and obese children. *BMC Public Health*. 2007;7:15.

23. Collins CE, Okely AD, Morgan PJ, Jones RA, Burrows TL, Cliff DP, *et al.* Parent diet modification, child activity, or both in obese children: an RCT. *Pediatrics*. 2011;127(4):619-27.

24. Burrows T, Warren JM, Baur LA, Collins CE. Impact of a child obesity intervention on dietary intake and behaviors. *Int J Obes*. 2008;32(10):1481-8.

25. Burrows T, Warren JM, Collins CE. The impact of a child obesity treatment intervention on parent child-feeding practices. *Int J Pediatr Obes*. 2010;5(1):43-50.

26. Marfell-Jones M, Olds T, Steward A, Carter L. International Standards for Anthropometric Assessment. Potchefstroom, South Africa: International Society for the Advancement of Kinanthropometry; 2006.

27. Berenson GS, Srnivasan SR, Bogalusa Heart Study G. Cardiovascular risk factors in youth with implications for aging: the Bogalusa Heart Study. *Neurobiol Aging*. 2005;26(3):303-7.

28. Nguyen QM, Srinivasan SR, Xu J-H, Chen W, Kieltyka L, Berenson GS. Utility of childhood glucose homeostasis variables in predicting adult diabetes and related cardiometabolic risk factors: the Bogalusa Heart Study. *Diabetes Care*. 2010;33(3):670-5.

29. Mattsson N, Ronnemaa T, Juonala M, Viikari JS, Raitakari OT. Childhood predictors of the metabolic syndrome in adulthood. The Cardiovascular Risk in Young Finns Study. *Ann Med*. 2008;40(7):542-52.

30. International Diabetes Federation. The IDF consensus definition of the Metabolic Syndrome in Children and Adolescents. Belgium: International Diabetes Federation, 2007.

31. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 2011;128 Suppl 5:S213-56.

32. Bergmann GG, Gaya A, Halpern R, Bergmann MLA, Rech RR, Constanzi CB, *et al.* Waist circumference as screening instrument for cardiovascular disease risk factors in schoolchildren. *J Pediatr (Rio J)*. 2010;86(5):411-6.

33. Kuczmarski R, Ogden C, Guo S. 2000 CDC Growth Charts for the United States: Methods and Development. Washington, DC.: National Center for Health Statistics; 2002.

34. Watson JF, Collins CE, Sibbritt DW, Dibley MJ, Garg ML. Reproducibility and comparative validity of a food frequency questionnaire for Australian children and adolescents. *Int J Behav Nutr Phys Act*. 2009;6:62.

35. Burrows TL, Warren JM, Colyvas K, Garg ML, Collins CE. Validation of overweight children's fruit and vegetable intake using plasma carotenoids. *Obesity (Silver Spring)*. 2009;17(1):162-8.

36. Burrows T, Berthon B, Garg ML, Collins CE. A comparative validation of a child food frequency questionnaire using red blood cell membrane fatty acids. *Eur J Clin Nutr*. 2012;66(7):825-9.

37. Burrows T, Janet WM, Collins CE. Long-term changes in food consumption trends in overweight children in the HIKCUPS intervention. *J Pediatr Gastroenterol Nutr*. 2011;53(5):543-7.

38. LP S. Stata Statistical Software: Version 12.1. 4905 Lakeway Drive, College Station, TX2012.

39. Burke V, Beilin LJ, Simmer K, Oddy WH, Blake KV, Doherty D, *et al.* Predictors of body mass index and associations with cardiovascular risk factors in Australian children: a prospective cohort study. *Int J Obes*. 2005;29(1):15-23.

40. Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics*. 1999;103(6 Pt 1):1175-82.

41. Barter P. The role of HDL-cholesterol in preventing atherosclerotic disease. *Eur Heart J Suppl*. 2005;7(F):F4-F8.

42. Juonala M, Magnussen CG, Venn A, Dwyer T, Burns TL, Davis PH, *et al.* Influence of age on associations between childhood risk factors and carotid intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study, the Childhood Determinants of Adult Health Study, the Bogalusa Heart Study, and the Muscatine Study for the International Childhood Cardiovascular Cohort (i3C) Consortium. *Circulation*. 2010;122(24):2514-20.

43. Lawlor DA, Benfield L, Logue J, Tilling K, Howe LD, Fraser A, *et al.* Association between general and central adiposity in childhood, and change in these, with cardiovascular risk factors in adolescence: prospective cohort study. *BMJ*. 2010;341:c6224.

44. Bel-Serrat S, Mouratidou T, Bornhorst C, Peplies J, De Henauw S, Marild S, *et al.* Food consumption and cardiovascular risk factors in European children: the IDEFICS study. *Pediatr Obes*. 2013;8(3):225-36.

45. Ambrosini GL, Huang RC, Mori TA, Hands BP, O'Sullivan TA, de Klerk NH, *et al.* Dietary patterns and markers for the metabolic syndrome in Australian adolescents. *Nutr Metab Cardiovasc Dis*. 2010;20(4):274-83.

46. Perichart-Perera O, Balas-Nakash M, Rodriguez-Cano A, Munoz-Manrique C, Monge-Urrea A, Vadillo-Ortega F. Correlates of dietary energy sources with cardiovascular disease risk markers in Mexican school-age children. *J Am Diet Assoc*. 2010;110(2):253-60.

47. Reinehr T, Toschke AM. Onset of puberty and cardiovascular risk factors in untreated obese children and adolescents: a 1-year follow-up study. *Arch Pediatr Adolesc Med*. 2009;163(8):709-15.

48. Reinehr T. Calculating cardiac risk in obese adolescents before and after onset of lifestyle intervention. *Expert Rev Cardiovasc Ther*. 2013;11(3):297-306.

49. Kelishadi R, Ardalan G, Adeli K, Motaghian M, Majdzadeh R, Mahmood-Arabi MS, *et al.* Factor analysis of cardiovascular risk clustering in pediatric metabolic syndrome: CASPIAN study. *Ann Nutr Metab*. 2007;51(3):208-15.

50. Berenson GS, Srinivasan SR, Bao W, Newman WP, 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *N Engl J Med*. 1998;338(23):1650-6.

51. Falkner B, Sadowski RH. Hypertension in children and adolescents. *Am J Hypertens*. 1995;8(12 Pt 2):106s-10s.

52. Kelly AS, Steinberger J, Jacobs DR, Hong CP, Moran A, Sinaiko AR. Predicting cardiovascular risk in young adulthood from the metabolic syndrome, its component risk factors, and a cluster score in childhood. *Int J Pediatr Obes*. 2011;6(2-2):e283-9.

53. Reaven GM. The metabolic syndrome: requiescat in pace. *Clin Chem*. 2005;51(6):931-8.

54. Andersen LB, Wedderkopp N, Hansen HS, Cooper AR, Froberg K. Biological cardiovascular risk factors cluster in Danish children and adolescents: the European Youth Heart Study. *Prev Med*. 2003;37(4):363-7.

55. Dixon DB, Kornblum AP, Steffen LM, Zhou X, Steinberger J. Implementation of lipid screening guidelines in children by primary pediatric providers. *J Pediatr*. 2014;164(3):572-6.

56. Johnson ST, Kuk JL, Mackenzie KA, Huang TT, Rosychuk RJ, Ball GD. Metabolic risk varies according to waist circumference measurement site in overweight boys and girls. *J Pediatr*. 2010;156(2):247-52 e1.

57. Update on the 1987 task force report on high blood pressure in children and adolescents. *Pediatrics*. 1996;98(4):649.

Chapter 5: Dietary patterns of adolescent girls attending schools in low-income communities highlight low consumption of core foods

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Schumacher TL, Dewar DL, Lubans DR, Morgan PJ, Watson J, Guest M, *et al.* Dietary patterns of adolescent girls attending schools in low-income communities highlight low consumption of core foods. *Nutrition & Dietetics*. 2014;71: 127-34. (Accepted June, 2013)

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5.1 Overview

This chapter is the second of the secondary data analyses used to assess diet-related CVD risk in young people with an existing CVD risk factors. The population investigated in this chapter are girls in early to mid-adolescence who attended school in a low socio-economic area. Socio-economic status was of interest as this is associated with increased risk of CVD. Data for this analysis was sourced from a randomized controlled trial that commenced in 2010.

5.2 Abstract

Aim: Overweight and obesity prevalence is high among adolescent girls of low socio-economic position and this increases their risk of cardiovascular disease and metabolic disorders in adulthood. The aim of this study was to describe the dietary patterns of adolescent girls in terms of the relative contribution of core food groups to overall diet and by weight status category.

Methods: Year 8 female students were recruited from schools in low-income communities. Weight status (i.e. underweight, healthy weight, overweight, obese) was determined using age- and sex-adjusted body mass index (*z* score). Dietary intakes were assessed using a validated semi-quantitative food frequency questionnaire. Individual foods were collated into core food group or energy-dense, nutrient-poor categories in line with the Australian Guide to Healthy Eating (AGHE) and the percentage contribution to total energy intake calculated.

Results: Participants (n=332) were (mean±SD) 13.7±0.4 years old with BMI z score 0.63±1.22. Few girls met AGHE core food group recommendations for daily serves; meat and substitutes 69.3%, vegetables 28.6%, fruit 23.8%, dairy 15.7%, and breads/cereals 5.7%. Total percentage energy derived from energy-dense, nutrient-poor foods was 46.6% (37.2-54.6%)[(median (interquartile range)], with takeaways 9.8% (7.0-13.6%), confectionery 7.0% (4.1-10.9%) and packaged snacks 6.8% (4.0-10.7%), with no significant differences by weight status.

Conclusions: Core food intakes are poor with excessive consumption of energy-dense, nutrient-poor foods in these adolescent girls. Nutrition education programs

targeting this population are needed to address this imbalance. Strategies could include substitution of unhealthy snacks for core food items and greater inclusion of core foods within main meals.

5.3 Introduction

Data reported by the Australian Bureau of Statistics show that the proportion of overweight adolescent girls increased from 12% to 20% in the years between 1985 and 2008 with the obesity rate staying constant at 6% (1). Overweight and obesity tracks from childhood into adolescence and the relationship with morbidity and mortality is well documented (2, 3). If obesity progresses into adulthood, it increases the risk of metabolic disorders, including type 2 diabetes, elevated triglyceride levels, hypertension and adult cardiovascular disease (CVD) (3-7). Many factors contribute to obesity and facilitate energy imbalance (7, 8). However limited evidence has been reported describing the association between weight status and dietary patterns in adolescence (9).

Emerging research from the Project Eat study (Minnesota, USA) has provided insight into the dietary habits and nutrient intakes of North American adolescents (10-13). It has shown that adolescent girls are likely to retain similar dietary patterns throughout adolescence and that intakes of fruit, vegetables and starchy foods are positively associated with socioeconomic position (SEP) (10, 11). This may be one reason why obesity levels are higher among adolescents living in low-income communities (14). Surprisingly few studies have explored the dietary patterns of Australian adolescent girls of low SEP, although international evidence suggests adolescent girls are more likely to report unhealthy eating behaviours, such as a skipping breakfast and snacking on energy-dense, nutrient-poor foods (15-17). Therefore, the primary aim of this investigation was to determine the relative contributions of core foods and energy-dense, nutrient-poor food groups to dietary intake in adolescent girls attending schools in low-income communities in an Australian population. Secondary aims were to investigate these dietary patterns by weight status, as determined by body mass index (BMI) category (i.e. underweight, healthy weight, overweight, obese) and related dietary behaviours. It was hypothesised that girls from low SEP would have low intakes of core foods,

and disproportionally high intakes of consumption of energy-dense, nutrient-poor foods and that this would vary based on weight status.

5.4 Methods

This is a secondary analysis of baseline data collected in 2010 as part of the Nutrition and Enjoyable Activity for Teen Girls (NEAT Girls) cluster randomised control trial (RCT) (18, 19). NEAT Girls is a 12-month obesity prevention program targeting teenage girls from schools in areas of social and economic disadvantage. Detailed methods have been published elsewhere (18).

The participants were recruited from government secondary schools located in low-income communities [based on the Socioeconomic Index for Areas (SEIFA)] in the Hunter and Central Coast regions in New South Wales, Australia (20). SEIFA is a measure of relative socioeconomic disadvantage of geographical areas, determined by education, employment, occupation-related skills and economic resources such as assets, income and expenditure. Schools were eligible to participate if located in areas with an allocated SEIFA index of ≤5 (bottom 50%). Girls were selected from Year 8, the second year of high school in Australia, and included if identified by their teachers as being disengaged from physical education classes and/or not participating in an organized team or individual sport. Approximately 30 girls from 12 schools were recruited (n=357).

Data were collected at each school. Written consent was obtained from school principals, parents and participants. Ethics approval was given by the University of Newcastle Human Research Ethics Committee and the New South Wales Department of Education and Training Human Research Ethics Committee. A portable stadiometer was used to obtain height measurements to the nearest 0.1cm and weight was measured to the nearest 0.1kg using portable digital scales. Body Mass Index (BMI) was calculated for each participant as weight (kg) divided by height (meters) squared and then ranked into underweight, healthy, overweight or obese categories according to the Cole cut-points.(21, 22). These were selected as they are derived from international data sets and represent the paediatric equivalent cut-points of adult BMI at <18.5, 25 and ≥30 kg/m^2 and were used to

categorise the students as BMI <18.5 underweight, ≥25 overweight and ≥30 obese. BMI *z* score was created using U.S. reference data (23). Physical activity was assessed using accelerometers and reported elsewhere (19, 24).

Participants completed the Australian Child and Adolescent Eating Survey (ACAES, version 1.2), a 120 question semi-quantitative food frequency questionnaire (FFQ) previously validated in nine to 16 year olds to estimate dietary intake data over the previous six months (25). The ACAES includes 15 supplementary questions related to eating behaviours, such as eating while viewing television, regular breakfast habits, number of dairy foods consumed daily and snacking occasions. Food serves are based on age group specific portion sizes derived from unpublished data in the 1995 National Nutrition Survey, and 'natural' food portions for items such as fruit (25). Nutrient intakes were derived from the questionnaire responses using FoodWorks (professional version 3.02.581, 2004, Xyris Software, Brisbane, Australia) and the following databases without modification (Australian AusNut, All Foods, Revision 14 and AusFoods, Brands, Revision 5, 1999 Food Standards Australia New Zealand, Canberra, Australia). Diet quality was measured using the Australian Recommended Food Score (ARFS), a validated measure of diet quality that aligns with national dietary guidelines (26).

For the purposes of this analysis, only individuals with complete data sets (height, weight, date of birth, completed FFQ) were included (n=356). Inaccurate dietary reporting was defined based on the methods of Field *et al.* with 24 reporters of <500kcal/day (2,090kJ) or >5000kcal/day (20,900kJ) per day removed before analysis as implausible (n=332 included) (27, 28).

5.4.1 Statistical analysis

Individual questions from the FFQ were aggregated into core and energy-dense, nutrient-poor food groups, defined according to the Australian Guide to Healthy Eating (AGHE), and used to derive food group total energy values and percentages (29). To determine differences in energy contributions by weight status, a Kruskal-Wallis equality of populations rank test was used at a statistical significance level of 5% as data were non-parametric. Fishers exact Chi squared Test was used to test equality in groups with dichotomous data. The statistical program used was

Stata/IC (version 11.2 for Windows 2012, StataCorp LP, College Station, TX, USA). Data are reported as median (interquartile range) unless otherwise indicated.

5.5 Results

Participants were 13.7 (13.4-13.9) years old, with 98% born in Australia and 99% speaking English at home. Most participants identified their cultural background as Australian (86%), inclusive of 11% Aboriginal and Torres Strait Islander (ATSI); small proportions identified as European (10%), Asian (1%) or other (3%).

Table 5.1 summarizes the anthropometric characteristics of the participants according to weight status. The number of participants meeting minimum core food group recommendation intakes and self-reported general eating habits are shown in Table 5.2. Only one of the 332 adolescent girls met all core food group recommendations. Participants were most likely to meet the daily serve recommendations for meat and vegetarian alternatives (69.3%) and least likely to meet the recommendations for dairy and breads/cereal servings (15.7% and 5.7% respectively). Participants reported low intake of dairy foods (0.6 (0.1-2.5) serves per day) and fruit (0.5 (0.2-2.0) pieces a day). Based on the ARFS, participants had poor diet quality with a mean of 24 (17-33) points scored from a maximum of 73. Almost equal percentages (median (IQR)) of energy were obtained from core (53.4% (45.4-62.8.%)) and energy-dense, nutrient-poor foods (46.6% (37.2-54.6%)), with no statistically significant difference by weight status group (P>0.05). Core food group intakes and energy-dense, nutrient-poor foods as a percentage of total energy (% energy) are summarised in Table 5.3 and indicate that takeaways (10% (7-14%)), confectionery (7% (4-11%)) and pre-packaged snacks (7% (4-11%)) account for a high percentage of total energy intake. The macronutrient distributions were protein 17% (15-19%), carbohydrate 50% (46-55%) and fat 34% (30-37%). Saturated fat contributed 15% (13-17%) of total energy, with 12% (10-13%) from monounsaturated fat and 4% (3-4%) from polyunsaturated fat.

Table 5.1: Baseline demographics, number and proportion of adolescent girls (n=332) from low socioeconomic schools by weight status categories [a].

	Underweight	Healthy	Overweight	Obese	All
	n (%)	n (%)	n (%)	n (%)	n (%)
	7 (2.1)	188 (56.6)	92 (27.7)	45 (13.6)	332 (100)
	← Median (interquartile range) →				
Weight (kg)	38.4 (35.5-41.6)	50.6 (46.1-54.2)	64.5 (59.9-67.8)	81.5 (76.6-89.4)	55.1 (49.0-65.9)
Height (m)	1.60 (1.51-1.63)	1.59 (1.56-1.64)	1.61 (1.56-1.64)	1.63 1.6-1.68)	1.61 (1.56-1.64)
BMI (kg/m^2)	15.6 (14.9-15.9)	19.8 (18.4-21.0)	24.9 (23.8-26.0)	30.6 (29.4-32.8)	21.7 (19.5-25.1)
BMI z score	-1.06 (-1.37- -0.9)	-0.09 (-0.48-0.24)	1.12 (0.78-1.42)	2.67 (2.23-3.38)	0.3 (-0.23-1.26)
kJ/kg/day	286 (209-433)	164 (129-229)	126 (84-171)	97 (68-126)	147 (102-204)

[a] Weight status is derived from adult limits for underweight (<18.5 kg/m2), overweight (≥25 kg/m2) and obesity (≥30 kg/m2)as defined by Cole *et al.* at age specific levels.(21, 22)

Table 5.2: Number (%) of girls meeting or exceeding minimum recommendation of daily servings of core food and results from self-reported eating behaviour questions in the Australian Child and Adolescent Eating Survey in adolescent girls (n=332) from low socioeconomic schools by weight status categories.

	Underweight 7 (2.1%)	Healthy 188 (56.6%)	Overweight 92 (27.7%)	Obese 45 (13.6%)	All 332 (100%)	P Value
CORE FOOD GROUPS [A]	← Girls meeting or exceeding core food recommendations[b] n (%) →					
Fruit	1 (14.3)	44 (23.4)	23 (25)	11 (24.4)	97 (23.8)	0.98
Vegetables	1 (14.3)	51 (27.1)	28 (30.4)	15 (33.3)	95 (28.6)	0.70
Meat & substitutes	7 (100)	134 (71.3)	64 (69.6)	25 (55.6)	230 (69.3)	0.08
Dairy	1 (14.3)	34 (18.1)	10 (10.9)	7 (15.6)	52 (15.7)	0.43
Breads & cereals	1 (14.3)	11 (5.9)	3 (3.3)	4 (8.9)	19 (5.7)	0.27
EATING BEHAVIOURS [C]	← Median (interquartile range) →					
Breakfast (days per week) [d]	6.0 (3.5-6)	3.5 (1.5-6.0)	6.0 (1.5-6.0)	3.5 (1.5-6.0)	3.5 (1.5-6.0)	0.17
Vegetables (days per week) [e]	5.5 (3.5-5.5)	3.5 (3.5-5.5)	5.5 (3.5-5.5)	5.5 (3.5-5.5)	3.5 (3.5-5.5)	0.24
Takeaway (days per week) [f]	0.5 (0.5-1.5)	0.5 (0.5-1.5)	0.5 (0.5-1.5)	0.5 (0.5-1.5)	0.5 (0.5-1.5)	0.94
Television (days per week) [g]	1.5 (0.5-7.0)	1.5 (0.5-3.5)	1.5 (0.3-6.3)	3.5 (0.5-5.5)	1.5 (0.5-5.5)	0.31
$AUD for snacks ($ per week) [h]	7.0 (1.0-7.0)	3.5 (1.0-7.0)	3.5 (1.0-9.5)	3.5 (1.0-7.0)	3.5 (1.0-7.0)	0.85
Fruit (pieces per day) [i]	0.5 (0.2-1.0)	0.5 (0.2-1.0)	0.5 (0.5-1.0)	0.8 (0.2-2.5)	0.5 (0.2-1.0)	0.75
Snacks (snacks per day) [j]	1.5 (1.5-3.5)	1.5 (1.5-3.5)	1.5 (1.5-3.5)	1.5 (1.5-3.5)	1.5 (1.5-3.5)	0.47
Dairy (dairy serves per day) [k]	0.6 (0.6-2.5)	0.6 (0.1-2.5)	0.6 (0.1-1.0)	1.0 (0.6-2.5)	0.6 (0.1-2.5)	0.07
Sweetened drinks (glasses per day) [l]	2.5 (0.5-2.5)	1.0 (0.5-2.5)	1.8 (0.5-2.5)	1.0 (0.5-2.5)	1.0 (0.5-2.5)	0.68
ARFS [m]	24 (20-29)	24 (17-33)	25 (17-34)	25 (19-31)	24 (17-33)	0.94

[a] P value obtained by Fisher's exact Chi-square test

Table 5.2: Number (%) of girls meeting or exceeding minimum recommendation of daily servings of core food and results from self-reported eating behaviour questions in the Australian Child and Adolescent Eating Survey in adolescent girls (n=332) from low socioeconomic schools by weight status categories.

	Underweight	Healthy	Overweight	Obese	All	P Value
	7 (2.1%)	188 (56.6%)	92 (27.7%)	45 (13.6%)	332 (100%)	

[b] Food servings per day based on self-report data compiled from relevant questions in the Australian Child and Adolescent Eating Survey and compared to minimum daily food group serving recommended in the 1998 Australian Guide to Health Eating for 12-18 year old girls (fruit 3, vegetables 4, meat or substitutes 1, dairy 3, breads or cereals 4).

[c] P value obtained by Kruskal-Wallis equality of populations with ties for non-parametric data for significance between group rankings.

[d] How many days per week do you usually have something to eat for breakfast?

[e] How many times a week do you eat vegetables with your meal at night?

[f] How often do you eat takeaway foods? E.g. Chinese, fish and chips, hamburger and chips/fries, pizza? (days per week)

[g] How many times a week do you eat your meal at night in front of the television (TV)?

[h] How much money are you given each week to buy food, including snacks and drinks?

[i] How many pieces of fruit do you eat? (per day)

[j] How many times a day do you eat snacks?

[k] Add up how many times a day you have a glass of milk, a tub of yoghurt or a slice of cheese.

[l] Add up how many glasses of soda or cordial you have each day *(all types)*.

[m] Australian Recommended Food Score; measures variety in dietary intake. Score range: 0 – 73.[26]

Table 5.3: Percentage energy from core and energy-dense, nutrient-poor food groups in adolescent girls (n=332) from low socioeconomic schools.§

	Underweight	Healthy	Overweight	Obese	All	P Value [a]
FOOD GROUP	← Median (interquartile range) →					
Core foods	50.6 (48.2-57.4)	53.7 (45.2-63.4)	52.8 (45.2-62.6)	53.6 (45.3-62.4)	53.4 (45.4-62.8)	0.96
Energy-dense, nutrient-poor foods	49.4 (42.6-51.8)	46.3 (36.6-54.8)	47.2 (37.4-54.8)	46.4 (37.6-54.6)	46.6 (37.2-54.6)	0.96
CORE FOOD GROUPS [B]						
Vegetables	3.9 (2.3-4.3)	4.5 (2.7-8.7)	4.9 (3.1-8.4)	5.7 (3.0-9.3)	4.9 (2.9-8.5)	0.20
Fruit	4.2 (1.8-7.8)	5.7 (2.7-9.4)	5.4 (3.3-9.5)	6.3 (3.1-10.4)	5.6 (2.8-9.4)	0.66
Meat	13.5 (10.1-17.1)	12.5 (8.9-19.1)	14.4 (9.8-19.8)	11.2 (6.7-15.1)	12.8 (8.7-18.9)	0.06
Meat alternatives	0.7 (0.5-2.8)	1.1 (0.4-1.9)	1.2 (0.5-2.4)	0.7 (0.3-1.4)	1.1 (0.4-2.0)	0.18
Grains and cereals	14.6 (12.6-20.4)	12.9 (7.8-18.0)	12.9 (8.7-18.7)	14.3 (9.8-20.5)	13.0 (8.5-18.8)	0.52
Dairy	6.9 (2.6-15.4)	9.0 (4.1-15.1)	8.3 (4.7-13.2)	10.3 (6.7-16.6)	8.9 (4.8-15.0)	0.33
COMBINATIONS OF CORE FOODS						
Meals with vegetables	6.0 (5.0-10.2)	6.4 (3.9-10.4)	7.6 (4.9-10.4)	5.7 (2.9-9.3)	6.7 (4.1-10.2)	0.21
Meals with no vegetables	3.6 (1.4-5.3)	3.9 (1.6-6.5)	2.9 (1.2-6.2)	2.2 (0.5-3.9)	3.4 (1.2-6.1)	0.02*
ENERGY-DENSE, NUTRIENT-POOR FOOD GROUPS						
Sweetened drinks [c]	8.7 (4.7-10.6)	5.9 (2.8-11.3)	6.9 (3.8-10.8)	8.1 (3.6-11.3)	6.4 (3.2-11.1)	0.59
Packaged snacks [d]	7.4 (5.7-15.4)	6.9 (4.2-11.2)	6.6 (4.2-11.2)	6.3 (3.2-9.8)	6.8 (4.0-10.7)	0.48
Confectionery [e]	10.0 (9.4-16.1)	6.7 (3.7-10.5)	6.7 (4.2-11.5)	7.8 (5.6-14.5)	7.0 (4.1-10.9)	0.07
Baked sweet products [f]	5.1 (3.5-11.0)	4.9 (3.0-8.3)	5.4 (3.1-8.2)	4.8 (3.1-7.8)	5.0 (3.1-8.3)	0.84
Takeaways and fried foods [g]	7.9 (3.6-11.5)	10.0 (6.7-13.5)	9.5 (7.1-14.4)	9.3 (6.5-12.1)	9.8 (7.0-13.6)	0.85
Spreads and sauces [h]	2.6 (0.7-3.4)	1.2 (0.7-2.1)	1.4 (0.9-1.8)	1.5 (0.6-2.5)	1.3 (0.7-2.1)	0.56
Fatty meats [i]	1.3 (0.7-1.6)	1.2 (0.6-2.2)	1.5 (0.7-2.7)	1.2 (0.8-1.9)	1.2 (0.7-2.3)	0.23

Table 5.3: Percentage energy from core and energy-dense, nutrient-poor food groups in adolescent girls (n=332) from low socioeconomic schools.§

	Underweight	Healthy	Overweight	Obese	All	P Value [a]
FOOD GROUP			← Median (interquartile range) →			
MISCELLANEOUS [J]	0.2 (0.1-1.3)	0.3 (0.0-0.9)	0.4 (0.1-0.7)	0.4 (0.0-0.6)	0.4 (0.1-0.8)	0.68

§ Percentage energy has been calculated as a percentage of total energy and reported as median (interquartile range) as data for these values are highly skewed. Therefore compilation of food groups are unlikely to match total values.

***** $p<0.5$

[a] P value obtained by Kruskal-Wallis equality of populations with ties for non-parametric data for significance between group rankings.

[b] Foods contributing to core foods were aligned with the Australian Guide to Healthy Eating [29]

[c] Comprised of beverages such as soft drinks, fruit juice based drinks and cordials

[d] Comprised of foods such as biscuit/spread combinations and snack noodles and bars

[e] Comprised of foods such as ice-creams and frozen yoghurts, chocolates and lollies

[f] Comprised of foods such as cakes, pastries and sweet biscuits

[g] Comprised of foods such as pies, pizzas, hamburgers, potato chips and crumbed chicken and fish

[h] Comprised of spread and sauces such as mayonnaise, jam, honey, vegemite and tomato sauce

[i] Comprised of foods such as sausages, salamis, bacon and devon

[j] Incorporates foods with no clear category such as clear soups, tea and coffee

In regards to weight status, there was no statistically significant difference (P>0.05) in the percentage of girls meeting healthy eating recommendations, in any eating behaviours or in % energy from food groups with the exception of % energy from meals with no vegetables (P=0.02) in core foods.

5.6 Discussion

This study highlights that the majority of adolescent girls from schools in low income communities in Australia do not have eating patterns that meet the AGHE recommended daily servings of each core food group (29). As hypothesised, a high proportion of their energy comes from energy-dense, nutrient-poor foods, however contrary to our hypothesis these dietary patterns did not vary with weight status.

The contribution of energy from core foods in this low income population (54%) is lower than that reported by Rangan *et al.* (62%) in adolescents of similar age in their analysis of the 2007 Australian National Children Nutrition and Physical Activity Survey data that included all income levels and males (30). Rangan *et al.* found the proportion of energy from core foods in a National representative population sample of 2-16 year old Australian children had increased from 59% (per capita) in 1995 to 65% in 2007. This suggests that eating habits in this SEP female population are much poorer than the pooled National data suggest.

Core food group intakes overall were poor, with no significant difference by weight status category. Similar to our findings, an online study by Savige *et al.* found that only 37% of adolescent girls ate fruit on a daily basis (31). This is of concern, as findings from Project EAT suggest that fruit intake decreases even further later in adolescence (12). However, the percentage of participants reporting consumption of four or more serves of vegetables per day (29%) was higher than that reported by Scully *et al.* where only 20% of 12-13 year olds met this target (32). One reason for low vegetable intake was highlighted in Table 2 where the majority of girls reported vegetable consumption with their evening meal 3-4 times a week or less.

Few girls met intake targets for dairy foods and breads and cereals, although more girls met recommendations for meat and substitutes. This is consistent with 2011

Cancer Council of Australia data, which found little difference in the proportion meeting dietary intake recommendations by weight status category (33). This mismatch between intake and adherence to core food intake targets increases the risk of diet related chronic diseases such as osteoporosis and CVD.

Dairy intake for Australian girls in this age group has been identified as a concern with only 11% of 12-13 year old and 18% of 14-16 year old girls in 2007 meeting the Australian estimated average requirements (EAR) for calcium, with a mean intake of 792mg/day (34). Our study found that 43% of girls were meeting their EAR of 1050mg for calcium, with a median intake of 946mg and similar to that found in Project EAT (997mg/day) (35). This is surprising, given that the participants reported less than one serve of dairy products per day.

Takeaway foods were the most commonly consumed energy-dense, nutrient-poor dietary sub-group (Table 3). It is noteworthy that the majority of girls reported having takeaway foods less than once a week in the general questions, yet the analysis found that approximately 10% of their daily energy intake came from takeaways and fried foods. This may indicate misperceptions as to what items are considered 'fast foods', or that consumption of these foods outside main meal times is not considered an eating away from home occasion. Alternatively it may indicate that foods similar to takeaways, such as chicken nuggets and chips or pizza are being routinely prepared and consumed at home. Data from the 2009/10 National Secondary Students' Diet and Activity (NaSSDA) survey found 34% of girls in a similar age group consumed takeaway at least once a week, with a higher frequency (40%) for lower SEP groups (33). NaSSDA also that found lower SEP groups were more likely to consume larger quantities of sweetened drinks and that this increased steadily with age (33).

Girls in the current study ate breakfast a median of 3.5 days per week, which is consistent with Delva *et al.* who found that adolescents from low SEP backgrounds ate breakfast less frequently than those of middle and high SEP (15). This is of concern, as skipping breakfast is associated with increased risk of weight gain between adolescence and young adulthood (36).

Overall, strategies are required to increase adolescent girls' intake of the core foods such as fruit, vegetable, dairy, breads and cereal. This should include improving the nutritional value of regularly consumed snacks. In addition, a greater emphasis could be placed on encouraging water as a preferred beverage and substitution of energy-dense, nutrient-poor snacks for those derived predominantly from core foods.

In terms of aims for nutrition education programs for this particular group of female adolescents, decreasing energy-dense, nutrient-poor foods and increasing core food intake is of major importance as a strategy to prevent development of diet-related chronic disease. Assistance with making nutrition-related decisions may be useful, such as choosing higher nutritional value snacks from core food groups, managing portion size and controlling eating occasions. Creating home and school environments where the easiest snack decision is a more nutritional one may be addressed through school education programs or engagement with parents. Emphasis may include reducing sweetened drinks, packaged snacks and confectionery intake and replacing with water, dairy, fruit, vegetable and wholegrain cereal serves. Hart *et al.* found improving fruit and reducing energy-dense snacks assisted in reducing BMI status in overweight teenagers (37). Those from the healthy weight range need to be encouraged to increase core foods, whilst decreasing frequency of energy-dense, nutrient-poor foods, to improve the nutrient intakes in this population group, thereby decreasing the risk of chronic disease (38).

A limitation of this study was the narrow sampling frame, as all participants included were initially selected based on criteria for non-engagement in physical activity. This may reflect a selection bias for poor diet as risk factors such as poor diet and low physical activity levels cluster in adolescents (39). A level of misreporting of energy intake was expected as low income, higher BMIs and females are associated with misreporting (40, 41). This was demonstrated to occur as shown by the difference between weight status groups in reported daily kilojoules per kilogram of body weight (see Table 1). Participants in the underweight category were more likely to over-report energy intake and those in

the obese category were more likely to under-report. In an effort to adjust for this, the data were reported as percentage of total energy intake.

In summary, core food intake was lower and high energy, low nutrient food intake was much greater than national recommendations for this group of low SEP adolescent females. There is a need to replace energy-dense foods with more nutrient-dense options. Dietary patterns by weight status category were consistent, with similar proportions of energy being derived from core foods and all adolescents needing to improve the nutrient density of their dietary intakes. Therefore we recommend nutrition programs that aim to improve intakes of core foods and decrease intakes of energy-dense, nutrient-poor foods are urgently required. Nutrition education should ideally focus on encouraging breakfast and snacks from core foods groups for those from lower socio-economic backgrounds.

References

1. Australian Bureau of Statistics. Austalian Social Trends: Children who are overweight or obese. September ed. Canberra2009.

2. Hughes AR, Sherriff A, Lawlor DA, Ness AR, Reilly JJ. Incidence of obesity during childhood and adolescence in a large contemporary cohort. *Prev Med*. 2011;52(5):300-4.

3. Lloyd LJ, Langley-Evans SC, McMullen S. Childhood obesity and adult cardiovascular disease risk: a systematic review. *Int J Obes*. 2010;34(1):18-28.

4. Bjorge T, Engeland A, Tverdal A, Smith GD. Body mass index in adolescence in relation to cause-specific mortality: a follow-up of 230,000 Norwegian adolescents. *Am J Epidemiol*. 2008;168(1):30-7.

5. Tailor AM, Peeters PH, Norat T, Vineis P, Romaguera D. An update on the prevalence of the metabolic syndrome in children and adolescents. *Int J Pediatr Obes*. 2010;5(3):202-13.

6. Wake M, Canterford L, Patton GC, Hesketh K, Hardy P, Williams J, *et al.* Comorbidities of overweight/obesity experienced in adolescence: longitudinal study. *Arch Dis Child*. 2010;95(3):162-8.

7. Biro FM, Wien M. Childhood obesity and adult morbidities. *Am J Clin Nutr*. 2010;91(5):1499S-505S.

8. Reilly JJ, Ness AR, Sherriff A. Epidemiological and physiological approaches to understanding the etiology of pediatric obesity: finding the needle in the haystack. *Pediatr Res*. 2007;61(6):646-52.

9. Ledoux TA, Hingle MD, Baranowski T. Relationship of fruit and vegetable intake with adiposity: a systematic review. *Obes Rev*. 2011;12(5):e143-50.

10. Cutler GJ, Flood A, Hannan P, Neumark-Sztainer D. Multiple sociodemographic and socioenvironmental characteristics are correlated with major patterns of dietary intake in adolescents. *J Am Diet Assoc*. 2011;111(2):230-40.

11. Cutler GJ, Flood A, Hannan P, Neumark-Sztainer D. Major patterns of dietary intake in adolescents and their stability over time. *J Nutr*. 2009;139(2):323-8.

12. Larson NI, Neumark-Sztainer D, Hannan PJ, Story M. Trends in adolescent fruit and vegetable consumption, 1999-2004: project EAT. *Am J Prev Med*. 2007;32(2):147-50.

13. Sherwood NE, Wall M, Neumark-Sztainer D, Story M. Effect of socioeconomic status on weight change patterns in adolescents. *Prev Chronic Dis*. 2009;6(1):A19.

14. Murasko JE. Trends in the associations between family income, height and body mass index in US children and adolescents: 1971-1980 and 1999-2008. *Ann Hum Biol*. 2011;38(3):290-306.

15. Delva J, O'Malley PM, Johnston LD. Racial/ethnic and socioeconomic status differences in overweight and health-related behaviors among American students: national trends 1986-2003. *J Adolesc Health*. 2006;39(4):536-45.

16. Matthys C, De Henauw S, Maes L, De Bacquer D, Verbeke W, De Backer G. Adolescents' education and their diet recorded by 7-day food records. *Sozial-und Pravent*. 2006;51(2):80-90.

17. Utter J, Scragg R, Mhurchu CN, Schaaf D. At-home breakfast consumption among New Zealand children: associations with body mass index and related nutrition behaviors. *J Am Diet Assoc*. 2007;107(4):570-6.

18. Lubans DR, Morgan PJ, Dewar D, Collins CE, Plotnikoff RC, Okely AD, *et al.* The Nutrition and Enjoyable Activity for Teen Girls (NEAT girls) randomized controlled trial for adolescent girls from disadvantaged secondary schools: rationale, study protocol, and baseline results. *BMC Public Health*. 2010;10:652.

19. Lubans DR, Morgan PJ, Okely AD, Dewar D, Collins CE, Batterham M, *et al.* Preventing Obesity Among Adolescent Girls: One-Year Outcomes of the Nutrition and Enjoyable Activity for Teen Girls (NEAT Girls) Cluster Randomized Controlled Trial. *Arch Pediatr Adolesc Med*. 2012;166(9):821-7.

20. NSW Labour Economics Office. *Socio-Economic Indexes for Areas* Sydney, Australia: Commonwealth of Australia; 2008 [updated November, 2008; cited 2011 October 11, 2011]. Available from: http://www.skillsinfo.gov.au/NR/rdonlyres/F57F6EB7-339A-41CF-8AF3-28A6ACF194DD/0/NSWSEIFA.pdf.

21. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ*. 2000;320(7244):1240-3.

22. Cole TJ, Flegal KM, Nicholls D, Jackson AA. Body mass index cut offs to define thinness in children and adolescents: international survey. *BMJ*. 2007;335(7612):194.

23. Kuczmarski R, Ogden C, Guo S. 2000 CDC Growth Charts for the United States: Methods and Development. Washington, DC.: National Center for Health Statistics; 2002.

24. Dewar DL, Morgan PJ, Plotnikoff RC, Okely AD, Batterham M, Lubans DR. Exploring changes in physical activity, sedentary behaviors and hypothesized mediators in the NEAT girls group randomized controlled trial. *J Sci Med Sport*. 2014;17(1):39-46.

25. Watson JF, Collins CE, Sibbritt DW, Dibley MJ, Garg ML. Reproducibility and comparative validity of a food frequency questionnaire for Australian children and adolescents. *Int J Behav Nutr Phys Act*. 2009;6:62.

26. Marshall S, Watson J, Burrows T, Guest M, Collins CE. The development and evaluation of the Australian child and adolescent recommended food score: a cross-sectional study. *Nutr J*. 2012;11:96.

27. Field AE, Austin SB, Gillman MW, Rosner B, Rockett HR, Colditz GA. Snack food intake does not predict weight change among children and adolescents. *Int J Obes Relat Metab Disord*. 2004;28(10):1210-6.

28. Field AE, Gillman MW, Rosner B, Rockett HR, Colditz GA. Association between fruit and vegetable intake and change in body mass index among a large sample of children and adolescents in the United States. *Int J Obes Relat Metab Disord*. 2003;27(7):821-6.

29. Kellet E, Smith A, Schmerlaib Y. *The Australian Guide to Healthy Eating. Background information for Consumers.* Canberra, Australia: Commonwealth of Australia; 1998.

30. Rangan AM, Kwan JS, Louie JC, Flood VM, Gill TP. Changes in core food intake among Australian children between 1995 and 2007. *Eur J Clin Nutr*. 2011;65(11):1201-10.

31. Savige GS, Ball K, Worsley A, Crawford D. Food intake patterns among Australian adolescents. *Asia Pac J Clin Nutr*. 2007;16(4):738-47.

32. Scully M, Dixon H, White V, Beckmann K. Dietary, physical activity and sedentary behaviour among Australian secondary students in 2005. *Health Promot Int*. 2007;22(3):236-45.

33. Cancer Council Australia, National Heart Foundation. Prevalence of Meeting Healthy Eating Guidelines in Australian Secondary Students: Cancer Council Australia; 2011 [updated 31 March, 2011; cited 2011 14 September]. Available from: http://www.cancer.org.au/File/NewsMedia/Media%20materials/Dietary_habits_research_memo_NaSSDA_updated.pdf.

34. CSIRO, University of South Australia. 2007 Australian National Children's Nutrition and Physical Activity Survey - Main Findings. In: Commonwealth Scientific Industrial Reseach Organisation Preventative Health National Research Flagship, Department of Health and Ageing, editors. Canberra: Commonwealth of Australia; 2008. p. 1-58.

35. Neumark-Sztainer D, Story M, Hannan PJ, Croll J. Overweight status and eating patterns among adolescents: where do youths stand in comparison with the healthy people 2010 objectives? *Am J Public Health*. 2002;92(5):844-51.

36. Niemeier HM, Raynor HA, Lloyd-Richardson EE, Rogers ML, Wing RR. Fast food consumption and breakfast skipping: predictors of weight gain from adolescence to adulthood in a nationally representative sample. *J Adolesc Health*. 2006;39(6):842-9.

37. Hart CN, Jelalian E, Raynor HA, Mehlenbeck R, Lloyd-Richardson EE, Kaplan J, *et al.* Early patterns of food intake in an adolescent weight loss trial as predictors of BMI change. *Eat Behav*. 2010;11(4):217-22.

38. Berenson GS, Srinivasan SR, Nicklas TA. Atherosclerosis: a nutritional disease of childhood. *Am J Cardiol*. 1998;82(10B):22T-9T.

39. Plotnikoff RC, Karunamuni N, Spence JC, Storey K, Forbes L, Raine K, *et al.* Chronic disease-related lifestyle risk factors in a sample of Canadian adolescents. *J Adolesc Health*. 2009;44(6):606-9.

40. Rangan AM, Flood VM, Gill TP. Misreporting of energy intake in the 2007 Australian Children's Survey: identification, characteristics and impact of misreporters. *Nutrients*. 2011;3(2):186-99.

41. Scagliusi FB, Ferriolli E, Pfrimer K, Laureano C, Cunha CS, Gualano B, *et al.* Characteristics of women who frequently under report their energy intake: a doubly labelled water study. *Eur J Clin Nutr*. 2009;63(10):1192-9.

Chapter 6: Feasibility of Recruiting Families into a Heart Disease Prevention Program Based on Dietary Patterns

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6.1 Overview

This chapter reports the feasibility of recruiting families who may be at increased risk of CVD to a dietary intervention. Families with a demonstrated history of CVD were of interest as they offer an opportunity for both primary and secondary prevention. The intervention provided dietary advice found to be efficacious in the literature review and was informed by findings in the systematic review. Documents required for ethics approval for this intervention can been seen from Appendix 9 to Appendix 17. Resources developed for this intervention can be seen in Appendix 18 and Appendix 19.

6.2 Abstract

Offspring of parents with a history of cardiovascular disease (CVD) inherit a similar genetic profile and share diet and lifestyle behaviors. This study aimed to evaluate the feasibility of recruiting families at risk of CVD to a dietary prevention program, determine the changes in diet achieved, and program acceptability. Families were recruited into a pilot parallel group randomized controlled trial consisting of a three month evidence-based dietary intervention study, based on the Mediterranean and Portfolio diets. Feasibility was assessed by recruitment and retention rates, change in diet by food frequency questionnaire, and program acceptability by qualitative interviews and program evaluation. Twenty one families were enrolled over 16 months, with fourteen families (n = 42 individuals) completing the study. Post-program dietary changes in the intervention group included small daily increases in vegetable serves (0.8 ± 1.3) and reduced usage of full-fat milk (−21%), cheese (−12%) and meat products (−17%). Qualitative interviews highlighted beneficial changes in food purchasing habits. Future studies need more effective methods of recruitment to engage families in the intervention. Once engaged, families made small incremental improvements in their diets. Evaluation indicated that feedback on diet and CVD risk factors, dietetic counselling and the resources provided were appropriate for a program of this type.

6.3 Introduction

The World Health Organization reported that 17.5 million deaths were attributed to cardiovascular disease (CVD) in 2012 (1). CVD risk factors include non-modifiable and modifiable factors, including genetic predisposition, metabolic conditions and lifestyle behaviors (2). Offspring of parents with CVD are at increased risk due to shared genetic profiles and lifestyle behaviors (3). In the Framingham Study, offspring with at least one parent with premature CVD had an increased age-adjusted risk of 2.3-2.6 (odds ratio) of developing CVD (4). The Bogalusa study found that children with at least one parent with coronary artery disease (CAD) had a higher mean body mass index (1.22kg.m^{-2}), total and LDL cholesterol (0.11mmol.L^{-1}, 0.14mmol.L^{-1}) and higher systolic blood pressure (1.63mmHg) compared with children with no parental CAD (5).

Dietary patterns are an important lifestyle factor influencing the development of CVD (6, 7). Dietary patterns and eating habits are fostered within families, with younger family members modelling consumption patterns of older family members (3, 8). The Mediterranean diet is an eating pattern associated with lower risk of CVD (6), and the Portfolio diet has been shown to be efficacious in lowering CVD risk factors such as the ratio of total cholesterol to HDL cholesterol and serum triglycerides (9, 10). The Mediterranean diet is high in vegetables, fruit, nuts and olive oil with moderate intakes of fish (6); the Portfolio diet combines foods with lipid-lowering efficacy such as soluble fibers, plant sterols and nuts (7). Both these food-group based dietary patterns have been shown to be more effective in improving lipid profiles than diets that emphasize specific nutrient intakes such as low fat or cholesterol diets (11, 12) and these food based approaches form the basis of current dietary guidelines for CVD prevention (13).

Families recruited into a dietary intervention study on the basis of one member having had an adverse CVD event, or being assessed as at high risk of CVD, may be more receptive to changing their diet. To test this hypothesis, the current study investigated: 1) the feasibility of recruiting and retaining families at increased risk of CVD into a dietary intervention program targeting alignment of existing eating

patterns with heart health recommendations, 2) the dietary changes made, and 3) the acceptability of the dietary CVD prevention program.

6.4 Experimental Section

Families were eligible to participate in the "Love your Food, Love your Heart, Love your Family" (FHF) study if at least one member (index recruit aged 18-70 years) had experienced an adverse CVD event or was classified as being at moderate-to-high risk using Australian cardiovascular risk charts (aged 18-80 years) (14), had no other medical conditions affecting dietary intake, and had internet access. Written informed consent was obtained from all family members, with those <18 years giving assent and having parental consent. Ethics approval was obtained from University of Newcastle Human Research Ethics Committee (H-2012-0246) and the Hunter New England Human Research Ethics Committee (HREC/12/HNE/140).

6.4.1 Study Design

The Protection Motivation Theory (15) proposes that health behaviors are the result of coping responses to perceived threats of vulnerability and severity. The theory posits that individuals look at the perceived benefits and usefulness of performing adaptive (helpful) behaviors, and their confidence to perform them. Therefore, the intervention sought to capitalize on the awareness of personal risk, incorporate strategies to build self-efficacy for helpful tasks, and give meaningful feedback on performing the recommended strategies.

The study was a pilot parallel group randomized controlled trial. Participating families were stratified by sex of the index member, CVD event (stroke/ischemic heart condition) and time of event (≤ 6months, >6 months) and randomized in blocks of six using QuickCalcs Software (16) to either the three-month intervention or feedback only control group, with assessors blinded to group allocation. Subjects received group assignment via the next available sealed envelope within their stratification.

Recruitment, baseline and follow-up assessments took place from December 2012 to May 2014. The intervention flow is summarized in Figure 6.1. After providing consent, families completed online questionnaires on demographics, medical history, smoking, and usual eating patterns. Fasting blood samples were analyzed for blood lipids prior to anthropometric assessments, and all individuals received a personalized feedback booklet containing lipid test results, anthropometric measures, and dietary intake analysis including macronutrient and micronutrient intakes and the percentage energy contributed by core (nutrient dense) and discretionary (energy-dense, nutrient-poor) foods. Randomization into intervention or control groups (feedback only) followed provision of the feedback booklet with those in the control group wait-listed for 3 months. Intervention group participants each received one 45-minute dietary counselling session with an Accredited Practising Dietitian (APD). To ensure consistency of the intervention delivery, a resource booklet specific to the intervention and a semi-structured education session for the counselling were used, which allowed for modification of strategies to cater for families' unique needs. Participants were asked to increase their intake of specific foods to more closely align with targets. The dietary intake targets used in the current intervention included: up to two serves (60g) of nuts per day; 2-3 daily serves (2-3g) of plant sterols; up to five daily serves (15g) of soluble fibers; up to seven daily (42g) serves of soy proteins; 2-3 serves of fish per week (170-450g, dependent on fish type); up to seven serves (approximately 650g) of legumes/pulses/lentils per week. Unsaturated fats were promoted whilst reducing saturated fats, as well as low-sodium food choices and general healthy eating guidelines (17).

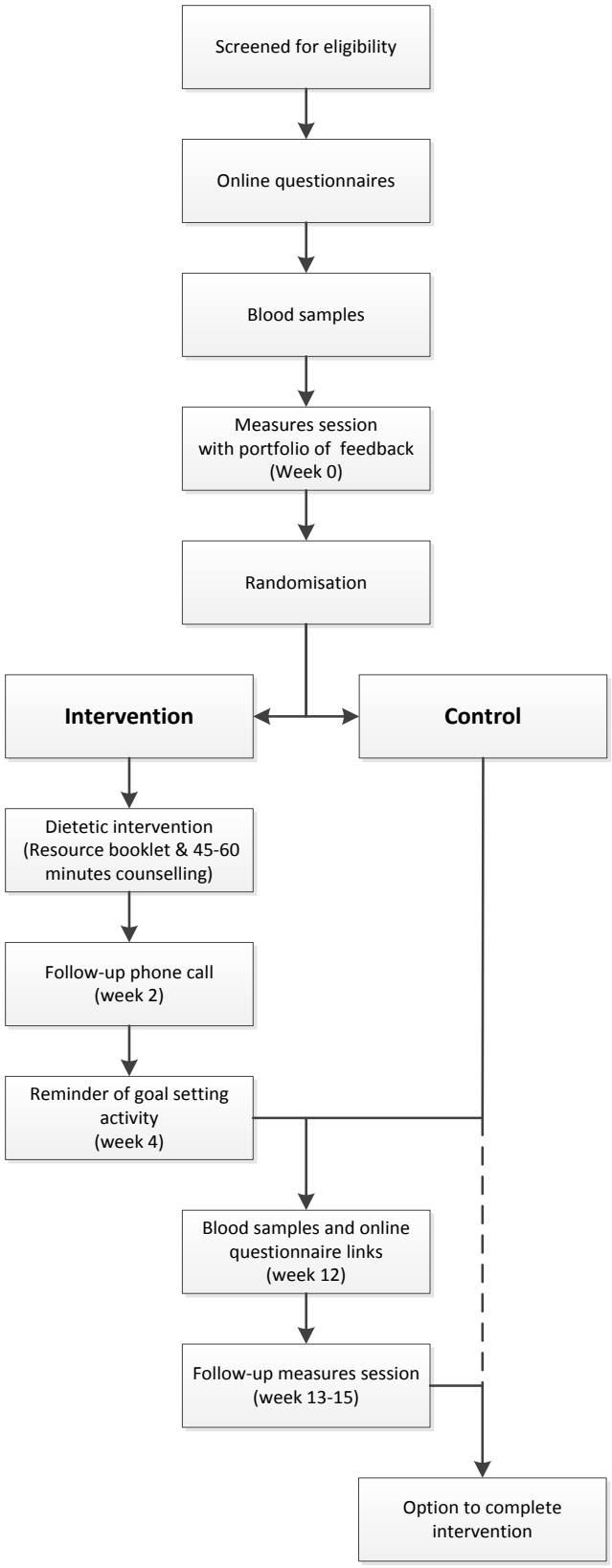

Figure 6.1: Intervention flow chart for study participants. Families randomized to the control group had the option of undergoing the intervention once the control period was completed

Dietary intake was assessed as frequency of usual intake over the past six months using the validated Australian Eating Survey (≥18 years) (AES) and Australian Child and Adolescent Eating Survey (ACAES), 120 item semi-quantitative food frequency questionnaires (FFQs) (18, 19). Nutrient intakes were computed using FoodWorks version 4.00.1158 (20) and the Australian AusNut 1999 nutrient database (All Foods, Revision 17) and AusFoods (Brands, Revision 5). Foods specific to the Mediterranean (6) and Portfolio diets (7) not included within the AES and ACAES were measured using a 72 question semi-quantitative FFQ that was specifically developed to assess intake of plant sterols, viscous fibers, soy proteins and provide specific details about fat type, sodium, legumes, nuts and fish intakes. The study-specific FFQ was comprised of 18 food habit questions and 35 intake questions with stated serve sizes aligned to the Australian Dietary Guidelines (21), Heart Foundation recommendations (22), or as natural portion sizes and 19 questions with portion size stated as "1 serving".

6.4.2 Qualitative measures to assess feasibility, dietary changes and program acceptability

All adult family members completing the intervention were invited by post to participate in a semi-structured telephone interview. Areas of enquiry explored by interview included motivation to participate, barriers to healthy eating and dietary changes made. Individuals were interviewed by a female research team member (TS) 1-6 months (mean 1.5 months) post completion using a semi-structured discussion framework developed by the researchers. Probes and prompts were used to expand and clarify responses. The interviews were digitally recorded with the participants' consent and transcribed verbatim. A computer program (NVIVO 10) was used to assist with the organizational aspects of data analysis. Qualitative analyses were conducted by an independent experienced qualitative researcher who was not part of the research team to reduce bias and ensure accuracy of themes identified. All index participants were asked to complete a process evaluation questionnaire after completing the three-month intervention. Questions were in regards to the suggested foods and eating patterns, resources provided, changes in behavior and general feedback.

6.4.3 Other CVD related health measures

Participants' height and weight were measured in light clothing to 0.1cm and 0.1kg, respectively using the Biospace BSM370 Automatic BMI Scale Stadiometer (Biospace Co. Ltd, Korea). Waist circumference was measured at the narrowest point between the lower costal (10^{th} rib) border and the top of the iliac crest using a non-extensible steel tape (KDFS10-02, KDS Corporation, Osaka, Japan). Brachial and central blood pressure and arterial stiffness measures (augmentation index) were obtained with the Pulsecor Cardioscope II (Pulsecor Ltd., New Zealand) using WelchAllyn FlexiPort reusable blood pressure cuffs. Participants were seated for five minutes before the first measurement occurred, and repeat measures were taken at two-minute intervals. Participants under the age of 18 years were also provided with a familiarization trial measure to reduce potential anxiety associated with this measurement. Physical activity in adults was assessed using the International Physical Activity Questionnaire long form (IPAQ) for the previous seven days.

Blood samples were assayed for markers of insulin resistance, inflammation and blood lipid concentrations (see Table 6.1) from adult family members after an overnight fast by trained phlebotomists and analyzed at a single accredited (National Association of Testing Authorities) pathology service.

Recruitment and retention data were measured as those enrolling and completing the intervention and by qualitative interview. Changes in dietary intakes, as measured by FFQ, are presented as mean ± standard deviation for normally distributed data and median (p25-p75) for non-normal data. An intention-to-treat analysis was used with last observation carried forward for missing data. As this was a feasibility trial, power calculations were not performed. Dietary intake themes from qualitative interviews were reported. Results for acceptability of the prevention program are summarized from program evaluation questionnaires.

Table 6.1: Baseline characteristics of the Love your food, Love your heart, Love your family study participants, inclusive of 15 families, presented as mean ± standard deviation, except where indicated.

	Children (<18 years)	Adults		All adults
	100% Female (*n* = 3)	Males (*n* = 20)	Females (*n* = 21)	(*n* = 41, 100%)
Age (years) median (p25–p75)	12.9 (7.9–16.7)	59.4 (46.0–67.8)	56.6 (42.7–64.0)	59.0 (42.7–66.5)
Height (cm)	151.1 ± 22.0	174.8 ± 6.1	162.8 ± 6.2	168.6 ± 8.6
Weight (kg)	41.9 ± 15.1	87.7 ± 15.6	75.9 ± 18.9	81.6 ± 18.2
BMI (kg/m^2)	17.8 ± 2.1	28.7 ± 5.1	28.6 ± 7.2	28.7 ± 6.2
Waist (cm)	60.7 ± 6.5	99.5 ± 15.1	86.9 ± 12.2	93.1 ± 14.9
Brachial BP (mmHg)				
systolic	100.3 ± 7.4 [a]	121.3 ± 15.6	114.3 ± 15.0	117.7 ± 15.5
diastolic	63.5 ± 7.1 [a]	71.8 ± 6.7	71.7 ± 7.8	71.7 ± 7.2
Central BP (mmHg)				
systolic	91.8 ± 7.4 [a]	114.1 ± 16.2	109.0 ± 14.7	111.5 ± 15.5
diastolic	65.3 ± 6.0 [a]	73.5 ± 7.0	72.8 ± 7.9	73.1 ± 7.4
Arterial stiffness	43 ± 11 [a]	75 ± 34	86 ± 35	81 ± 35
Level of physical activity [b]				
Low	N/A	10% (*n* = 2)	19% (*n* = 4)	15% (*n* = 6)
Moderate	N/A	60% (*n* = 12)	62% (*n* = 13)	61% (*n* = 25)
High	N/A	30% (*n* = 6)	19% (*n* = 4)	24% (*n* = 10)
Smoking status				
Current	N/A	*n* = 2 (10%)	*n* = 0 (0%)	*n* = 2 (5%)
Previous	N/A	*n* = 4 (20%)	*n* = 7 (33%)	*n* = 11 (27%)
Blood biomarkers				
Triglycerides (mmol/L)	N/A	1.3 ± 0.6	1.4 ± 0.9	1.3 ± 0.8
TC (mmol/L)	N/A	4.5 ± 1.1	5.3 ± 1.1	4.9 ± 1.2
LDL (mmol/L)	N/A	2.7 ± 1.1	3.3 ± 1.1	3.0 ± 1.1
HDL (mmol/L)	N/A	1.2 ± 0.3	1.5 ± 0.3	1.3 ± 0.4
Total: HDL ratio	N/A	3.9 ± 1.5	3.8 ± 1.0	3.9 ± 1.2
BGL (mmol/L)	N/A	5.0 ± 0.5	5.2 ± 0.8	5.1 ± 0.6
Insulin (IU/L)	N/A	8.8 ± 4.6	7.0 ± 3.4	7.9 ± 4.1
hsCRP (mg/L)	N/A	3.0 ± 3.4	2.4 ± 2.4	2.7 ± 2.9
ALT (U/L)	N/A	32.6 ± 10.8	22.5 ± 16.1	27.4 ± 14.5
AST (U/L)	N/A	30.0 ± 8.4	27.4 ± 25.8	28.7 ± 19.2
GGT (U/L)	N/A	28.0 ± 16.4	25.7 ± 25.6	26.8 ± 21.4

Abbreviations: BMI—Body mass index; Waist—waist circumference; BP—blood pressure; TC—total cholesterol; LDL—LDL cholesterol; HDL—HDL cholesterol; BGL—blood glucose level; hsCRP—high sensitivity C-Reactive Protein; [a] *n* = 2; [b] As categorized by the International Physical Activity Questionnaire

6.5 Results

6.5.1 Study participants

Twenty-one index participants enrolled with their families, totalling 59 participants across three generations. Fifteen families were retained until randomization, consisting of 41 adults and three children (Figure 6.2). Of the 39 adults who completed the main study, 16 adults from eight families (41%) plus one child who turned 18 during the study participated in qualitative interviews (age range 18-70 years, 47% male). Five index participants were interviewed and one other had a diagnosed CVD condition. The interviews indicated participant motivations to join the study included a long-term interest in improving diet, a desire to make positive changes in eating habits and health for self and extended family, and having existing heart health issues. Individual participants identified a key family member who drove their family's involvement, who was not necessarily the person with a CVD diagnosis.

Characteristics of the participants are summarized in Table 6.1. Sixteen participants (39%) reported knowing they had elevated serum cholesterol levels, with 18 (44%) taking lipid lowering medication. Twelve reported having high blood pressure (29%), with 17 (41%) on medication for this condition. Twelve (29%) had arthritis, with six taking medication (15%) and one had type 2 diabetes (medicated). Eleven of the 15 index recruits had experienced a prior CVD event; nine had been advised to attend cardiac rehabilitation, with seven having attending.

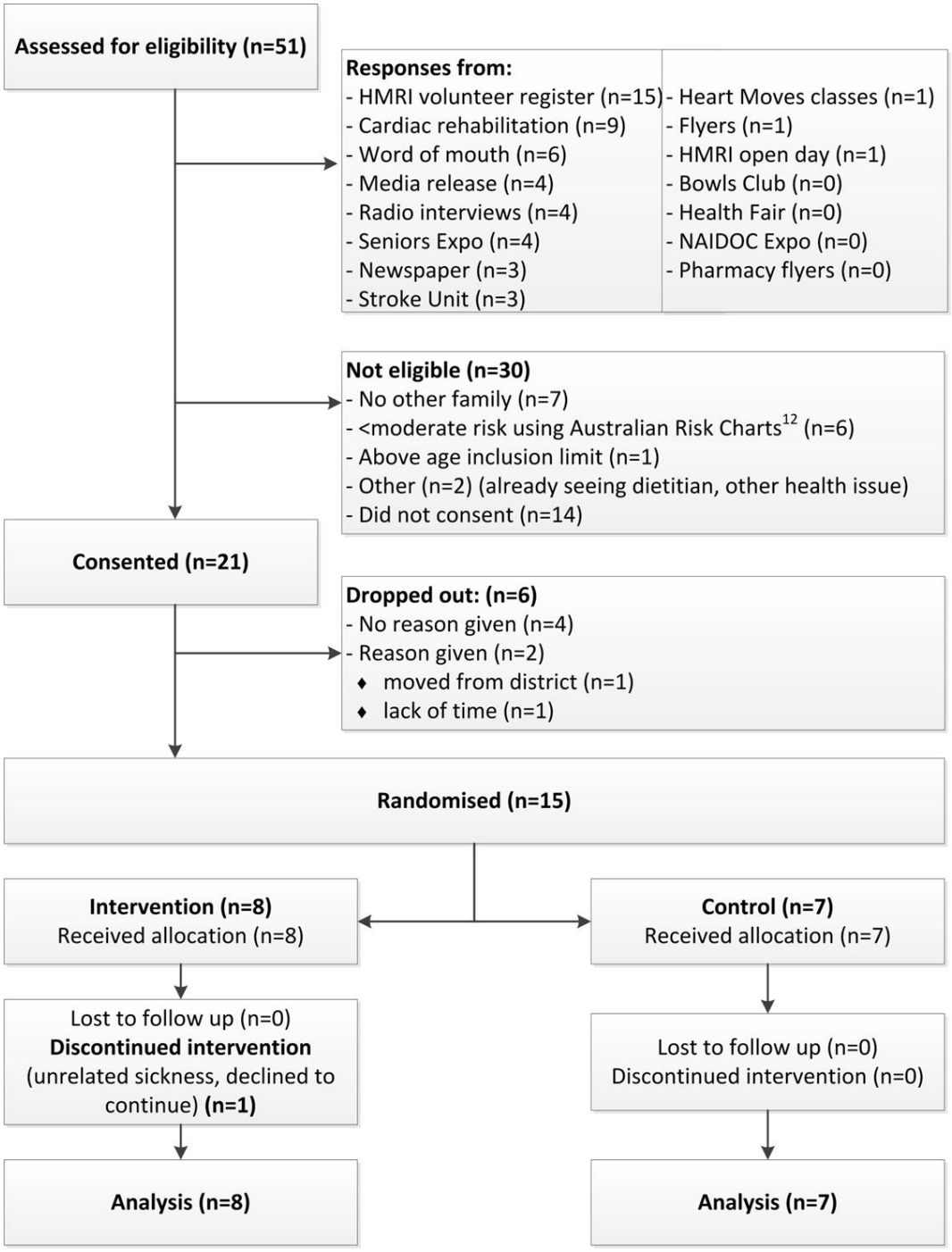

Figure 6.2: Flow chart showing the recruitment strategies used and number of participants assessed for eligibility and study retention.

6.5.2 Feasibility of recruiting and retaining families

Recruitment using a variety of methods (Figure 6.2) resulted in 51 index participants being assessed for eligibility over 16 months. Of 51 inquiries, 16 were not eligible and 14 did not return consent forms with the majority of those not returning consent forms recruited from cardiac rehabilitation and stroke units (n=6) (Figure 6.2). Highest enrolment rates came from word-of-mouth (50%). Retention rates were highest (9 eligible, 9 consented, 7 completions) among those recruited from the Hunter Medical Research Institute volunteer register and media releases, and lowest among those recruited from cardiac rehabilitation classes or stroke units (11 eligible, 5 consents, 2 completions).

6.5.3 Nature and extent of dietary changes made

Baseline dietary intakes indicate that 63±10% of energy came from nutritious, low energy-density (core) foods and 37±10% from energy-dense, nutrient-poor (discretionary) foods. There was no difference in reported total energy intake at baseline between the adults or children completing the study and those who did not ($p=0.34$ and $p=0.32$ respectively). Analysis of dietary intakes and key components of the Mediterranean and Portfolio Diets are summarized in Table 6.2. Mean time between baseline and follow up was 4.5 months (±1.1). Table 6.3 summarizes foods habits relating to CVD health and highlights reductions in full-fat types of dairy and meat products usually eaten in the intervention group. Results at three-month follow up indicate that both groups made changes to their dietary intakes. The proportion of energy from core food groups showed improvement, as did daily vegetable intakes (Table 6.1).

Table 6.2: Baseline and follow-up dietary intakes as assessed by the Australian Eating Survey (AES), Australian Child and Adolescent Eating Survey (ACAES) and additional food frequency questionnaire assessing foods specific to the Mediterranean and Portfolio diets. Data presented as mean ± standard deviation for 15 families.

	Baseline				Follow up			
	Children	Adults			Children	Adults		
		Adults	C	I		Adults	C	I
	(*n* = 3)	(*n* = 41)	(*n* = 18)	(*n* = 23)	(*n* = 3)	(*n* = 41)	(*n* = 18)	(*n* = 23)
Energy (kJ)	9967 ± 1619	10108 ± 2873	9582 ± 3197	10520 ± 2589	9565 ± 1942	9604 ± 2661	9253 ± 2878	9878 ± 2509
PE core foods (%)	57 ± 23	63 ± 10	66 ± 14	61 ± 6	63 ± 7	66 ± 11	68 ± 13	64 ± 9
PE discretionary foods (%)	43 ± 23	37 ± 10	34 ± 14	39 ± 6	37 ± 7	34 ± 11	32 ± 13	36 ± 9
PE protein (%)	20 ± 8	19 ± 4	21 ± 5	18 ± 2	20 ± 3	19 ± 3	21 ± 4	18 ± 2
PE CHO (%)	43 ± 8	44 ± 8	40 ± 8	47 ± 5	46 ± 8	43 ± 7	39 ± 7	47 ± 6
PE fats (%)	38 ± 1	33 ± 4	34 ± 4	32 ± 4	35 ± 7	33 ± 5	35 ± 6	31 ± 3
PE sat. fats (%)	18 ± 2	13 ± 2	14 ± 2	13 ± 2	15 ± 4	13 ± 2	14 ± 2	13 ± 2
Fiber (g)	25 ± 8	29 ± 11	28 ± 13	30 ± 8	27 ± 9	30 ± 9	27 ± 8	32 ± 9
Sodium (mg)	2067 ± 402	2321 ± 679	2197 ± 614	2418 ± 724	2067 ± 244	2259 ± 671	2275 ± 715	2246 ± 650
Fruit/day	2.0 ± 1.1	2.0. ± 1.7	1.8 ± 2.1	2.2 ± 1.3	2.4 ± 1.6	1.9 ± 1.1	1.5 ± 0.7	2.2 ± 1.2
Vegetables/day	4.6 ± 1.2	4.9 ± 2.1	5.2 ± 2.2	4.7 ± 2.1	4.3 ± 0.4	5.5 ± 1.7	5.6 ± 1.7	5.4 ± 1.7
ARFS [a]	28 ± 8	35 ± 10	35 ± 8	35 ± 12	29 ± 6	39 ± 9	37 ± 5	39 ± 9

Table 6.2: Baseline and follow-up dietary intakes as assessed by the Australian Eating Survey (AES), Australian Child and Adolescent Eating Survey (ACAES) and additional food frequency questionnaire assessing foods specific to the Mediterranean and Portfolio diets. Data presented as mean ± standard deviation for 15 families.

	Baseline				Follow up			
	Children	Adults			Children	Adults		
		Adults	C	I		Adults	C	I
	(*n* = 3)	(*n* = 41)	(*n* = 18)	(*n* = 23)	(*n* = 3)	(*n* = 41)	(*n* = 18)	(*n* = 23)
Frequency of foods specific to cardiovascular health (number of serves per day)								
FAT FROM ADDED SOURCES								
Saturated	N/A	0.3 ± 0.5	0.3 ± 0.7	0.2 ± 0.3	N/A	0.3 ± 0.7	0.5 ± 0.9	0.2 ± 0.3
Unsaturated	N/A	1.3 ± 0.8	1.4 ± 0.9	1.1 ± 0.8	N/A	1.2 ± 0.8	1.3 ± 0.9	1.1 ± 0.8
Nuts	0.2 ± 0.3	0.5 ± 0.6	0.5 ± 0.6	0.5 ± 0.5	1.1 ± 1.6	1.0 ± 1.1	0.9 ± 0.9	1.1 ± 1.2
Fish	0.3 ± 0.4	0.4 ± 0.5	0.5 ± 0.6	0.3 ± 0.3	0.5 ± 0.7	0.4 ± 0.3	0.5 ± 0.4	0.4 ± 0.3
Soy proteins	0.4 ± 0.7	0.1 ± 0.3	0.2 ± 0.4	0.1 ± 0.2	0.2 ± 0.3	0.2 ± 0.4	0.1 ± 0.3	0.3 ± 0.4
Legumes	0.1 ± 0.1	0.3 ± 0.4	0.3 ± 0.3	0.2 ± 0.5	1.2 ± 1.9	0.5 ± 0.7	0.7 ± 0.9	0.4 ± 0.5
Viscous fibers	0.2 ± 0.2	0.7 ± 0.7	0.7 ± 0.8	0.6 ± 0.6	0.3 ± 0.5	0.7 ± 0.7	0.4 ± 0.6	0.9 ± 0.8
Plant sterols	N/A	0.9 ± 1.1	1.2 ± 1.4	0.6 ± 0.9	N/A	0.7 ± 1.0	0.3 ± 0.7	1.0 ± 1.2

Abbreviations: C—Control group; I—Intervention group; N/A—Not assessed; PE—Percentage energy; ARFS—Australian Recommended Food Score; [a] measure of dietary variety.

Table 6.3: Reported eating habits of foods related to cardiovascular disease (CVD) health.

	Baseline			Follow up		
	All (*n* = 44)	**C (*n* = 20)**	**I (*n* = 24)**	**All (*n* = 44)**	**C (*n* = 20)**	**I (*n* = 24)**
Type of milk normally consumed						
Don't drink milk	7%	0%	13%	7%	0%	13%
Normal	25%	15%	33%	14%	15%	13%
Reduced fat	32%	30%	33%	43%	40%	46%
Skim	32%	50%	17%	34%	40%	29%
Other	5%	5%	4%	2%	5%	0%
Type of cheese normally eaten						
Don't eat this	7%	5%	8%	7%	5%	8%
Normal	55%	40%	67%	45%	45%	46%
Reduced fat	34%	55%	17%	32%	30%	33%
Low fat	2%	0%	4%	14%	15%	12%
Not sure	2%	0%	4%	2%	5%	0%
Type of meat						
Don't eat this	2%	5%	0%	5%	10%	0%
Normal	55%	65%	46%	45%	65%	29%
Reduced fat	30%	10%	46%	39%	15%	58%
Low fat	11%	15%	8%	11%	10%	13%
Not sure	2%	5%	0%	0%	0%	0%
Type of chicken						
Don't eat this	7%	5%	8%	7%	5%	8%
Fried	2%	0%	4%	0%	0%	0%
Crumbed	9%	5%	13%	7%	10%	4%
With skin	32%	40%	25%	25%	25%	25%
Skin removed	45%	45%	46%	55%	55%	54%
Not sure	5%	5%	4%	7%	5%	8%
Adding of salt to food						
Never add salt	25%	25%	25%	16%	15%	17%
During cooking	32%	35%	29%	36%	45%	29%
To meals	27%	30%	25%	25%	15%	33%
Both meals & cooking	14%	5%	21%	20%	20%	21%
Not sure	2%	5%	0%	2%	5%	0%

Table 6.3: Reported eating habits of foods related to cardiovascular disease (CVD) health.

	Baseline			Follow up		
	All (*n* = 44)	**C (*n* = 20)**	**I (*n* = 24)**	**All (*n* = 44)**	**C (*n* = 20)**	**I (*n* = 24)**
Take away per week *						
None	9%	15%	4%	11%	15%	8%
<once per week	48%	45%	50%	57%	50%	62%
1–2 per week	36%	25%	46%	30%	30%	29%
3–4 per week	7%	15%	0%	2%	5%	0%
>4 per week	0%	0	0%	0%	0%	0%

Abbreviations: C—Control group; I—Intervention group; * Take away described as chinese, fish and chips, hamburgers and chips/fries, pizza.

Results from the qualitative interviews indicate that prior to program involvement 14 of 17 participants rated the healthiness of their diet subjectively as 6-7 on an alpha-numeric rating scale, where 10 represents the most healthy. Only two participants rated their pre-study diets as below average at three out of 10. Participants appeared to use a cognitive balancing of 'good' versus 'bad' aspects of their diet to justify their ratings of their usual intake pre-study. Dietary habits they acknowledged as reducing their 'healthiness rating' included the consumption of fatty meats, low vegetable intakes, and snacking on sugary foods between meals. Dietary habits perceived as increasing their 'healthiness rating' were cutting back on red meat by eating chicken and fish, and exercising 'dietary moderation' described as 'nothing in excess'. These habits were perceived by some as making their diet healthier relative to a subjective 'average' to which they mentally compared their intakes. Although almost a third of participants acknowledged little change to the healthiness of their diet post-study due to persistence of major barriers (e.g. partner reluctance, personal preference and taste), the majority reported having made permanent changes to their dietary intake and food habits. Some households reported a subjective rating improvement of 2-3 out of 10 post-program participation, suggesting substantial changes were made.

These improvements were attributed to increased knowledge and awareness due to program participation and appeared to inspire greater experimentation with healthier options and purchasing of foods reflecting increased variety and nutritional quality.

"I decided I would make a lovely rice dish, and I put in some slivered almonds and a couple of herbs and some garlic and it was lovely, and a little bit of soy sauce...I think the main thing is, after this study, was just variety. Like, if I was to make a rice dish before that I wouldn't have thought to add in nuts."

Further examples of dietary improvements given were less impulsive food shopping, more variety in fruit and vegetable selection, lower sugar and fat options, use of legumes, lentils and soy products, healthier meat options, and elimination of energy-dense, nutrient-poor foods. For all participants, including those reporting little or no change in their diet post-study, involvement in the project appeared to have increased awareness of the different components of their diet. Examples given included the proportion of energy from discretionary foods, foods with a healthy heart tick, the healthiness of different types of fat, an increased awareness of processed foods and the importance of small changes. Indeed, one participant who only reported slight changes in his diet following the study described the cumulative impact of these small changes as evidence of a shift in his food behaviors and preferences;

"I just cut out more of the bad stuff, like I'm sort of thinking it was only marginal changes I made. Look when I ate poorly like snacks and things like that, I'd probably eat too much. Whereas when I have a snack or a treat now, actually I find that I can't eat as much anyway of it. I think my taste buds have changed a little bit. But again from the converse side of things, previously when I probably didn't eat as much good food. I'm eating more good food now...It's just those marginal shifts."

6.5.4 Acceptability of program to align current eating patterns with recommendations

Eleven of the 15 index participants (73%) returned program evaluation forms. These participants all agreed or strongly agreed that this type of diet was relevant to them, but they had mixed responses regarding the ease of integration into their lives (55% positive, 18% negative, 27% neutral). Six (55%) felt it impacted negatively on grocery costs. Ten participants (91%) agreed or strongly agreed they would recommend this type of eating pattern to people in a similar situation (n=1 neutral). Ten (91%) found the resource booklet easy to read and the information easy to understand, with the remainder (n=1) answering neutral to both questions.

Nine of the eleven participants (82%) read the booklet 2-3 or ≥3 times, with two participants (18%) reading it once. The individualized feedback booklets were similarly valued with eight participants (73%) reading it 2-3 or ≥3 times and three participants (27%) reading it once.

6.6 Discussion

The current study investigated whether families could be recruited and retained in a family CVD prevention program that was based on the Mediterranean and Portfolio eating patterns. Recruitment was challenging, with only 15 of 35 eligible families who initially expressed interest, engaging with the study through to the randomization stage. However, once randomized, the majority of these families completed the intervention. Those responding to media releases about the study and volunteer register invitations were more likely to be retained. Of interest is that amongst those families completing the trial, a key family member was found to drive the involvement and retention of the family. While overall dietary patterns were unaltered, participants made small, but incremental dietary changes, such as reducing discretionary foods and selecting fat-reduced versions of milk and cheese and fat-trimmed meats. Participants reported an increased awareness of their food habits and knowledge of food following the personalized dietary counselling they received from the study dietitian about their usual food and nutrient intakes. Evaluation of the program found that although participants noted some negatives, such as increases in grocery costs, these may have been offset by reductions in costs associated with takeaway foods. Evaluation of food costs in future studies is required. Participants used the resources and dietary feedback provided on multiple occasions and reported they would recommend the program to others in a similar situation.

A clear barrier to recruitment occurred between confirming eligibility of the index participant and the returning of consent forms from the family group, as shown by the limited number of returned consents (n=14) at this stage. This suggests that persuading a family member to participate was a substantial barrier. An additional seven interested participants were deemed ineligible because they could not identify a family member to accompany them in the study. A larger Canadian

family-based study had a similar focus, but recruited at-risk family members (n=426) through in-patients from a tertiary care cardiac center (23). While this study was able to randomize a greater proportion of their eligible participants, it had a 26% loss to follow-up. Recruiting using these methods may capitalize on a teachable moment, and lead to a change in lifestyle intentions (24), but does not necessarily imply a willingness to make permanent lifestyle changes amongst family members.

The lack of perceived CVD risk amongst those with actual increased risk is a significant barrier to program uptake as identified in the current study. The Protection Motivation Theory, on which the current study is based, identifies that a perception of risk must be present before any change in behavior can occur (15). However, risk of CVD events is often poorly perceived by those with a confirmed family history of CVD, and may not be sufficient to change or act on intentions suggesting other motivators are required (25). In the current study, some individuals lacked understanding of their medical risk factors, evidenced by the large proportion who were currently taking medications for lipid lowering or blood pressure control, but who did not identify when asked whether they had these conditions or any medical problems. Addressing appropriate awareness and management of risk is likely to be an important component in engaging people in CVD prevention programs. Future studies should consider identifying and engaging a key family member capable of influencing other family members. The recruitment approach for this study used the index person as the primary contact for the family in the first instance, but it may have been more advantageous to allow a key family member to engage on behalf of the high-risk participant.

The dietary components of this study were modelled on the Mediterranean and portfolio eating patterns as these have been shown to efficacious in reducing CVD risk. Participants commencing the study reported dietary patterns that did not align well to these eating patterns and had higher than recommended intakes of discretionary food choices. Comparison of the dietary intakes of participants in the current study to data from the 2011-12 Australian Health Survey (AHS) (26) indicates that this group were consuming higher energy intakes compared to the national average of 8672 kJ (value also obtained from 24 hour recall), both before

and after the intervention, while the proportion of energy from discretionary food choices was similar at 34.6% of total energy for adults. The macro-nutrient contributions appeared unchanged by the intervention and appears comparable to the national average, although small differences can be seen between the control and intervention groups. There was no apparent change in saturated fat intakes as analyzed by the FFQ, although questions on dietary habits (Table 6.3) indicate that saturated fat may have been decreased through the choosing of different cuts of meats. Within both this study and in a study of 426 family members of coronary artery disease patients by Reid *et al.* (23), participants were only able to make small increases in intakes of vegetables, showing this to be an area to be addressed in future work. Individually tailored dietary counselling immediately after personal dietary and risk biomarker feedback in the current study resulted in favorable changes in terms of selecting lower fat dairy products and fat-trimmed meat products, which may be due to capitalizing on the teachable moment the personalized feedback helped to facilitate. A possible strategy to enhance adherence in future studies includes the provision of feedback in an educational context, based on measured anthropometrics and blood lipids at an interim stage following initial dietary modifications, instead of at the end of the study as given here which may have increased motivation. Participants were contacted by a single telephone call during the three months follow-up period to discuss any difficulties they had encountered and to encourage maintenance of dietary changes made. This level of engagement was chosen and was comparable to a longer study by Jenkins *et al.* (27), which showed that more intensive follow-up did not greatly improve adherence in this type of diet.

The limitations of the current study include the recruitment of a small non-representative sample of families who volunteered. There may have been a seasonality bias influencing the reported dietary intakes impacting on both the control and intervention groups. The dietary modifications made may not be sufficient to show clinically important and statistically significant changes in serum lipids in the short term, but may benefit the individuals if continued long term (28) and a larger study with longer follow-up would be needed to evaluate this.

6.7 Conclusions

While the goal of primary prevention is to avert disease in high-risk individuals, the current study highlights there is little motivation to participate in CVD prevention programs when risk is poorly perceived and therefore insufficient to prompt behavior change. The program structure in the current study demonstrated promising results, but the challenges of recruitment need to be overcome. Once engaged, families were willing and able to make small incremental change in their dietary choices associated with CVD risk reduction in the long-term. Further research is needed to identify CVD-related motivators of dietary change, particularly those that engage individuals and have the ability to engage all family members in improving health behaviors.

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Author Contributions

Authors T.L.S., T.L.B., D.I.T., N.J.S, R.C. and C.E.C. conceived and designed the experiments, T.L.S. performed the intervention, analyzed the descriptive data and drafted the initial manuscript. All authors revised and approved the final manuscript.

Conflicts of Interest

Author C.E.C. received an honoraria as a member of the Novo Nordisk Obesity Advisory Board. All authors declare no other conflicts of interest

References

1. World Health Organization. Global status report on noncommunicable dieases 2014. Geneva: World Health Organization, 2015.

2. World Health Organization. Global Atlas on Cardiovascular Disease Prevention and Control. Geneva: World Health Organization, 2011.

3. Kral TV, Rauh EM. Eating behaviors of children in the context of their family environment. *Physiol Behav*. 2010;100(5):567-73.

4. Lloyd-Jones DM, Nam BH, D'Agostino RB, Sr., Levy D, Murabito JM, Wang TJ, *et al.* Parental cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults: a prospective study of parents and offspring. *JAMA*. 2004;291(18):2204-11.

5. Bao W, Srinivasan SR, Valdez R, Greenlund KJ, Wattigney WA, Berenson GS. Longitudinal changes in cardiovascular risk from childhood to young adulthood in offspring of parents with coronary artery disease: the Bogalusa Heart Study. *JAMA*. 1997;278(21):1749-54.

6. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, *et al.* Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013;368(14):1279-90.

7. Jenkins DJ, Josse AR, Wong JM, Nguyen TH, Kendall CW. The portfolio diet for cardiovascular risk reduction. *Curr Atheroscler Rep*. 2007;9(6):501-7.

8. Pachucki MA, Jacques PF, Christakis NA. Social network concordance in food choice among spouses, friends, and siblings. *Am J Public Health*. 2011;101(11):2170-7.

9. Jenkins DJ, Chiavaroli L, Wong JM, Kendall C, Lewis GF, Vidgen E, *et al.* Adding monounsaturated fatty acids to a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *CMAJ*. 2010;182(18):1961-7.

10. Jenkins DJ, Kendall CW, Faulkner DA, Nguyen T, Kemp T, Marchie A, *et al.* Assessment of the longer-term effects of a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *Am J Clin Nutr*. 2006;83(3):582-91.

11. Dalen JE, Devries S. Diets to prevent coronary heart disease 1957-2013: what have we learned? *Am J Med*. 2014;127(5):364-9.

12. Huang J, Frohlich J, Ignaszewski AP. The impact of dietary changes and dietary supplements on lipid profile. *Can J Cardiol*. 2011;27(4):488-505.

13. Anderson TJ, Gregoire J, Hegele RA, Couture P, Mancini GB, McPherson R, *et al.* 2012 update of the Canadian Cardiovascular Society guidelines for the diagnosis and treatment of dyslipidemia for the prevention of cardiovascular disease in the adult. *Can J Cardiol*. 2013;29(2):151-67.

14. National Heart Foundation of Australia (National Blood Pressure and Vascular Disease Advisory Committee). Guide to management of hypertension 2008. Updated December 2010. 2010.

15. Armitage CJ, Conner M. Social cognition models and health behaviour: A structured review. *Psychol Health*. 2000;15(2):173-89.

16. GraphPad Software Inc. QuickCalcs La Jolla2012 [updated 12 December 2012; cited 2012 12 December 2012]. 2012:[Random number generator]. Available from: http://graphpad.com/quickcalcs/randomN1.cfm.

17. Australian Government, NHMRC. Australian Guide to Healthy Eating: NHMRC; 2013 [cited 2014 3 November, 2014]. Available from: http://www.eatforhealth.gov.au/guidelines/australian-guide-healthy-eating.

18. Collins CE, Boggess MM, Watson JF, Guest M, Duncanson K, Pezdirc K, *et al.* Reproducibility and comparative validity of a food frequency questionnaire for Australian adults. *Clin Nutr*. 2014;33(5):906-14.

19. Watson JF, Collins CE, Sibbritt DW, Dibley MJ, Garg ML. Reproducibility and comparative validity of a food frequency questionnaire for Australian children and adolescents. *Int J Behav Nutr Phys Act*. 2009;6:62.

20. Xyris Software. FoodWorks. 4.00.1158 ed. Kenmore Hills, Australia: Xyris Software; 2005.

21. National Health and Medical Research Council. Australian Dietary Guidelines. In: National Health and Medical Research Council, editor. Canberra: Commonwealth of Australia; 2013.

22. National Heart Foundation of Australia. Position statement: Fish, fish oils, n-3 polyunsaturated fatty acids and cardiovascular health. National Heart Foundation of Australia, 2008 Contract No.: PRO-067.

23. Reid RD, McDonnell LA, Riley DL, Mark AE, Mosca L, Beaton L, *et al.* Effect of an intervention to improve the cardiovascular health of family members of patients with coronary artery disease: a randomized trial. *CMAJ*. 2014;186(1):23-30.

24. Cohen DJ, Clark EC, Lawson PJ, Casucci BA, Flocke SA. Identifying teachable moments for health behavior counseling in primary care. *Patient Educ Couns*. 2011;85(2):e8-15.

25. Imes CC, Lewis FM. Family history of cardiovascular disease, perceived cardiovascular disease risk, and health-related behavior: a review of the literature. *J Cardiovasc Nurs*. 2014;29(2):108-29.

26. Australian Bureau of Statistics. 4364.0.55.007 - Australian Health Survey: Nutrition First Results - Food and Nutrients, 2011-12. Canberra: Commonwealth of Australia; 2014.

27. Jenkins DJ, Jones PJ, Lamarche B, Kendall CW, Faulkner D, Cermakova L, *et al.* Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of

intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. *JAMA*. 2011;306(8):831-9.

28. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. *N Engl J Med*. 2011;364(25):2392-404.

Chapter 7: Preventative dietary patterns within the context of a family cardiovascular disease risk reduction intervention

This chapter was used to qualitatively assess factors involved in engaging families in a dietary risk reduction program.

The authors of this chapter are Tracy L. Schumacher, Tracy L. Burrows, Deborah I. Thompson, Robin Callister, Neil J. Spratt and Clare E. Collins. The work presented here was completed in collaboration with the co-authors (see Appendix 20).

7.1 Overview

The previous chapter described the challenges in recruiting families to a dietary CVD prevention program. This chapter is a qualitative investigation into factors that may have contributed to those challenges. As families completed the intervention phase of the program, they were invited to participate in a telephone interview on an individual basis, with no limit to the number of participants from each family. The interviews followed a semi-structured script, with the questions based on issues observed during family counselling sessions. This chapter reports the qualitative results from the interviews and discusses the impact these findings may have on health care practice. Documents relating to ethics approval can be seen from Appendix 21 to Appendix 23.

7.2 Abstract

Objectives: Diet is an essential strategy for the prevention of primary and secondary cardiovascular disease (CVD) events. The objective was to determine how families at increased risk of CVD perceive personal risk and their motivations, barriers and/or perceived need to make dietary modifications.

Methods: Individuals (>18 years) who completed a family-based CVD risk reduction program were invited to a semi-structured telephone interview. Responses were recorded, transcribed verbatim and analysed using a systematic deductive approach with coding derived from key concepts developed as part of the interview structure.

Results: Seventeen participants from eight families were interviewed (aged 18-70 years, 47% male, five with CVD diagnosis). Key themes from the areas of enquiry indicated both intrinsic and extrinsic motivations to improve heart health, variations in risk perception, recognition of the role diet plays in heart health, and the extent of family influences on eating patterns.

Conclusion: Discrepancies between perceived and actual CVD risk perception impacted on perceived "need" to modify current dietary patterns towards heart health recommendations. Therefore strategies not reliant on risk perception are

needed to engage those with low risk perception. This could involve identifying and accessing the family 'ringleader' to influence involvement and capitalising on personal accountability to other family members.

7.3 Introduction

Cardiovascular disease (CVD) is a leading cause of morbidity and mortality in Australia and worldwide (1, 2). Modifiable behaviours related to risk factors include diet, exercise, smoking, and alcohol consumption (3). Diet is effective in reducing CVD event rates (4, 5), but altering eating patterns can be challenging (6). Many believe their cardiovascular disease is due to bad luck or heredity (7), or is a short-term problem (8). Factors such as social support and unhelpful social contacts can affect the motivations of those with CVD, and contribute to poor adherence to dietary recommendations (9-11). Family interactions are also an important influence on adherence to chronic disease recommendations and may offer practical, emotional and social support, or be at a similar stage of change for improving health behaviours (10, 12-14). Alternatively, family interactions can act as a negative pressure and function as barriers to improvements (10, 12).

The inclusion of family members in dietary counselling for CVD risk reduction was trialled in the 'Love your Food, Love your Heart, Love your Family' (FHF) pilot randomised controlled trial, as described in Chapter 6 (15). This intervention was based on Protection Motivation Theory (PMT), as it has been shown to be applicable to CVD (16) prevention. PMT proposes that people act in accordance with their estimation of threat and coping appraisal (17). It was hypothesized that families with a moderate to high CVD risk would perceive their threat of adverse CVD events as high and be highly motivated to reduce their risk. Families were eligible to participate in the FHF study if at least one 18-70 year old family member (index person) had experienced an adverse CVD event or was classed at or above moderate risk using Australian cardiovascular risk charts (18). Families were required to attend two sessions (one baseline; one at 12 weeks) together as a group, that included assessments and feedback, with counselling provided in the first session only. Blood lipid profiles, anthropometric measures and current dietary intakes were assessed on all participating family members; these data were

used to provide feedback in a single 45-minute, family-based, dietetic counselling session with an Accredited Practising Dietitian. The dietary advice (1) recommended increased consumption of foods from the Mediterranean (4) and Portfolio diets (19) readily available in the Australian food supply (2) and reduced consumption of less healthy foods. Control families were waitlisted for three months and then were invited to undergo the intervention. Recruitment proved challenging; a total of 23 families were recruited over an 18-month period from a variety of sources, ranging from flyers displayed in the university setting, to media releases, accessing a volunteer register and attending cardiac rehabilitation classes and stroke clinics. Dietary changes made during the intervention aligned with the eating patterns recommended and were beneficial, though small or incremental in magnitude. These included reductions in usage of full-fat milks, cheese and meat products.

Follow-up interviews were conducted to gain qualitative insights from the index participants and their family members to inform future family-based studies. For example, gaining an understanding of the participants' risk perceptions and motivations to join a dietary CVD prevention study could be utilised to improve on future recruiting methods and assist with long-term behaviour change. Appreciation of and preparation for individual influences on family eating patterns may inform strategies clinicians undertake to support individuals and their families towards adopting dietary recommendations.

Therefore the aim of the current study was to examine the participants' 1) motivations for joining a dietary family-based study for CVD prevention, 2) perceptions of CVD risk in self and other family members, and 3) understanding of the role of diet and experiences of individual or family-based influences on eating patterns.

7.4 Methods

This study is a qualitative exploration of the FHF study (15), as more understanding was required in relation to risk perception and the role of family in eating patterns related to CVD health. A qualitative design was selected to elicit rich information and insights from participant experiences. Adults who completed

the intervention phase of the pilot FHF study were invited by mail to participate in individual semi-structured telephone interviews following collection of final data. Detailed methods for recruitment for the initial pilot phase have been described previously (15). The need for interviews was explained to the participants as "…the study raised other questions about barriers and family dynamics that investigators feel needs further investigating". Interviews were performed by one researcher (TS) post intervention at the convenience of the participants.

Telephone interviews were chosen as the preferred method of data collection as although there is an acknowledged loss of non-verbal data, a relationship was already established between the researcher and individual interviewees (20). Also, as participants were free to choose their own interview time and location for the telephone call, privacy from other family members was optimised and therefore they were more to be willing to disclose sensitive information (20). An interview guide was developed primarily by two authors (TS, DT) that included a prepared script to introduce the study; questions were based on issues that emerged from observation during the pilot study (20) (see Appendix 23). Probes and prompts were used to expand and clarify responses and notes were taken by the interviewer throughout to ensure all questions were answered (21). The areas of enquiry included motivations for participating in the FHF study, concerns about heart health, perceptions regarding the role of diet in heart health, barriers to healthier eating, and how family dynamics influenced dietary decisions. Interviews on a topic were continued until no new information emerged.

The interviews were digitally recorded with the participants' consent and transcribed verbatim. Qualitative analyses were conducted by an independent experienced qualitative researcher using NVIVO 10 and expanded on by lead author (TS) (22). A systematic deductive approach to data analysis was adopted. Initial codes were formulated from the overall study aims to develop a taxonomy of the domains that characterised the multifaceted experiences of participants and relationships within the data. This was revised and further expanded after coding of additional transcripts. Following coding of all the transcripts, codes were examined and collapsed into categories by an external qualitative researcher (V. H.). Themes were identified and summarised by the lead author (T.L.S.) in

consultation with the research team (T.L.B., D.I.T., R.C., N.J.S. and C.E.C.). Ethics approval was obtained from University of Newcastle Human Research Committee, H-2012-0246.

7.5 Results

Seventeen program participants from eight families were interviewed a mean of 1.5 months post intervention (acceptance rate of 59%). Participants were invited to participate in interviews as they completed their interventions until no new information emerged. Participants were aged 18 to 70 years (53±6 years), 47% men, with two to five members per family. Five interviewees were index participants, two of which had previously experienced adverse CVD events. The remaining three index participants had undertaken preventative surgery. The remainder of the interviewees were family members (five spouses, five children, one sibling and one grandchild); one family member also had a diagnosed CVD condition. Interviews lasted approximately 21 minutes (range 16-29 minutes). A summary of salient points that emerged from the areas of enquiry can be seen in Table 7.1.

Table 7.1: Salient points that emerged from the areas of enquiry.

Areas of enquiry	Salient points
Motivations	Importance of family unit
	Ringleaders and secondary recruits
Risk perceptions	CVD risk perception in index participants
	CVD risk perception in secondary recruits
Heart health and diet	Relative importance of diet
	Routine
	Accessibility to healthy foods
Influence of family on eating patterns	Working within confines of family roles and preferences
	Inferred, not discussed
	Overt and covert approaches
	Recognised boundaries of influence

7.5.1.1 *Motivations*

Motivations to join the study encompassed two broad areas: the importance of the study being inclusive of the family unit and involvement due to a more dominant family member.

Involvement of the family unit as a motivator for dietary and lifestyle changes was identified to have important benefits through demonstrating supportiveness or as mutually-implied inter-family accountability. It was alluded to that dietary changes for a single individual would be challenging and the role of support either given or received would be of value. As several participants said:

> *'There's not much point in one person being healthy in the family when there is heart disease in that side of the – the husband's side of the family. So it needed to be like a joint – a whole family thing.'*

> *'Because there were so many members in the family that had had heart issues, I felt that we would be a good source of information and we might be able to help each other.'*

> *'So any changes in your lifestyle, like I'm not good at that, but if I'm accountable to other people, I'm better at it.'*

All interviewed families clearly identified a family "ringleader" who was instrumental in getting their family to participate in the Love your food, Love your heart, Love your family intervention. *'... after having had a heart attack himself, he has been the driving force.'* "Ringleaders" were not from any particular family position: two fathers, one husband, one wife, two daughters and one grandfather were identified as performing this role within the family. "Ringleaders" varied in their motivations for participating, with reasons ranging from a long-term interest in improving their diet to a desire to bring about positive changes in health for themselves and their family.

> *'Oh my God, I was just so excited, I was so excited... this was just a great opportunity to...include some family members because some...weren't eating very well at all...Look my husband probably resisted the most...*

My mother was very excited. My daughter was okay; she just wanted to join in. My husband doesn't like change … he thought if it meant having to eat things he didn't like then he would never manage, but we talked him into it.'

While many of the "secondary recruits" were motivated to participate to support their family or help improve the health of a relative with heart health issues, they also enrolled for personal reasons, such as to reduce their personal risk of heart disease or other lifestyle diseases. In some instances, these 'secondary recruits' appeared to have a higher level of readiness to adopt dietary and lifestyle changes.

'Well, I just thought something that would improve my diet and assess my health was important, because I knew my diet wasn't very good'.

'…seeing both my parents and my in-laws, just the amount of medication that they take.'

7.5.1.2 *Risk perceptions*

Risk perceptions, as shown through concern levels about heart health for the index participants who had experienced adverse CVD events, varied substantially. One participant was very concerned about his heart health:

'Well it's close to maximum because I have had previous heart surgery…My father died with heart trouble so I'd want to be very conscious about the heart.'

In contrast, others expressed no real concern for heart health, despite previous surgery or CVD events, and viewed their heart health as of no more concern than those without a similar medical history. This appeared to be the result of confidence in their medical and surgical treatment, with little need for ongoing preventive strategies.

'[I'm] not concerned, I've sort of had my operations and all my medication, I'm quite happy with the way it appears to be going at the moment.'

Some diminished the severity of their condition due to their treatments, as suggested by the use of 'moderating words'.

> *'Well, it was a slight heart attack, they put a stent in which wasn't a great big operation and I am on medication now, that was three years ago, three and a half years ago and it has not annoyed me, in any way.'*

Another used moderating words to describe adverse CVD events of close family members and indicated he only recognised his individual risk following his own event:

> *Q: 'Were you aware of your risk?'*
>
> *A: '…I was aware through hearing of different family members having their little heart attacks or bypasses or dying from it, but did that register with me? No. ….As soon as it happened [his own adverse CVD event], then it was a reminder that "Oh well, you lost a cousin when he was 32 and don't forget your brother…had a heart attack, or had a bypass and your Dad did, and then your Aunty", and it's like "oh yeah, that's right. No wonder we're at risk.'*

Thus overall, for some participants, there was not a strong or consistent association between their perception of CVD risk and objective evaluation of risk based on assessments and medical history.

For the family members who also joined the study as secondary recruits, CVD risk perception was influenced by self-justification of lifestyle and heredity factors, although a healthy diet was generally acknowledged to play a role in decreasing CVD risk by all interviewees. Some family members rationalised their level of concern by factors such as the presence of symptoms or family history of heart-related problems:

> *'…No. I'm not very concerned about my heart. I seem to have kind of lowish to medium blood pressure and I don't seem to have any problems as yet. Nothing's come up on any tests or anything like that.'*

'…they've got some of the genes of their father, who's got the heart disease in the family. Maybe because I haven't, I'm just, you know, a bit more – not as concerned about me.'

For four other supportive family members, justification for reduced heart health concern was given as adherence to healthy dietary habits in combination with other preventive behaviours:

'I know that if I'm to remain healthy I've got to not only eat the right stuff, but I've got to keep moving.'

'Because I know I do eat well and I know I'm on medication and my husband's on medication, but I thought we had a reasonable diet. So I wasn't that concerned about heart problems.'

7.5.1.3 *Heart health and diet*

All participants rated the relationship between diet and heart health as moderate to high. All conveyed an awareness of the contribution of diet to heart health. The colloquial '*you are what you eat*' was a common analogy used to express this strongly-perceived association. Others believed in the importance of moderation and balance as the crucial element of a healthy diet: *'Too much of a good thing is not good for you'.*

Many specifically expressed an awareness of factors other than diet that influence CVD risk, such as exercise and genetics: *'…look I believe that there is some genetic factors involved, but I certainly believe that we can do a lot more with diet and exercise'.* Participants who rated diet as very important believed that diet is a significant moderator of other factors and is capable of overriding other significant risks in which personal actions shape health outcomes.

Very few participants believed that hereditary factors inevitably will prevail and counteract dietary and lifestyle intentions.

'I think sometimes no matter what your diet is, if you've got a problem genetically or anything, it can more or less kind of help, but sometimes you just have that problem and that's it, you know.'

Several participants mentioned the importance of routine in adhering to a healthier diet. Planning meals and purchases, as well as preparing meals at home, were some of the elements of routine which were considered conducive to healthy eating.

> *'Because I'm out so much every day and staying at different places, your diet just falls out of place...Routine [keeps me inline], because even waking up later and just having a late breakfast puts you out of place, but routine makes it easy and I always take fruit to school.'*

The most often-mentioned barrier to eating heart healthy foods was lack of convenience and the corresponding accessibility to healthy foods. Some mentioned lack of healthy options when eating out or while travelling.

> *'Particularly if you go out and eat, you're always getting lathered with chips. They're just tasty, so you do tend to eat them...even if you get salad you'll have steak and salad, you'll still get chips. Yeah, so I probably tend to order steak and vegetables...Because that eliminates the chips.'*

Another participant felt they had limited access to good quality healthy foods that they would like to consume:

> *'...look there's not everything there that is what we would eat normally. So certainly isn't your fish, the range of fish and things like that, that we like to eat... it's a standalone supermarket. So if it's not in the supermarket, it's very inconvenient to go and get it elsewhere.'*

The concept of accessibility and convenience appeared to be a powerful motivator for consuming highly processed and unhealthier foods. Being time-poor was presented as a rationale for less healthy eating patterns, due to the extra time involved in preparing healthier meals: *'Sometimes it's easier to grab something that's not necessarily totally nutritious and often the raw natural foods require a bit more preparation than the instant stuff.'* Portability and accessibility of 'instant' lower quality foods was emphasized by a younger working woman, who identified it as an important obstacle to the consumption of more healthy options.

Overall, there seemed to be an acceptance that life sometimes would 'get in the way' of a healthy diet. Indeed, most participants appeared to engage in a cognitive balancing act, where the benefits of quick and easy food choices were used to justify less desirable dietary habits.

> *'...everything I eat is not necessarily is what I would think is good for my heart. I mean, there's got to be some things that are not good for it. But life goes on and often you've got to have something – well, I feel you've got to have some enjoyment out of life, and sometimes that goes to living on the edge, I suppose.'*

7.5.1.4 *Family member influences on adoption of recommended eating patterns*

Participants reported a range of circumstances where individual members exerted influence over family eating behaviours, either through a willingness to change, or through indicating resistance and providing challenges to achieving change. Participants described family members who were champions that supported at least some of the recommended eating patterns, as well as some who acted as saboteurs.

Several participants made reference to tastes and preferences being an important barrier to healthier eating patterns. These barriers varied between preferences for unhealthier alternatives established through lifelong habits and cultural/traditional culinary heritage, to reluctance of either self or partner to try new healthier foods. Indeed, for most participants where food preferences were a barrier, their male partner's preference for other foods was the limiting factor.

Reluctance of a partner to change could have led to conflict but appeared to be resolved in one of two ways: either separate meals or conceding to the less healthy option. For one older woman, her husband's reluctance to change dictated which meals were served:

> *'...sometimes [wife's name] makes something for herself that I don't necessarily like, so she has another meal in the freezer for me or she will cook something different for me, you know?'*

Alternatively she would prepare and eat less nutritious meals, as the additional effort and time involved in cooking separate meals acted as a powerful barrier to adoption of her own healthier eating patterns:

> *'You tend to be a little bit lazy when you're doing the cooking just for two… it just makes it easier to cook the same thing.'*

Some family members identified advantages in compromising their preferences. One man indicated:

> *'…there might be some things that I don't particularly like taste wise, but I know they are probably good for me. So I will eat them without too many qualms. If I complain too much, then it won't happen and I will have to cook.'*

The advantage of following the lead of the ringleader in the presence of discrepancies between preferences and health behaviours was recognised by another husband:

> *'[If she wasn't as concerned as me, I would] probably become a little bit more complacent I would imagine. Having someone strong and up there to remind you is certainly beneficial... She's the driving force.'*

Related to this dyadic effect within households was a prominent issue relating to "bad foods" being brought into the home environment, creating temptations for others. In most cases, one person in the household was clearly identified, by self and others, as the "perpetrator":

> *'I'm probably, maybe, a bigger offender than…I mean, I do mainly buy the food that comes into the house.'*

The majority of participants perceived their family as being able to openly discuss diet-related issues, however interviewees revealed that conflict was more likely avoided by circumventing confrontation:

> *'I mean, after 30-odd years, you learn not to talk about things you don't need to talk about, don't you?'*

Another wife commented:

> *"I probably don't tell him [that he needs to lose weight]. He's the one who says 'oh, I need to lose a bit.' I think when he had his knees hurt or his ankles hurt, I might have suggested that losing a bit of weight might help. He just says 'I know'. So we don't - I think we need to do things together to help, to motivate each other as far as that goes.'*

In the few instances in which participants alluded to the presence of food-related topics capable of eliciting conflict, there appeared to be an implicit understanding of what and how to bring up certain issues. Indeed, one participant used the idiom 'walking on eggshells' to describe the sensitivity of food related issues around his obese sister, iterating the fact that the sensitive nature of food related topics, for some, will prevent open discussion around health promoting issues.

Cooking different meals and trialling new recipes were overt strategies used to try and facilitate dietary changes. Some tried a more inclusive partnership approach to increasing support for making dietary changes:

> *'He loves all the homemade stuff I make, all the soups that I make…he can't cook, but he's chopping up the vegetables for soup, so he can do that.'*

Others employed more covert strategies to facilitate adopting some of the dietary recommendations:

> *'…he is very defensive about changes to the diet, and if it's pointed out that something might be unhealthy he gets very defensive about it. I think he feels quite threatened that someone might change the status quo… because I work a lot, he does the shopping, so I have changed his shopping habits... But I have to be a bit clever about it… I just write him little lists and stuff like that and kind of plant the seed and I sort of display the fruits and things like that prominently around the place. So he knows that's what's getting eaten.'*

Several participants spoke of their boundaries of influence over the eating patterns of other family members. Beliefs relating to the extent of perceived influence guided their behaviours:

> *'If he doesn't like certain things, it's a bit hard to make something different – two different types of meals…But sometimes I say "Oh no, you can try this".'*

> *'There's times when I can influence that [the home environment] and times that I can't. Where I could I would.'*

> *'It goes in one ear and out the other when I say something. I think a lot of wives have the same problem.'*

Two participants recognised that their influence was not enough to achieve the desired behaviour change in their partners but identified a role for health professionals:

> *'The funny thing with him is that he will really listen to a professional. He probably listens to me a little bit less …so he came home [after a dietetic consultation] and he's actually been on a relatively low GI diet ever since….he really respects people who know what they're talking about.'*

> *'…I think they need a professional other than me saying all the time, "You need to do this. You need to do that." He needs it coming from some other person who's a professional.'*

7.6 Discussion

The current study intended to qualitatively explore risk perceptions and motivations to engage in dietary risk-reduction behaviours in families at increased risk of CVD to determine approaches that may be useful in engaging similar populations. The various influences of family members in adopting healthier eating patterns were also explored. Results indicate that risk perception alone may be inadequate to initiate and sustain dietary changes in this population. However, individual family members were shown to be both positive and negative influences

on dietary patterns and may be utilised to enhance adherence to dietary recommendations.

Heart health concern was explored with both index and secondary recruits. This concern directly relates to threat appraisal, a major construct of PMT. There was a disparity between perceived and actual risk, with participants rarely estimating their true level of risk. Cardiovascular risk tends to be inaccurately or optimistically viewed and these results are similar to those with established CVD, familial hypercholesterolemia and asymptomatic populations, who have all been shown to have poor agreement between perceived and true CVD risk, with those at high risk particularly underestimating their actual risk (23-25). This was most recently demonstrated in a multinational cohort of approximately 3,500 participants from United States and Spain who experienced an acute myocardial infarction before 56 years of age. Only half (53%) of the patients considered themselves at risk of CVD, even though almost all (98%) had at least one risk factor (26). Also, those experiencing CVD events may understand their condition as acute, not chronic, with the effects limited in time (8). According to PMT, for dietary changes to be made, an estimation of threat has to be perceived before coping options can be evaluated (27).

As an explanatory theory for behaviour relating to CVD health, the PMT provides that coping appraisal is used in conjunction with threat appraisal. This was explored in two ways. Firstly, response efficacy was explored within the relationship of heart health and diet and self-efficacy was explored within barriers faced and the influence of family. Coping appraisal within the PMT includes the belief that taking the recommended steps will produce a positive response (response efficacy) (27). In this study, many people believed that *"you are what you eat"*, described a relationship between diet and CVD within a more holistic understanding of lifestyle that included diet and exercise, but also apportioned a role for genetics.

It appears that many individuals believe they are already taking the necessary steps, such as taking medication and "eating well. Past surveys of the Healthy Eating Index, a guide to diet quality, has shown that approximately 40% of the

surveyed population optimistically perceived their eating habits to be of better quality than they actually were, with only 40% evaluating their diet quality accurately (28). A recent European study found that approximately one in five adults in their study sample overestimated their adherence to vegetable intake guidelines (29). Also, those who make small changes to their intake may consider it the appropriate level of change needed (9).

Factors such as social supports and unhelpful social contacts can affect the motivations of those with CVD (9, 10) and contribute to poor adherence to dietary recommendations (11). Although an individual's intention to perform an action may have been verbalised, whether the action was performed was likely to have been dependent upon other factors. The barriers to healthy eating encompassed a range of issues: the complex relationships with partners, the need to deal with partner's food preferences, individualised eating, with occasions of eating away from home being common. Family members were either a significant form of support or barrier to improving dietary patterns. These findings are similar to others: Aggarwal *et al.* showed that in family members of people with established CVD, those with low social support are less likely to adhere to a therapeutic diet (30). Other findings from health habits of married individuals demonstrate that when one spouse initiates a new health behaviour, it significantly increases the odds of the other in adopting the same behaviour, particularly for behaviours such as decreasing smoking (odds ratio (OR): 5.65 men, 5.21 women) and drinking (OR: 5.64 men, 5.58 women), and that readiness to change behaviour is similar between married couples (12, 14). Evans showed that family is a bigger influence on changes in dietary behaviours than friends for African American women, whether offering encouragement or criticisms (31). Social support can be extended to social contacts outside of the family unit, with these also creating a supportive or negative influence (10).

Eating away from the home, lack of time and individualised eating were some of the barriers mentioned in the results. These are aligned with food choice coping strategies and affect dietary quality and nutritional adequacy (32). Blake *et al.* showed that these coping strategies are related to work, marital status and the number of children. Those with a lack of time may miss meals or choose foods

requiring less preparation, leading to reduced intakes of quality grains and milk (32). Those preparing individualised meals tended to eat less dark green and orange vegetables (32). This is of concern in regards to populations looking to improve heart health, as unrefined and highly coloured plant and vegetable matter are associated with eating patterns such as the Mediterranean diet, which have been demonstrated to improve CVD outcomes (4, 33).

Strengths and limitations of this study include the qualitative design, which allowed participants to express in their own words their experiences of undertaking measures to prevent CVD by dietary means. The interviewer being known to participants is both a strength and a weakness, having developed contact during the study intervention period. Prior contact allowed rapport to be previously established between interviewer and interviewee thereby allowing the interviews to be performed by telephone, although it may also have biased the opinions that were expressed. Non-verbal cues that may have been incorporated in a face-to-face interview were also not possible. However, choice of time and location for the interview to take place allowed for the participant to choose a comfortable situation to increase their own privacy. The sample size interviewed was small and limited to a regional geographic area. Therefore these findings are not generalizable or representative of other populations. Also, participants previously participated in a CVD risk reduction intervention and should be expected to have an increased knowledge of the role of diet in CVD risk management.

7.7 Conclusion

Although the PMT is a suitable theory for this population at elevated risk for CVD due to family histories of CVD and shared environments, the results demonstrate the importance of understanding risk perception, as variation was seen between perceived and actual CVD risk. Family members may act as enablers or barriers to adherence dependent upon their understanding of risk and how they perceive the "healthiness" of their personal and/or shared current diet. Participants described a strong belief in the concept of moderation, but expressed persistent barriers to improving their dietary intakes. Inclusion of and accountability to family members

was reported as a strong motivator. This could be used in future interventions targeting CVD dietary recommendations and it is recommended that family members be included as part of CVD dietary counselling. Further to this, there is a need for future research to investigate methods of identifying influential family member/s, the extent of influence and how it affects dietary choices.

7.8 Implications for practice

This study has implications for practitioners aiming to improve adherence to dietary recommendations in those at increased risk of CVD and their families. Firstly, self-perception of risk may not be associated with objective risk and may be insufficient to engage families in dietary changes. Other more powerful motivators may be required. In these situations, counsellors may benefit from enlisting the support of individual family members, particularly the family "ringleader", the "enabler" or those members willing to adopt the recommended dietary patterns to capitalise on intra-family accountability. Alternately, it may be constructive to include those who act as barriers to improvements or the 'perpetrators' of unhealthier eating habits in the counselling sessions. The issues of accessibility to health foods, routine and being "time-poor" may be addressed concurrently by stressing the importance of planning ahead and developing new routines where necessary.

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References

1. World Health Organization. Global status report on noncommunicable dieases 2014. Geneva: World Health Organization, 2015.

2. Australian Bureau of Statistics. 3303.0 Causes of Death, Australia, 2013. Canberra: Commonwealth of Australia; 2015.

3. World Health Organisation. Global Atlas on cardiovascular disease prevention and control Geneva2011 [cited 2014 16 December]. Available from: http://whqlibdoc.who.int/publications/2011/9789241564373_eng.pdf?ua=1.

4. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, *et al.* Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013;368(14):1279-90.

5. de Lorgeril M, Salen P, Martin JL, Monjaud I, Delaye J, Mamelle N. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction: final report of the Lyon Diet Heart Study. *Circulation*. 1999;99(6):779-85.

6. Desroches S, Lapointe A, Ratte S, Gravel K, Legare F, Turcotte S. Interventions to enhance adherence to dietary advice for preventing and managing chronic diseases in adults. *Cochrane Database Syst Rev*. 2013;2:CD008722.

7. Perkins-Porras L, Whitehead DL, Steptoe A. Patients' beliefs about the causes of heart disease: relationships with risk factors, sex and socio-economic status. *Eur J Cardiovasc Prev Rehabil*. 2006;13(5):724-30.

8. Bradley F, Wiles R, Kinmonth AL, Mant D, Gantley M. Development and evaluation of complex interventions in health services research: case study of the Southampton heart integrated care project (SHIP). The SHIP Collaborative Group. *BMJ*. 1999;318(7185):711-5.

9. Doyle B, Fitzsimons D, McKeown P, McAloon T. Understanding dietary decision-making in patients attending a secondary prevention clinic following myocardial infarction. *J Clin Nurs*. 2012;21(1-2):32-41.

10. Cole JA, Smith SM, Hart N, Cupples ME. Do practitioners and friends support patients with coronary heart disease in lifestyle change? a qualitative study. *BMC Fam Pract*. 2013;14:126.

11. Ma Y, Li W, Olendzki BC, Pagoto SL, Merriam PA, Chiriboga DE, *et al.* Dietary quality 1 year after diagnosis of coronary heart disease. *J Am Diet Assoc*. 2008;108(2):240-6; discussion 6-7.

12. Falba TA, Sindelar JL. Spousal concordance in health behavior change. *Health Serv Res*. 2008;43(1 Pt 1):96-116.

13. Rosland AM, Heisler M, Piette JD. The impact of family behaviors and communication patterns on chronic illness outcomes: a systematic review. *J Behav Med*. 2012;35(2):221-39.

14. Franks MM, Shields CG, Lim E, Sands LP, Mobley S, Boushey CJ. I will if you will: similarity in married partners' readiness to change health risk behaviors. *Health Educ Behav*. 2012;39(3):324-31.

15. Schumacher TL, Burrows TL, Thompson DI, Spratt NJ, Callister R, Collins CE. Feasibility of Recruiting Families into a Heart Disease Prevention Program Based on Dietary Patterns. *Nutrients*. 2015;7(8):7042-57.

16. Plotnikoff RC, Higginbotham N. Predicting low-fat diet intentions and behaviors for the prevention of coronary heart disease - an application of Protection Motivation Theory among an Australian population. *Psychol Health*. 1995;10(5):397-408.

17. Plotnikoff RC, Higginbotham N. Protection motivation theory and the prediction of exercise and low-fat diet behaviours among Australian cardiac patients. *Psychol Health*. 1998;13(3):411-29.

18. National Heart Foundation of Australia (National Blood Pressure and Vascular Disease Advisory Committee). Guide to management of hypertension 2008. Updated December 2010. 2010.

19. Jenkins DJ, Kendall CW, Faulkner DA, Nguyen T, Kemp T, Marchie A, *et al.* Assessment of the longer-term effects of a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *Am J Clin Nutr*. 2006;83(3):582-91.

20. Novick G. Is there a bias against telephone interviews in qualitative research? *Res Nurs Health*. 2008;31(4):391-8.

21. Opdenakker R. Advantages and Disadvantages of Four Interview Techniques in Qualitative Research. *Forum Qual Soc Res*. 2006;7(4).

22. NVivo qualitative data analysis Software. Version 10. QSR International Pty Ltd.; 2012.

23. Davidson PM, Salamonson Y, Rolley J, Everett B, Fernandez R, Andrew S, *et al.* Perception of cardiovascular risk following a percutaneous coronary intervention: a cross sectional study. *Int J Nurs Stud*. 2011;48(8):973-8.

24. Claassen L, Henneman L, Kindt I, Marteau TM, Timmermans DR. Perceived risk and representations of cardiovascular disease and preventive behaviour in people diagnosed with familial hypercholesterolemia: a cross-sectional questionnaire study. *J Health Psychol*. 2010;15(1):33-43.

25. Katz M, Laurinavicius AG, Franco FG, Conceicao RD, Carvalho JA, Pesaro AE, *et al.* Calculated and perceived cardiovascular risk in asymptomatic subjects submitted to a routine medical evaluation: The perception gap. *Eur J Prev Cardiol*. 2015;22(8):1076-82.

26. Leifheit-Limson EC, D'Onofrio G, Daneshvar M, Geda M, Bueno H, Spertus JA, *et al.* Sex Differences in Cardiac Risk Factors, Perceived Risk, and Health Care Provider Discussion of Risk and Risk Modification Among Young Patients With Acute Myocardial Infarction: The VIRGO Study. *J Am Coll Cardiol*. 2015;66(18):1949-57.

27. Floyd DL, Prentice-Dunn S, Rogers RW. A meta-analysis of research on protection motivation theory. *J Appl Soc Psychol*. 2000;30(2):407-29.

28. Variyam JN, Shim Y, Blaylock J. Consumer misperceptions of diet quality. *J Nutr Educ*. 2001;33(6):314-21.

29. Dijkstra SC, Neter JE, Brouwer IA, Huisman M, Visser M. Misperception of self-reported adherence to the fruit, vegetable and fish guidelines in older Dutch adults. *Appetite*. 2014;82:166-72.

30. Aggarwal B, Liao M, Allegrante JP, Mosca L. Low social support level is associated with non-adherence to diet at 1 year in the Family Intervention Trial for Heart Health (FIT Heart). *J Nutr Educ Behav*. 2010;42(6):380-8.

31. Evans GL, McNeil LH, Laufman L, Bowman SL. Determinants of low-fat eating behaviors among midlife African American women. *J Nutr Educ Behav*. 2009;41(5):327-33.

32. Blake CE, Wethington E, Farrell TJ, Bisogni CA, Devine CM. Behavioral contexts, food-choice coping strategies, and dietary quality of a multiethnic sample of employed parents. *J Am Diet Assoc*. 2011;111(3):401-7.

33. Sofi F, Abbate R, Gensini GF, Casini A. Accruing evidence on benefits of adherence to the Mediterranean diet on health: an updated systematic review and meta-analysis. *Am J Clin Nutr*. 2010;92(5):1189-96.

Chapter 8: Effectiveness of a brief dietetic intervention for hyperlipidaemic adults using individually-tailored dietary feedback

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8.1 Overview

This chapter describes the translation of efficacious dietary advice to a hyperlipidaemic population in a single counselling session. This population was chosen as they self-identified as having serum cholesterol above desirable range. Ethics approval for this study can be seen in Appendix 25. Documents required for ethics approval can be seen from Appendix 26 to Appendix 28. Samples of resources used in this intervention can be seen in Appendix 29 and Appendix 30.

8.2 Abstract

Purpose: Dietary modifications can improve serum lipids and reduce CVD risk. However, attendance at multiple dietary consultations can be a barrier to achieving behaviour change. This study investigated the effectiveness of a brief dietetic intervention on CVD risk factors in hyperlipidaemic adults.

Methods: Adults with total cholesterol ≥5.0mmol/L or LDL cholesterol ≥4.0mmol/L and not currently taking lipid-lowering medication were eligible to participate in a minimum 6-week dietary intervention based on Protection Motivation Theory. Dietary intake data (Australian Eating Survey completed online) and blood lipids were acquired prior to a single dietetic counselling session with an Accredited Practising Dietitian. Participants were provided with individually tailored feedback and advice on modifying diet to improve serum lipids and reduce CVD risk. Dietary advice was informed by evidence from the Mediterranean and Portfolio diets and supported by written documentation. CVD risk factors and dietary intakes (two three-pass 24-hour recalls) were used to assess pre-post intervention change using linear mixed model regression analyses.

Results: Thirty-nine participants (59.3±11.1 years, n=28 female) were analysed. Mean±SD follow-up from baseline time was 9.5±2.5 weeks. Significant ($p<0.05$) reductions in total cholesterol (-0.51mmol/L), total:HDL ratio (-0.27mmol/L), triglycerides (-0.38mmol/L), total energy (-870kJ/day), energy from nutrient-poor foods (-1006kJ/day) and sodium (-325mg/day), and improved dietary fat quality (-5.1% of energy/day saturated, +5.0% of energy/day polyunsaturated) and body mass index (-0.4kg/m^2) were achieved.

Conclusion: A brief dietetic intervention incorporating targeted, personalised dietary feedback and education in a single counselling session can improve lipid profiles in adults with hyperlipidaemia.

8.3 Introduction

The World Health Organisation identified cardiovascular disease (CVD) as the leading worldwide cause of mortality (46.2%) in 2012. Dietary intakes are a modifiable risk factor for CVD (1, 2) with dietary patterns, such as the Mediterranean diet shown to reduce incidence of cardiovascular events within five years of adhering to the pattern (3). The United States Preventive Services Task Force deemed the likelihood of harm to be small to none for dietary modification, with adequate evidence of benefits from intensive counselling in populations with multiple risk factors for CVD (2).

However, achieving dietary change in populations at increased risk of CVD can be difficult (4, 5). Barriers to implementation of dietetic counselling include the time and cost of multiple dietetic consultations for those at risk, as well as reluctance of medical practitioners to refer at risk patients if the practitioner believes the patient is unwilling or unable to attend dietetic consultations or that medication is going to be as effective (6). Evidence for the effectiveness of less intensive dietary interventions, .i.e. fewer consultations, is lacking and trials assessing such approaches are needed (2). Given these barriers and lack of evidence for the effectiveness of brief interventions, a study design more pragmatic than explanatory may be of value. Pragmatic study designs mimic or are suitable for normal practice, use participants that would typically be receiving the treatment, and target outcomes relevant to the stakeholders: i.e. the patients, general practitioners and healthcare providers (7).

Key components of behavioural interventions shown to contribute to effectiveness include individualised assessment, goal setting, education and feedback, and provision of written information to reference as verbal information is commonly forgotten or incorrectly recalled (8-10). Also, provision of food items or subsiding healthier food products have been shown to be effective in weight-loss interventions and in modifying dietary behaviours (11, 12)

The aim of the current study was to trial the effectiveness of a brief dietetic intervention on diet-related CVD risk factors in hyperlipidaemic adults. The intervention translated the best-available dietary evidence into a manual for participants and included targeted feedback and individualised CVD-health counselling strategies in a single session provided by an Accredited Practising Dietitian (APD). It was hypothesised that the dietetic intervention would improve lipid profiles.

8.4 Methods

8.4.1 Study Design

The study was a pre-post dietary intervention in hyperlipidaemic adults. The study protocol is summarised in Figure 8.1. Participants completed the Australian Eating Survey (AES) food frequency questionnaire and provided a fasting blood sample for analysis prior to attending a single dietetic counselling session where they received personalised feedback on their current diet, CVD risk, and counselling on dietary strategies. The dietary advice focused on evidence from the Mediterranean and Portfolio diets, which have been shown to be effective in lowering CVD events and risk factors (3, 13). A study-specific education manual was provided to assist participants adopt the recommended eating patterns. Assessments of diet (24-h recalls) and CVD risk (blood lipids, blood pressure, anthropometric measures) were conducted prior to counselling and after a minimum 6 weeks follow-up, at the participant's convenience. Primary outcome measures were dietary changes and blood lipids.

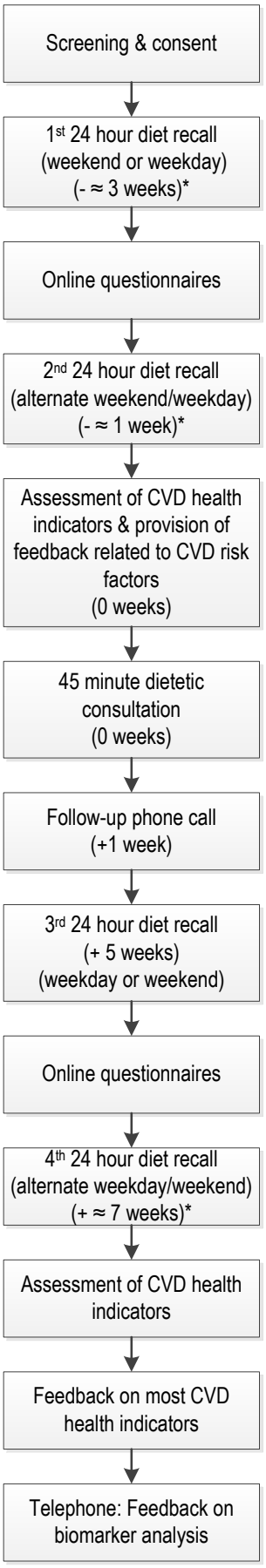

Figure 8.1: Study timeline. *Note that time between recalls and counselling was dependent on completion of online questionnaires, day of original recall and scheduling of appointments.

8.4.2 Participants

Individuals were eligible to participate if they were aged between 18-75 years, had internet access, were not on lipid lowering medication and had one or more of the following: LDL cholesterol ≥4.00mmol/L; total:HDL ratio ≥5; total cholesterol ≥5.00mmol/L (14). Participants currently on medication for hyperlipidaemia were eligible if their treating medical practitioner provided written clearance to halt medication for the duration of the study, and participants undertook a six-week washout period. Those with medical conditions affecting dietary intake (such as coeliac disease) or requiring medication for thyroid conditions were excluded. Written consent was obtained from all participants. Ethics approval was obtained from the University of Newcastle Human Research Ethics Committee (H-2013-0420). Participants were recruited via media releases from the University of Newcastle and the Hunter Medical Research Institute (HMRI), advertising on notice boards within the university setting, invitations through the HMRI volunteer research register, and by word of mouth.

8.4.3 Baseline screening

Online questionnaires were used to obtain demographic data, health characteristics, usual dietary intake and preferences for food products related to CVD health. To inform dietary feedback and counselling, usual dietary intakes over the last 6 months were derived from the Australian Eating Survey (AES) (15, 16). The AES is a validated 120 item semi-quantitative food frequency questionnaire (FFQ) that assesses frequency of usual intake over the past six months (15, 16). AES nutrient intakes were computed using FoodWorks version 4.00.1158 (Xyris Software, 2005), the Australian AusNut 1999 nutrient database (All Foods, Revision 17) and AusFoods (Brands, Revision 5).

8.4.4 Dietary outcome measures

To determine intervention effects on diet, dietary intake was assessed using a three-pass 24-hour recall on two occasions (one weekend day; one week day) at both baseline and follow-up, and was performed by one researcher (TS) (17-19). Prior to baseline dietary assessment, each participant was issued with a booklet of

two dimensional food models that included a reference scale to assist with portion size estimation. Entries obtained from 24-hour recalls were matched to a food from the AUSNUT 2011-13 food nutrient database by one research dietitian and assessed for comparability by another. Foods not found in the database were matched to foods of similar nutrient content. Quantities were converted to gram measures using values from the AUSNUT food measures database where necessary.(20, 21) Energy-dense, nutrient-poor foods were categorised according to the Australian Health Survey discretionary food list.(22) Nutrient intake values for 24-hour recalls were calculated using Stata/IC 13.1 by multiplying gram measures by nutrient values for each individual food (23).

8.4.5 CVD-related health outcome measures

Blood samples collected after an overnight fast were assayed for blood lipid concentrations, glucose, insulin, and inflammatory markers at an accredited pathology service laboratory (National Association of Testing Authorities, Newcastle, Australia) at baseline and follow-up, except insulin, which was measured only at baseline unless a result ≥10mIU/L was found. Brachial and central blood pressure and arterial stiffness measures (augmentation index) were obtained with the Pulsecor Cardioscope II (Pulsecor Ltd., New Zealand) using WelchAllyn FlexiPort reusable blood pressure cuffs. Participants were seated for five minutes before the first measurement occurred and repeat measures were taken at two-minute intervals until two consistent measures were obtained. Participants' height and weight were measured in light clothing to 0.1cm and 0.1kg, respectively using the Biospace BSM370 Automatic BMI Scale Stadiometer (Biospace Co. Ltd, Korea) and used to calculate body mass index (BMI, kg/m^2). Waist circumference was measured at the narrowest point between the lower costal (10^{th} rib) border and the top of the iliac crest using a non-extensible steel tape (KDFS10-02, KDS Corporation, Osaka, Japan). Physical activity was assessed using the International Physical Activity Questionnaire (IPAQ) long form for the previous seven days (24, 25).

8.4.6 Intervention theoretical framework

The intervention was based on a Protection Motivation Theory (PMT) framework, which describes health behaviours as responses to perceptions of threats of vulnerability and their severity (26). The theory posits that individuals determine the benefits and/or usefulness of performing adaptive (helpful) or mal-adaptive (harmful) behaviours, then assess their confidence to perform the given action (26). The study sought to use awareness of personal CVD risk as motivation and integration of behaviour change strategies to build self-efficacy for helpful dietary intake tasks, such as increasing fish and reducing energy-dense, nutrient-poor foods, and to provide meaningful feedback on performing the recommended strategies.

8.4.7 Intervention

Following baseline assessments, written feedback was provided in the form of a booklet, including the individual's serum cholesterol results, anthropometric measures, and analysis of their usual dietary intake. Dietary feedback included macronutrient and micronutrient intakes, and percentage of energy contributed by core (nutrient dense) and energy-dense, nutrient-poor foods. Verbal feedback was provided in regard to CVD risk factors identified, such as serum lipids, and was linked to dietary intake. Dietetic counselling immediately followed in a 45-minute session, which used a semi-structured presentation to ensure consistent delivery of the dietary components and goal setting strategies, whilst allowing for individualised counselling by the APD (Table 8.1). Participants were educated on and given a choice of dietary components informed by healthy eating guidelines and foods demonstrated to reduce CVD risk (3, 13, 27, 28). The Mediterranean diet is characterised by a high monounsaturated:saturated fat ratio, high intakes of legumes, fruits, vegetables, wholegrains, minimally processed cereals, regular intakes of fish and nuts, low intakes of red meats, and moderate intakes of dairy and alcohol (3, 29, 30). The Portfolio diet is a predominantly vegetarian diet with the addition of plant-based foods with lipid-lowering properties such as plant sterols, soy and vegetable protein, nuts and soluble fibres (13). Additional resources included a nutrition education manual specifically designed to

complement and extend the dietary information delivered as part of the intervention and included supplementary detail, food guides and recipes for targeted foods. Participants also received a grocery bag containing samples of shelf-stable recommended food products valued at approximately $50 AUD. This was to encourage participants to try foods that were not part of their usual diet, such as soy milk, low-sodium canned beans/legumes and canned oily fish. In the week following the intervention, participants received one follow-up phone call (approximately 10 minutes) from the APD to discuss progress and to troubleshoot any difficulties faced.

Table 8.1: Foods recommended and behaviour change strategies incorporated in the intervention. Foods targeted for dietary knowledge were informed by evidence for reducing CVD risk (3, 24) or improving serum lipids (10).

Dietary knowledge	Quantity	Example foods
Nuts	25-30g/day	Unsalted almonds, walnuts
Fish & omega 3 fats	2-3 serves/week	Fresh/canned salmon or tuna
Soy proteins	Up to 7 serves/day	Soy milk, tofu, tempeh
Lentils & legumes	Up to 7 serves/week	Kidney beans, lentils, chick peas
Soluble fibre	Up to 15g/day	Psyllium husk, oat bran, fruit, vegetables
Plant sterols	2-3 serves/day	Margarines/milk/cheese with added sterols
Healthy eating	As given by the Australian Guide to Healthy Eating (42)	
Behavioural technique (43)	**Illustration from intervention**	
Provide information about behaviour-health link	General information given about the types of foods and nutrients that increase blood triglyceride levels	
Prompt intention formation	Participants were provided with pantry items of recommended foods and recipes for their use	
Prompt specific goal setting	Participants were required to generate three specific personal goals	
Provide instruction	All participants were provided with 63-page resource manual with recommended serving sizes of food groups and food preparation methods	
Model or demonstrate the behaviour	Study participants were given breakfast upon completion of fasting study measures, with choices offered from a menu of items consistent with the recommended dietary advice	
Provide feedback on performance	Participants were given dietary feedback based on analysis of current intake	

*Other behaviour change techniques such as relapse prevention, time management or prompt barrier identification, may have been incorporated within the individual counselling session dependent on needs identified by accredited practising dietitian.

8.4.8 Follow-up feedback

Participants were provided feedback on their diet and physical health changes at the follow-up assessment session. Individual results of the post-intervention blood biomarker analyses were provided to participants via telephone when available.

8.4.9 Statistical analysis

A sample size of 33 participants was required to detect a 0.4 mmol/L change in the primary outcome measure of total cholesterol, based on a standard deviation of 0.8, power of 80% and an alpha of 0.05 (33). Allowing for 20% loss to follow-up, the study aimed to recruit 40 participants. Data were analysed as intention-to-treat by mixed model linear regression using Stata/IC 13.1(23). Analyses for total cholesterol, HDL and total:HDL ratio were adjusted for sex, BMI and physical activity. Analyses for triglycerides, LDL cholesterol, inflammation markers, glucose, blood pressure, and augmentation index were adjusted for sex and BMI. Analyses for dietary measures were adjusted for recall on weekend/weekday and sex, with percentage energy values adjusted for weekend/weekday recall only. Categorical data were analysed by using multinomial logistic regression, with standard errors adjusted for clustering to account for multiple time points.

8.5 Results

Participant flow through the trial is summarised in Figure 8.2, with 42 participants eligible and 39 participating in the intervention from February to December 2014. Time between baseline and follow-up was (mean ±SD) 9.5±2.5 weeks. Participants were aged 59.3 ±11.1 years (range 25-73), 72% female, 28 had completed education to higher than Year 10 or equivalent, and 18 had household incomes below $1000 AUD per week. Six participants discontinued their lipid-lowering medication prior to starting the trial, with one restarting after baseline results (follow-up lipid results excluded from analysis). Thirty-four participants reported one or more health conditions: high cholesterol (n=27), high blood pressure (n=13), arthritis (n=10), depression (n=4), asthma (n=3), type 1 and 2 diabetes (n=1 each). No participants were current smokers, but 20 were former smokers.

Physical activity levels were categorised by IPAQ as low (n=6), moderate (n=21) or high (n=12) at baseline and low (n=4), moderate (n=24) or high (n=10) at follow-up.

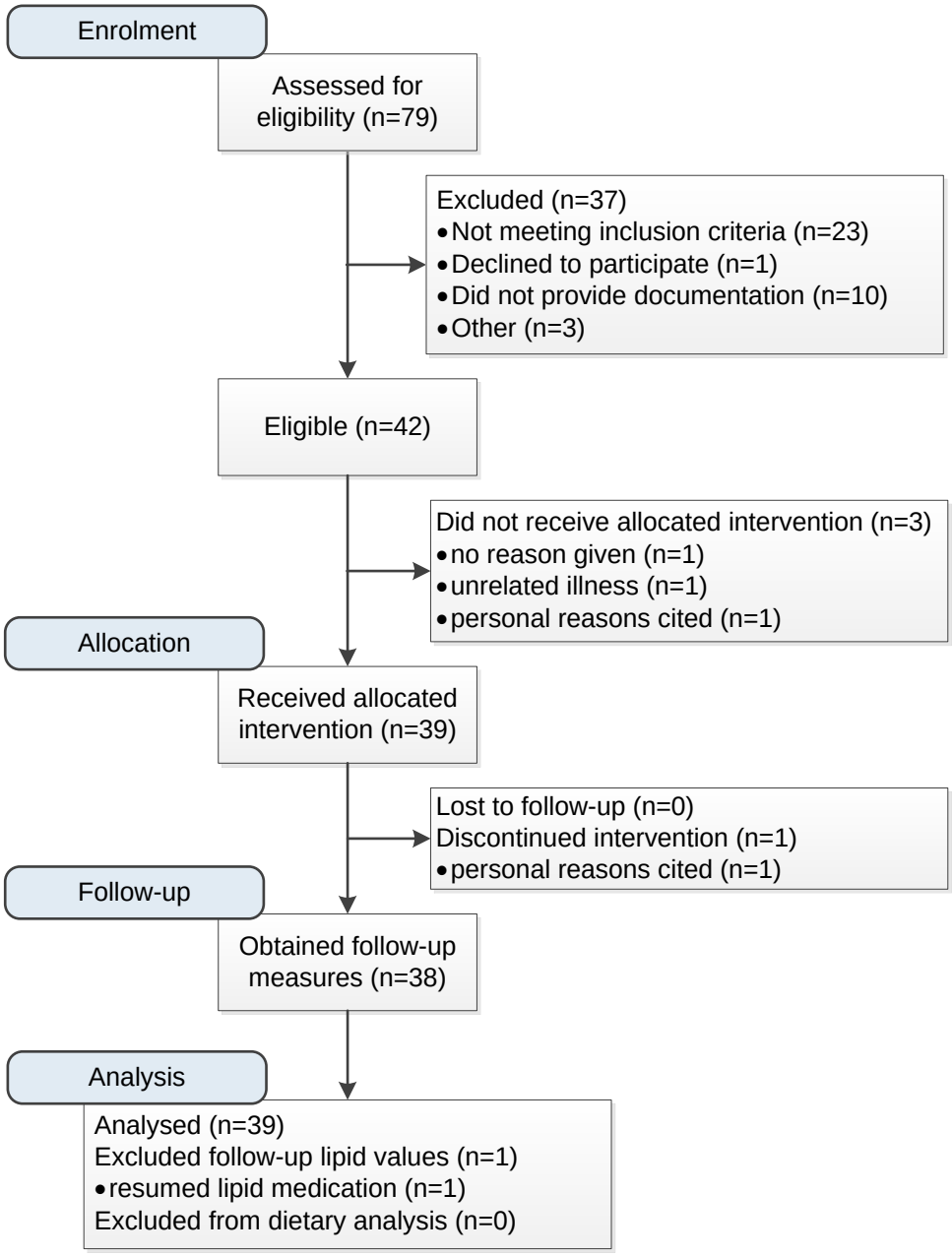

Figure 8.2: Recruitment, allocation and analysis flowchart

Table 8.2 summarises baseline, follow-up, and changes in CVD health indicator measures. Significant post-intervention reductions in triglycerides, LDL and total cholesterol, total:HDL cholesterol ratio were observed, including a small reduction in HDL cholesterol. For serum lipids, total cholesterol decreased by 7%, LDL-C by 6% and HDL by 3%, while the ratio of HDL:total cholesterol decreased by 5% and

triglycerides by 24%. Significant reductions in BMI, waist circumference and brachial blood pressure were also observed. No change was seen in inflammatory markers (high sensitivity C-reactive protein) or liver function (ALT, AST, GGT).

Table 8.2: Changes in cardiovascular risk factors following the dietary intervention. Data reported as mean±standard deviation, with 95% confidence intervals (CI) reported for change.

CVD health indicators	(Ref. range[π])	Units	Baseline (n=39)	Follow up (n=39)	Change[ϫ]	95% CI	P value
Triglycerides	<1.5	(mmol/L)	1.60±1.27	1.19±0.51	-0.38	-0.73, -0.03	0.03
Total cholesterol	<4.00	(mmol/L)	6.79±1.10	6.27±1.00	-0.51	-0.77, -0.24	<0.01
LDL cholesterol	<2.5	(mmol/L)	4.60±1.04	4.30±0.97	-0.28	-0.53, -0.04	0.02
HDL cholesterol	>1.0	(mmol/L)	1.46±0.34	1.43±0.35	-0.05	-0.10, -0.00	0.05
Total:HDL ratio			5.02±1.96	4.63±1.44	-0.27	-0.51, -0.04	0.02
hsCRP	<5.0	(mg/L)	2.25±2.26	2.22±2.23	0.1	-0.40, 0.60	0.70
ALT	0-45	(U/L)	25.9±15.1	27.8±14.7	2.0	-0.1, 4.1	0.07
AST	0-41	(U/L)	25.9±5.9	27.6±7.1	1.9	-0.5, 4.2	0.12
GGT	0-45 (F) 0-70 (M)	(U/L)	32.5±37.0	30.1±29.5	-1.1	-5.2, 3.1	0.62
BGL	3.0-6.0	(mmol/L)	5.16±0.57	4.94±0.59	-0.20	-0.32, -0.08	<0.01
Insulin[§]	<10	(mIU/L)	7.62±5.62				
HOMA IR score[Φ]		(mmol/L)	1.04±0.74				
BMI		(kg/m2)	28.1±5.7	27.6±5.7	-0.4	-0.7, -0.2	<0.01
Waist circumference		(cm)	92.3±15.9	90.2±14.5	-1.5	-2.3, -0.7	<0.01
Blood pressure[ɣ]		(mmHg)					
Brachial							
Systolic			125.9±17.2	121.1±16.0	-4.8	-8.3, -1.2	<0.01
Diastolic			75.9±7.3	73.2±8.6	-2.4	-4.1, -0.7	<0.01
Central							
Systolic			120.2±17.0	116.6±14.7	-3.6	-7.5, 0.2	0.07
Diastolic			77.2±7.3	75.0±8.7	-2.0	-4.0, 0.4	0.06
Augmentation index			108.4±56.8	99.8±34.8	-9.6	-23.1, 3.9	0.16

[π] Reference range (Australian) (44, 45)

[ϫ] Regression model includes sex & BMI. Coefficient reported for change. Regression model for HDL includes sex, BMI & PA.

[§] n=38 (type 1 diabetic excluded)

[Φ] n=36 (based on HOMA2 calculations; n=2 insulin values too low for calculation validity (46)). No follow-up measures taken if baseline value <10mIU/L

[ɣ] Regression model includes sex & BMI

BGL, blood glucose level; BMI, body mass index; Coef., Coefficient; F, females; HDL, high density lipoprotein; LDL, low density lipoprotein; M, males; PA physical activity; Ref, reference range

Table 8.3 summarises dietary intake measured by 24-hour recalls, indicating a substantial reduction in the proportion of energy from energy-dense, nutrient-poor foods. Macronutrient contributions remained relatively stable, although small but significant changes were found in protein, saturated and polyunsaturated fat, and sodium intakes. Fruit and vegetable intakes remained sub-optimal relative to national recommendations.

Table 8.3: Changes in dietary intakes of hyperlipidaemic participants obtained by 24-hour recall. Data presented as mean± standard deviation and 95% confidence interval for reported for change. Foods specific to cardiovascular health included.

	Baseline (n=39)	Follow up (n=39)	Change ⁑	95%CI	P value
Total energy (kJ/day)	9580±2695	8712±2614	-870	-16611, -130	0.02
Discretionary energy (kJ/day)Φ	3231±2058	2223±1821	-1006	-1563, -450	<0.001
% Discretionary energy	31.7±16.6	24.1±15.1	-7.5	-12.4, -2.7	<0.01
% Protein	17.4±4.4	18.9±4.6	1.5	0.1, 2.9	0.04
% CHO	45.0±10.0	43.6±9.9	-1.4	-4.4, 1.6	0.35
% Fats	33.6±8.7	32.9±9.7	-0.7	-3.7, 2.3	0.64
% sat. fat	11.4±4.0	9.8±4.5	-1.6	-2.9, -0.2	0.02
% mono. fat	13.5±5.2	12.9±5.2	-0.7	-2.4, 1.1	0.46
% poly. fat	6.0±2.9	7.2±2.8	1.3	0.48, 2.1	<0.01
Fibre (g)	29.2±10.2	29.3±10.0	0.1	-2.2, 2.5	0.91
Sodium (mg)	2764±1397	2410±1184	-349	-646, -53	0.02
Foods specific to cardiovascular health					
Fruit (g/day)	128±134	146±148	17	-20, 55	0.36
Vegetable (g/day)	239±212	229±186	-8	-58, 42	0.75
Nuts (g/day)	16.3±32.3	17.6±25.6	1.3	-7.7, 10.3	0.78
Fish (g/day)	29.4±62.9	44.0±68.4	13.5	-5.4, 32.5	0.16
Soy proteins (g/day)¥	1.0±2.9	2.9±4.3	2.0	0.9, 3.3	0.001
Legumes (g/day)	8.3±28.0	20.9±54.4	12.9	-0.33, 26.1	0.06
Fibre from oats/psyllium/linseed (g/day)*	1.7±3.0	2.4±3.5	0.7	-0.3, 1.8	0.16
Plant sterols (mg/day)	257±582	604±885	353	84, 623	0.01

⁑ Regression model includes adjustment for recall on weekend/weekday and sex. Coefficient reported for change. Regression model for percentage energy adjusted for recall on weekend/weekday only.

Φ Energy-dense, nutrient-poor foods classed as discretionary according to the Australian Health Survey discretionary food list (19)

¥ Only grams of soy protein reported as this is posited as the factor having lipid-lowering abilities

* Only grams of fibre reported as this is posited as the mechanism for lowering serum lipids

PE, percentage energy; CHO, carbohydrates; sat. fats, saturated fats; mono. fats, monounsaturated fats; poly. fats, polyunsaturated fats; g/day, grams per day; mg/day, milligrams per day.

Table 8.4 indicates that some dietary habits related to CVD health, such as using reduced fat cheese, were amendable to change ($p<0.01$).

Table 8.4: Changes in reported eating habits of foods related to CVD health

	Baseline % (n=39)	Follow up % (n=38)	P value *
TYPE OF MILK NORMALLY CONSUMED			
Don't drink milk	7.7 (n=3)	7.7 (n=3)	<0.05
Normal / whole / full cream	15.4 (n=6)	2.6 (n=1)	
Reduced fat	53.9 (n=21)	41.0 (n=16)	
Skim	12.8 (n=5)	15.4 (n=6)	
Soy	7.7 (n=3)	30.8 (n=12)	
Other/Not sure	2.6 (n=1)	2.6 (n=1)	
TYPE OF CHEESE NORMALLY EATEN			
Don't eat cheese	5.1 (n=2)	5.1 (n=2)	<0.01
Normal / full fat	56.4 (n=22)	20.5 (n=8)	
Reduced fat	28.2 (n=11)	43.6 (n=17)	
Low fat	10.3 (n=4)	30.8 (n=12)	
TYPE OF MEAT			
Don't eat meat	5.1 (n=2)	5.1 (n=2)	0.23
Normal / untrimmed	38.5 (n=15)	23.1 (n=9)	
Reduced fat / semi-trimmed	35.9 (n=14)	43.6 (n=17)	
Low fat / fully-trimmed	20.5 (n=8)	28.2 (n=11)	
ADDING OF SALT TO FOOD			
Never add salt	43.6 (n=17)	51.2 (n=20)	0.45
During cooking	23.1 (n=9)	23.1 (n=9)	
To meals	23.1 (n=9)	23.1 (n=9)	
Both meals & cooking	10.3 (n=4)	2.6 (n=1)	
PURCHASING SALT-REDUCED FOODS			
Never	18.0 (n=7)	12.8 (n=5)	0.05
Sometimes	66.7 (n=26)	51.3 (n=20)	
Always	15.4 (n=6)	35.9 (n=14)	

* p value obtained from multinomial logistic regression, with standard errors adjusted for clustering.

8.6 Discussion

Significant reductions in CVD risk factors were achieved through dietary changes implemented by hyperlipidaemic adults following a brief dietetic intervention based on individualised feedback and advice. Key intervention components were prior assessment of dietary intake using a purpose-designed online questionnaire

and access to current blood lipid results so that the single counselling session could be personally targeted, with a focus on providing feedback and education, with support via purposed designed resources. Results suggest that a brief dietetic intervention, suitable for widespread use in primary care settings, can have a significant impact on diet-related CVD risk factors.

Participants achieved clinically meaningful improved serum lipid profiles, which can be attributed to dietary change, given that other confounding variables, such as physical activity, medication and smoking status were accounted for in the statistical analysis. Findings from the Cholesterol Treatment Trialists' Collaboration indicated that a 1.00 mmol/L reduction in serum LDL cholesterol following statin prescription reduced the major vascular events rate ratio by 0.62, 0.69 and 0.79 in those at <5%, ≥5 to<10% and ≥10 to <20% five-year risk, respectively (37). An additional benefit of achieving reductions in LDL cholesterol secondary to dietary change is the concomitant reduction in risk for other chronic conditions (38). Reductions in LDL (6%) and total cholesterol (7%) in the current study are similar to those achieved using a single-session community-based intervention by Gaetke *et al.,* where participants were free to choose their own food based on advice provided,(39) but were less than the 26% and 34% reductions reported by Tovar *et. al* in an intervention that specified menu plans and provided prescribed foods (40).

The dietary change responsible for the greatest reduction in energy intake in the current study was the reduction in energy-dense, nutrient-poor food consumption from 32% of total energy at baseline to 24% at follow up. Increased intakes of core foods were small and individually variable. There were no group differences in intakes of recommended foods, such as fruit and vegetables, and only small increases in legumes and soy. The significant increases in soy and plant sterol containing foods indicate that many were willing to increase intakes of these foods, but further strategies are needed to reach the recommended efficacious levels (41-43). There were no significant group improvements in reported intakes of nuts, fish, fruit, fibre or vegetables. However with the exception of vegetables, for which legumes may have been substituted, the point estimates were in the direction of improvement. Individual participants made changes in some but not all of the food

recommendations, and this varied based on personal preferences and baseline consumption. The current study provides evidence for the cumulative benefit of multiple small improvements in intakes of foods with known cardio-protective effects being sufficient to provide CVD-risk protection, even though individual food changes were not detected as statistically significant.

The PREDIMED study was a large scale intervention that provided evidence of the long-term impact of a Mediterranean style eating pattern on the primary prevention of CVD (3). Total fat intakes (33% energy) of those in the current study were substantially less than those in the PREDIMED study (41% post trial), although they were still higher than Australian adults at 31% (3, 44). Consequently there were slightly higher protein (3%) and carbohydrate (3%) intakes compared to PREDIMED. Total fibre intakes in the current study were adequate, although the types of foods consumed were unlikely to contain the level or viscosity of soluble fibres required to reduce risk factors for CVD (45, 46). Future quantification of soluble and insoluble fibre intakes could be a valuable tool to use in personalised feedback for those at elevated risk of CVD.

Strengths of the current study include the provision of specific, targeted and individually tailored feedback by a dietitian, made possible through the prior online assessment of dietary intake, provision of current blood lipid results, and an educational counselling session supplemented by a purpose-designed nutrition education manual. Feedback related the individual's current intakes to appropriate reference ranges and suggested targets in both written and verbal forms, allowing participants time to ask questions and consider results. The use of the Protection Motivation Theory as the intervention framework was appropriate, as participants self-identified as having high serum cholesterol levels (threat vulnerability) and were counselled to achieve self-efficacy through behavioural techniques (Table 8.1).

Limitations of the current study include the pragmatic study design, relatively small sample size and hence risk of type 2 error. An explanatory study design, such as a randomised controlled trial, may have increased internal validity and given greater proof of causality. However, this study has informed power calculations for

future intervention sample sizes, with the primary outcome based on changes in percentage energy of discretionary food choices. The study design was also not truly pragmatic, as an intervention suitable for everyday practice would not have been able to provide shelf-stable grocery products to induce experimentation with unfamiliar products. Given that the greatest dietary change was an approximate 1,000kJ/day reduction in discretionary food choices, experimentation with food choices as a behaviour change may not necessarily be of high value in a brief intervention. Changes in foods recommended for consumption several times a week, such as fish, may not have been accurately captured using the 24-hr recall dietary method, even though both a weekday and weekend day were assessed at each time point. It is possible that this also impacted on legume intake given that they may not be consumed every day (47). Misreporting is common when assessing dietary intake, although changes in biochemical variables and reduction in BMI in the current study provide support for the relative accuracy of reported dietary intakes (48). Although the age of the study population reflects those with elevated LDL cholesterol levels within the general population and heart disease currently has the highest mortality rate for women in Australia, this sample was overly represented by females relative to males (49, 50). The average nine week intervention period is not indicative of long-term dietary patterns. Moderating contextual factors include the university setting leading to an inherent higher level of trust in the dietary advice, and the majority of participants being recruited from a research volunteer register, hence potentially more likely to comply with recommendations. The number of contacts due to dietary recalls and screening for study entry meant that a relationship was already established between the dietitian and participant, thereby decreasing the time needed for this during the counselling session.

These findings support the usefulness of including well-structured dietetic counselling as an initial approach to manage increased risk of CVD conferred by diet. Participants significantly reduced their intakes of energy-dense, nutrient-poor foods, although few significant changes were seen in intakes of specific foods associated with serum lipid reduction. Individuals with hyperlipidaemia can make cumulative small changes to their diet based on the personalised feedback leading

to improved CVD risk markers. Recommendations for future practice include utilising validated methods of assessing usual intake prior to counselling sessions in this population and use of behaviour change strategies to tailored interventions to individuals.

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References

1. World Health Organization. Global status report on noncommunicable dieases 2014. Geneva: World Health Organization, 2015.

2. LeFevre ML, Force USPST. Behavioral counseling to promote a healthful diet and physical activity for cardiovascular disease prevention in adults with cardiovascular risk factors: U.S. Preventive Services Task Force Recommendation Statement. *Ann Intern Med*. 2014;161(8):587-93.

3. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013;368(14):1279-90.

4. Reid RD, McDonnell LA, Riley DL, Mark AE, Mosca L, Beaton L, et al. Effect of an intervention to improve the cardiovascular health of family members of patients with coronary artery disease: a randomized trial. *CMAJ*. 2014;186(1):23-30.

5. Imes CC, Lewis FM. Family history of cardiovascular disease, perceived cardiovascular disease risk, and health-related behavior: a review of the literature. *J Cardiovasc Nurs*. 2014;29(2):108-29.

6. Pomeroy SE, Cant RP. General practitioners' decision to refer patients to dietitians: insight into the clinical reasoning process. *Aust J Prim Health*. 2010;16(2):147-53.

7. Bhattacharyya OK, Estey EA, Zwarenstein M. Methodologies to evaluate the effectiveness of knowledge translation interventions: a primer for researchers and health care managers. *J Clin Epidemiol*. 2011;64(1):32-40.

8. Kessels RP. Patients' memory for medical information. *J R Soc Med*. 2003;96(5):219-22.

9. Prestwich A, Kellar I, Parker R, MacRae S, Learmonth M, Sykes B, et al. How can self-efficacy be increased? Meta-analysis of dietary interventions. *Health Psychol Rev*. 2014;8(3):270-85.

10. Artinian NT, Fletcher GF, Mozaffarian D, Kris-Etherton P, Van Horn L, Lichtenstein AH, et al. Interventions to promote physical activity and dietary lifestyle changes for cardiovascular risk factor reduction in adults: a scientific statement from the American Heart Association. *Circulation*. 2010;122(4):406-41.

11. Jeffery RW, Wing RR, Thorson C, Burton LR, Raether C, Harvey J, et al. Strengthening behavioral interventions for weight loss: A randomized trial of food provision and monetary incentives. *J Consult Clin Psychol*. 1993;61(6):1038-45.

12. An R. Effectiveness of subsidies in promoting healthy food purchases and consumption: a review of field experiments. *Public Health Nutr*. 2013;16(07):1215-28.

13. Jenkins DJ, Jones PJ, Lamarche B, Kendall CW, Faulkner D, Cermakova L, et al. Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. *JAMA*. 2011;306(8):831-9.

14. National Cholesterol Education Program Expert Panel on Detection Evaluation Treatment of High Blood Cholesterol in Adults. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106(25):3143-421.

15. Collins CE, Boggess MM, Watson JF, Guest M, Duncanson K, Pezdirc K, et al. Reproducibility and comparative validity of a food frequency questionnaire for Australian adults. *Clin Nutr*. 2014;33(5):906-14.

16. Burrows TL, Hutchesson MJ, Rollo ME, Boggess MM, Guest M, Collins CE. Fruit and vegetable intake assessed by food frequency questionnaire and plasma carotenoids: a validation study in adults. *Nutrients*. 2015;7(5):3240-51.

17. Haines PS, Hama MY, Guilkey DK, Popkin BM. Weekend eating in the United States is linked with greater energy, fat, and alcohol intake. *Obes Res*. 2003;11(8):945-9.

18. Nelson M, Erens B, Bates B, Church S, Boshier T. Protocol for the completion of a food consumption record: Individual 24-hour recall London: King's College; [cited 2014 January]. Available from: http://dapa-toolkit.mrc.ac.uk/documents/en/24h/24hr_Protocol_LIDNS.pdf.

19. Human Nutrition Research Centre. Nutrition in the Newcastle 85+ study. Overview of the 24hr Multiple pass recall United Kingdom: Newcastle University; [cited 2014 January]. Available from: http://dapa-toolkit.mrc.ac.uk/documents/en/Tra/Training_day_-_24hr_multiple_pass_recall.pdf.

20. Food Standards Australia New Zealand. AUSNUT 2011-2013 Food nutrient database file. 2014.

21. Food Standards Australia New Zealand. AUSNUT 2011-13 Food measures database file. 2014.

22. Australian Bureau of Statistics. Australian Health Survey: Users Guide, 2011-13 — Discretionary Food List. Canberra: Australian Bureau of Statistics; 2014.

23. StataCorp LP. Stata/IC 13.1 for Windows. College Station, Texas USA: StataCorp LP; 2014.

24. Craig CL, Marshall AL, Sjostrom M, Bauman AE, Booth ML, Ainsworth BE, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc*. 2003;35(8):1381-95.

25. IPAQ. Guidelines for Data Processing and Analysis of the International Physical Activity Questionnaire (IPAQ) - Short and Long forms, revised on November 2005. 2005.

26. Armitage CJ, Conner M. Social cognition models and health behaviour: A structured review. *Psychol Health*. 2000;15(2):173-89.

27. National Health and Medical Research Council. Australian Dietary Guidelines. In: National Health and Medical Research Council, editor. Canberra: Commonwealth of Australia; 2013.

28. Sofi F, Abbate R, Gensini GF, Casini A. Accruing evidence on benefits of adherence to the Mediterranean diet on health: an updated systematic review and meta-analysis. *Am J Clin Nutr*. 2010;92(5):1189-96.

29. Rees K, Hartley L, Flowers N, Clarke A, Hooper L, Thorogood M, et al. 'Mediterranean' dietary pattern for the primary prevention of cardiovascular disease. *Cochrane Database Syst Rev*. 2013;8:CD009825.

30. Trichopoulou A, Martinez-Gonzalez MA, Tong TY, Forouhi NG, Khandelwal S, Prabhakaran D, et al. Definitions and potential health benefits of the Mediterranean diet: views from experts around the world. *BMC Med*. 2014;12(1):112.

31. Australian Government, NHMRC. Australian Guide to Healthy Eating: NHMRC; 2013 [cited 2014 3 November, 2014]. Available from: http://www.eatforhealth.gov.au/guidelines/australian-guide-healthy-eating.

32. Abraham C, Michie S. A taxonomy of behavior change techniques used in interventions. *Health Psychol*. 2008;27(3):379-87.

33. Jenkins DJ, Kendall CW, Faulkner DA, Nguyen T, Kemp T, Marchie A, et al. Assessment of the longer-term effects of a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *Am J Clin Nutr*. 2006;83(3):582-91.

34. Tonkin A, Barter P, Best J, Boyden A, Furler J, Hossack K, et al. National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand: position statement on lipid management--2005. *Heart Lung Circ*. 2005;14(4):275-91.

35. LabInfo Test Directory: Pathology North/Health Pathology; 2015 [updated 7th May, 2015; cited 2015 21st May]. Available from: http://www.palms.com.au/php/labinfo/index.php?site=JHH.

36. Hines G, Kennedy I, Holman R. HOMA 2 Calculator. 2.2.3 ed. University of Oxford: Diabetes Trial Unit; 2013.

37. Cholesterol Treatment Trialists Collaborators. The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomised trials. *The Lancet*. 2012;380(9841):581-90.

38. Chiuve SE, Fung TT, Rimm EB, Hu FB, McCullough ML, Wang M, et al. Alternative dietary indices both strongly predict risk of chronic disease. *J Nutr*. 2012;142(6):1009-18.

39. Gaetke LM, Stuart MA, Truszczynska H. A single nutrition counseling session with a registered dietitian improves short-term clinical outcomes for rural Kentucky patients with chronic diseases. *J Am Diet Assoc*. 2006;106(1):109-12.

40. Tovar J, Nilsson A, Johansson M, Ekesbo R, Aberg AM, Johansson U, et al. A diet based on multiple functional concepts improves cardiometabolic risk parameters in healthy subjects. *Nutr Metab (Lond)*. 2012;9:29.

41. Jenkins DJ, Mirrahimi A, Srichaikul K, Berryman CE, Wang L, Carleton A, et al. Soy protein reduces serum cholesterol by both intrinsic and food displacement mechanisms. *J Nutr*. 2010;140(12):2302S-11S.

42. Bazzano LA. Effects of soluble dietary fiber on low-density lipoprotein cholesterol and coronary heart disease risk. *Curr Atheroscler Rep*. 2008;10(6):473-7.

43. Patch CS, Tapsell LC, Williams PG, Gordon M. Plant sterols as dietary adjuvants in the reduction of cardiovascular risk: theory and evidence. *Vasc Health Risk Manag*. 2006;2(2):157-62.

44. Australian Bureau of Statistics. 4364.0.55.007 - Australian Health Survey: Nutrition First Results - Food and Nutrients, 2011-12. Canberra: Commonwealth of Australia; 2014.

45. National Health and Medical Research Council. Nutrient reference values for Australia and New Zealand. In: Health Do, editor. Canberra: Commonwealth of Australia; 2006.

46. Vuksan V, Jenkins AL, Rogovik AL, Fairgrieve CD, Jovanovski E, Leiter LA. Viscosity rather than quantity of dietary fibre predicts cholesterol-lowering effect in healthy individuals. *Br J Nutr*. 2011;106(9):1349-52.

47. Mitchell DC, Lawrence FR, Hartman TJ, Curran JM. Consumption of dry beans, peas, and lentils could improve diet quality in the US population. *J Am Diet Assoc*. 2009;109(5):909-13.

48. Rasmussen LB, Matthiessen J, Biltoft-Jensen A, Tetens I. Characteristics of misreporters of dietary intake and physical activity. *Public Health Nutr*. 2007;10(3):230-7.

49. National Heart Foundation of Australia. Women and Heart Disease Forum Report. 2011.

50. Australian Bureau of Statistics. Australian Health Survey: Biomedical results for Chronic Diseases, 2011-12. Canberra: Commonwealth of Australia; 2013.

Chapter 9: Comparison of fatty acid intakes assessed by a cardiovascular-specific food frequency questionnaire with red blood cell membrane fatty acids in hyperlipidaemic Australian adults: A validation study

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9.1 Overview

This chapter describes the validation of a food frequency questionnaire designed to capture dietary intakes of foods specific to cardiovascular health. This dietary intake tool was developed as limited methods exist that can capture comprehensive information and quantify change in dietary patterns in Australian populations at increased risk of CVD. Ethics approval for this study can be seen in Appendix 25. Sample data relating to construction of the CVD-AES food frequency questionnaire can be seen in Appendix 33.

9.2 Abstract

Background: Limited dietary intake tools have been validated specifically for hyperlipidaemic adults. The Australian Eating Survey (AES) Food Frequency Questionnaire (FFQ) was adapted to include foods with cardio-protective properties (CVD-AES). The aims were to estimate dietary fatty acid (FA) intakes derived from the CVD-AES and AES and compare to red blood cell (RBC) membrane FA content.

Methods: Dietary intake was measured using the semi-quantitative 120-item AES and 177-item CVD-AES. Nutrient intakes were calculated using AUSNUT 2011-13. Fasting RBC membrane FAs were assessed using gas chromatography. Extent of agreement between intakes estimated by AES or CVD-AES and RBC membrane composition (% of total fatty acids) for linoleic acid (LA), alpha-linolenic acid (ALA), eicosapentanoic acid (EPA), docosapentaenoic acid (DPA), and docosahexaenoic acid (DHA) were assessed using Spearman's correlation coefficients, adjusted linear regressions, and Kappa statistics.

Results: Data from 39 participants (72% female, 59.3±11.1 years) indicate stronger positive correlations between RBC membrane FAs and CVD-AES dietary estimates compared to the AES. Significant ($p<0.05$) moderate-strong correlations were found between CVD-AES FAs and FA proportions in RBC membranes for EPA ($r=0.62$), DHA ($r=0.53$) and DPA ($r=0.42$), with a moderate correlation for LA ($r=0.39$) and no correlation with ALA. Significant moderate correlations were found with the AES for DHA ($r=0.39$), but not for LA, ALA, EPA, or DPA.

Conclusion: The CVD-AES provides a more accurate estimate of long chain fatty acid intakes in hyperlipidaemic adults, compared with AES estimates. This indicates that a CVD-specific FFQ should be used when evaluating FA intakes in this population.

9.3 Introduction

Diet influences hyperlipidaemia (1), which is an acknowledged risk factor for the development of cardiovascular disease (CVD) (2). Assessment of dietary intakes in free-living individuals is challenging due to the inherent limitations of self-report methods (3), therefore validation of dietary assessment tools is essential to provide an objective measure of dietary intake. Food frequency questionnaires (FFQs) are commonly used to estimate usual dietary intakes, as they are relatively inexpensive to administer and have lower respondent burden compared to other methods, such as food diaries. Disadvantages of FFQs include errors in the estimation of portion size and lack of specificity to target populations, such as those with hyperlipidaemia, due to the use of finite food lists (3). To address these limitations, it is important to customise dietary assessment by including additional food items specific to cardiovascular health, and following with validation of the modified FFQ.

One method of FFQ validation compares dietary intakes with blood or tissue biomarkers as an objective measure of the nutrient(s) of interest (4). In CVD, this includes long chain omega-3 fatty acids (LCn3 FA), which can be assayed in red blood cell (RBC) membranes as an indicator of dietary FA intakes (5). Importantly the essential FAs linoleic (LA) and α-linoleic acid (ALA) cannot be produced endogenously (6), therefore biological levels reflect dietary intake. ALA is the parent FA from which the LCn3 eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA) and docosahexaenoic acid (DHA) are derived but conversion of ALA to these LCn3 is limited (7). Therefore these LCn3 are of primary interest in dietary validation studies as biological markers for LCn3, including levels in plasma, red blood cell (RBC) membranes and adipose tissues, as the biomarker levels have been shown to be dose-dependent and reflect specific time scales of incorporation (5, 8). The Omega-3 Index reflects the amount of EPA and DHA found in the RBC

membrane compared to the total FAs (9). It is a recognised index that has been proposed as both a risk marker and risk factor for CVD, with Index levels <4% suggested as undesirable, 4-8% as intermediate risk and >8% as a target level for those at increased risk of CVD (9).

Higher intakes of LCn3 from food sources and dietary supplements predict lower risk of primary CVD events (10), although prevention of secondary events is inconclusive (11). The 2012 European Guidelines for CVD prevention recommend LCn3 supplementation in those with elevated triglycerides (12) and the Australian CVD secondary prevention guidelines recommend supplementation as a way to meet daily EPA and DHA intake targets, in combination with dietary choices (13). Consequently a proportion of those at increased risk of CVD are likely to take fish oil supplements (14). Therefore, it is important to include both dietary and supplementary sources of EPA and DHA within dietary validation studies specific to this population.

The Australian Eating Survey (AES) is a FFQ designed to assess dietary intakes in healthy populations (15). We have developed 66 supplementary questions specific to foods and nutrients related to cardiovascular health (16, 17), used in conjunction with the AES, to create the CVD-AES for hyperlipidaemic populations. Therefore, the aims of the current study were to determine and compare the validity of both the AES and CVD-AES in estimating FA intakes from all dietary sources using both the AES and CVD-AES compared to the FA content of RBC membranes in hyperlipidaemic adults.

9.4 Subjects and methods

9.4.1 Subjects and study design

This cross-sectional analysis was conducted in adults with hyperlipidaemia. Individuals were eligible to participate if they were aged between 18-75 years, had access to the internet, not currently on statins, and had serum levels of one or more of the following: LDL cholesterol ≥4.00mmol/L; total:HDL ratio ≥5; total cholesterol ≥5.00mmol/L. Exclusion criteria were medical conditions affecting dietary intake, for example coeliac disease or nut allergies, or requiring medication

for thyroid conditions, as soy foods are suggested to affect thyroid medication absorption (18). Participants were recruited via media releases from the University of Newcastle and the Hunter Medical Research Institute (HMRI), invitations through the HMRI research volunteer register, advertising on notice boards in the University setting and by word of mouth. Written consent was provided by all participants. Ethics approval was obtained from the University of Newcastle Human Research Ethics Committee (H-2013-0420).

Participants completed both the AES and CVD-AES questions consecutively online, and provided a fasting blood sample for RBC membrane FA analysis. The order of questionnaire completion (AES versus CVD-AES specific questions) was randomised using QuickCalcs Software (19), with participants stratified by sex and self-reported weight status (healthy weight/overweight/obese) and blinded to FFQ order.

9.4.2 Food Frequency Questionnaires

Usual dietary intake over the previous six months was assessed using both the AES and the CVD-AES. The AES is a 120-item semi-quantitative FFQ (15, 20). The CVD-AES contains 111 unmodified AES items, with nine AES items modified, replaced or expanded to become 39 questions, and a further 27 items added to assess foods not currently covered by the AES. The CVD-AES was developed to specifically assess intakes of nutrients and foods related to cardiovascular health, such as those from the Mediterranean (16) and Portfolio diets (17). This included food preparation methods, use of salt-reduced foods, foods containing plant sterols, soluble fibres, soy and vegetable proteins, as well as specific details about lean and oily fish consumption and types of fat consumed (12, 13, 16, 17).

The AES nutrient and FA analyses were updated to use the AUSNUT 2011-2013 database (21) and applied original frequencies and portion sizes to pre-existing AES questions. The CVD-AES analyses utilised the same database for nutrients and FAs. CVD-AES food preference questions were also used to modify some nutrient intakes derived from the AES FFQ and incorporated into the CVD-AES analysis (see Supplementary Table 1). For example, the CVD-AES questioned usual chicken preparation methods (fried, crumbed, skin on, skin removed), which were applied

to relevant chicken questions in the AES analysis for this study. All nutrient analyses were calculated using Stata/IC 13.1 for Windows.

Dietary supplement use was assessed via a question in the online demographic and health questionnaire (defined as an over-the-counter medication/multivitamin/ herbal supplement, with example answers of multivitamin and fish oil supplements provided) completed before each FFQ and dosage confirmed via telephone by a research dietitian. Individual nutrient values for EPA, DHA and DPA derived from LCn3 supplements are not available in either the AUSNUT food nutrient database 2011-2013 (21) or AUSNUT dietary supplement database (22). Consequently, supplement dosages of EPA and DHA were standardised as 180mg and 120mg respectively to fit within the LCn3 total provided by the AUSNUT supplement database with approximately 8% DPA applied, a level consistent with that found by Khoomrung et al (23).

9.4.3 Blood sample collection and RBC membrane fatty acid analysis

Blood samples were collected after an overnight fast by trained phlebotomists at an accredited pathology service (National Association of Testing Authorities, Newcastle, Australia). Samples for red blood cell (RBC) membrane fatty acid (FA) analysis using gas chromatography (GC) were collected into vials containing EDTA and were separated by centrifuging for 10 minutes at 3800 rpm at ambient room temperature. The RBC were transferred into Eppendorf tubes and stored at -80°C until analysed. RBC membranes were prepared for FA analysis by washing 500µL of RBCs with 12mL of 0.25M glucose solution and 12mL hypotonic tris buffer before storing on ice for 5 minutes, then centrifuging at 10000 rpm at 4°C for 10 minutes. Supernatant was removed by pipette and discarded. The RBC pellet was resuspended in 12mL glucose solution and 12mL hypotonic tris buffer, again stored on ice for five minutes and re-centrifuged for 10 minutes at 12000rpm. This process was repeated once before re-centrifuging at 15000rpm for 20minutes, with final supernatant removed by pipette and discarded. The dried pellet was resuspended in 250µL of glucose and 250µL of tris buffer, vortexed and stored in amber glass vial at -80°C until required for methylation. FAs were methylated and

total FA concentration determined using the validated method established by Lepage and Roy (24) as described previously (25). A mixture of methanol/toluene (4:1 v/v), containing C13:0 and C19:0 and BHT (0.12g/L) was added to the membrane sample. FAs were methylated by adding acetyl chloride drop-wise while vortexing and heating to 100°C for 1 hour. The sample was cooled and 6% K_2CO_3 added to stop the reaction. The sample was centrifuged at 3000rpm and 4°C for 10 minutes before the upper toluene layer was collected for GC analysis. GC analysis of the FA methyl esters was performed using a 30m x 0.25mm (DB-225) fused carbon-silica column, coated with cyanopropylphenyl (J & W Scientific, Folsom, CA). Sample FA methyl ester peaks were identified by comparing their retention times with a standard mixture of FA methyl esters and quantified using a Hewlett Packard 6890 Series Gas Chromatograph with Chemstations software (version A.04.02, Hewlett-Packard, Palo Alto, CA). The quantity of individual RBC membrane FAs was expressed as a percentage of total RBC membrane FAs. Omega-3 Index was calculated as the proportion of EPA+DHA as a percentage of total RBC membrane FAs (9).

9.4.4 CVD related health measures

Height and weight were measured in light clothing to 0.1cm and 0.1kg (respectively) using the Biospace BSM370 Automatic BMI Scale Stadiometer (Biospace Co. Ltd, Korea). Waist circumference was measured at the narrowest point between the lower costal (10^{th} rib) border and the top of the iliac crest using a non-extensible steel tape (KDFS10-02, KDS Corporation, Osaka, Japan). Brachial blood pressure was obtained with the Pulsecor Cardioscope II (Pulsecor Ltd., New Zealand) using WelchAllyn FlexiPort reusable blood pressure cuffs. Participants were seated for five minutes before the first blood pressure measurement, followed by repeated measures at two-minute intervals until two measures consistent within 10mmHg systolic and 5mmHg diastolic pressure were obtained, for a maximum of five measures.

9.4.5 Statistical analysis

All dietary data are reported as mean (95% confidence interval). Total energy intake was examined to determine whether randomisation order influenced reported dietary intake. The aim of the statistical analysis was to determine which FFQ was a better predictor of LCn3 found in RBC membranes. This was investigated through the strength of associations between each FFQ and RBC membrane FA levels, and examined in four ways. 1) Spearman's correlation coefficients were used due to non-normal distribution of FAs in RBC membranes (exceptions of linoleic acid and DHA with normal distributions). Correlation strength was described as poor <0.20, moderate 0.2-0.6 and strong >0.6. 2) Linear regressions using Hubert-White robust standard errors were used with 95% confidence intervals to explain the relationship between LCn3 in RBC membranes and corresponding nutrients from dietary intakes, while adjusting for other known influential factors. The *a priori* factors adjusted for in the regression models were total energy intake, age, sex, BMI and use of fish oil supplements. A backward stepwise approach was used whereby variables were removed if inclusion did not improve data fit and removal did not change the coefficients. R^2 values and coefficients (95% confidence interval) are reported. Crude and adjusted values for linear regressions are reported, with crude values only reported for correlations as adjustment made no difference. 3) The third analysis was to assess whether the FFQs were able to correctly classify participants into tertiles. The precision of the agreement of the categorical assessments of each FFQ and RBC membrane values was tested with weighted Kappa (K_w) statistics with both sets of data categorised into tertiles. 4) The Bland Altman method was used to examine the agreement in total LCn3 intakes between the two FFQs. The difference between the CVD-AES and AES (CVD-AES - AES) was plotted on the *y* axis and the mean plotted on the *x* axis ((CVD-AES + AES)/2), with 95% limits of agreements (LOA) provided. Analyses were performed using individual nutrient intakes in grams/milligrams, as assessed from each FFQ, and the corresponding percentage of FAs from total RBC membrane FAs, in Stata/IC 13.1 using an alpha level of 0.05.

9.5 Results

Seventy-five people applied for the study, of whom 33 were assessed and deemed as ineligible. Primary reasons for ineligibility were: did not provide proof of current serum lipid status (n=13), currently on lipid-lowering medication (n=8) and not hyperlipidaemic (n=7). Forty-two individuals consented to the study. Three participants dropped out prior to baseline measures being taken (no reason given (n=1), personal reasons cited (n=1), developed unrelated medical condition (n=1)). Included participant (n= 39) characteristics are summarised in Table 9.1. There was no significant difference in reported total energy intake based on randomisation order of FFQ completion (p=0.20).

Table 9.1: Characteristics of participants (n=39)

Characteristic	Mean ± SD or n (%)
Age (years)	59.3 ± 11.1
Female (n (%))	28 (72%)
BMI (kg/m^2)	28.1 ± 5.7
Weight (kg)	77.8 ± 17.6
Waist circumference (cm)	92.3 ± 15.9
Brachial blood pressure (mmHg)	
Systolic	125.9 ± 17.2
Diastolic	75.9 ± 7.3
Total cholesterol (mmol/L)	6.79 ± 1.10
HDL cholesterol (mmol/L)	1.5 ±0.34
HDL:total cholesterol ratio	5.02 ± 1.96
Triglycerides (mmol/L)	1.60 ± 1.27
Omega-3 supplement consumer (n (%))	16 (41%)
Smoking status (n (%))	
Currently smoking	0 (100%)
Previous smoker	20 (51%)
Never smoked	19 (49%)
Highest qualification achieved (n(%))	
University degree or higher	9 (23%)
Certificate or diploma	8 (21%)
Trade or apprenticeship	7 (18%
Year 10 or equivalent	7 (18%)

Table 9.2 summarises dietary intakes, including FA intakes, as assessed by the AES and CVD-AES and the corresponding percentage of total RBC membrane FAs. Participants had Omega-3 Indices ranging from 4-8% indicating they were in the intermediate CVD risk category.

Table 9.2: Estimated dietary intakes assessed by the AES (Australian Eating Survey) and the CVD-AES (with the additional cardiovascular disease (CVD) specific questions) and corresponding percentage distribution of fatty acids in red blood cell membranes. Data are presented as mean and 95% confidence interval.

	Estimated daily dietary intake (n=39)				% RBC membrane FA composition	
	AES		CVD-AES			
	Mean	(95% CI)	Mean	(95% CI)	Mean	(95% CI)
Energy (kJ)	9736	(8866 - 10605)	10944	(9944 - 11945)	-	-
Protein (% of energy)	17.5	(16.6 - 18.3)	17.7	(16.8 - 18.7)	-	-
CHO (% of energy)	47.2	(45.5 - 48.8)	44.5	(42.5 - 46.4)	-	-
Fibre (g)	33.2	(30.2 - 36.2)	38.4	(33.2 - 43.5)	-	-
Fats (% of energy)	31.3	(29.9 - 32.6)	32.6	(31.0 - 34.2)	-	-
- saturated	11.3	(10.5- 12.1)	11.8	(10.9 - 12.7)	-	-
- monounsaturated	12.7	(12.1 - 13.4)	13.7	(12.8 - 14.6)	-	-
- polyunsaturated	4.4	(4.1 - 4.6)	6.9	(6.0 - 7.9)	-	-
LA (g)	8.9	(8.1 - 9.8)	14.0	(11.6 - 16.3)	8.34	(7.99 - 8.69)
ALA (g)	1.1	(1.0 - 1.2)	4.9	(3.3 - 6.4)	0.16	(0.14 - 0.18)
EPA (mg)	104	(83 - 125)	350	(243 - 457)	1.42	(1.04 - 1.79)
DPA (mg)	75	(67 - 83)	139	(115 - 162)	2.63	(2.48 - 2.77)
DHA (mg)	156	(124 - 187)	383	(274 - 490)	5.48	(5.07 - 5.89)
Total LCn3 (mg)	335	(279 - 390)	897	(652 - 1141)	9.53	(8.71 - 10.35)
Omega-3 Index [a]	260	(208 - 312)	733	(520 - 945)	6.90	(6.17 -7.62)

RBC, red blood cell; FA, fatty acid; CHO, carbohydrates; LA, linoleic acid; ALA, alpha linolenic acid; EPA, eicosapentaenoic acid; DPA, docosapentaenoic acid; DHA, docosahexaenoic acid; LCn3, total long chain Omega-3 fatty acids; [a] Omega-3 Index calculated as EPA + DHA (mg) for dietary intake and percentage of (EPA+DHA) / total RBC membrane fatty acids.

Table 9.3 summarises crude Spearman's correlations and crude and adjusted linear regression analyses between percentage FAs in RBC membranes and dietary FAs as assessed by the AES and CVD-AES. Correlations were much stronger using the CVD-AES than the AES. Moderate to strong correlations were found for EPA, DHA and LCn3 FA using the CVD-AES whereas only small correlations were found for the AES FFQ. The CVD-AES correlations for the Omega-3 Index also exceeded those found in the AES. Poor correlations were found for LA with both FFQs. Regression coefficients were also stronger with the CVD-AES compared to the AES. Adjustment for use of fish oil supplements increased the amount of variation in RBC membrane FAs (R^2) explained by the AES (see Table 3, crude and adjusted R^2 values).

Table 9.3: Spearman crude correlations, and crude and adjusted regression analyses between participant (n=39) RBC membrane fatty acids and dietary fatty acids, as determined by the AES FFQ and the CVD-AES FFQ.

	Spearman's	Crude regression			Adjusted regression		
	ρ	β	(95% CI)	R^2	β	(95% CI)	R^2
Australian Eating Survey FFQ							
LA (g)	0.20	0.06	(-0.09, 0.21)	0.02	0.03	(-0.14, 0.19)	0.06[a]
ALA (g)	-0.07	-0.01	(-0.07, 0.05)	0.00	-0.04	(-0.12, 0.03)	0.07[b]
EPA (g)	0.25	2.10	(-2.52, 0.01)	0.01	0.87	(-3.27, 5.01)	0.29[c]
DPA (g)	0.11	0.64	(-4.79, 6.06)	0.00	0.24	(-5.17, 5.65)	0.22[c]
DHA (g)	0.39*	3.56	(-0.45, 7.57)	0.08	2.73	(-0.56, 6.01)	0.35[b]
Total n3 (g)	0.27	2.76	(-1.51, 7.03)	0.03	1.72	(-1.55, 4.99)	0.33[b]
Omega 3 Index [d]	0.37*	3.05	(-0.94, 7.03)	0.05	2.09	(-1.13, 5.31)	0.33[b]
CVD- Australian Eating Survey FFQ							
LA (g)	0.39*	0.06	(-0.00, 0.13)	0.19	0.05	(-0.03, 0.13)	0.23[a]
ALA (g)	0.05	-0.00	(-0.00, 0.00)	0.00	-0.00	(-0.01, 0.00)	0.12[b]
EPA (g)	0.62***	2.85***	(1.72, 3.97)	0.66	3.01***	(1.62, 4.40)	0.67[c]
DPA (g)	0.42**	3.02**	(1.04, 5.00)	0.23	2.34*	(0.04, 4.64)	0.32[c]
DHA (g)	0.53***	2.21***	(1.36, 3.07)	0.35	1.64**	(0.58, 2.693)	0.52[b]
LCn3 (g)	0.61***	2.54***	(1.66, 3.42)	0.58	2.28**	(1.08, 3.49)	0.62[b]
Omega 3 Index[d]	0.60***	2.53***	(1.66, 3.39)	0.55	2.24**	(1.02, 3.46)	0.62[b]

* p <0.05, ** p<0.01, *** p<0.001; [a] adjusted for energy, FOS and BMI; [b] adjusted for energy, FOS, BMI and sex; [c] adjusted for energy, FOS, BMI and age; [d] Omega-3 Index calculated as EPA + DHA (mg) for dietary intake and percentage of (EPA+DHA) / total RBC membrane fatty acids. Abbreviations: RBC, red blood cell; FA, fatty acid; LA, linoleic acid; ALA, alpha linolenic acid; EPA, eicosapentaenoic acid; DPA, docosapentaenoic acid; DHA, docosahexaenoic acid; LCn3, total long chain Omega-3 fatty acids; FOS, fish oil supplementation.

Table 9.4 reports the extent of agreement between tertiles of absolute FA intakes derived from the AES or CVD-AES and tertiles of percentage of total FAs in RBC membranes. Level of agreement, indicated by Kappa statistics, was greater with the CVD-AES than the AES, with non-significant values for ALA and DPA with both FFQs.

Table 9.4: Extent of the agreement between tertiles of absolute nutrient values derived from the AES or CVD-AES FFQs and tertiles for the corresponding percentage of fatty acids in RBC membranes.

FFQ / Variable	Same tertile	Adjacent tertile	Misclassified[ɤ]	Kappa (K_w)	p
		n=39 (100%)			
AES					
LA	18 (46%)	16 (41%)	5 (13%)	0.25	0.02
ALA	14 (36%)	14 (36%)	11 (28%)	-0.04	0.62
EPA	15 (38%)	18 (46%)	6 (15%)	0.13	0.14
DPA	13 (33%)	18 (46%)	8 (21%)	0.02	0.44
DHA	21 (54%)	12 (31%)	6 (15%)	0.31	<0.01
LCn3	15 (38%)	20 (51%)	4 (10%)	0.19	0.06
Omega 3 Index	19 (49%)	16 (41%)	4 (10%)	0.31	<0.01
CVD-AES					
LA	20 (51%)	14 (36%)	5 (13%)	0.31	<0.01
ALA	17 (44%)	14 (36%)	8 (21%)	0.13	0.14
EPA	20 (51%)	16 (41%)	3 (8%)	0.37	<0.01
DPA	15 (38%)	18 (46%)	6 (15%)	0.13	0.14
DHA	20 (51%)	14 36(%)	5 (13%)	0.31	<0.01
LCn3	25 (64%)	12 (31%)	2 (5%)	0.54	<0.001
Omega 3 Index	24 (62%)	12 (31%)	3 (8%)	0.48	<0.001

[ɤ] Misclassified: Classified at extreme categories; LA, linoleic acid; ALA, alpha linolenic acid; EPA, eicosapentaenoic acid; DPA, docosapentaenoic acid; DHA, docosahexaenoic acid; LCn3, total long chain Omega-3 fatty acids;

Figure 9.1 indicates that at lower levels of intakes there was only a small difference between the total LCn3 intakes measured by both FFQs, with greater differences observed at higher intakes, particularly amongst those taking LCn3 supplements. However, in the non-supplemented group, all values fell within the 95% LOA.

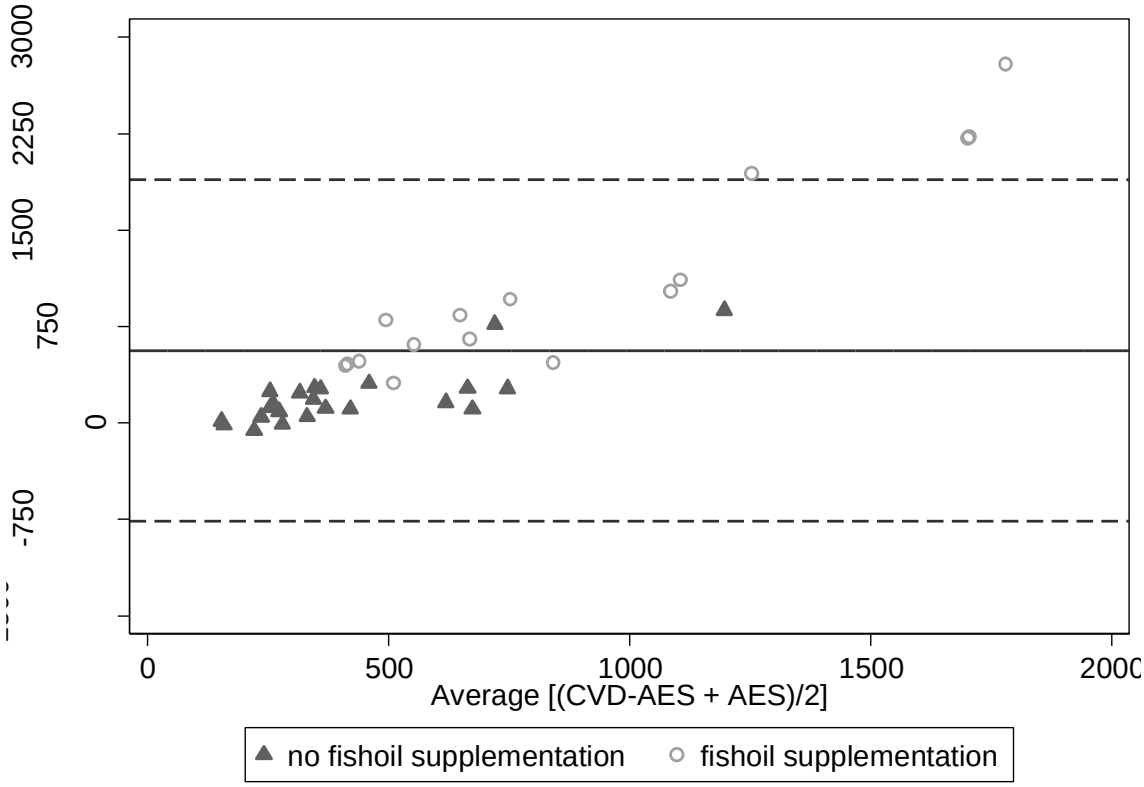

Figure 9.1: Bland Altman plot of the difference in total long chain omega 3 fatty acids as measured by both the CVD-AES FFQ and AES FFQ. The solid line in the centre represents the mean difference between the two FFQs, with the 95% limits of agreement represented by the dashed lines. Values from individuals taking fish oil supplements have been differentiated (see key above) to highlight the contribution of LCn3 from fish oil supplements.

9.6 Discussion

The current study investigated whether the addition of specific food items, associated with CVD risk (16, 17), to a standard FFQ (AES) to create a CVD-specific FFQ (CVD-AES) provided a better estimate of FA intakes in hyperlipidaemic adults. Using RBC membrane FA content as a biomarker of FA intake verified that this CVD-specific FFQ provided a better estimate of FA intake as shown by the stronger correlations and a reduction in the proportion misclassified by tertiles of intakes. An additional benefit of using the CVD-AES in this population was that it attempts to capture supplementary LCn3 intake by including questions about usage of fish oil supplements, which 41% reported consuming regularly. Those reporting LCn3 supplementation were provided with a standard LCn3 value that was applied to the respondent's frequency of consumption. The food items included in the CVD-

AES (for example, intake of oily fish) are specifically related to CVD risk (26). These additions are important and indicate that long chain FA intake would not be adequately captured using the AES FFQ, which was designed for use in the general population (see Supplementary Table 1 in Appendix 33). This potentially allows for the use of this FFQ in dietary interventions specifically targeting modification of long chain fatty acid intakes, which could be used to provide more appropriate and personally tailored dietary advice and monitoring.

A systematic review by Serra-Majem *et al.* (26) reported the range of significant correlations between FFQ and RBC membrane fatty acids in various populations, with crude and adjusted Spearman and Pearson correlations ranging for EPA (0.23-0.40), DHA (0.39-0.56) and total Omega-3 (0.41-0.50), with weaker correlations reported for ALA (0.18) and DPA (0.01). Subsequently, Turcot *et al.* (27) modified an existing 14-item FFQ for use in 137 participants (62% with hyperlipidaemia). They reported adjusted Spearman's correlations for ALA of 0.23 and 0.18 for total n3, with values for EPA and DHA of 0.29 and 0.41 respectively, which were consistent with the range of correlations found by Serra-Majem *et al.* More recently Allaire *et al.* (28) reported higher adjusted Spearman correlations of 0.59, 0.59 and 0.55 for EPA, DHA and LCn3, respectively ($p<0.01$) using a larger range of questions (136 questions with 40 sub-questions) in a sample of 60 men with prostate cancer. The results from the current study are similar to those of Allaire *et al.*, as would be expected based on the greater number of questions used to identify food intakes related to CVD, as in the current study. Patterson *et al.* (29) developed a 37-item FFQ to assess dietary sources of LCn3, including those from functional foods and supplements available in the Canadian food supply. RBC and plasma were analysed together as whole blood in 78 healthy participants to evaluate EPA and DHA intakes with statistically significant ($p<0.01$) unadjusted Pearson correlations of 0.31 for EPA and 0.42 for DHA. This provides further support for the use of a CVD specific FFQ when seeking to measure dietary intake relative to CVD risk.

Strengths of the current study include the use of the most recent and extensive nutrient database in Australia and the use of RBC membrane FAs as biomarkers of dietary fat intake. However the study has some limitations that need to be

acknowledged. Firstly, the relatively small sample size reduces the external validity of the results, but can be contextualised to adults with hyperlipidaemia. Intakes of functional foods containing LCn3 were not assessed, although limited items currently exist in the Australian food supply (21). Difficulties inherent in accounting for fish oil supplement usage have been highlighted by a review of the LCn3 content in currently available supplements (23, 30, 31). Actual quantities of EPA and DHA have been shown to be approximately 68% of the labelled supplement values (30) and can vary according to brand and country of origin (23, 30, 31). Whilst it is feasible to assess regular consumption of LCn3 supplements, it is beyond the scope of an FFQ to account for this type of variation. This will impact on the strength of correlations with FFQ data as incorporation of LCn3 into RBC membranes is dose responsive (8). Despite this, the CVD-AES detected significant relationships with most of the LCn3 FAs, that are also found in fish oil supplements. The length of time over which participants were asked to report usual intake was six months and this time period is sufficient to allow for incorporation of LCn3 into RBC membranes (5). Finally, having participants complete two FFQs consecutively may have introduced a source of bias, although no difference was found based on randomisation order.

This study demonstrated that a CVD specific FFQ that captures food items relevant to CVD risk is a better estimate of RBC membrane fatty acid content than a general FFQ in adults with hyperlipidaemia. The tailored CVD-AES FFQ improves accuracy in assessing dietary intakes relative to CVD risk in those with hyperlipidaemia. Further evaluation in the context of interventions to lower diet-related CVD risk is warranted.

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Conflict of interest

The authors declare no conflict of interest.

References

1. Franklin BA, Durstine JL, Roberts CK, Barnard RJ. Impact of diet and exercise on lipid management in the modern era. *Best Pract Res Clin Endocrinol Metab*. 2014;28(3):405-21.

2. Lewington S, Whitlock G, Clarke R, Sherliker P, Emberson J, Hasley J, *et al.* Blood cholesterol and vascular mortality by age, sex, and blood pressure: ci meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. *Lancet*. 2007;370(9602):1829-39.

3. Illner AK, Freisling H, Boeing H, Huybrechts I, Crispim SP, Slimani N. Review and evaluation of innovative technologies for measuring diet in nutritional epidemiology. *Int J Epidemiol*. 2012;41(4):1187-203.

4. de Vries J, Antoine JM, Burzykowski T, Chiodini A, Gibney M, Kuhnle G, *et al.* Markers for nutrition studies: review of criteria for the evaluation of markers. *Eur J Nutr*. 2013;52(7):1685-99.

5. Katan MB, Deslypere JP, vanBirgelen APJM, Penders M, Zegwaard M. Kinetics of the incorporation of dietary fatty acids into serum cholesteryl esters, erythrocyte membranes, and adipose tissue: an 18-month controlled study. *J Lipid Res*. 1997;38(10):2012-22.

6. Arab L. Biomarkers of fat and fatty acid intake. *J Nutr*. 2003;133 Suppl 3(3):925S-32S.

7. Arterburn LM, Hall EB, Oken H. Distribution, interconversion, and dose response of n-3 fatty acids in humans. *Am J Clin Nutr*. 2006;83(6 Suppl):1467S-76S.

8. Flock MR, Skulas-Ray AC, Harris WS, Etherton TD, Fleming JA, Kris-Etherton PM. Determinants of erythrocyte omega-3 fatty acid content in response to fish oil supplementation: a dose-response randomized controlled trial. *J Am Heart Assoc*. 2013;2(6):e000513.

9. Harris WS. The omega-3 index: clinical utility for therapeutic intervention. *Curr Cardiol Rep*. 2010;12(6):503-8.

10. Delgado-Lista J, Perez-Martinez P, Lopez-Miranda J, Perez-Jimenez F. Long chain omega-3 fatty acids and cardiovascular disease: a systematic review. *Br J Nutr*. 2012;107 Suppl 2:S201-13.

11. Kwak SM, Myung SK, Lee YJ, Seo HG, Korean Meta-analysis Study G. Efficacy of omega-3 fatty acid supplements (eicosapentaenoic acid and docosahexaenoic acid) in the secondary prevention of cardiovascular disease: a meta-analysis of randomized, double-blind, placebo-controlled trials. *Arch Intern Med*. 2012;172(9):686-94.

12. Perk J, De Backer G, Gohlke H, Graham I, Reiner Z, Verschuren M, *et al.* European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). The Fifth Joint Task Force of the European Society of Cardiology

and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts). *Eur Heart J*. 2012;33(13):1635-701.

13. National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand. Reducing risk in heart disease: an expert guide to clinical practice for secondary prevention of coronary heart disease. Melbourne: National Heart Foundation of Australia; 2012. p. 1-19.

14. Dickinson KM, Delaney CL, Allan R, Spark I, Miller MD. Validation of a Brief Dietary Assessment Tool for Estimating Dietary EPA and DHA Intake in Australian Adults at Risk of Cardiovascular Disease. *J Am Coll Nutr*. 2015;34(4):333-9.

15. Burrows TL, Hutchesson MJ, Rollo ME, Boggess MM, Guest M, Collins CE. Fruit and vegetable intake assessed by food frequency questionnaire and plasma carotenoids: a validation study in adults. *Nutrients*. 2015;7(5):3240-51.

16. Rees K, Hartley L, Flowers N, Clarke A, Hooper L, Thorogood M, *et al.* 'Mediterranean' dietary pattern for the primary prevention of cardiovascular disease. *Cochrane Database Syst Rev*. 2013;8:CD009825.

17. Jenkins DJ, Jones PJ, Lamarche B, Kendall CW, Faulkner D, Cermakova L, *et al.* Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. *JAMA*. 2011;306(8):831-9.

18. Jonklaas J, Bianco AC, Bauer AJ, Burman KD, Cappola AR, Celi FS, *et al.* Guidelines for the treatment of hypothyroidism: prepared by the american thyroid association task force on thyroid hormone replacement. *Thyroid*. 2014;24(12):1670-751.

19. GraphPad Software Inc. QuickCalcs La Jolla2012 [updated 12 December 2012; cited 2012 12 December 2012]. 2012:[Random number generator]. Available from: http://graphpad.com/quickcalcs/randomN1.cfm.

20. Collins CE, Boggess MM, Watson JF, Guest M, Duncanson K, Pezdirc K, *et al.* Reproducibility and comparative validity of a food frequency questionnaire for Australian adults. *Clin Nutr*. 2014;33(5):906-14.

21. Food Standards Australia New Zealand. AUSNUT 2011-2013 Food nutrient database file. 2014.

22. Food Standards Australia New Zealand. AUSNUT 2011-2013 Dietary supplement database. 2014.

23. Khoomrung S, Raber G, Laoteng K, Francesconi KA. Identification and characterization of fish oil supplements based on fatty acid analysis combined with a hierarchical clustering algorithm. *Eur J Lipid Sci Technol*. 2014;116(7):795-804.

24. Lepage G, Roy CC. Direct Transesterification of All Classes of Lipids in a One-Step Reaction. *J Lipid Res*. 1986;27(1):114-20.

25. Wood LG, Fitzgerald DA, Garg ML. Hypothesis: vitamin E complements polyunsaturated fatty acids in essential fatty acid deficiency in cystic fibrosis. *J Am Coll Nutr*. 2003;22(4):253-7.

26. Serra-Majem L, Nissensohn M, Overby NC, Fekete K. Dietary methods and biomarkers of omega 3 fatty acids: a systematic review. *Br J Nutr*. 2012;107 Suppl 2(SupplementS2):S64-76.

27. Turcot V, Brunet J, Daneault C, Tardif JC, Des Rosiers C, Lettre G. Validation of fatty acid intakes estimated by a food frequency questionnaire using erythrocyte fatty acid profiling in the Montreal Heart Institute Biobank. *J Hum Nutr Diet*. 2015;28(6):646-58.

28. Allaire J, Moreel X, Labonte ME, Leger C, Caron A, Julien P, *et al.* Validation of the omega-3 fatty acid intake measured by a web-based food frequency questionnaire against omega-3 fatty acids in red blood cells in men with prostate cancer. *Eur J Clin Nutr*. 2015;69(9):1004-8.

29. Patterson AC, Hogg RC, Kishi DM, Stark KD. Biomarker and dietary validation of a Canadian food frequency questionnaire to measure eicosapentaenoic and docosahexaenoic acid intakes from whole food, functional food, and nutraceutical sources. *J Acad Nutr Diet*. 2012;112(7):1005-14.

30. Albert BB, Derraik JG, Cameron-Smith D, Hofman PL, Tumanov S, Villas-Boas SG, *et al.* Fish oil supplements in New Zealand are highly oxidised and do not meet label content of n-3 PUFA. *Sci Rep*. 2015;5:7928.

31. Yi T, Li SM, Fan JY, Fan LL, Zhang ZF, Luo P, *et al.* Comparative analysis of EPA and DHA in fish oil nutritional capsules by GC-MS. *Lipids Health Dis*. 2014;13(1):190.

Chapter 10: Final discussion and recommendations for research and practice

10.1 Overview

The final chapter of this thesis begins with a summary of findings in relation to the hypotheses, then provides a brief discussion of each chapter and closes with making recommendations for practice and future research.

10.2 Review of chapter hypotheses and findings

10.2.1 Literature review: the role of diet in CVD prevention and treatment

The literature review sought to identify 1) diet and nutrition factors that impacted on the development of atherosclerosis, 2) methods that could quantify dietary patterns that were protective from CVD, 3) populations at increased CVD risk, and 4) the contribution of diet and nutrition to CVD prevention and treatment guidelines.

Nutrition is an important modifiable lifestyle factor in the development of atherosclerosis. Biochemical interactions exist between macro and micronutrients, which contribute to the development of atherosclerosis. Of particular importance is the quality of fats and carbohydrates. Overall dietary patterns are important, with those that include higher quantities of unrefined plant-based foods, and foods with a high nutrient density and relatively lower energy density, being protective in terms of CVD risk. Two such dietary patterns are the Mediterranean and Portfolio diets (1-3).

Although eating patterns recommended for health in general populations and eating patterns recommended to achieve a reduction in CVD-related risk factors

are similar for the most part, there are some differences specific to CVD. These include increased emphasis on nutrients such as LCn3 and sodium, high quality carbohydrates, including soluble fibres, as well as an increased ratio of unsaturated to saturated fats. Limited Australian tools existed to quantify efficacious dietary patterns associated with long-term CVD risk reduction with reasonable accuracy, low participant burden, wide affordability and validated specifically in a population with CVD or at high risk of CVD.

Behavioural risk factors such as smoking, physical activity and nutrition are well known risk factors and feature in CVD risk assessment and prevention guidelines (4-7). Risk factors such as ethnicity, sex, and a family history of premature CVD are commonly used to screen for genetic risk in the clinical setting. However, the future may well include individual genetic screening and gene expression for identifying CVD risk, and although not currently used in routine clinical practice, are likely to become more common place as technology advances (8).

10.2.2 Systematic review of dietary knowledge translation interventions for populations with CVD

The purpose of the systematic review was to identify aspects of successful translation studies for diet-related CVD prevention and treatment that could be replicated in clinical settings.

Hypothesis: Successful health service nutrition translation studies in CVD will follow a clearly defined protocol, yet allow flexibility in the delivery.

This hypothesis was not supported with results inconclusive due to translation studies being very heterogeneous. No studies were identified that had the primary outcome of translating nutrition knowledge, although this was inferred in studies through the study design and measures used. A wide array of outcome measures were used in populations which ranged from MI and heart failure patients through to health professionals likely to treat CVD patients.

Hypothesis: Methodology of dietary translation studies will be of low translation quality.

This hypothesis was supported. Few details were provided from the dietary studies as to how researchers translated their dietary knowledge to study participants, in a manner that would be of value to a practising clinician. This outcome was anticipated given that knowledge translation (KT) is a relatively recent area of published research and many of the major dietary CVD studies were published prior to KT becoming a specific research focus. Also, the quality of reported interventions was inconsistent, with many published prior to the release of the CONSORT guidelines (9). The consequences of inconsistent reporting of methodology and translation strategies were that although intervention programs clearly employed strategies to modify short and long-term dietary patterns, the strategies were not described in detail. This limited the identification of effective strategies and hence the opportunity for replication in a clinical setting.

10.2.3 Dietary patterns in overweight and obese children

Chapter 4 was a secondary data analysis that evaluated the association between CVD risk factors, nutrient intakes and overweight and obesity in primary school aged children enrolled in a larger RCT. Results indicated that boys in particular may be at elevated risk of CVD due to their eating patterns. Total fat, sugar and carbohydrate intakes were estimated to be significantly associated with having multiple risk factors for CVD beyond overweight and obesity in boys, whereas no relationship was seen in girls. This study highlights the importance of lifestyle interventions in overweight and obese children that target family dietary patterns, given this age group would be consuming a significant proportion of their intakes in the home environment. Further research is needed in a larger sample to evaluate sex-specific associations between nutrient intakes and CVD risk factors in pre-pubertal children.

Hypothesis: Overweight and obese children will be at increased risk of developing CVD.

The findings presented partially supported this hypothesis. This analysis showed that approximately 50% of the boys and 46% of the girls had at least one risk factor for CVD in addition to high BMI and waist circumference.

Hypothesis: Children in high CVD risk categories will have significantly poorer nutrient intakes compared to children in low CVD risk categories.

This hypothesis was partially supported as significantly different nutrient intakes were found in boys, but not in girls. In particular, boys at high risk had significantly higher total intakes of energy, fat, sugar and sodium compared to lower CVD risk boys.

Hypothesis: Sub-optimal anthropometrics related to adiposity will have significant associations with biomedical risk factors.

This hypothesis was supported by the study findings. Waist circumference and BMI were shown to have significant association with other CVD risk factors in both boys and girls. For example, boys with a waist circumference from a lower versus higher risk percentile band, as described by Mellerio *et al.*, were 14 times less likely to have additional CVD risk factors such as lipid abnormalities, with girls approximately five times less likely (10).

Overall, these results show the necessity of early intervention for these children if their risk of CVD is to be reduced. Targeting intakes of discretionary foods that are high in energy, fat, sugar and sodium is likely to be of value for boys. Decreasing total energy intake and increasing nutrient density will be of benefit to both sexes to assist with reduction of waist circumference and BMI.

10.2.4 Dietary patterns in adolescent girls

Chapter 5 was a secondary data analysis of the eating patterns of adolescent girls from areas of low socioeconomic status. The analysis showed that a high proportion(47%) of the foods the girls consumed were of low nutritional quality and not consistent with national dietary recommendations to prevent the

development of CVD and other chronic disease in later life (11). Populations who are socioeconomically disadvantaged are more likely to experience premature mortality due to chronic disease (7, 12). These findings indicate that dietary patterns are a likely contributing factor and highlights the vulnerability of young people with risk factors that predispose them for CVD, such as low income status and poor dietary patterns.

Hypothesis: Girls from low socioeconomic positions would have low intakes of core foods and disproportionally high intakes of energy-dense, nutrient-poor foods.

This was supported by the study findings as the contribution to total energy intake from nutritious core foods in this sample of girls (54%) was substantially less that the Australian average for girls of similar age (62%).

Hypothesis: Dietary behaviours in adolescent girls attending schools in low-income communities will vary based on weight status.

This hypothesis was not supported. No differences in dietary patterns were found based on weight status in this sample population. Approximately 57% of girls were assessed as healthy weight, with 2% assessed as underweight, 28% overweight and 14% obese. All weight status groups had similar proportions of total energy contributed by core and energy-dense, nutrient-poor, discretionary foods.

These findings indicate that a CVD risk reduction intervention in teenage girls may be more effective if it were tailored to accommodate the specific barriers to good nutrition and particular needs of those from low income communities, rather than focusing on weight status.

10.2.5 Feasibility of conducting a family-based dietary CVD intervention

This chapter investigated the feasibility of a dietary CVD prevention program for families with a member who had either experienced an adverse CVD event, or was at increased risk of doing so. The evidence-based intervention provided personally tailored dietary advice for secondary prevention of CVD events and primary

prevention for the accompanying younger members of the family who were at elevated risk of developing CVD based on shared genetic profiles, family environments and eating patterns. The intervention was based on findings from the literature review on efficacious dietary approaches to lowering CVD risk and incorporated dietary advice from the Mediterranean and Portfolio diets. The intervention also built on findings from the systematic review on possible strategies to translate this dietary knowledge. This study was challenging for a number of reasons. Firstly, the study was based on Protection Motivation Theory which states that people act in accordance to perceived threats. Similar to the findings in the AHS (2011-2012) (13), a proportion of participants in the FHF study did not identify as having CVD risk factors, such as adverse blood lipids or hypertension. Participants were difficult to recruit to the study and this suggests that these biomedical threats are unrecognised or insufficient to initiate dietary change and additional motivators may be required. Secondly, an initial assumption was made that a proportion of families volunteering for the study would have young children. However, most of the study recruits participated with children from much older age groups, who often had children of their own (i.e. grandparent and adult children). This indicated that those most interested in an intervention of this type were mature adults, aged 40-65 years for women and 45-70 years for men. Appointments for the family groups presented logistic challenges in regards to scheduling times convenient to all individuals. Also, whilst the intervention was structured so as to provide individualised feedback and counselling, the time was still limited in the face-to-face consultation to provide counselling specific to individuals.

Hypothesis: Families recruited into a dietary intervention study on the basis of one member having had an adverse CVD event, or being assessed at high risk of CVD, may be more receptive to changing their diet.

This hypothesis was not supported. Those identified at most risk of future CVD events, i.e. those attending cardiac or stroke rehabilitation, were least likely to consent to and/or complete the study. Highest retention rates came from those listed on a volunteer register or who responded to media releases, which suggests that these groups may be inherently more motivated and receptive to dietary

changes. As this hypothesis was not supported, it was further investigated by qualitative interviews with participants and these results were reported separately.

Hypothesis: Families will make small, incremental changes to a number of food groups to align dietary intakes with heart health recommendations.

The study did not recruit sufficient families to evaluate this hypothesis with confidence. This hypothesis was subsequently investigated in a study targeting individuals. Overall, no major dietary changes were seen, although improvements in intakes of core food groups and changes in some food habits, such as the reduction in full-fat dairy types and meat products, were reported. A study investigating which dietary recommendations targeting CVD risk reduction are most difficult to adhere to indicated that limiting consumption of red meats and full cream dairy products, as well as consuming the appropriate daily servings of fruits and vegetables, were difficult for people to achieve (14). Conversely, recommendations easier to comply with involved the use of unsaturated oils and controlling salt usage (14). Sex and age were factors that explained difficulty with compliance to recommendations. Males over the age of 34 found it particularly difficult to limit red meat consumption, whereas women found it more difficult to consume the required fruit and vegetable recommendations (14).

Qualitative data for this study showed that participants justified the healthiness of their dietary intakes in terms of 'good' foods and 'bad' foods. Some households felt their diet generally improved following the intervention by 2-3 points out of 10, which they believed was due to a range of factors such as increased knowledge and awareness of healthier eating patterns, more thoughtful shopping habits, more variety in fruits and vegetables, and experimentation with different food types.

Hypothesis: Families will find the overall program acceptable and highlight areas where improvements may be achieved.

This hypothesis was supported. Overall, participants felt the program provided relevant information, they utilised the resources supplied on multiple occasions and would recommend the program to others in a similar situation. An area

identified for improvement was managing the cost of an eating plan as a proportion of the household budget.

10.2.6 Qualitative investigation of preventative dietary patterns in the context of a family-based CVD prevention program

This chapter was a qualitative investigation into factors that may have impacted on achieving desired changes in dietary patterns identified by the previous pilot study. On completion of the pilot study, participants were invited to participate in telephone interviews to describe their experiences of the study, the impact of having their families participate with them, perceptions of their heart health, and how they felt heart health was affected by diet.

Hypothesis: Risk perception will correlate with actual risk in those identified at high risk of CVD.

This hypothesis was not supported. The interviews showed that although some participants at increased CVD risk recognised and acted on an accurate perception of their increased CVD risk, others with a similar level of risk were only moderately concerned.

Hypothesis: Healthy eating patterns will be recognised as an important component in reducing CVD risk.

This hypothesis was supported. All participants conveyed an understanding of the relationship between heart health and diet, although most thought that other lifestyle factors such as physical activity also played a similar role.

Hypothesis: Family will have considerable influence over dietary patterns.

This hypothesis was supported. Family members acted as both supports for and barriers to healthy eating. Although little actual confrontation was reported, many personal preferences were being catered for, to the detriment of some family members. Individuals were identified by other family members as “ringleaders”, who drove the involvement of family in healthful behaviours, while others acted as

"saboteurs", who made healthful behaviours more challenging for those around them.

These findings indicate perception of risk is unlikely to be of adequate strength to initiate and maintain dietary modification and that family are more likely to be either enablers or barriers to long-term behaviour change. Correctly identifying and accounting for the family member or members that act as barriers or enablers may assist in achieving dietary modification for individuals.

10.2.7 Brief dietetic intervention for hyperlipidaemia

Chapter 8 describes the efficacy of a brief dietetic intervention in a hyperlipidaemic population to reduce diet-related CVD risk factors. The intervention format was similar to that presented to families in the earlier chapters and adapted based on previous findings from the families. Key changes in testing the efficacy of the intervention were the removal of several barriers. Recruitment was changed to target individual adult participants, based on self-identified risk factors. Participants were able but not required to have a family member accompany them, and the focus on behaviour change strategies was increased during the counselling session. Most questionnaires were moved to an online format so they could be completed from home or work, at the convenience of the participants, before the face-to-face session, and intervention flow was redesigned by rationalising key CVD measures.

Changes in dietary patterns, such as elimination of energy-dense, nutrient-poor foods and more thoughtful shopping decisions that were referred to in the qualitative interviews in the previous intervention, were similarly repeated here following the single counselling session. Substantial reductions in energy-dense, nutrient-poor food intakes were achieved and changes in the types of dairy foods usually consumed occurred. Small, cumulative changes were achieved in intakes of core food groups and bioactive foods specific to CVD health. Total cholesterol decreased by approximately 7.5%, LDL cholesterol by 6% and TGs by 24%. Anthropometrics improved concurrently, with small reductions seen in BMI (0.4 kg/m^2), waist circumference (1.5 cm), and blood pressure (4.8 mmHg systolic and 2.4 mmHg diastolic). Blood glucose levels were also reduced by 4%.

Hypothesis: A brief dietetic intervention using best-available dietary evidence will improve lipid profiles.

This hypothesis was supported. Within an average 9.5±2.5 week timeframe, participants were able to reduce their total cholesterol by approximately 7.5% (0.51mmol/L), LDL cholesterol by 6% (0.28mmol/L) and triglycerides by approximately 24% (0.38mmol/L). The Cholesterol Treatment Trialists Collaboration showed that for each 1mmol/L reductions in LDL cholesterol by statins rates of major vascular events could be reduced by between 0.62 and 0.79, dependent on level of assessed risk (15).

These findings support the value of providing well-structured individually-tailored dietetic counselling sessions to populations at increased risk of CVD to address risk conferred by diet.

10.2.8 Validation of a CVD-specific food frequency questionnaire

The purpose of chapter 9 was to determine whether existing dietary intake tools were sufficient to capture changes in long-term dietary intakes associated with CVD health. A food frequency questionnaire previously validated in a healthy population (AES FFQ) was adapted to include an additional food list specifically related to CVD health (CVD-AES). It also accounted for LCn3 supplement use, as this population were expected to consume higher amounts than the general population. Both the AES and additional food list were completed by a sample population of hyperlipidaemic adults and the resulting dietary analysis compared to the fatty acid composition of RBC membranes as an objective validation marker. An FFQ was chosen in preference to other dietary intake methods based on the ability to 1) capture change in individual long-term eating patterns with low participant and administrator burden, and 2) identify intakes of concern at relatively low cost. These characteristics are relevant to consistent use and replication by clinicians in CVD populations.

Hypothesis: Existing dietary intake tools designed for healthy populations will require modification to improve dietary intake estimates in populations at increased risk of CVD.

This hypothesis was supported. The FFQ used in conjunction with the additional food list provided a better estimate of fatty acid intakes compared to the FFQ designed for capturing dietary intakes of healthy populations. One of the primary reasons for this was the addition of extra questions that focused on obtaining information relating to usual intake of food and supplementary sources of dietary fats, especially long chain omega 3 fats.

Limitations associated with the addition of extra questions include the propensity to over-estimate intakes and an additional time burden. This is offset by the extra detail captured about nutrients and foods important to individual CVD risk. Further research is required to validate the CVD-AES FFQ with additional questions for other areas shown to be of importance to CVD health, namely, fruit and vegetables, quantities of discretionary food choices, quality of carbohydrates and dietary variety.

10.3 Implications of the body of research

10.3.1 Implications for practice

The implications for practice from this body of research relate to health practitioners who are in a position to offer dietary advice for the prevention and treatment of CVD. This includes general practitioners as the health professionals most likely to initially assess CVD risk and prescribe treatments, health educators in clinical and outpatient services as these professionals are expected to have the most contact with individuals experiencing adverse CVD events, and dietitians as the professionals most likely to prescribe individual dietary treatments.

Although education is a core behaviour change strategy for health professionals, it is unlikely to lead to initiation and maintenance of long-term dietary behaviour change if used in isolation. An understanding of behaviour change theories relevant to CVD will assist in promoting lifestyle changes in high-risk individuals.

Risk awareness and readiness for change are two key factors related to behaviour change theory that will impact on the effectiveness of a prescribed treatment plan. As risk awareness may not be compatible with actual risk, it would be appropriate to assess an individual's current understanding and use this in conjunction with their readiness stage as part of developing an individualised treatment plan.

Family members may be either sources of support or negative influences in the process of adapting eating patterns to comply with heart healthy recommendations. Therefore, influential family members are encouraged to attend counselling sessions, so that they too may be assessed for risk perception and readiness for change, with the extent of their influence factored into the individualised treatment plan.

Results from the intervention studies undertaken as part of this thesis showed that personalised feedback in combination with other behaviour change strategies was effective in initiating awareness of diet-related risk. The feedback included references to what would be considered "healthy" or "desirable". For example, individual serum lipid results were related to reference values consistent with low risk of CVD and dietary intakes were related to recommended guidelines or estimated average requirements. The timeliness of this feedback was possible through the administration of an electronic FFQ which allowed for a minimal time lapse between data collection and transformation into meaningful data. This too has clinical implications for practitioners as detailed dietary data could be collected online prior to initial consultations, thereby reducing the time burden required for assessment of current dietary patterns.

Provision of comprehensive resources containing both individualised and generic information in combination with a brief follow-up session by telephone was another key strategy for initiating appropriate dietary behaviour changes. There is a natural limit to an individual's ability to process and retain information within a set time, such as in consultations. A brief follow-up session allows information from both the health professional and resources to be processed and considered, while providing an opportunity to clarify advice and recommendations. This also

provides the practitioner with an opportunity to assess barriers to change and counsel accordingly.

Results from the family-based prevention program highlighted the logistical challenges of multiple family members attending consultations together. Practice implications for overcoming this issue include utilising current and evolving telehealth technologies. Developing rapport with clients is an essential component of any therapeutic relationship and has traditionally been established using face-to-face contact. However, evolving communication technologies may be satisfactory substitute for clients with transportation, geographical or time-restraint challenges and improve access to specialist advice and services.

10.3.2 Implications for research

Implications for research include both primary and secondary prevention areas.

The systematic review showed that details presented in publications about dietary CVD treatment and prevention are limited in terms of how researchers assisted participants with initiating and maintaining changes in eating patterns aligned with the intervention recommendations. A consequence of this is that although a dietary pattern may be shown to improve outcomes for CVD patients, the methods by which the positive results were attained cannot be replicated in a clinical setting.

This research also identified that opportunities exist to target pre-pubescent overweight and obese children, particularly boys, to reduce their risk of developing CVD as adults. Teenage girls from areas of socio-economic disadvantage also had dietary patterns that increased their risk of CVD. However, the pilot study targeting families at increased risk of CVD described in this thesis showed that engaging this population was challenging.

It was shown that changes in dietary patterns could be initiated in a population with hyperlipidaemia, but it is not known whether these changes and their effects on serum lipids were sustained in the long-term. Resources designed for this type of intervention, specifically to assist in attaining these changes, were developed and found to be highly utilised in the family-based study and were then refined for

the subsequent study in hyperlipidaemic individuals. This showed there was a desire for comprehensive and trustworthy information of this nature, delivered by an APD. A FFQ specific to this population was also trialled and compared to a FFQ designed for healthy populations. The CVD-specific FFQ (CVD-AES) provided a better estimate of dietary intakes of LCn3 for this particular population, but validation for other CVD dietary risk factors such as sodium and fibre are required.

Therefore, research encompassing the following aspects of primary CVD prevention through dietary interventions is recommended:

- More detailed description of behaviour change strategies in CVD dietary intervention publications so as to allow replication of successful strategies in a clinical setting
- Further research into how to engage parents and their young offspring who may be at increased risk of CVD in preventive eating patterns
- Long-term assessment of dietary patterns following a single individualised dietetic counselling session
- Development of a set of easily-accessible food-based CVD prevention resources based on behavioural change strategies and appropriate for high-risk populations
- Validation of the CVD-specific FFQ in a larger population for other nutrients related to CVD risk factors.

This thesis also reviewed dietary models of care recommended by national guidelines for secondary prevention of CVD at the health service level. Improvements may be possible in the provision of lifestyle advice during admission as a hospital inpatient following an initial ACS diagnosis. Time admitted as an inpatient is limited, with most patients discharged within a week (16). An average ACS patient is in contact with a number of health professionals during this time, ranging from cardiologists and nurses, through to social workers, physiotherapists, and indigenous health workers. In addition, it may be a challenging time emotionally, and which may influence their understanding of the condition (17). However, risk perception following discharge has been shown to diminish in as little as three months following an event (17).

The current model of care provides for referral to CR services where a number of activities are undertaken to assist an individual to preserve or resume optimal functioning within their community (18). Dietary advice is one such service provided. However, little information exists on eating patterns prior to the ACS admittance and dietary changes made following completion of CR services. Newly released core components of CVD secondary prevention and cardiac rehabilitation services recommend documenting the percentage of patients receiving dietary education and the percentage of those who made changes in their dietary pattern (18). The challenge here is that dietary patterns for CVD health are difficult to quantify for those not trained in the area. Additionally, dietary education is usually performed in group settings in CR (19). Research provided in this thesis showed that individualised counselling is effective in initiating dietary changes, but it is uncertain whether the same level of dietary change can be achieved in a group setting for the same population.

Further research in these areas will add to the body of evidence in terms of the extent of the role that dietary patterns play in the recovery from primary events and prevention of secondary occurrences:

- Assessment of long-term dietary patterns during hospitalisation for patients admitted under an initial ACS diagnosis to be used as baseline for evaluating changes in dietary patterns
- Quantification of patients undertaking individual or group-based nutrition counselling based on referral, uptake and completion of services
- Collection and utilisation of dietary core component data from CR services to inform areas of future research, such as patients requiring a different approach to improve dietary patterns or identifying those that may require more intense or individual attention
- Comparison of changes in dietary patterns in individuals:
    - Not opting to utilise CR services
    - Undertaking group CR dietary education sessions
    - Referred to specialist dietary counselling
    - In receipt of dietary education whilst an inpatient

- Develop a model of care suitable for CR services that incorporate provision of personalised information and feedback
- The cost effectiveness of modification of dietary patterns for the prevention of secondary CVD events.

10.4 Concluding remarks

The research presented in this thesis indicates that there are a number of areas where improvements in translating current evidence into clinical practice for the prevention of CVD through diet and nutrition are possible and needed. Lifestyle, including diet and nutrition, is a major factor in the development of CVD, yet it remains one of the more challenging lifestyle aspects to modify in terms of CVD prevention. National dietary guidelines and recommendations are not adequately followed by a large proportion of the Australian population, leading to increased risk of CVD and other chronic lifestyle diseases, which is potentially modifiable. Dietary assessment and counselling can be an effective adjunct to CVD prevention, and thereby should be included routinely along with other factors, such as physical activity and smoking cessation, and medical treatments.

References

1. Sofi F, Abbate R, Gensini GF, Casini A. Accruing evidence on benefits of adherence to the Mediterranean diet on health: an updated systematic review and meta-analysis. *Am J Clin Nutr*. 2010;92(5):1189-96.

2. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, *et al.* Primary prevention of cardiovascular disease with a Mediterranean diet. *N Engl J Med*. 2013;368(14):1279-90.

3. Jenkins DJ, Jones PJ, Lamarche B, Kendall CW, Faulkner D, Cermakova L, *et al.* Effect of a dietary portfolio of cholesterol-lowering foods given at 2 levels of intensity of dietary advice on serum lipids in hyperlipidemia: a randomized controlled trial. *JAMA*. 2011;306(8):831-9.

4. National Vascular Disease Prevention Alliance. Guidelines for the assessment of absolute cardiovascular disease risk. National Heart Foundation of Australia; 2009.

5. Anderson TJ, Gregoire J, Hegele RA, Couture P, Mancini GB, McPherson R, *et al.* 2012 update of the Canadian Cardiovascular Society guidelines for the diagnosis and treatment of dyslipidemia for the prevention of cardiovascular disease in the adult. *Can J Cardiol*. 2013;29(2):151-67.

6. J.B.S. Board. Joint British Societies' consensus recommendations for the prevention of cardiovascular disease (JBS3). *Heart*. 2014;100 Suppl 2(Suppl 2):ii1-ii67.

7. Perk J, De Backer G, Gohlke H, Graham I, Reiner Z, Verschuren M, *et al.* European Guidelines on cardiovascular disease prevention in clinical practice (version 2012). The Fifth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of nine societies and by invited experts). *Eur Heart J*. 2012;33(13):1635-701.

8. Tikkanen E, Havulinna AS, Palotie A, Salomaa V, Ripatti S. Genetic risk prediction and a 2-stage risk screening strategy for coronary heart disease. *Arterioscler Thromb Vasc Biol*. 2013;33(9):2261-6.

9. Begg C, Cho M, Eastwood S, Horton R, Moher D, Olkin I, *et al.* Improving the quality of reporting of randomized controlled trials. The CONSORT statement. *JAMA*. 1996;276(8):637-9.

10. Mellerio H, Alberti C, Druet C, Capelier F, Mercat I, Josserand E, *et al.* Novel modeling of reference values of cardiovascular risk factors in children aged 7 to 20 years. *Pediatrics*. 2012;129(4):e1020-9.

11. NHMRC. Australian Guide to Healthy Eating Canberra: Commonwealth of Australia; [cited 2013 8 April]. Available from: http://www.eatforhealth.gov.au/guidelines/australian-guide-healthy-eating.

12. Australian Institute of Health and Welfare. Premature mortality from chronic disease. In: Australian institute of Health and Welfare, editor. Canberra: AIHW; 2010.

13. Australian Bureau of Statistics. Australian Health Survey: Biomedical results for Chronic Diseases, 2011-12. Canberra: Commonwealth of Australia; 2013.

14. Henson S, Blandon J, Cranfield J, Herath D. Understanding the propensity of consumers to comply with dietary guidelines directed at heart health. *Appetite*. 2010;54(1):52-61.

15. Cholesterol Treatment Trialists Collaborators. The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomised trials. *The Lancet*. 2012;380(9841):581-90.

16. Redfern J, Hyun K, Chew DP, Astley C, Chow C, Aliprandi-Costa B, *et al.* Prescription of secondary prevention medications, lifestyle advice, and referral to rehabilitation among acute coronary syndrome inpatients: results from a large prospective audit in Australia and New Zealand. *Heart*. 2014;100(16):1281-8.

17. Broadbent E, Leggat A, McLachlan A, Kerr A. Providing cardiovascular risk management information to acute coronary syndrome patients: a randomized trial. *Br J Health Psychol*. 2013;18(1):83-96.

18. Woodruffe S, Neubeck L, Clark RA, Gray K, Ferry C, Finan J, *et al.* Australian Cardiovascular Health and Rehabilitation Association (ACRA) core components of cardiovascular disease secondary prevention and cardiac rehabilitation 2014. *Heart Lung Circ*. 2015;24(5):430-41.

19. Briffa TG, Kinsman L, Maiorana AJ, Zecchin R, Redfern J, Davidson PM, *et al.* An integrated and coordinated approach to preventing recurrent coronary heart disease events in Australia. *Med J Aust*. 2009;190(12):683-6.

Appendices

Appendix 1. Statement of contribution and collaboration for Chapter 3

I attest that Research Higher Degree candidate Tracy Leigh Schumacher contributed to the following paper:

Schumacher T, Burrows T, Neubeck L, Redfern J, Callister R, Collins C. How dietary evidence for the prevention and treatment of CVD is translated into practice in those with or at high risk of CVD: A systematic review. Submitted to *Public Health Nutrition.*

Tracy L Schumacher contributed to study design and was primarily responsible for screening and reviewing titles, abstracts and full texts for inclusion, data extraction and manuscript preparation. Dr Lis Neubeck and Associate Professor Julie Redfern contributed to study design and manuscript preparation. Dr Tracy Burrows, Professor Robin Callister and Professor Clare Collins contributed to the study design, screening and reviewing of full texts for inclusion and manuscript preparation. All authors approved the final manuscript.

Ms Tracy Schumacher (25th November, 2015)

Dr Tracy Burrows (25th November, 2015)

Dr Lis Neubeck (4th December, 2015)

Associate Professor Julie Redfern (3rd December 2015)

Professor Robin Callister (3rd December, 2015)

Professor Clare Collins (25th November, 2015)

Professor Robert Callister (7th December, 2015)

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Appendix 2. Search strategy for systematic review

Table 1: Abbreviations and symbols used in search strategy

Abbreviations and symbols used below:	
/	After a term indicates that the MESH subject heading is used
*	After a term indicates that the term is truncated and will retrieve all words beginning with that set of letters
exp	Before an index term indicates that the term is exploded
.tw	Indicates a search for a term in the title and abstract fields
?	In the middle of a term indicates the use of a wildcard – used to identify alternate spellings
adj	Indicates a search for two or more terms where they appear adjacent to one another

Knowledge Translation Terms

Knowledge translation terms in Table 2were sourced from:

1. Armstrong R, Waters E, Dobbins M, Lavis JN, Petticrew M, Christensen R. Knowledge translation strategies for facilitating evidence-informed public health decision making among managers and policy-makers. The Cochrane Library. 2011.
2. Scott SD, Albrecht L, O'Leary K, Ball GD, Dryden DM, Hartling L, *et al.* A protocol for a systematic review of knowledge translation strategies in the allied health professions. Implement Sci. 2011;6: 58.
3. Scott SD, Albrecht L, O'Leary K, Ball GDC, Hartling L, Hofmeyer A, *et al.* Systematic review of knowledge translation strategies in the allied health professions. Implement Sci. 2012;7: 70.

Table 2: Search terms identified from relevant knowledge translation systematic reviews

TERM	COMMENTS
(Knowledge adj2 (application or broke* or creation or diffus* or disseminat* or exchang* or implement* or management or mobili* or translat* or transfer* or uptake* or utili*)).tw	
(Evidence* adj2 (exchang* or translat* or transfer* or diffus* or disseminat* or exchang* or implement* or management or mobil* or uptake* or utili*)).tw	
(KT adj2 (application or brok* or diffuse* or disseminat* or decision* or exchang* or implement* or intervent* or mobili* or plan* or policy or policies or strategy* or translat* or transfer* or uptake* or utili*)).tw	
(Research adj2 (diffus* or disseminat* or exchang* or transfer* or translation* or application or implement* or mobili* or transfer* or uptake* or utili*)).tw	
("research findings into action" or "research to action" or "research into action" or "evidence to action" or "evidence to practice" or "evidence into practice").tw	
("research utili*" and ("decision mak*" or decisionmak* or "decision-mak*" or "policy mak*" or "policy-mak*" or "policy decision*" or "health* polic*" or practice or action*).tw	
technology transfer.tw	
Technology transfer/	MESH Heading scope - Spread and adoption of inventions and techniques from one geographic area to another, from one discipline to another, or from one sector of the economy to another.
Diffusion of innovation/	MESH Heading scope - The broad dissemination of new ideas, procedures, techniques, materials, and devices and the degree to which these are accepted and used.
(Diffusion adj2 innovation).tw	
(systematic review* or "knowledge synthes*") adj2 ("decision mak*" or decisionmak* or "decision-mak*" or "policy mak*" or "policy-mak*" or "policy decision*" or "health* polic*" or application or implement* or utili*).ti,ab	
Research utili?ation.tw	
("evidence base*" or "evidence inform*") adj5 (decision* or plan* or policy or policies or practice or action*)	
Exp motivation/	MESH Heading scope - Those factors which cause an organism to behave or act in either a goal-seeking or satisfying manner. They may be influenced by physiological

Table 2: Search terms identified from relevant knowledge translation systematic reviews

TERM	COMMENTS
	drives or by external stimuli. Exploded includes: achievement, aspirations, conflict, drive, exploratory behaviour, food deprivation , goals, handling, instinct, intention, power, water deprivation
~~Exp~~ self efficacy/	MESH Heading scope - cognitive mechanism based on expectations or beliefs about one's ability to perform actions necessary to produce a given effect. It is also a theoretical component of behavior change in various therapeutic treatments. Cannot be exploded
Exp organizational innovation/	MESH Heading scope - Introduction of changes which are new to the organization and are created by management. Exploded will include: entrenreneurship
Exp diffusion of innovation/	MESH Heading scope - The broad dissemination of new ideas, procedures, techniques, materials, and devices and the degree to which these are accepted and used. Exploded will include: technology transfer
((research or evidence or guideline*) adj3 (implementation or utilization or utilisation or diffusion or translation)).tw	
(increase adj2 implementation).tw	
(predisposing or enabling or reinforcing) adj factor*).tw	
((support or imped*) adj change*)).tw	
((behavio?r adj2 change*)).tw	
(knowledge adj2 (utilization or utilisation or uptake or transfer* or implementation or dissemination or diffusion* or translation)).tw	
(implementation adj2 (program or strategy or strategies)).tw	
Decision making/	MESH Heading scope - The

Table 2: Search terms identified from relevant knowledge translation systematic reviews

TERM	COMMENTS
	process of making a selective intellectual judgment when presented with several complex alternatives consisting of several variables, and usually defining a course of action or an idea.
Research uptake	
Implementation research	Term found in …Bero L, Grilli R, Grimshaw JM, Harvey E, Oxman AD, Thomson MA. Closing the gap between research and practice: An overview of systematic reviews of interventions to promote implementation of research findings by health care professionals. *BMJ* 1998; 317:465–468.
KTE (knowledge translation and exchange)	
professional practice evidence based	

Nutrition Terms

Nutrition terms used in the search are shown in Table 3 below:

Table 3: Nutrition terms used within the search strategy

TERM	COMMENTS
Nutrition*.mp	Keyword search for all occurrences of nutrition, nutritional, etc
Nutrient*.mp	Keyword search for all occurrences of nutrient and nutrients
Feeding behaviour/	MESH Heading scope - Behavioral responses or sequences associated with eating including modes of feeding, rhythmic patterns of eating, and time intervals.
Eating/	MESH Heading scope - The consumption of edible substances.
Food/	MESH Heading scope - Any substances taken in by the body that provide nourishment.
diet*.mp	Keyword search for all occurrences of diet, diets, dietary, dietary intake
(Calor* or fat or carbohydrate* or protein* or energy or diet*) adj intake.tw	Keyword search for any occurrence of the words in brackets adjacent to intake
Fats/ Exploded to include **dietary fats** (butter, dietary cholesterol, ,margarine) and **unsaturated fats** (castor oil, cod liver oil, corn oil, cottonseed oil, linseed oil, safflower oil, sesame oil, soybean, triolein).	MESH Heading Scope - The glyceryl esters of a fatty acid, or of a mixture of fatty acids. They are generally odorless, colorless, and tasteless if pure, but they may be flavored according to origin. Fats are insoluble in water, soluble in most organic solvents. They occur in animal and vegetable tissue and are generally obtained by boiling or by extraction under pressure. They are important in the diet (DIETARY FATS) as a source of energy. (Grant & Hackh's Chemical Dictionary, 5th ed)
Dietary Fats, Unstaurated/ Exploded to include cod liver oil, corn oil, cottonseed, omega 3 fatty acids, safflower, sesame soybean oils)	MESH Heading Scope - Unsaturated fats or oils used in foods or as a food.
Nuts/	MESH Heading scope - Botanically, a type of single-seeded fruit in which the pericarp enclosing the seed is a hard woody shell. In common usage the term is used loosely for any hard, oil-rich kernel. Of those commonly eaten, only hazel, filbert, and chestnut are strictly nuts. Walnuts, pecans, almonds, and coconuts are really drupes. Brazil nuts, pistachios, macadamias, and cashews are really seeds with a hard shell derived from the testa rather than the pericarp.

Table 3: Nutrition terms used within the search strategy

TERM	COMMENTS
Phytosterols/ Exploded to include Brassinosteroids, Ecdysteroids, Ergosterol, Sitosterols, Stigmasterol	MESH Heading scope - A class of organic compounds known as STEROLS or STEROIDS derived from plants.
plant sterols.mp	Keyword search
Fruit/	MESH Heading Sope - The fleshy or dry ripened ovary of a plant, enclosing the seed or seeds.
Vegetables/	
Dietary Fiber/ Exploded to include prebiotics.	MESH Heading scope - The remnants of plant cell walls that are resistant to digestion by the alimentary enzymes of man. It comprises various polysaccharides and lignins.
Diet, Mediterranean/	

Cardiovascular Terms

Cardiovascular terms in Table 4were identified from two Cochrane systematic reviews relating to cardiovascular disease:

1. Hartley L, Flowers N, Lee MS, Ernst E, Rees K. Tai chi for primary prevention of cardiovascular disease. *The Cochrane Library*. 2014.
2. Hooper L, Bartlett C, Davey Smith G, Ebrahim S. Advice to reduce dietary salt for prevention of cardiovascular disease. *The Cochrane Library*. 2004.

Table 4: Cardiovascular terms identified as search terms

TERM	COMMENTS
Cardiovascular diseases/	Pathological conditions involving the CARDIOVASCULAR SYSTEM including the HEART; the BLOOD VESSELS; or the PERICARDIUM. Search using MESH heading. Not exploded as additional terms are too specific (pregnancy complications). However, relevant terms are included below.
cardio*.tw	
cardia*.tw	
heart*.tw	
coronary*.tw	
angina*.tw	
ventric*.tw	
myocard*.tw	
pericard*.tw	
isch?em*.tw	
emboli*.tw	
arrhythmi*.tw	
thrombo*.tw	
atrial fibrillat*.tw	
tachycardi*.tw	
endocardi*.tw	
exp Stroke/	MESH Heading scope - A group of pathological conditions characterized by sudden, non-convulsive loss of neurological function due to BRAIN ISCHEMIA or INTRACRANIAL HEMORRHAGES. Stroke is classified by the type of tissue NECROSIS, such as the anatomic location, vasculature involved, etiology, age of the affected individual, and hemorrhagic vs. non-hemorrhagic nature.
(stroke or stokes).tw.	
cerebrovasc*.tw	
cerebral vascular.tw.	

Table 4: Cardiovascular terms identified as search terms

TERM	COMMENTS
apoplexy.tw	
(sick adj sinus).tw.	
(brain adj2 accident*).tw.	
((brain* or cerebral or lacunar) adj2 infarct*).tw.	
exp Hypertension/	MESH Heading scope - Persistently high systemic arterial BLOOD PRESSURE. Based on multiple readings (BLOOD PRESSURE DETERMINATION), hypertension is currently defined as when SYSTOLIC PRESSURE is consistently greater than 140 mm Hg or when DIASTOLIC PRESSURE is consistently 90 mm Hg or more. Exploded includes Hypertension, Malignant, Hypertension, Pregnancy-Induced, Hypertension, Renal, Hypertensive Retinopathy, Masked Hypertension, White Coat Hypertension
hypertensi*.tw	
peripheral arter* disease*.tw.	
((high or increased or elevated) adj2 blood pressure).tw.	
exp Hyperlipidemias/	MESH Heading scope - Conditions with excess LIPIDS in the blood. Exploded includes Hypercholesterolemia, Hyperlipidemia, Familial Combined; Hyperlipoproteinemias; Hypertriglyceridemia
hyperlipid*.tw	
hyperlip?emia*.tw	
hypercholesterol*.tw	
hypercholester?emia*.tw	
hyperlipoprotein?emia*.tw	
hypertriglycerid?emia*.tw	
exp Arteriosclerosis/	MESH Heading scope - Thickening and loss of elasticity of the walls of ARTERIES of all sizes. There are many forms classified by the types of lesions and arteries involved, such as ATHEROSCLEROSIS with fatty lesions in the ARTERIAL INTIMA of medium and large muscular arteries. Exploded includes Arteriosclerosis Obliterans, Coronary Artery Disease, Intracranial Arteriosclerosis, Intermittent Claudication
cholesterol.tw	
“coronary risk factor*”.tw.	
Blood Pressure/	MESH Heading scope - PRESSURE of the BLOOD on the ARTERIES and other BLOOD VESSELS
blood pressure.tw.	

The following tables show results from the search strategies relating to individual databases used.

Table 5: Medline search terms and results

DATABASE: MEDLINE Name of Host: OVID Number of results: **1238 (1103 after de-duplication)** Date searched: 6th August 2013		
Set #	**Search terms**	**No of results**
1	(knowledge adj2 (application or broke* or creation or diffus* or disseminat* or exchang* or implement* or management or mobili* or translat* or transfer* or uptake* or utili*)).tw	6503
2	(evidence* adj2 (exchang* or translat* or transfer* or diffus* or disseminat* or exchang* or implement* or management or mobil* or uptake* or utili*)).tw	7098
3	(KT adj2 (application or brok* or diffuse* or disseminat* or decision* or exchang* or implement* or intervent* or mobili* or plan* or policy or policies or strategy* or translat* or transfer* or uptake* or utili*)).tw	179
4	(research adj2 (diffus* or disseminat* or exchang* or transfer* or translation* or application or implement* or mobili* or transfer* or uptake* or utili*)).tw	10747
5	("research findings into action" or "research to action" or "research into action" or "evidence to action" or "evidence to practice" or "evidence into practice").tw	6624
6	("research utili*" and ("decision mak*" or decisionmak* or "decision-mak*" or "policy mak*" or "policy-mak*" or "policy decision*" or "health* polic*" or practice or action*)).tw	413
7	technology transfer.tw	703
8	Diffusion of innovation/	13787
9	(Diffusion adj2 innovation).tw	282
10	(("systematic review*" or "knowledge synthes*") adj2 ("decision mak*" or decisionmak* or "decision-mak*" or "policy mak*" or "policy-mak*" or "policy decision*" or "health* polic*" or application or implement* or utili*)).ti,ab	148
11	research utili?ation.tw	539
12	(("evidence base*" or "evidence inform*") adj5 (decision* or plan* or policy or policies or practice or action*)).tw	11606
13	exp motivation/	127738
14	self efficacy/	11818
15	exp organizational innovation/	22260
16	exp diffusion of innovation/	15314
17	((research or evidence or guideline*) adj3 (implementation or utilization or utilisation or diffusion or translation)).tw	9101
18	(increase adj2 implementation).tw	119

Table 5: Medline search terms and results

DATABASE: MEDLINE
Name of Host: OVID
Number of results: **1238 (1103 after de-duplication)**
Date searched: 6th August 2013

Set #	Search terms	No of results
19	((predisposing or enabling or reinforcing) adj factor*).tw	12083
20	((support or imped*) adj change*).tw	881
21	(behavio?r adj2 change*).tw	12493
22	(knowledge adj2 (utilization or utilisation or uptake or transfer* or implementation or dissemination or diffusion* or translation)).tw	2759
23	(implementation adj2 (program or strategy or strategies)).tw	3422
24	Decision making/	67934
25	Research uptake.tw	21
26	Implementation research.tw	291
27	(KTE or "knowledge translation and exchange").tw	41
28	("professional practice" adj2 "evidence based").tw	3
29	Technology transfer/	1717
30	1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29	296154
31	nutrition*.mp	249936
32	nutrient*.mp	76272
33	Feeding behavior/	39084
34	Eating/	41955
35	Food/	23229
36	diet*.mp	489018
37	((Calor* or fat or carbohydrate* or protein* or energy or diet*) adj intake).tw	40347
38	exp Fats/	80303
39	exp Dietary Fats, Unsaturated/	24238
40	exp Fatty Acids, Omega-6/	13428
41	omega 9 fatty acids.mp	20
42	Nuts/	1956
43	exp Phytosterols/	7448
44	stanols.mp	291
45	plant sterols.mp	893
46	Fruit/	25497
47	Vegetables/	16351
48	exp Dietary Fiber/	13077
49	((soluble or insoluble) adj (fiber or fibre)).tw	817

Table 5: Medline search terms and results

DATABASE: MEDLINE
Name of Host: OVID
Number of results: **1238 (1103 after de-duplication)**
Date searched: 6th August 2013

Set #	Search terms	No of results
50	Diet, Mediterranean/	1367
51	31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43 or 44 or 45 or 46 or 47 or 48 or 49 or 50	817030
52	Cardiovascular diseases/	98944
53	cardio*.tw	477648
54	cardia*.tw	420111
55	heart*.tw	598617
56	coronary*.tw	284669
57	angina*.tw	44275
58	ventric*.tw	301960
59	myocard*.tw	283215
60	pericard*.tw	29381
61	isch?em*.tw	259370
62	emboli*.tw	81113
63	arrhythmi*.tw	63432
64	thrombo*.tw	254680
65	atrial fibrillat*.tw	34473
66	tachycardi*.tw	44849
67	endocardi*.tw	36828
68	exp Stroke/	82702
69	(stroke or strokes).tw	136690
70	cerebrovasc*.tw	35581
71	cerebral vascular.tw	4583
72	apoplexy.tw	2157
73	(sick adj sinus).tw	1850
74	(brain adj2 accident*).tw	121
75	((brain* or cerebral or lacunar) adj2 infarct*).tw	17333
76	exp Hypertension/	209235
77	hypertensi*.tw	301643
78	peripheral arter* disease*.tw	6878
79	((high or increased or elevated) adj2 blood pressure).tw	23278
80	exp Hyperlipidemias/	55691
81	hyperlipid*.tw	20768
82	hyperlip?emia*.tw	2143

Table 5: Medline search terms and results

DATABASE: MEDLINE
Name of Host: OVID
Number of results: **1238 (1103 after de-duplication)**
Date searched: 6th August 2013

Set #	Search terms	No of results
83	hypercholesterol*.tw	25581
84	hypercholester?emia*.tw	583
85	hyperlipoprotein?emia*.tw	4126
86	hypertriglycerid?emia*.tw	8943
87	exp Arteriosclerosis/	129403
88	cholesterol.tw	171520
89	"coronary risk factor*".tw	2966
90	Blood Pressure/	238344
91	"blood pressure".tw	210961
92	52 or 53 or 54 or 55 or 56 or 57 or 58 or 59 or 60 or 61 or 62 or 63 or 64 or 65 or 66 or 67 or 68 or 69 or 70 or 71 or 72 or 73 or 74 or 75 or 76 or 77 or 78 or 79 or 80 or 81 or 82 or 83 or 84 or 85 or 86 or 87 or 88 or 89 or 90 or 91	2428545
93	30 and 51 and 92	1937
94	limit 93 to english language	1822
95	limit 94 to humans	1325
96	limit 95 to yr="1985 -Current"	1238

Appendix 3. Systematic review quality checklist

Additional file 2: Quality checklist

Item	Description
RELEVANCE QUESTIONS	
1	Would implementing the studied intervention or procedure (if found successful) result in improved outcomes for the patients/clients/population group?
2	Did the authors study an outcome or topic that the patients/clients/population group would care about?
3	Is the focus of the intervention a common issue of concern to dietetics practice?
4	Is the intervention feasible?
VALIDITY QUESTIONS	
A	Was the research question clearly stated? Identification and clarity of stated independent and dependent variables considered, as well as the target population and setting.
B*	Was the selection of study participants free from bias? Inclusion and exclusion criteria were considered, as well as representativeness of sample population.
C*	Were the study groups comparable? Considered randomisation techniques, distribution of disease states and type of control group/s. Not applicable (N/A) to interventions without a comparison group.
D	Was the method of withdrawals described? Considered follow-up methods, loss to follow-up and reasons for withdrawals.
E	Was blinding used to prevent introduction of bias? Considered blinding of subjects, clinicians, investigators and data collectors. N/A to interventions without comparison group.
F*	Were procedures for both intervention and controls described in detail? Were intervening factors described? Considered intensity and duration of intervention applied, as well as patient exposure/compliance, co-interventions and unplanned treatments
G*	Were outcomes clearly defined and the measurements valid and reliable? Considered the relevance of the primary and secondary endpoints, the consistency, validity and precision of tools used for measurements, length of follow-up and effects of other factors.
H	Was the statistical analysis appropriate for the study design and outcomes? Considered description and adequacy of statistical tests used, e.g. intention to treat, adjusting for confounders etc., power calculation, reporting methods and clinical significance.
I	Are the conclusions supported by the results with biases and limitations taken into consideration? Considered biases and limitations identified and discussed by authors.
J	Is bias due to study funding or sponsorship unlikely? Considered sources of funding, declaration of investigator affiliations and sources of apparent conflict.

Additional file 2: Quality checklist

Item	Description
KNOWLEDGE TRANSLATION STRATEGY QUESTIONS	
K§	Did the authors provide a detailed description of the intervention? Considerations included details provided about: intervention deliverers; intervention recipients; setting details; mode of delivery; intensity and duration of intervention; adherence to study protocol; and the intervention content provided to each study group.
L§	Did the authors provide clarification of assumed change process and design principles? Considerations included details provided on the development of the intervention, change techniques used and causal processes.
M	Is there easy access to the materials used in the intervention?

***** indicates questions of high importance according to the ADA Evidence Analysis Manual quality criteria checklist [28]

§ indicates questions of high importance to the replication of the knowledge translation strategy [29].

Appendix 4. Methodological quality scores for systematic review

Additional file 3: Methodological quality scores and risk of bias assessment in nutrition knowledge translation studies for cardiovascular disease

Study	Relevance	Validity	KT strategy Overall
CARDIOVASCULAR DISEASE DIAGNOSIS			
Aish (1996), [30] Aish (1996) [31]	✓	×	×
Allen (1996) [32]	✓	Ø	✓
Arntzenius (1985) [33] Kromhout (1986) [34]	✓	×	×
Billings (2000) [35] Koertge (2003) [36]	✓	×	×
Campbell (1998) [37]	✓	Ø	×
Dalgard (2001) [[38]	✓	Ø	Ø
de Lorgeril (1994) [39] de Lorgeril (1999) [40]	✓	✓	×
Evon (2004) [41]	✓	Ø	×
Frost (2004) [[42]	✓	Ø	×
Giannuzzi (2008) [43]	✓	Ø	×
Gleason (2002) [44]	×	Ø	Ø
Goodwin (2012) [45]	✓	Ø	✓
Hofman-Bang (1999) [46]	×	×	×
Jackson (2005) [47] Jackson (2005) [48]	✓	Ø	×
Luszczynska (2007) [49]	✓	Ø	✓
Masley (2001) [50]	✓	Ø	×
Mildevtveldt(2007) [51]	✓	Ø	×
Shenberger (1992) [52]	✓	Ø	×
Singh (2002)][53]	✓	✓	×
Sundin (2003) [54]	×	Ø	×
Timlin (2002) [55]	✓	Ø	×
Toobert (1998) [56]	×	✓	×
Vale (2003) [57]	✓	✓	×
Van Elderen-van Kemenade (1994) [58]	✓	×	×
Vestfold (2003) [59]	×	✓	×
Wallner (1999) [60]	✓	×	×

Additional file 3: Methodological quality scores and risk of bias assessment in nutrition knowledge translation studies for cardiovascular disease

Study	Relevance	Validity	KT strategy Overall
HIGH RISK OF CVD			
Gorder (1986) [61] Van Horn (1997) [62]	✓	×	×
Siero (2000) [63]	✓	Ø	×
HEART FAILURE			
Donner Alves (2012) [64]	✓	✓	×
Philipson (2010) [65]	✓	Ø	×
Powell (2010) [66]	✓	✓	Ø
HEALTH PROFESSIONALS			
Banz (2004) [67]	✓	×	×
Carson (2002) [68]	✓	Ø	×
Perry (2000) [69] Perry (2003a) [70] Perry (2003b) [71]	✓	Ø	×
Van Der Weijden (1998) [72]	✓	Ø	×

✓positive; × negative; Ø neutral

Appendix 5. Intervention content for systematic review included studies

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
CARDIOVASCULAR DISEASE DIAGNOSES					
Aish, 1996 [30] Aish, 1996 [31]	Feedback on current diet compared to healthy heart diet nutritional goals by telephone. Recognition & encouragement of health self-care behaviour at home visit, 3 follow up telephone calls.	Booklet on how to modify lifestyle following MI.	Individual	Baseline interview in hospital, home visit (week 1), 3 telephone calls over 6 weeks, home visit (week 7)	Untargeted activity
Allen (1996) [32]	Component 1: (pre-discharge) Revision of HCP instructions, directions for resources usage, counselled on low-confidence situations. Component 2: (at home) Dietary feedback (fat, cholesterol, fibre, F&V). Strategies for dietary goals. Short-term goals mutually established. Follow up: (clinic) Further instructions. Counselling based on patient progress & (phone): encouragement & reinforcement of adherence	Video, workbook from Active Partnership Program from American Heart Association	Individual	T1: (pre-discharge), unspecified duration T2: (home) + 2 weeks, unspecified duration T3: (clinic) + 1 month, unspecified duration T4: (phone) + 2 months, unspecified duration	Standard care
Arntzenius,1985 [33] Kromhout,1986 [34]	Individualised advice based on prior 24 hour recall of food intake	Not stated	Individual	15 contact points with either cardiologist or dietitian over 24 months, included two full examinations, no further information	Pre-, Post

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
Billings, 2000 [35] Koertge, 2003 [36]	Group-based Ornish Program. Didactic & experiential learning, intense program immersion.	Unclear. Group meals	Group, unclear size	Stage1: Two day intensive workshop plus 12 weeks program (2 x 4 hours/week (inclusive group meal) plus 1x 2 hour/week). Stage 2: 1 x 4hr session for 40 weeks. Includes group meal. Stage 3: self-directed.	Pre-, Post
Campbell,1998 [37]	Diet included in behavioural risk assessment in initial & follow-up clinic visits: behavioural change negotiated	"One step at a time" leaflet	Individual	One year program .Initial visit (30-60min) plus follow-up (10-30 minutes) every 2-6 months. Diet included in 4th stage of each visit	Unclear
Dalgard, 2001 [38]	Brief dietary counselling (BDC): 5 pieces of dietary behaviour advice provided with Plate Model Comprehensive dietary counselling (CDC): Based on NCEP step I. Included diet history, diet influences on blood cholesterol, strategies for achieving goals, individualised information & follow-up counselling with feedback.	Unclear	Individual	BDC: Single 10 minute session CDC: Dietary consultation: 50-60 minutes. Follow-up (week 12): 40-50 minutes.	Other KT intervention (CDC)

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
de Lorgeril, 1994 [39] de Lorgeril, 1999 [40]	Dietary advice by cardiologist & dietitian (↑bread, ↑ root & green vegetables, ↑ fish, ↓ meat, fruit each day, replace butter & cream with margarine (supplied). Moderate alcohol consumption). Individual detailed advice.	Unclear	Individual	1 hour consult at baseline, dietary survey at each visit (8 weeks after initial visit, then annually)	Standard care
Evon, 2004 [41]	2 x25 minute seminars by dietitian, option for private consultation with dietitian with family member.	Unclear	Group, unclear size	Dietary focus: 2 x 25 minute group sessions, optional private consultation. Included as part of 3 x 1 hour sessions per week for 12 weeks	No control group
Frost, 2004 [42]	Baseline: Instruction & counselling on randomised diet by dietitian. Provided with written advice & targets. Weeks 4 & 8: dietetic visit, new goals set.	Written advice	Individual	5 visits over 12 weeks. Dietetic support via telephone between each visit. Visit & call length unclear.	Other KT intervention
Giannuzzi, 2008 [43]	Counselling on lifestyle & risk factors & reinforcement of preventive interventions	Booklet	Individual	Standard cardiac rehabilitation classes for 1 month plus intervention (2 hour sessions at 6 & 12 months then yearly until 3 years (unclear time dedicated to diet)).	Standard care
Gleason, 2002 [44]	Individualised provision of meals & snacks (4 weeks) developed by dietitian. Substitutions of own lunch post- week 4.	Meals & snacks provided, information pack regarding energy requirements, program kit, rotating weekly menu, snack list	Individual	Initial contact by dietitian to establish preferences, participants contacted weekly by telephone x 8 weeks, 3 study visits	No control group

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
Goodwin, 2012 [45]	Acceptance-based behaviour therapy sessions: psychoeducation- behavioural techniques for adhering to heart-healthy lifestyle, mindfulness & distress tolerance	Low-calorie/low fat recipes, educational material from American Heart Association (Getting Healthy, 2009)	Medium group (5-20)	4x 90 minute group therapy sessions	No control group
Hofman-Bang, 1999 [46]	Intense health education, training of practical skills & habit rehearsal. Curriculum included physical activity, food preparation, applied relaxation, self-observation, Type A behavioural drills.	4 week residential stay, seminars, lectures, 11 month structured maintenance program	Medium group (5-20)	4 week residential stay, 11 month with regular contact based on individual goals set (time unclear).	Standard care
Jackson, 2005 [47] Jackson, 2005 [48]	TPB only group: demonstrated portion of F&V, goal of two extra portions of F&V each day for next 3 months plus TPB questionnaire. TPB + intention group: as per TPB group, plus intervention (state specific implementation intention).	Postcards demonstrating c a serve of F&V	Individual	Single visit to researcher	Other KT intervention, Untargeted activity
Luszczynska, 2007 [49]	Implementation Intentions training (II): instructions, completion of II training form with feedback & compliments.	nutrition guidelines, II form	Individual	1 x 10-20 minute intervention in addition to cardiac rehabilitation classes	Untargeted activity
Masley, 2001 [50]	Group classes: cooking demonstrations, setting of specific goals. Additional recipes included & physical activity. Significant others encouraged to participate. Dietary goal setting provided.	Textbook: The 28-Day Anti-oxidant Program (included shopping lists, menu plans, food-monitoring sheets)	Group, unclear size	14 x 90 minute sessions over 12 months. Weekly for first month, monthly afterwards	Standard care

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
Mildestvedt , 2007 [51]	Standard treatment: Didactic, group-based dietary & smoking cessation counselling. Intervention: Standard treatment plus 2 x individual sessions of goal setting activities, personalised strategies to overcome barriers + 2 x telephone calls (follow up) focusing on selected goals	Not stated	Individual, Group, unclear size	Unclear, 2 individual sessions during rehabilitation stay, follow up telephone calls at 6 & 24 months	Standard care
Shenberger, 1992 [52]	Dietetic counselling sessions (including goal setting, feedback on lipid profile)	Not stated	Not clear	3 x counselling sessions, length unclear, 1 month apart	Pre-, Post
Singh, 2002 [53]	Unclear, approximately 10 sessions (week 4, 8, 12, then 12 week intervals), met with dietitian to review food diaries, given explanation of usefulness of foods & motivation to adhere to diet.	Not stated	Individual	Approximate 10 visits with dietitian, length unclear	Other KT intervention
Sundin, 2003 [54]	Two phases in 1 year program. Residential included diet in lectures & discussions, skills training & habit rehearsal. Maintenance: self-observation & recording of lifestyle behaviours, regular follow-up contacts with nurse, verbal feedback, problem-solving & re-planning discussions.	Unclear. Written information regarding value of lifestyle changes	Medium group (5-20), Individual	4-week residential phase, 11 month structured maintenance, diet 18/64 hours per week? Intensity of maintenance not stated. Contact successively stepped down.	Other KT intervention, Standard care
Timlin, 2002 [55]	Group nutrition education classes led by dietitian. Learning objectives based on developing behavioural capability to comply with AHA Step 2.	Not stated	Group, unclear size, Individual	2 x 1 hour education classes + 1 individual session within 6 week cardiac rehab program	Standard care

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
Toobert, 1998 [56]	Retreat: cooking classes, stress-reduction /relaxation classes, daily PA, unstructured group sessions for group support. Follow up meetings: PA, relaxation, catered dinner & group discussions.	1hr audiocassette tape for relaxation, unclear	Group, unclear size	Retreat: 7 days Follow up: 4 hour meetings twice weekly for 15 months, fortnightly for 6 months, then monthly for 3 months.	Standard care
Vale, 2003 [57]	Coached to continuous improvement by encouraging responsibility in managing risk factors for condition. Negotiating plans & targets.	Coach program package. Risk factor targets provided to individual & usual HCP. Written reports after each coaching session.	Individual	1 initial phone call <2 weeks post-randomisation + 3 follow-up calls at 6 week intervals + 5th call to arrange final assessments.	Standard care
van Elderen-van Kemenade, 1994 [58]	Counselling by nurse, education on PA by physiotherapist, healthy eating by dietitian	Unclear. Written information provided	Individual, Group, unclear size	2 initial counselling sessions as inpatients, 2 group educations as inpatients, 6 follow up phone calls (1 week apart)	Standard care
Vestfold Heartcare Study, 2003 [59]	Heart school: supervised PA plus bi-weekly group meetings (dietary advice, smoking cessation, PA counselling, psychosocial management & health education). Individual counselling offered. Followed up: Supervised PA & group meetings. Group meetings conducted by nurse, physician, physiotherapist & dietitian. Agenda led by patients priorities.	Unclear. Exercise diary.	Medium group (5-20), Individual	Heart School: six weeks bi-weekly group meetings + PA Follow-up: 9 week bi-weekly PA. Group meetings every three months for remaining 2 years.	Standard care

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
Wallner, 1999 [60]	Dietary & lifestyle advice to align with AHA step II diet by study nutritionist. Individualised instructions.	Not stated	Individual	1 hour session/week for first month, then fortnightly for 2 months, then monthly for remaining 9 months.	Standard care
HIGH RISK OF CVD					
Gorder, 1986 [61] Van Horn, 1997 [62]	Phase 1 (intensive): risk factor modification & expected benefits. Application of behaviour change principles. Phase 2 (extended): individualised content, some formation of group topics e.g. obesity & smoking. Educational programs for nutrition. Annual medical examination.	Unclear	Group, size unclear	Phase 1: 10 group sessions, unknown intensity & duration. Phase 2: unclear	Standard care
Siero, 2000 [63]	Group A & B: information on Mediterranean eating patterns, 10 nutritional guidelines, strategies for following recommendations. Group B only: targeted information based on stage of behaviour change	A & B: Psychological questionnaire, margarine supplied, film on shopping for food, knowledge quiz, information and recipe booklets, homework tasks. B only: Tailored responses to psychological questionnaire	Medium group (5-20)	A & B: 3x 2 hour group sessions, time points unclear. B only: tailored feedback between session 2-3	Untargeted activity

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
HEART FAILURE					
Donner Alves, 2012 [64]	Standard care + session with nutritionist (food groups, macro- & micro-nutrients, dietary fibre, water intakes, dietary sources of sodium, cholesterol and triglycerides, goal setting for dietary adherence & motivation	Calendar with graphic representation of information provided	Individual	1 hour with nutritionist	Standard care
Philipson, 2010 [65]	Individualised training from dietitian or trained nurse on how to reduce sodium intake to 2-3g/day & formulated plan to restrict fluid to 1.5 L/day. Follow up telephone call including 24 hour dietary recall & further counselling.	Unclear	Individual	Initial counselling session + 3 x follow-up (telephone) (unclear length of time) 2-3 weeks sessions.	Standard care
Powell, 2010 [66]	Self-management + education treatment: group-based education counselling on problem-solving and self-management skills (self-monitoring, environmental restructuring, elicitation of support from family & friends, cognitive restructuring & relaxation).	18 Heart Failure tip sheets from AHA	Medium group (5-20)	18 x 2 hour meetings over 12 months	Standard care + attention control
HEALTH PROFESSIONALS					
Banz, 2004 [67]	Lectures on soy & CVD, demonstrations on how to include soy into diet, including example of soy-based breakfast & lunch	Grab-bag of soy information & samples	Group, size unclear	1 workshop, length unclear	No intervention

Additional file 3: Intervention content

Author , year (country)	Intervention content	Intervention resources	Recipient delivery size	Intervention intensity	Control group
Carson, 2002 [68]	Incorporation of cardiovascular nutrition into existing ACR framework; 1 hour class discussion; two web-based cases including multiple choice questions with feedback & explanations, including role modelling from physicians & use of a dietitian's nutrition therapy note.	Web-based case study; Pocket reference cards	Group, size unclear	1 hour class discussion, Unclear	Pre-, post + historical control
Perry & McLaren, 2000, 2003a, 2003b [69-71]	Establishment of project team with opinion leader, education & training for relevant personnel (screening using validated tool, nutrition risk & monitoring using validated tool, early initiation of nutrition support, effective artificial nutrition support). Opinion leader trained in change management approaches. Formal & informal teaching & practical sessions.	User-friendly guidelines, unclear other	Group, size unclear	Unclear	Pre-, post
Van der Weijden, 1998 [72]	Plenary session with local opinion leader (lecture, small group discussion, example case studies), recorded cholesterol consultations, feedback, 2 x individual outreach visits + additional feedback	Cholesterol guidelines, consultation registration forms, desktop flowchart of guidelines, patient education leaflets	Group, size unclear	3 hour plenary x 1, 2 x outreach visits, time points unclear	Untargeted activity

AHA American Heart Association; F&V fruit and vegetables; HCP health care provider; PA physical activity; TBP: theory of planned behaviour;

Appendix 6. Statement of contribution and collaboration for Chapter 4

I attest that Research Higher Degree candidate Tracy Leigh Schumacher contributed to the following paper:

Schumacher T, Burrows T, Cliff D, Jones R, Okely A, Baur L, Morgan P, Callister R, Boggess M, Collins C. Dietary intake is related to multifactor risk score in obese boys. Healthcare. 2014;2:282-298 DOI:10.3390/healthcare2030282.

Tracy Schumacher interpreted the data and drafted the initial manuscript. Dr Tracy Burrows, Dr Dylan Cliff, Dr Rachel Jones, Professor Anthony Okely, Professor Louise Baur, and Professor Philip Morgan were original investigators from which this data is sourced, contributed to study design, reviewed and revised the manuscript. Professor May Boggess performed the statistical analysis, drafted the results and revised the manuscript. Professor Robin Callister contributed to conceptualising the hypothesis, revised and reviewed the manuscript. Professor Clare Collins was an original investigator, conceptualised the hypothesis, contributed to the study design, revised and reviewed the manuscript. All authors approved the final manuscript.

Ms Tracy Schumacher (25th November, 2015)

Dr Tracy Burrows (25th November, 2015)

Dr Dylan Cliff (26th November, 2015)

Dr Rachel Jones (4th December, 2015)

Professor Anthony Okely (26th November, 2015)

Professor Louise Baur (27th November, 2015)

Dr May Boggess (2nd December, 2015)

Professor Robin Callister (3rd December, 2015)

Professor Philip Morgan (26th November, 2015)

Professor Clare Collins (3rd December, 2015)

Professor Robert Callister (7th December, 2015)

(Assistant Dean Research Training)

Appendix 7. Statement of contribution and collaboration for Chapter 5

I attest that Research Higher Degree candidate Tracy Leigh Schumacher contributed to the following paper:

Schumacher T, Dewar D, Lubans D, Morgan P, Watson J, Guest M, Callister R, Collins C. Dietary patterns of adolescent girls attending schools in low-income communities highlight low consumption of core foods. *Nutrition and Dietetics*. 2014;71(2):127-134 DOI:10.1111/1747-0080.12084.

Tracy Schumacher performed the data analysis, interpreted the data and drafted the initial manuscript. Dr Deborah Dewar, Professor David Lubans and Professor Philip Morgan were original investigators from which this data is sourced, contributed to study design, reviewed and revised the manuscript. Dr Maya Guest and Dr Jane Watson assisted with data analysis, reviewed and revised the manuscript. Dr Tracy Burrows and Professor Robin Callister contributed to conceptualising the hypothesis, revised and reviewed the manuscript. Professor Clare Collins was an original study investigator from which this data was sourced, contributed to study design and conceptualising the hypothesis, assisted with data analysis, reviewed and revised the manuscript. All authors approved the final manuscript.

Ms Tracy Schumacher (25th November, 2015)

Dr Deborah Dewar (7th December, 2015)

Professor David Lubans (26th November, 2015)

Professor Philip Morgan (26th November, 2015)

Dr Jane Watson (1st December, 2015)

Dr Maya Guest (3rd December, 2015)

Dr Tracy Burrows (25th November, 2015)

Professor Robin Callister (3rd December, 2015)

Professor Clare Collins (25th November, 2015)

Professor Robert Callister (7th December, 2015)

(Assistant Dean Research Training)

Appendix 8. Statement of contribution and collaboration for Chapter 6

I attest that Research Higher Degree candidate Tracy Leigh Schumacher contributed to the following paper:

Schumacher T, Burrows T, Thompson D, Spratt N, Callister R, Collins C. Feasibility of recruiting families into a heart disease prevention program based on dietary patterns. Nutrients. 2015;7(8):7042-57 DOI:10.3390/nu7085323.

Tracy L Schumacher was primarily responsible for and contributor to resource development, data collection, data analysis, manuscript preparation and contributed to study design. Dr Deborah Thompson contributed to qualitative data collection and manuscript preparation. Dr Neil Spratt contributed to quantitative data collection and manuscript preparation. Dr Tracy Burrows, Professor Robin Callister and Professor Clare Collins contributed to the study design, resource development, data collection, data analysis and manuscript preparation. All authors approved the final manuscript.

Ms Tracy Schumacher (25th November, 2015)

Dr Tracy Burrows (25th November, 2015)

Dr Deborah Thompson (25th November, 2015)

Dr Neil Spratt (25th November, 2015)

Professor Robin Callister (25th November, 2015)

Professor Clare Collins (25th November, 2015)

Professor Robert Callister (7th December, 2015))

(Assistant Dean Research Training)

Appendix 9. Ethics approval: Hunter New England Local Health District

13 June 2012

Professor Clare Collins
Room 310
Level 3 ATC Building
University of Newcastle

Dear Professor Collins,

Re: Love your Food, Love your Heart, Love your Family (12/05/16/4.01)

HNEHREC Reference No: 12/05/16/4.01
NSW HREC Reference No: HREC/12/HNE/140

Thank you for submitting the above protocol for single ethical review. This project was first considered by the Hunter New England Human Research Ethics Committee at its meeting held on **16 May 2012.** This Human Research Ethics Committee is constituted and operates in accordance with the National Health and Medical Research Council's *National Statement on Ethical Conduct in Human Research (2007)* (National Statement) and the *CPMP/ICH Note for Guidance on Good Clinical Practice*. Further, this Committee has been accredited by the NSW Department of Health as a lead HREC under the model for single ethical and scientific review. The Committee's Terms of Reference are available from the Hunter New England Local Health District website: http://www.hnehealth.nsw.gov.au/Human_Research_Ethics.

I am pleased to advise that following acceptance under delegated authority of the requested clarifications and revised Information Statements by Dr Nicole Gerrand Manager, Research Ethics & Governance, the Hunter New England Human Research Ethics Committee has granted ethical approval of the above project.

The following documentation has been reviewed and approved by the Hunter New England Human Research Ethics Committee:

- For the Study Advertisement (Version 1.0 dated 26 April 2012);
- For the Participant Information Statement (Version 2 dated 8 June 2012);
- For the Children's Information Statement (Version 2 dated 13 June 2012);
- For the Adolescent's Information Statement (Version 2 dated 13 June 2012);
- For the Consent Form – Primary Participant (Version 1 dated 27 April 2012);
- For the Consent Form – Participant 2 (Partner) (Version 1 dated 27 April 2012);
- For the Consent Form – Participant 3 (Child) (Version 1 dated 27 April 2012);
- For the Appendix II – Telephone Eligibility Screen;
- For the Health Questionnaire (Version 1.0 dated March 2012);
- For the Process Evaluation – Index Person (Version 1.0 dated March 2012);
- For the Program Evaluation – 3 months; and
- For the Guide to helping you and your family to healthy heart diet (Draft Version dated April 2012)

Hunter New England Research Ethics & Governance Unit
(Locked Bag No 1)
(New Lambton NSW 2305)
Telephone (02) 49214 950 Facsimile (02) 49214 818
Email: hnehrec@hnehealth.nsw.gov.au
http://www.hnehealth.nsw.gov.au/research_ethics_and_governance_unit

For the protocol: **Feasability of targeting parents with heart disease to improve their hearty health & that of their children – Love your Food, Lover your Heart, Love your Family**

Approval from the Hunter New England Human Research Ethics Committee for the above protocol is given for a maximum of **3** years from the date of this letter, after which a renewal application will be required if the protocol has not been completed.

The *National Statement on Ethical Conduct in Human Research (2007),* which the Committee is obliged to adhere to, include the requirement that the committee monitors the research protocols it has approved. In order for the Committee to fulfil this function, it requires:

- A report of the progress of the above protocol be submitted at 12 monthly intervals. Your review date is **June 2013.** A proforma for the annual report will be sent two weeks prior to the due date.

- A final report must be submitted at the completion of the above protocol, that is, after data analysis has been completed and a final report compiled. A proforma for the final report will be sent two weeks prior to the due date.

- All variations or amendments to this protocol, including amendments to the Information Sheet and Consent Form, must be forwarded to and approved by the Hunter New England Human Research Ethics Committee prior to their implementation.

- The Principal Investigator will immediately report anything which might warrant review of ethical approval of the project in the specified format, including:

    - any serious or unexpected adverse events

        - Adverse events, however minor, must be recorded as observed by the Investigator or as volunteered by a participant in this protocol. Full details will be documented, whether or not the Investigator or his deputies considers the event to be related to the trial substance or procedure. These do not need to be reported to the Hunter New England Human Research Ethics Committee

        - Serious adverse events that occur during the study or within six months of completion of the trial at your site should be reported to the Manager, Research Ethics & Governance, of the Hunter New England Human Research Ethics Committee as soon as possible and at the latest within 72 hours.

        - All other safety reporting should be in accordance with the NHMRC's Safety Monitoring Position Statement – May 2009 available at http://www.nhmrc.gov.au/health_ethics/hrecs/reference/_files/090609_nhmrc_position_statement.pdf

        - Serious adverse events are defined as:

            - Causing death, life threatening or serious disability.
            - Cause or prolong hospitalisation.
            - Overdoses, cancers, congenital abnormalities whether judged to be caused by the investigational agent or new procedure or not.

Hunter New England Research Ethics & Governance Unit
(Locked Bag No 1)
(New Lambton NSW 2305)
Telephone (02) 49214 950 Facsimile (02) 49214 818
Email: hnehrec@hnehealth.nsw.gov.au
http://www.hnehealth.nsw.gov.au/research_ethics_and_governance_unit

- Unforeseen events that might affect continued ethical acceptability of the project.

- If for some reason the above protocol does not commence (for example it does not receive funding); is suspended or discontinued, please inform Dr Nicole Gerrand, as soon as possible.

You are reminded that this letter constitutes ethical approval only. You must not commence this research project at a site until separate authorisation from the Chief Executive or delegate of that site has been obtained.

A copy of this letter must be forwarded to all site investigators for submission to the relevant Research Governance Officer.

Should you have any concerns or questions about your research, please contact Dr Gerrand as per the details at the bottom of the page. The Hunter New England Human Research Ethics Committee wishes you every success in your research.

Please quote **12/05/16/4.01** in all correspondence.

The Hunter New England Human Research Ethics Committee wishes you every success in your research.

Yours faithfully

For: Associate Professor M Parsons
Chair
Hunter New England Human Research Ethics Committee

Appendix 10. Ethics approval: University of Newcastle

HUMAN RESEARCH ETHICS COMMITTEE

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

Notification of Expedited Approval

To Chief Investigator or Project Supervisor:	**Professor Clare Collins**
Cc Co-investigators / Research Students:	**Doctor Neil Spratt** **Miss Tracy Schumacher** **Doctor Tracy Burrows** **Professor Robin Callister**
Re Protocol:	**Feasibility of targeting parents with heart disease to improve their heart health and that of their children**
Date:	**24-Jul-2012**
HREC Reference No:	**H-2012-0246**
External HREC Reference No:	**12/05/16/4.01**
Date of Initial Approval:	**17-Jul-2012**

Thank you for your **Initial Application** submission to the Human Research Ethics Committee (HREC) seeking approval in relation to the above protocol.

Your submission was considered under **Expedited Review of External Approval** review by the Chair/Deputy Chair.

I am pleased to advise that the decision on your submission is **External HREC Approval Noted** effective **17-Jul-2012**.

In approving this protocol, the Human Research Ethics Committee (HREC) is of the opinion that the project complies with the provisions contained in the *National Statement on Ethical Conduct in Human Research, 2007*, and the requirements within this University relating to human research.

As the approval of an External HREC has been "noted" the approval period is as determined by that HREC.

The full Committee will be asked to note this decision at its next scheduled meeting. A formal *Certificate of Approval* will be available upon request. Your approval number is **H-2012-0246**.

PLEASE NOTE:
As the HREC has "noted" the approval of an External HREC, progress reports and reports of adverse events are to be submitted to the External HREC only. In the case of Variations to the approved protocol, or a Renewal of approval, you will apply to the External HREC for approval in the first instance and then Register that approval with the University's HREC.

Linkage of ethics approval to a new Grant

HREC approvals cannot be assigned to a new grant or award (ie those that were not identified on the application for ethics approval) without confirmation of the approval from the Human Research Ethics Officer on behalf of the HREC.

Best wishes for a successful project.

Professor Allyson Holbrook

Chair, Human Research Ethics Committee

For communications and enquiries:

Human Research Ethics Administration

Research Services
Research Integrity Unit
HA148, Hunter Building
The University of Newcastle
Callaghan NSW 2308
T +61 2 492 18999
F +61 2 492 17164
Human-Ethics@newcastle.edu.au

Linked University of Newcastle administered funding:

Funding body	Funding project title	First named investigator	Grant Ref

Appendix 11. Participant screening script

Name:_____

Date:_____

Found out about study through: _____

I will now just give you a bit more detail about what we are trying to do.

The purpose of the study is to test a specifically designed diet in a family setting study to assess whether or not it is successful in reducing risks for heart attacks and strokes.

It is currently being conducted as a small trial, and if successful will lead to a larger study and may lead to a national program for reducing family risk factors associated heart disease or stroke for families where an adults has already experienced heart disease or stroke.

You and your family would be expected to follow the test diet for a total of 3 months. Not included in the three months is the time taken to fill forms and arrange consultations.

The entire process is expected to take no longer than four months.

Participation in the study is entirely voluntary and if you do decide to participate, you may withdraw from the study at any time without giving a reason.

I will now ask you a series of screening questions to determine your family's eligibility for the study. Included in this are a number of procedures that you and your family must be willing to consent to.

Heart or stroke event: When: Stent / MI / CABG etc:
Age:
Partner or child willing to participate with you:

<table>
<tr><td colspan="5">No heart or stroke event:</td></tr>
<tr><td>Male / Female</td><td colspan="2">Age:</td><td>Smoker:</td><td>Diabetes:</td></tr>
<tr><td colspan="5">Blood pressure:</td></tr>
<tr><td colspan="5">Checked: Yes (see below) / No (default 140mmHg)</td></tr>
<tr><td rowspan="4">What was it?</td><td colspan="4">Don't know:</td></tr>
<tr><td rowspan="3">Ever been told it's high?</td><td colspan="3">No: Default 140mmHg</td></tr>
<tr><td colspan="3">Yes:</td></tr>
<tr><td>Medicated?
Default:
140mmHg</td><td colspan="2">Not medicated?
Default:
160mmHg</td></tr>
<tr><td colspan="5">BP Medication:</td></tr>
<tr><td colspan="5">Cholesterol:</td></tr>
<tr><td colspan="5">Checked: Yes (see below) / No (default ratio 5:1)</td></tr>
<tr><td rowspan="4">What was it?</td><td colspan="4">Don't know:</td></tr>
<tr><td rowspan="3">Ever been told it's high?</td><td colspan="3">No: Default ratio 5:1</td></tr>
<tr><td colspan="3">Yes:</td></tr>
<tr><td colspan="2">Medicated?
Default:
Ratio 5:1</td><td>Not Medicated?
Default:
Ratio 6:1</td></tr>
<tr><td colspan="5">Ancestry:
Aboriginal or Torres Strait Islander
South Asian (India, Pakistan, Afghanistan, Sri Lanka, Tibet, Iran, Burma)
Chinese, Japanese, Hispanic, African descent</td></tr>
<tr><td>Height:</td><td colspan="2">Weight:</td><td colspan="2">BMI:</td></tr>
<tr><td colspan="5">Exercise: Yes / No
More than 30 min MODERATE x 5/week or
More than 20 min VIGOROUS x 3/week</td></tr>
<tr><td colspan="5">Family member from different generation willing to participate? Yes / No</td></tr>
<tr><td colspan="5">Eligible / Not eligible</td></tr>
</table>

Speak and understand English: **Yes / No**
Access to internet and emails: **Yes / No**
Food allergies: **Yes / No**
Food intolerances: **Yes / No**
Pre-existing medical conditions: **Yes / No**

As previously outlined, as part of the study you will be required to:

- Attend 2 assessments sessions, to be held at the University of Newcastle.
- The whole family will have height, weight, body composition (fat free mass/fat mass), blood pressure and skin colour analysis performed. Adults will undergo an additional test for arterial stiffness.
- Blood tests for you and your partner will be required. These will measure blood fats (cholesterol and triglyceride levels, as well as insulin resistance (insulin and glucose) and other risk factors (such as markers for inflammation).
- You and your partner will need to complete questionnaires about your medical history, general health and demographics, dietary intake and physical activity.
- Your child/ren will need to complete questionnaires in regards to their dietary intake and their levels of physical activity.

Consent: **Yes / No**

Contact details:

Full name: _____

Partner: **Yes / No**

Other family members: **Yes / No**

Info statements required: _____Adult _____Adolescent _____Child

Address: _____

_____

Suburb: _____ Postcode: _____

Phone numbers:

Work: _____

Home: _____

Mobile: _____

Email address: _____

Appendix 12. Child information statement

Priority Research Centre
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
49217053 (FAX)
FoodHeartFamily@newcastle.edu.au

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

Children's Information Statement for the Research Project:

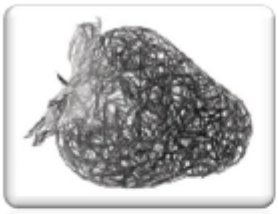
LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY

Program Study

Prof Clare Collins, Prof Robin Callister, Dr Tracy Burrows, Dr Neil Spratt,
Ms Tracy Schumacher (student researcher)

Version 4: 6th November, 2012

You and your family are being asked to join a study. This study is about helping families eat foods that make healthier hearts. It is being run by a team of researchers at the University of Newcastle.



What do researchers do?

Our job is to find the best foods for people to eat if they would like a healthy heart. We will do this by taking some measures, asking you about what you are eating now and making some suggestions.

Why are we asking you?

We have asked your family to join us as your parents would like to find out more about how to make the hearts in your family healthier.

What will you have to do?

There are a few things that we would ask you to do. The first is to go online with your parents and fill in a quiz that we have set up for you. The quiz asks you lots of questions about the foods you eat. When this has been done, your parents will make a time for you and your family to come to the University to meet some of the people from our team.

The team will ask you if they can:

Measure your height

You would do this by standing under our special height measuring machine.

Measure your blood pressure

We have a machine that you stick your arm into and it squeezes very tight. It doesn't feel nice, but it shouldn't hurt.

Measure your weight and muscle

We have a very special machine to do this. You would be asked to stand on the silver plate and hold onto two handles. It doesn't hurt, but you will have to stand very quietly for a minute or two.

Have your waist measured

One of the people from our team would measure your middle.

What if I don't want to do these things?

Then you won't have to. You can tell your parents how you feel and you won't have to join our study. You can even change your mind later. You can tell the person who is measuring you that you don't want to do this anymore and they will stop.

Where do we have to go?

Your parents would arrange for your family to meet our team at the University. We would take around 2-3 hours to take the measures talk with your family. We also have new snacks for you to taste and some activities planned for you while we talk to your parents.

What would happen after we visited the University?

Your parents will be asked to make some changes to what you are eating as a family. There may be some new foods bought into the house for you to try. You may also have some different kinds of snacks packed for your lunch at school. After three months, your parents will arrange for your family to come back to the University, where we can take more measures. These are to check what changes have happened. For example, we will check how much you have grown.

Have you talked to your parents about this?

It is important that you sit down with your parents to talk about this as a family. This is to make sure that everyone gets to say why they would or wouldn't like to join the study. You can also ask your parents any questions you might have. If you are upset about anything that happens while doing this research, tell your parents and they will know who to talk to.

Would you like to ask the team at the University a question?

If you still have more questions, you can talk to one of the team. You can call Tracy S at the University yourself or ask your parents to do it for you. The number to call is 4921 6259. Or maybe you would like to email us at FoodHeartFamily@newcastle.edu.au.

Thank you for thinking about joining our study.

On behalf of our team:

- ***Prof Clare Collins***
- ***Prof Robin Callister***
- ***Dr Tracy Burrows***
- ***Dr Neil Spratt***
- ***Tracy Schumacher***

Appendix 13. Adolescent information statement

Priority Research Centre
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
49217053 (FAX)
FoodHeartFamily@newcastle.edu.au

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

Adolescent's Information Statement for the Research Project:

LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY Program Study

Prof. Clare Collins, Prof. Robin Callister, Dr Tracy Burrows, Dr Neil Spratt,
Ms Tracy Schumacher (student researcher)

Version 6: 25 March, 2013

You and your family are invited to join the research project shown above. It is being run by researchers from the University of Newcastle. The researchers are Professor Clare Collins, Professor Robin Callister, Dr Tracy Burrows, Dr Neil Spratt and Ms Tracy Schumacher. We have been given money by the Hunter Medical Research Institute to run this study.

Why are we researching heart health in Families?

Heart disease affects a lot of families in the Hunter Region. Members of families where one person already has heart disease are at a higher risk than others because of shared genes and / or living together. The home environment is important because this is where growing families pick up their eating and exercise habits.

What are we trying to find out?

We are trying to find the best dietary advice that can be given to families to help to improve heart health in all family members, including children and adolescents. We will review your eating patterns and then provide you with feedback on how to eat for a healthy heart.

Who are we asking to participate in the research?

Any interested family can participate in this project if they:

- Have a parent or grandparent who has had a heart disease or stroke episode or who is at risk of heart disease.
- Have children of any age.
- This means all family members who live in the same household and want to participate and provide consent can participate.
- Can attend 2 assessment sessions at the university.

This study is not suitable for you and your family if you:

- Are not able to speak or read English
- Cannot make it to the University for assessments
- Have family members with food allergies.

What choice do you have?

It is your choice to join this study. Only those parents and children who give informed consent will be included. Whether or not you and your family decide to join, it will not disadvantage you or your family in any way. If you choose to drop out of the study at any time, you don't have to give a reason and have the option of withdrawing any data you have provided.

What would you and your family be asked to do?

Families who agree to participate in the study will be asked to complete a number of online tasks. You would then be provided with feedback on what you are eating when you attend the university assessment. You will also be given general advice for the family about eating for a healthy heart.

You would be required to participate in the assessments listed below:

- **Weight:** measured in light clothing without shoes.
- **Height:** measured without shoes.
- **Waist circumference**: measured using a metal tape measure.
- **Body composition including fat mass and fat free mass (muscle and bone)**: measured using a specialised machine where you stand on a metal plate and hold onto two rails. A very small electrical current is passed through the body. It won't do any harm and you won't feel anything. This will tell us your percentage body fat and muscle mass.
- **Skin colour analysis:** this analyses the colours in your skin that are affected by health and nutrition. It does this by bouncing white light off the skin and measures each colour that is reflected back.
- **Blood Pressure:** a series of measurements will be taken to obtain blood pressure. We have a tube that you put your arm into, and this then is inflated around your arm. It may be a little uncomfortable, but it shouldn't hurt.
- **Arterial stiffness:** this measures how elastic your blood vessels are in your body. It is measured in a similar way to blood pressure, as a cuff is inflated around your upper arm.

What happens next?

The study will take place during 2012. Families will be randomly divided into one of two groups. One group will be provided with feedback on their eating habits and given advice immediately. The other group will be given advice in their second assessment session.. Families will have an equal chance of being in either group. Regardless of the group you are in, you will be asked to attend one assessment session August/September and a second session 12 weeks later. If you are to be given dietary advice in your second session , you will also be invited to attend a third session 12 weeks later.

What happens at the sessions?

Assessment sessions must be attended by the family members who said they were willing to join the study. They will be held at the University of Newcastle Nutrition and Dietetics Laboratory, which is in the Hunter Building on the Callaghan Campus. The sessions will last between 2-3 hours and include taking the measures listed above, receiving feedback and dietary advice from a dietitian, as well as trying examples of foods that are considered to help heart health.

What are the risks and benefits of joining the study?

There is some risk that you may feel uncomfortable having some of the measures taken. The benefits of joining the study are that you:

- Get personal advice on how to choose foods that are considered to be healthy for your heart.
- Find out the composition of your body in terms of bone, muscle and fat stores

How will your privacy be protected?

We will take great care to protect your information. We will take your measures when you are in a private setting, such as behind a screen. We will only give information, such as your weight, to your parents with your permission. Anything we write down on paper will be put into a locked filing cabinet and anything that is electronic will be password protected.

How will the information collected be used?

All the data collected during the study will be analysed and may be reported at national and international conferences, in scientific publications, and as part of the research thesis for Tracy Schumacher's PhD studies. You will not be identified in any reports arising from the study. At the conclusion of the study you will receive a brief summary of the results from the Chief Investigator, Professor Collins.

Have you talked to your parents about this?

It is very important that you sit down with your parents to talk about this as a family. This is to make sure that everyone gets to say why they would or wouldn't like to do this and to make sure that you understand what is asked of you. If you are upset about anything that happens while doing this research, tell your parents and they will know who to talk to.

Would you like to ask the team at the University a question?

If you would like to ask a question, you can call Tracy Schumacher at the University yourself or ask your parents to do it for you. Our number is 4921 6259. Maybe you would like to email us at FoodHeartFamily@newcastle.edu.au.

What do you need to do to participate?

Please read this Information Statement carefully and be sure you understand its contents before you agree to join. You may also choose to read the information statement that was given to your parents which contains more detail. If there is anything you do not understand, or you have questions, contact the researcher (Tracy Schumacher) yourself or have your parents do it for you.

If you would like to join the study, please complete the accompanying consent form and return it to the researchers in the envelope provided.

Thank you for thinking about joining our study.

- ***Prof Clare Collins***
- ***Prof Robin Callister***
- ***Dr Tracy Burrows***
- ***Dr Neil Spratt***
- ***Tracy Schumacher***

Appendix 14. Adult information statement

Priority Research Centre
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
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FoodHeartFamily@newcastle.edu.au

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

Information Statement for the Research Project:

LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY Program Study

Prof. Clare Collins, Prof. Robin Callister, Dr Tracy Burrows, Dr Neil Spratt, Ms Tracy Schumacher (student researcher)

Version 6: 25th March, 2013

You and your family are invited to participate in the research project identified above, which is being conducted by researchers from the University of Newcastle. The researchers are Professor Clare Collins, Professor Robin Callister, Dr Tracy Burrows, Dr Neil Spratt and Ms Tracy Schumacher. The project is funded by a grant from the Hunter Medical Research Institute.

Why is the research being done?

Heart disease affects a lot of families in the Hunter Region. Most of those discharged from hospital each year following a heart disease event or a stroke have partners and children. The families of those affected are also at a higher risk of cardiovascular disease (CVD) because of shared genetics and/or a shared home environment.

We are trying to determine whether providing the best available dietary advice to families after an adult member is discharged from hospital after a heart event or stroke can help to reduce the CVD risk in all family members, including children. The dietary advice is to be provided after evaluating your current dietary patterns and is designed to create a foundation for a long-term healthy lifestyle.

Who can participate in the research?

We are accepting two groups of people into this study: families where an adult has already experienced a heart or stoke event and families where at least one adult is at moderate or high risk of cardiovascular disease.

<u>If a family member has already experienced a heart or stroke event:</u>

The family can participate if:

- An adult has in their medical history a diagnosed heart event and is now aged between 25-70 years or if they have a diagnosed stroke episode, they are aged 25-60 years.

- All family members living with the person are willing to participate with them. If the person who had the episode is a grandparent and they wish to involve their children and grandchildren, the grandparents need not be part of the same household.

<u>If a family member is at moderate or high risk of cardiovascular disease:</u>

The family can participate if:

- The family includes an adult who has been assessed to have a moderate or high risk of cardiovascular disease and the person must be aged between 35 - 80 years.
- The family must include members of at least 2 generations. This means that parents and children OR grandparents and grandchildren must be willing to participate.

ALL families must:

- Currently live in the Newcastle Region.
- Be available to attend 2 assessment sessions at the university. It will also be necessary to attend 2 appointments at a local HAPS community clinic.
- Consent to having their child/ children's measurements taken if under 18 years (see below for details).

This study is not suitable for you and your family if you are:

- Not able to speak or read English,
- Unwilling or unable to travel to the University for assessments
- Have any family member who has food allergies.

Please note: You should explain the purpose and requirements of the study to children under the age of 18 before agreeing to participate and give them this information statement to read. There are also age appropriate information statements for children.

What choice do you have?

Participation in this research is entirely by choice. Only those parents and children who give their informed consent will be included in the project. Whether or not you and your family decide to participate, it will not disadvantage you or your family. If you decide to participate you may withdraw from the project at any time without giving a reason and also have the option of withdrawing any data you have provided. Before any child agrees to participate, please advise them that they can choose to withdraw from the study at any stage and that this will not disadvantage them.

What would you and your family be asked to do?

Families who agree to participate in the study will be asked to complete a number of online and over the phone questionnaires to provide information on the current eating habits of each participant. You will then be provided with feedback on your diet, as well as specific recommendations for a heart-healthy diet. You will also be required to participate in assessments, which are described in Table 1.

The study will take place during 2012 and 2013. Families will be randomly allocated to one of two groups; one group will be provided with their dietary advice in the first assessment session and the other group will receive this 12 weeks later. Both groups will receive dietary feedback in the first assessment session. Families will have an equal chance of being in the group that begins immediately or 12 weeks later. Regardless of the group you are in, you will be asked to attend one assessment session upon enrolment and a second session 12 weeks later. If you are in the later group, you will also be invited to attend a third assessment session 24 weeks later. Assessment sessions must be attended by all family member participants (primary participant, partner and child /children). The feedback on your current diet and dietary advice will be provided by a dietitian.

The amount of time needed for the initial assessment will depend on the group allocation and the number of family members attending the session, although it is expected that the session will take approximately 2-3 hours. Nutritious refreshments will be offered to participants during this session.

Adults will be asked to have blood samples taken at their local community HAPS clinic before the initial assessment session. The appropriate forms for this will be supplied to participants to take with them to the chosen collection centre. You will be asked **not** to eat (i.e. have an overnight fast) for the 12 hours before the blood test to ensure accurate measurements. A reminder text will be sent to you for this purpose.

Table 1 outlines the measurements that are to be completed ☑ by each study participant at session 1.

Table 1.	**Primary Participant**	**Partner & other adults**	**Child/ children**
Weight: measured in light clothing without shoes and accurate to 0.1kg	☑	☑	☑
Height: measured without shoes using a portable stadiometer.	☑	☑	☑
Waist circumference: measured using a metal tape measure.	☑	☑	☑
Body composition including fat mass and fat free mass: measured using a bioelectrical impendence analyser whereby you stand on a metal plate and hold onto two rails and a very small electrical current of no harm is passed through the body. This will tell us the percentage body fat and muscle mass.	☑	☑	☑
Skin colour analysis: this analyses the colours in your skin that are affected by health and nutrition. It does this by bouncing white light off the skin and measures each colour that is reflected back.	☑	☑	☑
Blood Pressure: a series of measurements will be taken to obtain blood pressure.	☑	☑	☑
Blood test: a fasting blood test of approximately 5ml will be collected. Your family may choose to have the sample taken at the university or at an earlier, more convenient time.	☑	☑	☒

Central arterial pressure: a test measuring blood pressure near the heart. This test is performed in a similar method to blood pressure, with a cuff being inflated around the upper arm.	☑	☑	☑
Questionnaires: You, your partner and your child / children will be asked to complete online questionnaires with questions about your demographics (e.g. age, gender, etc.), eating and physical activity habits.	☑	☑	☑
Cardiac questionnaire and process evaluation about experience / satisfaction with the program	☑	☒	☒

☑ Denotes the measurement will be undertaken for participant,

☒ denotes the measurement will not be taken for the participant

How much time will it take?

You, your partner and your children will be involved in the study for three months if you are allocated to the immediate dietary advice group or for six months if you are allocated to the dietary advice in session 2 group. We will try to arrange all sessions at a time convenient to your family.

What are the risks and benefits of participating?

All the measures used in the study (height, weight, body composition and questionnaires) have been widely used in research studies conducted by our group and are considered standard measurement tools. All participants will have their height and weight accurately measured by a trained professional. There have been no reported adverse effects from assessing body fat with the body composition analyser, however this measurement is not suitable for anyone with a cardiac pacemaker.

Potential benefits from participating include: Family members will receive feedback about their dietary intakes and guidance on how to improve their dietary patterns to reduce their future cardiovascular disease risk.

How will your privacy be protected?

Data collected by the research team will be collected either electronically or in paper-based form. Any data collected in non-electronic form and will be stored in a locked filing cabinet. Data in electronic form will be in a password protected computer file to ensure the security and confidentiality of any identified data. Only the research team will have access to the raw data. The researchers will enter this raw data into a statistics program. As there is a need to be able to identify individual data due to multiple data entry points, the identifiers will be removed and replaced with a code. Data used for analysis will be de-identified before entry into the statistical program. Once this information is entered on the data file, all raw data will be shredded and no person will be identifiable in the data files or published reports. The data will be kept for 10 years after the study in a password-protected location. A list of names and codes will be stored separately to data files in the chief investigator's office.

How will the information collected be used?

Your blood will be tested for cardiovascular health and nutritional biomarkers, for example: cholesterol, triglycerides and glucose, and stored. All the data collected during the study will be analysed and may be reported at national and international conferences, in scientific publications, and as part of the research thesis for Tracy Schumacher's PhD studies. You will not be identified in any reports arising from the study. At the conclusion of the study you will receive a brief summary of the results from the Chief Investigator, Professor Collins.

What do you need to do to participate?

Please read this Information Statement and be sure you understand its contents before you consent to participate. If there is anything you do not understand, or you have questions, contact the researcher on the details below.

If you would like to participate, please complete the accompanying consent form and return it to the researchers in the reply paid envelope provided. If you consent, you will then be contacted to confirm a time convenient for you to complete the first assessment session and dietetic consultation at the University.

Complaints about this research

This research has been approved by the Hunter New England Human Research Ethics Committee of Hunter New England Local Health District, Reference: 12/05/16/4.01

Should you have concerns about your rights as a participant in this research, or you have a complaint about the manner in which the research is conducted, it may be given to the researcher, or, if an independent person is preferred, to Dr Nicole Gerrand, Manager Research Ethics and Governance, Hunter New England Local Health District, Locked Bag 1, New Lambton NSW 2305, telephone (02) 49214950, email Hnehrec@hnehealth.nsw.gov.au

Further information

If you would like further information please contact the researchers on 49216259 or email FoodHeartFamily@newcastle.edu.au.

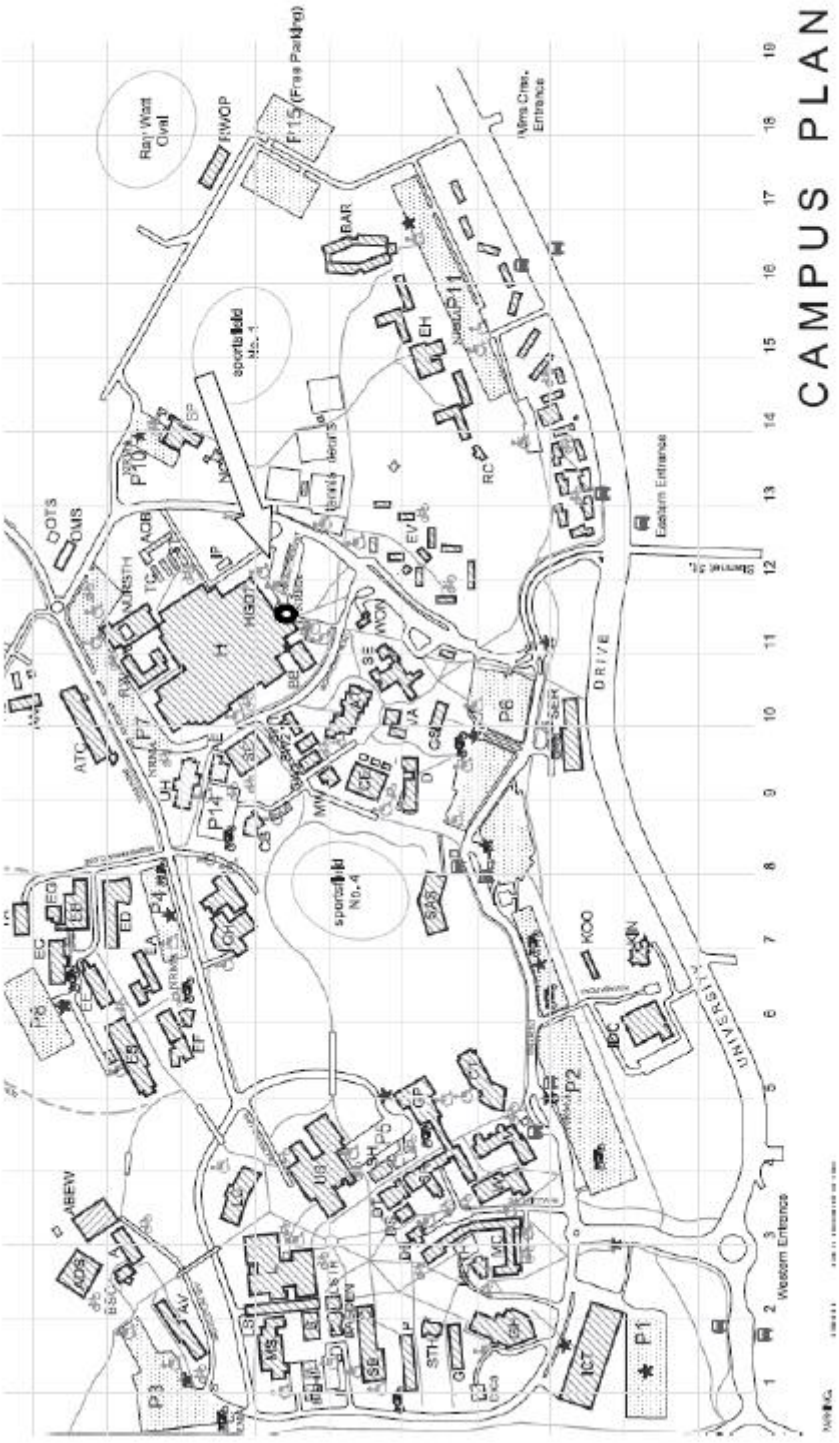
Thank you for considering this invitation.

Research team for the Love your Food, Love your Heart, Love your Family study.

- ***Prof Clare Collins***, School of Health Sciences, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Prof Robin Callister***, School of Biomedical Sciences and Pharmacy, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Dr Tracy Burrows***, School of Health Sciences, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Dr Neil Spratt,*** School of Biomedical Sciences and Pharmacy, Priority Research Centre for Translational Neuroscience and Mental Health, The University of Newcastle.
- ***Tracy Schumacher***, (PhD candidate) School of Health Sciences, University of Newcastle.

An alternate version of the map can be found at:

http://www.newcastle.edu.au/Resources/Locations/Newcastle/campus-map.pdf



Appendix 15. Index participant consent form

Priority Research Centre in Physical Activity and Nutrition
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
4927053 (FAX)
FoodHeartFamily@newcastle.edu.au

Consent form for the Research Project:

LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY Study

Version 4: 25th March, 2013

PRIMARY PARTICIPANT

I give my consent to participate in the project. I understand that the project will be conducted as described in the Information Statement, a copy of which I have retained. I understand that I can withdraw from the project at any time and do not need to give any reason for withdrawing.

I consent to completing the measurements outlined in the Information Statement; attending sessions (1 of which includes a dietetic consultation) all of these with my partner / child / children / grandchild / grandchildren.

- Participating in a 12 week dietary intervention to reduce heart disease risk.
- Having my height, weight, waist circumference, body fat, skin colour analysis, blood pressure and arterial stiffness measured.
- Completing food frequency questionnaires, usual intake records, health and demographic questionnaires, as well as study and program evaluations, some of which will be completed online and over the phone prior to the assessment session.
- Having a blood test to evaluate my health status at each assessment.

YES **NO**

I allow the research team to access my medical records relating to this project.

YES **NO**

I understand that my personal information will remain confidential to the researchers and that data collected from my participation will be used in journal publications and conference presentations. If I decide not to participate or withdraw from the study, it will not affect my relationship with the University of Newcastle. I have had the opportunity to have questions answered to my satisfaction. By signing below I am indicating my consent to participate in the research project.

Your name: _____ **Your child's name:** _____

Your partners name: _____ Birthdate: _____

Signature:_____ **Date:** _____

Contact Details: Phone (Home) _____ (Mobile)_____

e-mail_____

Please return the completed consent in the prepaid envelope enclosed.

Appendix 16. Adult consent form

Priority Research Centre in Physical Activity and Nutrition
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
4927053 (FAX)
FoodHeartFamily@newcastle.edu.au

Love Your Food
Love Your Heart
Love Your Family

Consent form for the Research Project:

LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY Study

Version 4: 25th March, 2013

ACCOMPANYING ADULT

I give my consent to participate in the project. I understand that the project will be conducted as described in the Information Statement, a copy of which I have retained. I understand that I can withdraw from the project at any time and do not need to give any reason for withdrawing.

I consent to completing the measurements outlined in the Information Statement; attending sessions (1 of which includes a dietetic consultation) all of these with my partner / child / children / grandchild / grandchildren.

- Participating in a 12 week dietary intervention to reduce heart disease risk.
- Having my height, weight, waist circumference, body fat, skin colour analysis, blood pressure and arterial stiffness measured.
- Completing a food frequency questionnaire and usual intake record, study and program evaluation questionnaires, some of which will be completed online or over the phone prior to the assessment session.
- Having a blood test to evaluate my health status at each assessment.

YES **NO**

I understand that my personal information will remain confidential to the researchers and that data collected from my participation will be used in journal publications and conference presentations.
If I decide not to participate or withdraw from the study, it will not affect my relationship with the University of Newcastle.
I have had the opportunity to have questions answered to my satisfaction. By signing below I am indicating my consent to participate in the research project.

Your name: _____ **Your child's name:** _____

Your partners name: _____ Birthdate:_____

Signature: _____ **Date:** _____

Contact Details: Phone (Home) _____ (Mobile)_____

E-mail to be sent to: _____

Please return the completed consent in the prepaid envelope enclosed.

Appendix 17. Child and adolescent consent form

Priority Research Centre in Physical Activity and Nutrition
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
4927053 (FAX)
FoodHeartFamily@newcastle.edu.au

Love Your Food
Love Your Heart
Love Your Family

Consent form for the Research Project:

LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY Study

Version 4: 25th March, 2013

PARTICIPANT 3 (Child)

I give my consent to participate in the project. I understand that the project will be conducted as described in the Information Statement, which has been explained to me by my parent or care giver, a copy of which my parent or care giver has retained. I understand that I can withdraw from the project at any time and do not need to give any reason for withdrawing.

I consent to completing the measurements outlined in the Information Statement; attending sessions (1 of which includes a dietetic consultation) all of these with my parents / care givers.

- Participating in a 12 week dietary intervention to reduce heart disease risk.
- Having my height, weight, waist circumference, body fat, skin colour analysis, arterial stiffness and blood pressure measured.
- Completing a food frequency questionnaire and usual intake record, study and program evaluation questionnaires, some of which will be completed online or over the phone prior to the assessment session.

YES **NO**

I understand that my personal information will remain confidential to the researchers and that data collected from my participation will be used in journal publications and conference presentations. If I decide not to participate or withdraw from the study, it will not affect my relationship with the University of Newcastle. I have had the opportunity to have questions answered to my satisfaction.

(Parent) By signing below I am indicating my consent and that of my child to participate in the research project.

Child's name: _____ **Birthdate:** _____

Child Signature: _____ **Date:** _____

Parents name _____

Parent Signature _____ **Date:** _____

Contact Details: Phone (Home) _____ (Mobile)_____

E-mail to be sent to: _____

Please return the completed consent in the prepaid envelope enclosed.

Appendix 18. Sample pages of individual feedback reports

Summary Report

Date: *20th June, 2013*

Dear Jessica,

Thank you for participating in our **Food, Heart, Family Study** which tested whether a family could adopt specific food components that improve heart health. This is an important study that will enable further research into family eating habits and ways to make it easier for people to adopt heart healthy eating patterns. We asked you and your family to undertake many measures and we appreciate your efforts and openness. We have been processing all the information you provided to us and would like to provide you with some feedback on your first set of results. Data collection is underway and we aim to release a report early next year.

Jessica's Report **Study ID: 54932**

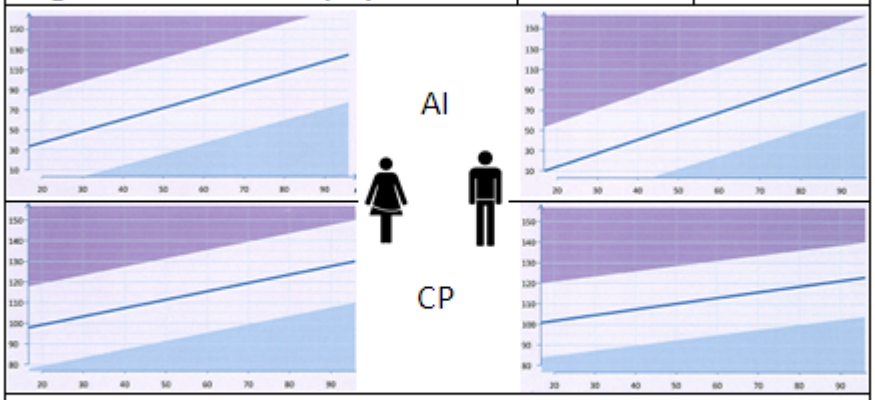
Baseline measurement session date: 20 / 5 / 2013

Follow up measurement session date: / /

Anthropometric Data	Baseline	Follow up
Height (cm):	167.4	
Weight (kg):	70.2	
Body Mass Index (kg/m^2):	25.1	
Your weight classification is ... This BMI range is...		
Waist Circumference (cm):	79.9	
For most people, the following waist measurements are associated with increased risk of chronic disease. ***Increased risk:*** Men: >94 cm, Women: > 80 cm ***Greatly increased risk:*** Men: >102 cm, Women: > 88 cm		
Skeletal Muscle Mass (kg):	26	
This is the amount of muscle in your body. Higher muscle mass helps with strength, stability and weight control.		
Body fat (%):	31	
The comparison reference ranges for adults are 8-24% for males and 21-35% for females.		
Visceral fat (cm^2):	90	
This is the amount of fat that is covering your internal organs. Risk of chronic disease increases with higher visceral fat, but the absolute value of risk is unknown.		
Total body water (l):	36	
This is the amount of water that is within your body.		

Where to get more information about body measurements

The *Measure Up* website at www.measureup.gov.au

Heart rate & blood pressure results	Baseline	Follow up
Heart rate (beats per minute):	65	
A healthy heart rate for an adult is between 60-100 beats per minute, although under 70 is ideal.		
Blood pressure (mmHg): (Systolic/diastolic)	129/69	
Your blood pressure category is... This blood pressure range is... An ideal blood pressure is to have a reading less than 120/80.		
Arterial Stiffness Measures	**Baseline**	**Follow up**
Central blood pressure (mmHg): (Systolic/diastolic)	113/69	
Augmentation index (AI)	128	
		
The augmentation index is a measure of arterial stiffness. Higher central blood pressures and stiffer arteries will result in higher AI values.		

Blood test results

The results that are given below are from the blood tests that you had taken on:

Baseline test date: 10 / 5 /2013

Follow up test date:

Test	Baseline	Follow up
Triglycerides:		
Triglycerides (mmol/L) Low risk: Less than 1.5mmol/L	.84	
Cholesterol:		
LDL cholesterol (mmol/L) Low risk: Less than 3.0mmol/L High risk: More than 4.0mmol/L	2.84	
HDL cholesterol (mmol/L) Low risk: More than 1.0mmol/L	1.7	
Total cholesterol (mmol/L) Low risk: Less than 5.5mmol/L	4.9	
Total:HDL ratio Low risk: Less than 3.0 Average risk: 3.0 – 5.0	2.9	
Insulin:		
Fasting blood glucose level (mmol/L) Normal: 3.5 – 6.0mmol/L Impaired: 6.0 – 7.0mmol/L Diabetic: More than 7.0mmol/L	5	
Insulin (IU/L) (Adult Fasting): <15mIU/L	3.7	

Jessica's Dietary Intake

The table below shows your average daily intake and the Estimated Average Requirement **(EAR)**. The EAR is a daily nutrient level estimated to meet the daily requirements of half the healthy individuals in a particular life stage and gender group. Your intake is based on the answers given on food frequency questionnaire, completed as part of this study, about your usual food habits over the past 6 months.

Other references values for your nutrient intake may be an Acceptable Macronutrient Distribution Range **(AMDR)** or Adequate Intake **(AI)**. The **AMDR** is matched to an average persons need for protein, carbohydrates and fats. **AI** is the daily amount consumed that is associated with good health.

Nutrient	EAR, AMDR or AI	Baseline	Follow up
Energy	-	10582kJ	8459kJ
% energy from core foods	-	54%	55%
% energy from non-core foods	-	46%	45%
Protein	-	95g	89g
% energy from Protein	AMDR: 15-25%	15%	18%
Carbohydrate	-	337g	245g
% energy from Carbohydrate	AMDR: 45-65%	54%	50%
Total Fat	-	74g	62g
% energy from Fat	AMDR: 20-35%	27%	28%

Appendix 19. Sample pages of education booklet

Love Your Food
Love Your Heart
Love Your Family Diet

The food you eat can help keep your heart healthy. The role it plays can be complex and confusing and this can make hard to decide what is best for you and your family. Our "Love your Food, Love your Heart, Love your Family" eating plan is designed for families at increased risk of heart disease and stroke. It has been specifically developed to take advantage of the positive effects food can have on heart health.

The science behind the Love Your Food, Love your Heart, Love Your Family diet...

We have taken the latest research and translated it into a healthy eating plan that the whole family can enjoy.

Research has shown that food affects heart health in a number of ways. These are:

A high fat/ high energy eating habit increases total and "bad" cholesterol (LDL cholesterol) and lowers levels of "good" or HDL cholesterol in the blood

High intakes of saturated fat increase LDL cholesterol

High fibre can help lower cholesterol absorption in the gut.

Recently it has been shown that adding other dietary components can further enhance heart health. These components are soluble fibre, foods enriched with plant stanols/sterols and soy protein.

The Love your Food, Love your Heart, Love your Family diet has 6 key features. It has:

1. Low saturated fat
2. Foods high in soluble fibres
3. Plant sterols
4. Soy products
5. Nuts
6. Fish, particularly those high in omega 3 oils

These components work best when used with other healthy eating habits. Healthy eating habits means having a lot of variety in the foods that are eaten, including a range of fruit and vegetables, low fat dairy products, lean meats and a range of wholegrain breads and cereals.

Soy Proteins

Soy products are available as either ready to eat, or as a base in other meals. They are found in the chilled section of supermarkets, or as pantry items in "health" sections. Soy milks are generally found with other long-life milks or chilled products. You might like to try the following ways of including soy into your eating habits:

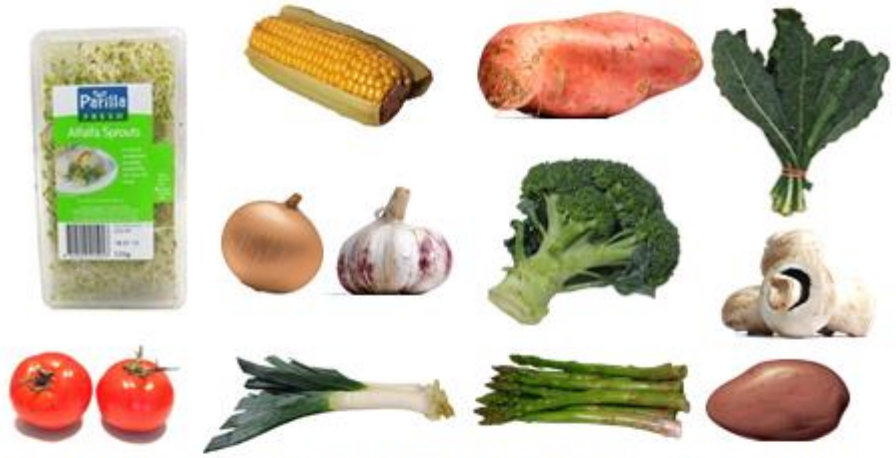
- Mince dishes with half beef and half soy mince substitutes will have the same texture and flavour. Soy mince does not have a lot of flavour on its own, and will absorb the flavours of the sauce surrounding it. You may like to try a pinch of chilli, cayenne or black pepper, garlic or fresh herbs such as parsley or chives.
- Silken tofu is very mild in flavour and blends very easily into any soft textured dish. For example, scrambled eggs with silken tofu blended in will retain the same texture and flavour, but extend the dish further.
- Flavoured tofu can be used in stir-fries, Asian soups and rolled into sushi.
- Use your favourite marinade on kebabs with half chicken cubes and half firm tofu cubes either for the BBQ or in a stir-fry.
- Vegetarian soy products can be directly substituted, such as BBQ or curried sausages or added to a pasta bake (see recipe section).
- Some people find the mild nuttiness of soy milk a pleasant addition to breakfast mueslis and cereals, but be aware that the flavour changes when used in tea and coffee. Try a soy banana smoothie with a pinch of nutmeg and drop of vanilla essence.

- Soy milks can also easily be incorporated into home-baked products, such as cakes, batters and muffins.
- Soy yoghurts can be used as day time snacks or as accompaniments to desserts such as stewed fruit.

Vegetables

This is one of the most important core groups for good health. They are high in vitamins and minerals, and low in kilojoules, saturated fat and cholesterol. Eating the recommended daily amount of vegetables potentially reduces high blood pressure, risk of cardiovascular disease and some kinds of cancer, and is also useful in promoting weight loss.

Serves	Adult Serving size	Child Serving size
Up to 5 a day	½ Cup of cooked vegetables 1 medium potato 1 cup of salad vegetables	¼ Cup of cooked vegetables 1 small potato ½ Cup of salad vegetables

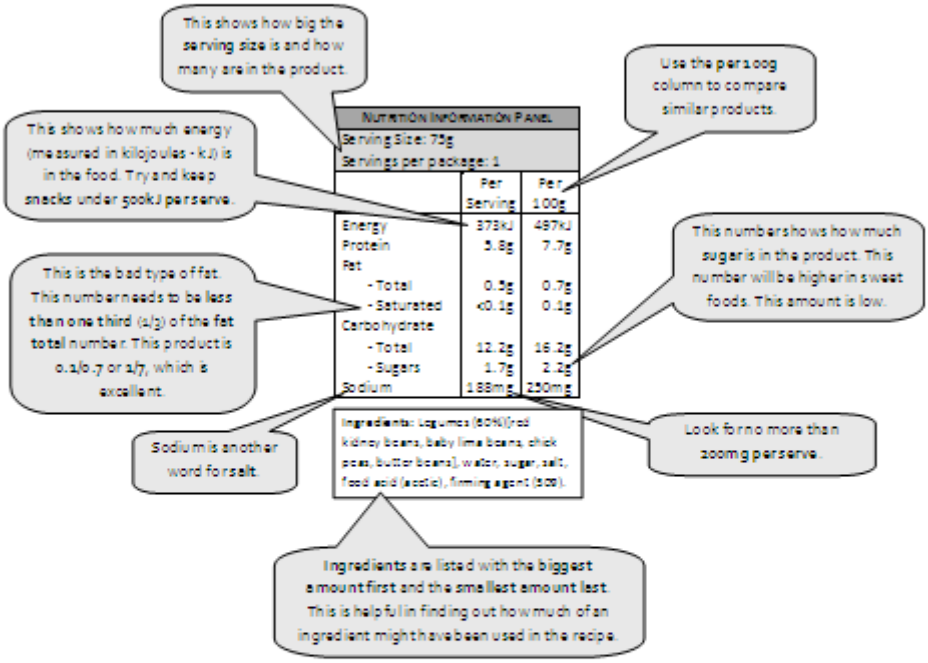


Vegetables should form the basis of most of your meals, and can be easily incorporated into snacks as well *(see recipe section for ideas on how to use vegetables)*. It is important to plan how you are going to use your vegetables when you buy them, as they are commonly thrown out because they have been left in the fridge too long. Try to source a regular supply of vegetables, and frequently check your fridge to ensure that they are not being wasted, as this can become a very expensive habit.

Frozen and low salt canned vegetables are a great alternative to fresh vegetables, and can often be cheaper. Use a mix of both to take advantage of cost and availability. As with fruit, greater variety is better. This can be achieved by using many different coloured groups of vegetables. For example, red, green, purple, yellow, orange and white can be made into a salad by using tomato, spinach, beetroot, corn, carrot and mushrooms.

Compare Products

There are many products available that might be suitable for you and your family. Reading the nutrition panel and ingredients list on commercial products will help you to identify the best choices.



The diagram shows the minimum amount of information that needs to be on a nutrition panel. However, manufacturers commonly list more than they need to. Other nutrients commonly listed that are useful in deciding whether a product is heart smart or not are listed here, with targets to aim for:

Fibre or dietary fibre	Cholesterol	Trans Fats	Plant Sterols
3 grams per serve	Less than 25mg per serve in packaged products	Less than 0.5g per 100g	0.5-1.0g per serve

Shopping List

Below is a list of foods that may help when shopping for foods that are heart healthy. Not all the foods contained in the booklet are listed, although this list can be used as a starting point. Some of these foods are pantry items, such as canned kidney beans and rolled oats. These can be stored for longer periods of time.

Don't forget that herbs and spices can make a big difference to the enjoyment of your foods. They can be dried, fresh or grown in your own garden.

Fresh Foods		Pantry Items	Chilled foods
Summer	Autumn	Breakfast cereals containing soluble fibre	Cheese with sterols
Apricots	Apples	Canned tuna, sardines or mackerel	Cooking oils with sterols
Beetroot	Berries	Cannellini beans	Eggs
Berries	Broccoli	Chick peas	Fresh or frozen fish
Eggplant	Kiwifruit	Grains: rice, cous cous, etc	Kangaroo sausages
Mushrooms	Mushrooms	Herbs and spices:	Lite or skim milk
Strawberries	Pears	Kidney beans	Lite cheese
Sweet corn	Strawberries	Lentils	Low fat mince
Tomato	Tomato	Lite soy milk (long life)	Low fat yoghurt
Winter	Spring	Nuts: almonds, walnuts, pecans, macadamias etc	Margarine with sterols
Broccoli	Broccoli	Psyllium husks or oat bran	Soy cheese or yoghurt
Lebanese cucumbers	Leek	Rolled oats	Soy proteins: sausages, deli luncheon slices
Leek	Mushrooms	Textured vegetable protein	Tofu
Mushrooms	Oranges	Wholegrain breads and pastas	
Oranges	Spinach		
Potato	Strawberries		
Spinach	Tomato		

Recipe

Bake in the Bag Fish

Ingredients:

4 fillets of your favourite fish, such as salmon, gemfish, dory or whiting (around 150g each)

4 cups of baby spinach leaves

1 red capsicum, deseeded and cut into strips

2 carrots, thinly sliced on the diagonal

½ Spanish (red) onion, thinly sliced

1 lemon, quartered

Fresh herbs, such as thyme, oregano or coriander

Drizzle of olive oil

Pepper to taste

Contains:
Fish & seafood
Vegetables

Nutrition Information		
	Per Serve	Per 100g
Energy (kJ)	1000	347
Protein (g)	394	13.7
Fat		
- Total (g)	6.6	2.3
- Saturated fat (g)	1.2	0.4
Carbohydrate		
- Total (g)	4.3	1.5
- Sugars (g)	4.3	1.5
- Fibre (g)	3.7	1.3
Sodium (mg)	135	47

Method:

1. Preheat oven to 240°C.
2. Cut 4 pieces of aluminium foil to the length of 50cm. Each piece of foil will contain ¼ of the ingredients above.
3. Put 1 cup of baby spinach leaves in the middle third of the foil, and 4 cm from the outside edges.
4. Lay the fish fillet on top of the spinach. Season with pepper and olive oil.
5. Top with herbs, onion, capsicum and carrots.
6. Squeeze the juice of a lemon quarter onto the vegetables.

7. Tightly fold the short edges of the foil together, leaving a small space above the fish and vegetables. Fold the remaining edges of the foil together, making an airtight bag.
8. Bake in oven for approximately 10-12 minutes. Thicker fillets may require longer cooking times.
9. Serve with rice, homemade potato wedges or on a bed of couscous.

Appendix 20. Statement of contribution and collaboration for Chapter 7

I attest that Research Higher Degree candidate Tracy Leigh Schumacher contributed to the following chapter:

Schumacher T, Burrows T, Thompson D, Callister R, Spratt N, Collins C. Preventative dietary patterns within the context of a family cardiovascular disease risk reduction intervention.

Tracy L Schumacher was primarily responsible for data collection, data analysis, manuscript preparation and contributed to study design. Dr Deborah Thompson, Dr Tracy Burrows, Professor Robin Callister, Dr Neil Spratt and Professor Clare Collins contributed to the study design and manuscript preparation. All authors approved the final chapter.

Ms Tracy Schumacher (25th November, 2015)

Dr Tracy Burrows (25th November, 2015)

Dr Deborah Thompson (26th November, 2015)

Professor Robin Callister (3rd December, 2015)

Dr Neil Spratt (25th November, 2015)

Professor Clare Collins (26th November, 2015)

Professor Robert Callister (3rd December, 2015)

(Assistant Dean Research Training)

Appendix 21. Information statement for qualitative interviews

Priority Research Centre
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
49217053 (FAX)
FoodHeartFamily@newcastle.edu.au

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

Information Statement for INTERVIEWS for the Research Project:

LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY Program Study

Prof. Clare Collins, Prof. Robin Callister, Dr Tracy Burrows, Dr Neil Spratt, Ms Tracy Schumacher (student researcher)

Version 2: 11th November, 2013

You and your family have previously provided consent to participate in the research project identified above, which is being conducted by researchers from the University of Newcastle. The researchers Professor Clare Collins, Professor Robin Callister, Dr Tracy Burrows, Dr Neil Spratt and Ms Tracy Schumacher would like to thank you for your involvement in the study to date. The project is funded by a grant from the Hunter Medical Research Institute.

Why is the research being done?

We would like to investigate the importance of eating heart healthy foods within families at increased risk of heart disease. We are conducting structured telephone interviews with participants who have already completed the study.

The major aim of the telephone interview with participants is to explore the importance of heart healthy eating habits and what situations can aid or act as barriers in families from an individual's point of view.

Who can participate in the research?

You can participate in this interview if you have already completed the Love your Food, Love your Heart, Love your Family study.

What choice do you have?

Participation in this telephone interview is entirely by choice. You will only be included in the interview if you have given your informed consent. Whether or not you decide to participate, it will not disadvantage you or your family. If you decide to participate you may withdraw from the project at any time without giving a reason and also have the option of withdrawing any data you have provided. This decision will not disadvantage you in any way.

What would you be asked to do?

You will be invited to participate in a telephone interview with the lead student researcher, Ms Tracy Schumacher. The following topics will be explored:

- Why you chose to participant in the study with your family

- The difficulties in trying to eat heart healthy foods
- How heart health is viewed and managed in your family
- The role of the health professional in food-related topics that are sensitive in nature

The telephone interviews will take place during 2013 and 2014.

Transcriptions

All interview audio recordings will be transcribed by an transcription service, using only de-identified data.

How much time will it take?

The telephone interviews will take approximately 20-30 minutes.

What are the risks and benefits of participating?

There is the potential risk for participants to experience emotional distress, particularly when exploring issues which touch on their family relationships. Participants experiencing emotional distress as a result of the interview will be provided with contact details for a counselling service, such as Lifeline (phone 131114, http://www.lifeline.org.au/)

The information provided by participants will help inform how other family-based studies are designed, which is hoped will have the long-term benefit in improving heart health in families in the future.

How will your privacy be protected?

Your name for this telephone interview will be replaced by a pseudonym of your choosing and this is the name that will be recorded. The pseudonym will be used during transcription and in any further data analysis, or publications or reports where you are quoted. Information that links your pseudonym and your study identification number will be stored securely and accessed only by the researcher, unless you consent otherwise, except as required by law. The digital recording of the interview will be stored securely on a password protected computer for 5 years.

How will the information collected be used?

All the data collected during the telephone interviews will be analysed and may be reported at national and international conferences, in scientific publications, and as part of the research thesis for Tracy Schumacher's PhD studies. You will not be identified in any reports arising from the study. At the conclusion of the study you will receive a brief summary of the results from the Chief Investigator, Professor Collins.

What do you need to do to participate?

Please read this Information Statement and be sure you understand its contents before you consentto participate. If there is anything you do not understand, or you have questions, contact the researcher on the details below.

If you would like to participate, please complete the accompanying consent form and return it to the researchers in the reply paid envelope provided. If you consent, you will then be contacted to confirm a time convenient for you to complete the first assessment session and dietetic consultation at the University.

Complaints about this research

This research has been approved by the Hunter New England Human Research Ethics Committee of Hunter New England Local Health District, Reference: 12/05/16/4.01

Should you have concerns about your rights as a participant in this research, or you have a complaint about the manner in which the research is conducted, it may be given to the researcher, or, if an independent person is preferred, to Dr Nicole Gerrand, Manager Research Ethics and Governance, Hunter New England Local Health District, Locked Bag 1, New Lambton NSW 2305, telephone (02) 49214950, email Hnehrec@hnehealth.nsw.gov.au

Further information

If you would like further information please contact the researchers on 49216259 or email FoodHeartFamily@newcastle.edu.au.

Thank you for considering this invitation.

Professor Clare Collins
Chief Investigator

Tracy Schumacher
Student researcher

Research team for the Love your Food, Love your Heart, Love your Family study.

- ***Prof Clare Collins***, School of Health Sciences, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Prof Robin Callister***, School of Biomedical Sciences and Pharmacy, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Dr Tracy Burrows***, School of Health Sciences, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Dr Neil Spratt,*** School of Biomedical Sciences and Pharmacy, Priority Research Centre for Translational Neuroscience and Mental Health, The University of Newcastle.
- ***Tracy Schumacher***, (PhD candidate) School of Health Sciences, University of Newcastle.

Appendix 22. Consent forms for qualitative interviews

Priority Research Centrein Physical Activity and Nutrition
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
4927053 (FAX)
FoodHeartFamily@newcastle.edu.au

Love Your Food
Love Your Heart
Love Your Family

Consent form for INTERVIEWS the Research Project:
LOVE YOUR FOOD, LOVE YOUR HEART, LOVE YOUR FAMILY Study
Version 1: 4th November, 2013

I give my consent to participate in the telephone interviews for the project given above. I understand that the project will be conducted as described in the Information Statement, a copy of which I have retained. I understand that I can withdraw from the project at any time and do not need to give any reason for withdrawing.

I consent to undertaking an individual telephone interview to explore the following topics:

- Why I chose to participate in the Love your Food, Love your Heart, Love your Family study
- The difficulties in trying to eat heart healthy foods
- How heart health is viewed and managed in my family
- The role of the health professional in food-related topics that are sensitive in nature

YES **NO**

I understand that my personal information will remain confidential to the researchers and that data collected from my participation will be used in journal publications and conference presentations. If I decide not to participate or withdraw from the study, it will not affect my relationship with the University of Newcastle. I have had the opportunity to have questions answered to my satisfaction. By signing below I am indicating my consent to participate in the research project.

Your name: _____

Signature:_____ **Date:** _____

Contact Details: Phone (Home) _____ (Mobile)_____

e-mail_____

Please return the completed consent in the prepaid envelope enclosed.

Appendix 23. Script for qualitative interview

Qualitative questions, including prompts and probes

Version 1: 4 November, 2013

Background:

This interview is designed for individual family members who have already completed the first 3 months of the study. Ideally, this would be done over the phone at the convenience of the people we are calling.

Introduction:
Hi xxxxx, thanks for speaking with me tonight. As you know, we have asked people that joined the Food, Heart, Family for further help as the study has raised other questions about barriers and family dynamics that we feel need investigating. So tonight I will be asking a series of questions about your reasons for joining the study, problems that you may encounter when you try to eat a healthier diet, how your family addresses health topics, and the role of a health professional when there is a food-related health problem in your family. We are planning on using this information to help engage other people in family-based studies and to ensure that future studies include the information that people want, and are delivered in a way that meets their needs. This interview should take approximately 20-30 minutes. With your permission, we would like to record the interview, and this is to speed things up and to ensure we have all the information that you give us and that we use it in the context it was given. We haven't started recording yet, and before we do, I will ask you to choose a different name for the interview so that your privacy is protected at all times. Your name will not appear in any reports or papers, and any information that we do collect will be keep private and confidential in a secure location. I would also ask that if you refer to any other member of your family, that you call them by their relationship, rather than by name. For example, your son, daughter or partner. Do you have any questions before we start recording? Do I have your permission to start recording?

Topic: Why did you volunteer?

1a) Let's start by choosing a name other than your real name. We will use this in the interview instead of your real name to protect your privacy. Any name you want to use is ok. It could be your favourite actor, your favourite cartoon character, etc.

1b) What name would you like to be called? Could you tell me something about your reasons for choosing that name? [keep brief]

2) Ok. [insert name]. Let's start by talking about how you heard about the study?

3a) What were your reasons for signing up?

Probe:

- Was there one family member that wanted to do this more than others? *If yes, who?*

4) What made you want to do this with your family?

Prompts that can be used:

- *social*
- *health*
- *support or concern for other family members*
- *encouraged by someone else in the family*

5) How would you rate your concern about your heart health, if 1 was not concerned and 5 was very concerned?

Probe:

*-Why have you chosen **X**?*

6) What impact, if any, do you think eating habits have on your heart health, if 1 was what I eat makes no difference to my heart health, and 5 was everything I eat makes a difference to my heart health?

Probe:

*-Why have you chosen **X**?*

Topic: Eating well – is it a problem?

7. a) How would you define "healthy food"?
b) What makes food healthy or unhealthy?
c) Can you give me examples of healthy foods or unhealthy foods?

8. What barriers, if any, make it hard for your family to eat heart healthy foods?

Probe:

- Are any unhealthy foods purchased and bought into the house that think shouldn't be there? *(give examples)*
- Do you think this is a problem? Why?

9. How would you rate the healthiness of your food patterns before you started the study, based on a scale of 1 to 10 with 10 being the healthiest?

10. And how would you rate it now?

Probe:

- Please tell me the reasons your rating has changed / stayed the same?
- **If rating has increased:** What have you done to make your diet healthier?
- Has the study changed the way you think about what a healthy diet is? *(give examples)*

Topic: Family dynamics

11. On a scale of 1 to 5, with one being not concerned and 5 being very concerned, how concerned are others in your immediate family about what they eat?

12. On the same rating scale, how concerned are others in your family about their heart health?

Probe:

- Why have you given them these ratings?

If different level of concern as given in Question 5:

13) What do you do when your significant others are not as concerned about their heart health or food intake as you are?

If same level of concern as given in Question 5:

13) What would you do if any of your significant others were not as concerned as you are their heart health?

Prompts that can be used:

- E.g. address the issue with the person by talking about it or change the home environment

14. Are there eating topics that are avoided in your household because they might cause conflict? *Yes / No*

Prompts that can be used:

- E.g. the types or amounts of certain foods, or foods that someone thinks might be damaging to a another person's health

Prompts that can be used if YES:

- Can you tell me what are the topics?
- What usually happens when these topics are bought up?

15. When someone in the family has a health problem, what does your family usually do?

Prompts that can be used:

- Is it discussed, avoided, plan changes…
- Is there someone in the family that other family members go to, to talk about their health issues? (why?)
- What do **YOU** do if someone in the family has a health problem?

Topic: Role of the health professional in topics that may be sensitive in nature

16. If there was a health problem related to food in your family that could cause potential conflict, is there one kind of health professional that you would choose over another to help with this?

Prompts that can be used:

- Do you think it makes a difference which health professional addresses the issue? For example, GP, dietitian, nurse or pharmacist?
- Is there a health professional that you would trust more than another in this situation?

Probe:

- Why do you think *(X health professional)* will be the best person for this sensitive topic?
- How do you think that talking this over with *(X health professional)* will change the way this problem is dealt with within your family?

Topic: Summary

So, to summarise all that we talked about tonight, you've said… **[summarise briefly]**. Is this accurate or is there something that you would like to change or clarify? Have we missed anything that you would like to talk about and haven't had a chance to say?…

…Thank you for your time tonight, it is very much appreciated. You will receive your gift voucher in the mail sometime in the next few days. Have a good evening.

Appendix 24. Statement of contribution and collaboration for Chapter 8

I attest that Research Higher Degree candidate Tracy Leigh Schumacher contributed to the following paper:

Schumacher T, Burrows T, Rollo M, Spratt N, Callister R, Collins C. Effectiveness of a brief dietetic intervention for hyperlipidaemic adults using individually-tailored dietary feedback. Submitted to *Journal of Cardiopulmonary Rehabilitation and Prevention*.

Tracy Schumacher was primarily responsible for data collection, data analysis, initial manuscript preparation and contributed to study design. Dr Neil Spratt was assisted with data collection and contributed to manuscript drafting. Dr Tracy Burrows, Dr Megan Rollo, Professor Robin Callister and Professor Clare Collins contributed to the study design, nutrient and data analysis and manuscript preparation. All authors approved the final manuscript.

Ms Tracy Schumacher (25th November, 2015)

Dr Tracy Burrows (25th November, 2015)

Dr Megan Rollo (7th December, 2015)

Dr Neil Spratt (25th November, 2015)

Professor Robin Callister (25th November, 2015)

Professor Clare Collins (25th November, 2015)

Professor Robert Callister (7th December, 2015)

(Assistant Dean Research Training)

Appendix 25. Ethics approval: University of Newcastle

HUMAN RESEARCH ETHICS COMMITTEE

Notification of Expedited Approval

To Chief Investigator or Project Supervisor:	**Professor Clare Collins**
Cc Co-investigators / Research Students:	**Associate Professor Lisa Wood** **Doctor Megan Rollo** **Doctor Neil Spratt** **Professor Robin Callister** **Doctor Tracy Burrows** **Miss Tracy Schumacher**
Re Protocol:	**Cardiovascular Health Eating Questionnaire Using Propensities (CHEQ UP) validation study**
Date:	**17-Jan-2014**
Reference No:	**H-2013-0420**
Date of Initial Approval:	**17-Jan-2014**

Thank you for your **Response to Conditional Approval (minor amendments)** submission to the Human Research Ethics Committee (HREC) seeking approval in relation to the above protocol.

Your submission was considered under **Expedited** review by the Ethics Administrator.

I am pleased to advise that the decision on your submission is **Approved** effective **17-Jan-2014**.

In approving this protocol, the Human Research Ethics Committee (HREC) is of the opinion that the project complies with the provisions contained in the National Statement on Ethical Conduct in Human Research, 2007, and the requirements within this University relating to human research.

Approval will remain valid subject to the submission, and satisfactory assessment, of annual progress reports. *If the approval of an External HREC has been "noted" the approval period is as determined by that HREC.*

The full Committee will be asked to ratify this decision at its next scheduled meeting. A formal *Certificate of Approval* will be available upon request. Your approval number is **H-2013-0420**.

If the research requires the use of an Information Statement, ensure this number is inserted at the relevant point in the Complaints paragraph prior to distribution to potential participants You may then proceed with the research.

Conditions of Approval

This approval has been granted subject to you complying with the requirements for *Monitoring of Progress*, *Reporting of Adverse Events*, and *Variations to the Approved Protocol* as detailed below.

PLEASE NOTE:
In the case where the HREC has "noted" the approval of an External HREC, progress reports and reports of adverse events are to be submitted to the External HREC only. In the case of Variations to the approved protocol, or a Renewal of approval, you will apply to the External HREC for approval in the first instance and then Register that approval with the University's HREC.

- ***Monitoring of Progress***

Other than above, the University is obliged to monitor the progress of research projects involving human participants to ensure that they are conducted according to the protocol as approved by the HREC. A progress report is required on an annual basis. Continuation of your HREC approval for this project is conditional upon receipt, and satisfactory assessment, of annual progress reports. You will be advised when a report is due.

- ***Reporting of Adverse Events***

1. It is the responsibility of the person **first named on this Approval Advice** to report adverse events.
2. Adverse events, however minor, must be recorded by the investigator as observed by the investigator or as volunteered by a participant in the research. Full details are to be documented, whether or not the investigator, or his/her deputies, consider the event to be related to the research substance or procedure.
3. Serious or unforeseen adverse events that occur during the research or within six (6) months of completion of the research, must be reported by the person first named on the Approval Advice to the (HREC) by way of the Adverse Event Report form (via RIMS at https://rims.newcastle.edu.au/login.asp) within 72 hours of the occurrence of the event or the investigator receiving advice of the event.
4. Serious adverse events are defined as:
    - Causing death, life threatening or serious disability.
    - Causing or prolonging hospitalisation.
    - Overdoses, cancers, congenital abnormalities, tissue damage, whether or not they are judged to be caused by the investigational agent or procedure.
    - Causing psycho-social and/or financial harm. This covers everything from perceived invasion of privacy, breach of confidentiality, or the diminution of social reputation, to the creation of psychological fears and trauma.
    - Any other event which might affect the continued ethical acceptability of the project.
5. Reports of adverse events must include:
    - Participant's study identification number;
    - date of birth;
    - date of entry into the study;
    - treatment arm (if applicable);
    - date of event;
    - details of event;
    - the investigator's opinion as to whether the event is related to the research procedures; and
    - action taken in response to the event.
6. Adverse events which do not fall within the definition of serious or unexpected, including those reported from other sites involved in the research, are to be reported in detail at the time of the annual progress report to the HREC.

- ***Variations to approved protocol***

If you wish to change, or deviate from, the approved protocol, you will need to submit an *Application for Variation to Approved Human Research* (via RIMS at https://rims.newcastle.edu.au/login.asp). Variations may include, but are not limited to, changes or additions to investigators, study design, study population, number of participants, methods of recruitment, or participant information/consent documentation. **Variations must be approved by the (HREC) before they are implemented** except when Registering an approval of a variation from an external HREC which has been designated the lead HREC, in which case you may proceed as soon as you receive an acknowledgement of your Registration.

Linkage of ethics approval to a new Grant

HREC approvals cannot be assigned to a new grant or award (ie those that were not identified on the application for ethics approval) without confirmation of the approval from the Human Research Ethics Officer on behalf of the HREC.

Best wishes for a successful project.

Professor Allyson Holbrook
Chair, Human Research Ethics Committee

For communications and enquiries:
Human Research Ethics Administration

Research Services
Research Integrity Unit
The Chancellery
The University of Newcastle
Callaghan NSW 2308
T +61 2 492 17894
F +61 2 492 17164
Human-Ethics@newcastle.edu.au

RIMS website - https://RIMS.newcastle.edu.au/login.asp

Linked University of Newcastle administered funding:

Funding body	Funding project title	First named investigator	Grant Ref
Hunter Medical Research Institute/Cardiovascular Grant(**)	Validation of a Food Frequency Questionnaire to detect changes in diet-related cardiovascular disease risk	Collins Clare,	G1301346

Appendix 26. Information statement

Professor Clare Collins
C/O Priority Research Centre for Physical Activity and Nutrition
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49215646 (PH)
49217053 (FAX)
Clare.Collins@newcastle.edu.au

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

Information Statement for the Research Project:

C.H.E.Q. U.P. Validation Study

(the Cardiovascular Health Eating Questionnaire Using Propensity)

Prof. Clare Collins, Prof. Robin Callister, A/Prof. Lisa Wood, Dr. Neil Spratt, Dr. Tracy Burrows, Dr. Megan Rollo, Ms Tracy Schumacher (student researcher)

Version 4: 15th August, 2014

You are invited to participate in the research project identified above, which is being conducted by researchers from the University of Newcastle. The researchers are Professor Clare Collins, Professor Robin Callister, Associate Professor Lisa Wood, Dr. Neil Spratt, Dr. Tracy Burrows, Dr. Megan Rollo and Ms Tracy Schumacher. The project is funded by a grant from the Hunter Medical Research Institute.

Why is the research being done?

Heart disease affects a lot of people in the Hunter and New England regions of New South Wales. An accurate method of measuring the quality of eating patterns is needed for people with or at risk of heart disease to ensure targeted lifestyle advice is given by their health professionals.

Dietary advice can reduce a person's risk of heart disease by increasing the quality of the diet and targeting foods known to improve risk factors associated with heart disease. We are trying to validate a questionnaire to measure the types and amounts of foods people eat that are associated with increased heart health. This questionnaire is designed to be completed online by people who have experienced a cardiovascular disease event, such as a heart attack or stroke, or who have been identified as being at increased risk of heart disease. The tool is to evaluate current eating patterns in terms of heart health and be used in conjunction with dietary advice that is designed to create a foundation for a long-term healthy lifestyle.

Who can participate in the research?

Any person who is aged between 18 and 75 years and has a blood cholesterol sample that has been taken within the last 6 months that shows ANY of the following is eligible to join:

- LDL cholesterol > 4.00 mmol/L
- Total / HDL ratio > 5.00
- Total cholesterol > 5 mmol/L

ALL participants must:

- Provide a copy of a blood cholesterol sample that has been taken within the last 6 months
- Currently live in the Newcastle or Tamworth region.
- Be available to attend 2 assessment sessions at either the University of Newcastle's Callaghan campus or the Department of Rural Health in Tamworth.
- Have access to telephone, emails and the internet

This study is not suitable for you if you are:

- Not able to speak or read English
- Unwilling or unable to travel to the University for assessments
- Have no medical conditions that restrict the types of foods you can eat, such as coeliac disease or nut allergies
- Are on medication for thyroid conditions
- Pregnant or planning to become pregnant within the next 6 months

What choice do you have?

Participation in this research is entirely by choice. Only those people who give their informed consent will be included in the project. Whether or not you decide to participate, it will not disadvantage you in any way. If you decide to participate you may withdraw from the project at any time without giving a reason and also have the option of withdrawing any data you have provided.

What would you be asked to do?

People who agree to participate in the study will be asked to complete a number of online and over the phone questionnaires to provide information on their current eating habits. They will also be requested to provide access to cholesterol blood test records that have been taken in the previous 6 months. Participants will then be provided with feedback on their current diet, as well as specific recommendations for a heart healthy diet. A summary of all assessments participants will be required to undertake can be found on page 3.

The study will take place during 2014. Participants will be asked to fill out online questionnaires about what they are currently eating, their activity levels and general health, including any medical history that is related to heart health. Participants will also be contacted by phone, where they will be asked about what they ate on the day before.

Participants will be asked to attend two assessment sessions that are scheduled a minimum of six weeks apart. The assessment sessions will include having fasting blood samples and physical measures taken. In the first session, participants will receive feedback on their current diet and be advised by an Accredited Practicing Dietitian about dietary patterns that improve heart health and reduce blood cholesterol. As the measurement sessions involve having fasting blood samples taken, participants are requested not to eat or drink any beverages, excepting water, for the 12 hours prior to their appointment. Reminders not to eat will be offered to all participants.

The amount of time needed for the initial assessment will take approximately 1.5 hours. Food and beverages will be offered to participants during this session after initial measures and fasting blood samples are taken.

After the initial consultation, participants are expected to follow the recommended eating patterns for a period of six weeks. At the end of this six week period, participants will be asked to again fill out online questionnaires, and participate in phone calls where they are again asked about what they ate the day before. They will then return to the University of Newcastle to undertake physical measures and have blood samples taken. They will receive feedback on their progress at this time and after results from their blood tests have been obtained.

Participants will be asked to undertake the following measures:

- **Weight:** measured in light clothing without shoes and accurate to 0.1kg
- **Height:** measured without shoes using a portable stadiometer.
- **Waist circumference**: measured using a metal tape measure.
- **Body composition including fat mass and fat free mass**: measured using a bioelectrical impendence analyser whereby you stand on a metal plate and hold onto two rails and a very small electrical current of no harm is passed through the body. This will tell us the percentage body fat and muscle mass.
- **Blood Pressure and central arterial pressure:** a test measuring blood pressure near the heart. This test is performed by a cuff being inflated around the upper arm.
- **Blood test:** a fasting blood test of approximately 15ml will be collected. This will be collected during your appointments at the University of Newcastle.
- **Questionnaires:** You will be asked to complete online questionnaires with questions about your demographics (e.g. age, gender, etc.), eating and physical activity habits and general health.
- **24hr diet recalls:** You will be asked about what you ate the day before on 4 separate occasions over the phone at a time of your convenience.

How much time will it take?

You will be involved in the study for six weeks, not including time taken to complete online questionnaires, undertake phone calls and schedule appointments. We will try to arrange all measurement sessions at a time convenient to you. It is expected that each phone call regarding what was eaten on the day before will take less than 30 minutes. The length of time needed for individual online questionnaires will vary between approximately 20-30 minutes.

What are the risks and benefits of participating?

All the measures used in the study (height, weight, body composition and questionnaires) have been widely used in research studies conducted by our group and are considered standard measurement tools. All participants will have their height and weight accurately measured by a trained professional. There have

been no reported adverse effects from assessing body fat with the body composition analyser, although this measurement is not suitable for anyone with a cardiac pacemaker. Those with pacemakers are still eligible to join the study but must declared this condition to the researchers. Participants may also be asked to increase their intake of nuts and / or soy, which has the potential to cause allergic reactions in sensitive people.

Potential benefits from participating include: participants will receive feedback about their current dietary intakes and a 45 minute dietary counselling session by an Accredited Practicing Dietitian that advises on how to improve dietary patterns to reduce their future cardiovascular disease risk using the best available evidence. Participants will also be provided with a grocery pantry pack of foods to try when initially starting the eating plan. There are no costs associated with any of the tests performed and participants will receive a copy of their results for future reference.

How will your privacy be protected?

Data collected by the research team will be collected either electronically or in paper-based form. Any data collected in non-electronic form and will be stored in a locked filing cabinet. Data in electronic form will be in a password protected computer file to ensure the security and confidentiality of any identified data. Only the research team will have access to the raw data. The researchers will enter this raw data into a statistics program. As there is a need to be able to identify individual data due to multiple data entry points, the identifiers will be removed and replaced with a code. Data used for analysis will be de-identified before entry into the statistical program. Once this information is entered on the data file, all raw data will be shredded and no person will be identifiable in the data files or published reports. The data will be kept for 10 years after the study in a password-protected location. A list of names and codes will be stored separately to data files in the chief investigator's office.

How will the information collected be used?

Your blood will be tested for cardiovascular health and nutritional biomarkers, for example: cholesterol, triglycerides and glucose, and stored. All the data collected during the study will be analysed and may be reported at national and international conferences, in scientific publications, and as part of the research thesis for Tracy Schumacher's PhD studies. You will not be identified in any reports arising from the study. At the conclusion of the study you will receive a brief summary of the results from the Chief Investigator, Professor Collins. The funding body (Hunter Medical Research Institute) will receive brief updates as to the progress of the study and a short summary of results.

What do you need to do to participate?

Please read this Information Statement and be sure you understand its contents before you consent to participate. If there is anything you do not understand, or you have questions, contact the researcher on the details below.

If you would like to participate, please complete the accompanying consent form and return it to the researchers in the reply paid envelope provided. If you consent, you will then be contacted to confirm a time convenient for you to complete the first assessment session and dietetic consultation at the University.

Complaints about this research

This project has been approved by the University's Human Research Ethics Committee, Approval No. H-2013-0420.

Should you have concerns about your rights as a participant in this research, or you have a complaint about the manner in which the research is conducted, it may be given to the researcher, or, if an independent person is preferred, to the Human Research Ethics Officer, Research Office, The Chancellery, The University of Newcastle, University Drive, Callaghan NSW 2308, Australia, telephone (02) 49216333, email Human-Ethics@newcastle.edu.au.

Further information

If you would like further information please contact the researchers on 49216259 or email Tracy.Schumacher@uon.edu.au.

Thank you for considering this invitation.

Professor Clare Collins
Chief Investigator
C/O Priority Research Centre for Physical Activity & Nutrition
Room 310 ATC Building
University of Newcastle
Callaghan NSW 2308
Clare.Collins@newcastle.edu.au
Ph: 49215646

Tracy Schumacher
Student Investigator

Research team for the C.H.E.Q. U.P. validation study.

- ***Prof Clare Collins***, School of Health Sciences, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Prof Robin Callister***, School of Biomedical Sciences and Pharmacy, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Associate Professor Lisa Wood,*** School of Biomedical Sciences and Pharmacy, Priority Research Centre for Asthma & Respiratory Diseases, The University of Newcastle.
- ***Dr Neil Spratt,*** School of Biomedical Sciences and Pharmacy, Priority Research Centre for Translational Neuroscience and Mental Health
- ***Dr Tracy Burrows***, School of Health Sciences, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Dr Megan Rollo,*** School of Health Sciences, Priority Research Centre for Physical Activity and Nutrition, The University of Newcastle.
- ***Tracy Schumacher***, (PhD candidate) School of Health Sciences, University of Newcastle.

Appendix 27. Participant consent form

Priority Research Centre in Physical Activity and Nutrition
Faculty of Health
The University of Newcastle
Callaghan NSW 2308
49216259 (PH)
4927053 (FAX)
Tracy.Schumacher@uon.edu.au

Consent form for the Research Project:

C.H.E.Q. U.P validation Study

Version 1: 29th October, 2013

I give my consent to participate in the project. I understand that the project will be conducted as described in the Information Statement, a copy of which I have retained. I understand that I can withdraw from the project at any time and do not need to give any reason for withdrawing.

I consent to completing the measurements outlined in the Information Statement and attending two sessions at my nearest University of Newcastle campus (Callaghan or UDRH, Tamworth).

- Participating in a six week dietary intervention to reduce heart disease risk.
- Having my height, weight, waist circumference, body fat, blood pressure and arterial stiffness measured.
- Completing food frequency questionnaires, usual intake records, general health, physical activity and demographic questionnaires, some of which will be completed online and over the phone prior to the assessment sessions.
- Having a blood test to evaluate my health status at each assessment.

YES **NO**

I understand that my personal information will remain confidential to the researchers and that data collected from my participation will be used in journal publications and conference presentations. If I decide not to participate or withdraw from the study, it will not affect my relationship with the University of Newcastle. I have had the opportunity to have questions answered to my satisfaction. By signing below I am indicating my consent to participate in the research project.

Your name: _____

Signature:_____ **Date:** _____

Personal Details: *Birthdate:* _____

Phone (Home): _____ *(Mobile):*_____

*e-mail:*_____

*home address:*_____

Please return the completed consent in the prepaid envelope enclosed.

Appendix 28. Participant screening script

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

Version 2: 10th June, 2104
Telephone Eligibility Screen for C.H.E.Q. U.P. validation Study

Date of enquiry: _____

Name: _____

Postcode: _____

Sex: **Male** / **Female**

Please read the following script for the screening:

*Hi, this **insert name** speaking. Are you calling in relation to the C.H.E.Q. U.P. validation study?*

*May I ask your name? Are you interested in participating in the study? **YES / NO***

Ok, so I will first give a brief description of the study, and if you are still interested, I will go through some screening questions with you to check that the study is suitable for you. If we pass that stage, I will then send out further details for you to read over in your own time and decide if this is something that you want to be part of.

Let me give you a brief description of the study and what you would be expected to do. We are measuring the answers people give us about what they are eat and comparing this to markers of their cardiovascular health. We ask people to follow a heart healthy diet for 6 weeks and compare their before and after results to their answers about their diet.

The purpose of the study is to test a questionnaire we have designed that checks what people at increased risk of heart disease are actually eating.

You would be expected to follow the test diet for 6 weeks and not included in this is the time taken to fill forms and arrange consultations. The entire process is expected to take no longer than two months.

Participation in the study is entirely voluntary and if you do decide to participate, you may withdraw from the study at any time without giving a reason.

Next are a series of questions that will determine your suitability for the study. Included in this are a number of procedures that you must be willing to consent to.

PART A:			
Question:	**Response:**	**Inclusion criteria:**	**Action:**
May I ask your age?		18-75 years	
Can you speak and understand English?	**Yes / No**		
Are you able and willing to travel to the university for 2 assessments?	**Yes / No**	Yes	
Do you have any food intolerances or allergies such as coeliac disease, irritable bowel syndrome, Crohn's disease, reactions to nut or soy, or intolerances to specific types of fruit or vegetables?	**Yes / No** If yes, list here:	No coeliac disease, soy or nut allergies, IBS or Crohns disease, or intolerances that may impact on intake of recommended foods	
Do you have access to internet and email?	**Yes / No**	Yes	
When was the last time you remember you had a blood sample taken for cholesterol?	**< 6 months** **> 6 months**	<6 months	

If >6 months:

Unfortunately we need people to have had a recent blood cholesterol sample taken. Would you be able to go to your general practitioner to arrange to have a blood cholesterol sample taken?

Yes / No

If yes:

That's great. I can send out an information package that contains a letter for you to take to your doctor that explains what the study is about and the routine blood samples that we require. You are also able to ask for a copy of your blood results to be sent to you. The information package will also include a self-addressed envelope to post those results to check if you are eligible to join our study.

If no:

Ineligible.

PART A:			
Question:	**Response:**	**Inclusion criteria:**	**Action:**
If <6 months:			
Do you remember what any of your values for cholesterol were?	**Yes / No / Unsure** List values here if known:	LDL≥4.00mmol/L OR total/HDL ≥5.00 OR total chol.≥5.00	
Are you currently on medication to control your blood cholesterol?	**Yes / No**	No	
If yes: You are ineligible to join as we require that people are not currently on medication for blood cholesterol. If you are considering stopping your medication, this is something that needs to be discussed with your General Practitioner. I can provide you with an information statement and letter to take to your doctor that explains the purpose of the study and the dietary information that we will provide. If your doctor consents to you joining the study, you will need to provide us with a signed consent form from your General Practitioner and have not taken your medication for 6 weeks prior to having your first blood sample taken. Would you like me to post out to you information to take to your General Practitioner? **Yes / No**			
We require people who are interested in joining our study to provide a copy of their most recent blood tests. Do you have a copy of your latest results or would you be able to contact your GP to send us a copy of your latest results? We will provide you with the information and forms required.	**Yes / No**	Yes	
Are you on medication for thyroid conditions?	**Yes / No**	Ineligible	
Do you have any pre-existing conditions or taking any other medications:	**Yes / No**		
List:			

As previously outlined, as part of the study you will be required to:

- Attend 2 assessments sessions, to be held at the University of Newcastle, which for you will be the xxxxxxx campus. You will have your height, weight, waist circumference, body composition (fat free mass/fat mass), blood pressure and arterial stiffness measured.
- We will require two blood tests from you that will measure blood fats (cholesterol and triglyceride levels), as well as insulin resistance (insulin and glucose) and other risk factors (for example, inflammation markers).
- You will need to complete questionnaires about your medical history, general health and demographics, dietary intake and physical activity.
- You will need to participate in 4 telephone interviews where we ask you to recall the foods you ate the previous day.

Consent:	Yes / No

INELIGIBLE SCRIPT
If the participant is not eligible for inclusion:
Thank you for your interest in the study. Would you like us to forward you the website address of the Heart Foundation?

ELIGIBLE SCRIPT
If the participant is eligible for inclusion:

Thank you for your interest in the study. I will send an information pack out to you in the mail. Please read the information supplied carefully. If you wish to participate in the study, please sign the consent form and return it to us in the reply paid envelope provided. If you have any questions, you will find a contact number and email address on the forms.

Once we receive your signed consent forms, we will email you individual log on details for questionnaires to be filled out online. Once these are completed, we will contact you to complete the next stage of the study.

Collect contact details:

Full Name: _____

Date of Birth:

Address: _____

_____

Suburb:_____ **Postcode:** _____

Phone Contact: Day _____ Evening _____

Mobile: _____

E-mail: _____ (confirm email address)

Name of General Practitioner:_____

Information to post out:

	Information:	Date posted:
☐	GP permission to stop medications letter + information statement	
☐	Letter to obtain copy of latest blood samples	
☐	Information statement and consent forms	

Appendix 29. Sample pages of individual feedback

CHEQ UP

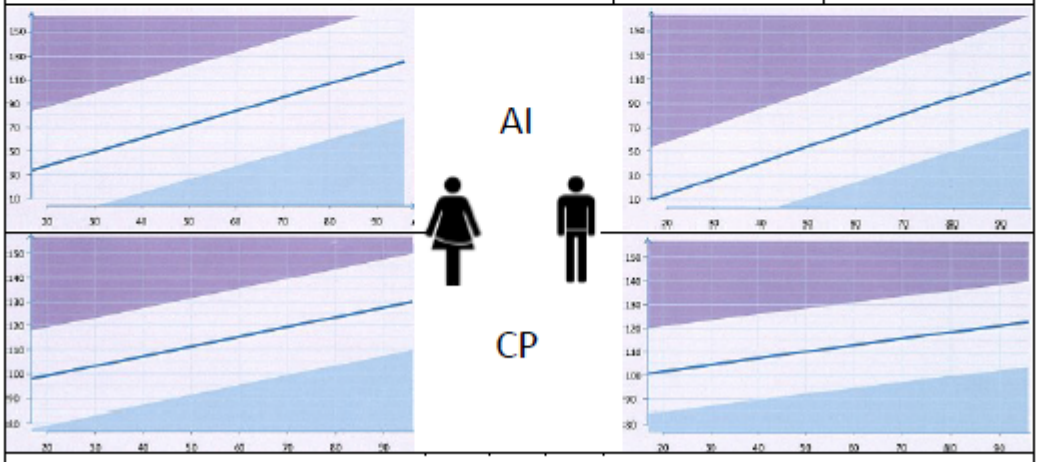
Cardiovascular Health Eating Questionnaire Using Propensities

Summary Report

HMRI Hunter Medical Research Institute

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

PRIORITY RESEARCH CENTRE FOR PHYSICAL ACTIVITY AND NUTRITION

Heart rate & blood pressure results	Baseline	Follow up
Heart rate (beats per minute):		
A healthy heart rate for an adult is between 60-100 beats per minute, although under 70 is ideal.		
Blood pressure (mmHg): (Systolic/diastolic) Your blood pressure category is… This blood pressure range is… An ideal blood pressure is to have a reading less than 120/80.		
Arterial Stiffness Measures	**Baseline**	**Follow up**
Central blood pressure (mmHg): (Systolic/diastolic)		
Augmentation index (AI)		
		
The augmentation index is a measure of arterial stiffness. Higher central blood pressures and stiffer arteries will result in higher AI values.		

Jodie's Dietary Intake

The table below shows your average daily intake and the Estimated Average Requirement **(EAR)**. The EAR is a daily nutrient level estimated to meet the daily requirements of half the healthy individuals in a particular life stage and gender group. Your intake is based on the answers given on food frequency questionnaire, completed as part of this study, about your usual food habits over the past 6 months.

Other references values for your nutrient intake may be an Acceptable Macronutrient Distribution Range **(AMDR)** or Adequate Intake **(AI)**. The **AMDR** is matched to an average persons need for protein, carbohydrates and fats. **AI** is the daily amount consumed that is associated with good health.

Nutrient	EAR, AMDR or AI	Baseline	Follow up
Energy	-	6924kJ	
% energy from core foods	-	61%	
% energy from non-core foods	-	39%	
Protein	-	83g	
% energy from Protein	AMDR: 15-25%	21%	
Carbohydrate	-	157g	
% energy from Carbohydrate	AMDR: 45-65%	39%	
Total Fat	-	65g	
% energy from Fat	AMDR: 20-35%	37%	

Appendix 30. Sample pages of education booklet

CHEQ UP

Cardiovascular Health Eating Questionnaire Using Propensities

Are you eating foods that keep your heart healthy?

Give your diet a C.H.E.Q. U.P.!

A guide to choosing and preparing foods that are scientifically proven to improve heart health

What effect does this diet have?

What you eat can affect your risk of having heart disease or a stroke. The C.H.E.Q. U.P. diet combines foods known to reduce the risk of heart disease or a stroke. High levels of LDL cholesterol in the blood is one factor known to contribute to heart and stroke risk. Recent research has shown that strict use of diets similar to this may reduce LDL cholesterol levels by as much as 30%. This is similar to some medications.

How does it work?

There are a number of ways to improve heart health through changing the foods you usually eat. Some foods with specific properties have been shown to be beneficial to those who have experienced heart disease or a stroke. These foods have been put together into a single eating plan: the C.H.E.Q. U.P. diet. They work together to improve health by changing the way cholesterol is absorbed and transported in the body. Extra information on this can be found further on in the booklet.

- Sterols and stanols help to stop cholesterol being absorbed from within the digestive tract. They are similar in shape and size to cholesterol that our body makes and so they can block absorption of this pre-made cholesterol in the gut.
- Fibre can be divided into two types: soluble and insoluble. Soluble fibre can bind to cholesterol and help eliminate it from the body. Insoluble fibre passes through the digestive tract unchanged. When it reaches the large intestine, the colon, it can absorb water and is also broken down by the healthy bacteria that live in the gut. The C.H.E.Q. U.P. diet is high in soluble and insoluble fibre.
- Soy proteins reduce the amount of LDL cholesterol the liver makes, although the way this happens is not fully understood. The liver is responsible for making most of the LDL cholesterol that is found in your blood and only a small part of it comes pre-made from the food we eat. Soy foods also contain a mix of compounds that help to reduce cholesterol absorption.
- There are many types of fats, but large amounts of one type in particular, saturated fat, can lead to high levels of LDL cholesterol. Reducing the amount of saturated fat in the diet, and replacing it with healthier unsaturated fats leads to lower LDL levels and higher HDL cholesterol levels.
- Nuts such as almonds, walnuts, hazelnuts, pecans and pistachios contain a mix of soluble fibres, sterols and healthy unsaturated fats, all which work together to reduce LDL cholesterol.
- Omega-3 oils are found in cold water fish such as salmon, mackerel and sardines. They can also be found in nuts and seeds such as linseed and walnuts. These oils have protective benefits associated with heart disease and stroke.

The following sections of the booklet looks at each of the six key features of the diet, explaining why they are important and how to mix them with the foods you are already eating. There is also general information given on healthy eating and ideas for daily menus and recipes that use many of the foods from the key features.

VEGETABLE PROTEIN

This group of foods are low in kilojoules and high in nutrients. The contain protein and carbohydrate, and are good sources of soluble fibre. These foods are also naturally very low in saturated fats.




Serves: Up to 7 per week
½ cup cooked kidney beans or baked beans
½ cup lentils or chickpeas

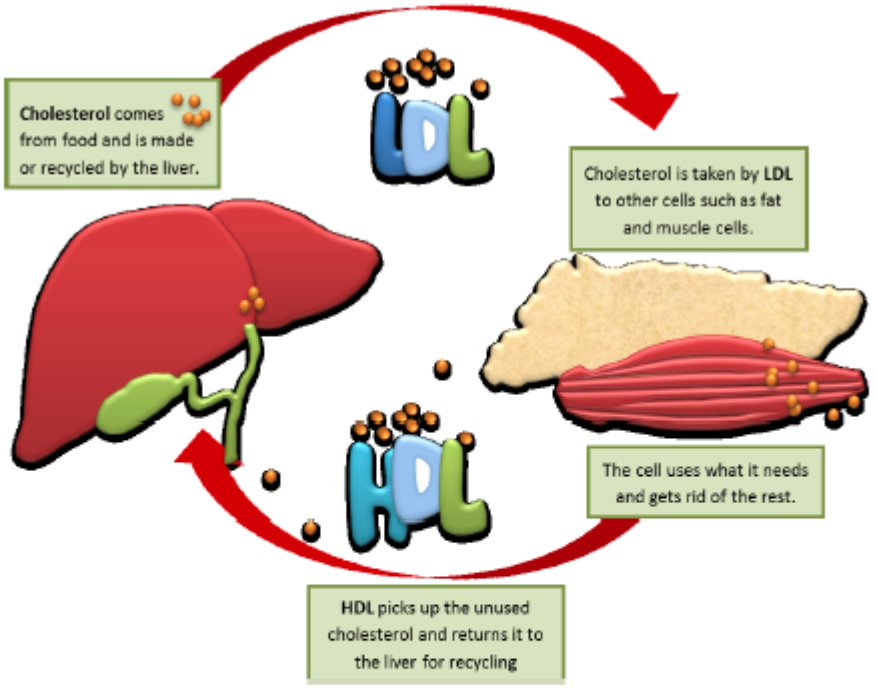
Rather than making these foods the focus of a meal or snack, they can be added to other foods to enhance flavour and texture. Canned beans, lentils and legumes are a convenient alternative to the fresh or dried varieties. Those with No Added Salt are the best to choose, although they may not always be available. However, the salt of any canned bean / lentil / legume can be easily reduced by draining the beans of the liquid in the can and rinsing them with fresh water. This method can reduce almost half of the salt in the product. Adding beans and legumes into dishes that contain meat is an excellent way of extending a meal and decreasing costs. They are also helpful in reducing the number of kilojoules or calories found in the food.

Vegetable protein may be used in the following ways:

- Baked beans can be used on wholegrain toast as a warm and filling winter breakfast
- Chickpeas and four bean mix can be used to give summer salads added texture or added into casseroles and soups (see recipe section)
- Lentils are easily used to extend any favourite mince dish, such as bolognaise or lasagne
- Kidney beans can be used in tacos, enchiladas and burritos or as a tasty addition to toasted sandwiches
- Beans can be puréed and eaten in dips or blended into rissoles and vegie patties
- Chickpeas can be roasted and used as snacks or mixed with herbs and spices and cooked with vegetables.

Cholesterol in the Body

1. Most cholesterol is made by the body with only a small amount in our body coming from food.
2. Cholesterol from food is absorbed in the intestine and taken to the liver.
3. The liver is responsible for many processes. One role it plays is the joining of cholesterol to a carrier that allows it to move throughout the body. These carriers are called Lipoproteins. When the liver sends cholesterol to the rest of the body, it joins it to a carrier called Low Density Lipoprotein (LDL cholesterol). This allows it to travel out of the liver to other cells, such as muscle and fat.
4. Cells away from the liver take in the LDL and use the cholesterol. Cholesterol that is not needed leaves the cell, where it is picked up by a different carrier, this time called High Density Lipoprotein or HDL cholesterol. HDL takes the cholesterol back to the liver for more recycling.
5. When there is too much LDL cholesterol traveling through the blood, distributing the cholesterol to parts of the body, it can sometimes leave cholesterol in places where it is not necessarily needed, for instance, in blood vessel walls. If there are plenty of HDL carriers around, they can pick up this cholesterol and take it back to the liver. However, if there is too few of them, the cholesterol is left behind.



Appendix 31. Sample breakfast menu

Breakfast options:

Water	Tea	Coffee
[ ] Tap	[ ] English Breakfast	[ ] Mocha Kenyan
[ ] Chilled	[ ] Earl Grey	[ ] Espresso
	[ ] Lady Grey	[ ] Skim cappuccino
	[ ] Orange Pekoe	[ ] Decaf cappuccino
	[ ] Prince of Wales	
	[ ] Traditional Afternoon	

Milk:	Cereal:
[ ] Skim	[ ] Wholegrain Weetbix
[ ] Soy	[ ] Uncle Tobys Plus omega 3
	[ ] Uncle Tobys Oats with Oat and Linseed fibre

Toast and toppings:	
[ ] Wholemeal wholegrain Toast	[ ] Tomato and onion Tuna
[ ] Pumpkin and sunflower seed Toast	[ ] Smoked Tuna
[ ] Proactive light Margarine	[ ] Lemon pepper Tuna
[ ] Proactive Cream Cheese	[ ] Baked Beans
[ ] Proactive cheese slices	

Appendix 32. Statement of contribution and collaboration for Chapter 9

I attest that Research Higher Degree candidate Tracy Leigh Schumacher contributed to the following paper:

Schumacher T, Burrows T, Rollo M, Wood L, Callister R, Collins C. Comparison of fatty acid intakes assessed by a cardiovascular-specific food frequency questionnaire with red blood cell membrane fatty acids in hyperlipidaemic Australian adults: A validation study. Submitted to *European Journal of Clinical Nutrition*.

Tracy L Schumacher was primarily responsible for data collection, programming and calculations for nutrient analysis, data analysis, initial manuscript preparation and contributed to study design. Dr Lisa Wood was responsible for fatty acid analysis and manuscript drafting. Dr Tracy Burrows, Dr Megan Rollo, Professor Robin Callister and Professor Clare Collins contributed to the study design, nutrient and data analysis and manuscript preparation. All authors approved the final manuscript.

Ms Tracy Schumacher (25th November, 2015)

Dr Tracy Burrows (25th November, 2015)

Dr Megan Rollo (7th December, 2015)

Dr Lisa Wood (24th November, 2015)

Professor Robin Callister (23rd November, 2015)

Professor Clare Collins (20th November, 2015)

Professor Robert Callister (7th December, 2015)

(Assistant Dean Research Training)

Appendix 33. Supplementary table for Chapter 9

Supplementary Table 1

Questions from the CVD-AES affecting AES questions.

CVD-AES supplementary questions	AES questions impacted on:
What type of cheese do you usually eat?	DF8 and DF9 (Cheese and cottage cheese)
What type of chicken do you usually eat?	M9, M10 (Chicken with/out vegetables)
What type of meat do you usually eat?	M2, M3, M4, M5, M13, M14 and O15 (Beef, lamb and pork questions with/out vegetables)
CVD-AES substitution question	**AES question replaced**
SFBR5 rolled oats	B2 cooked porridge
N2-N9 Questions relating to common nut types	O9 Nuts
SF3-8 Questions relating to fresh, frozen and canned seafood, including crustaceans	M16-M19 (Fish, fresh, crumbed, battered and canned)
SP1-11 Questions relating to sources of soy proteins	F22 Soybeans and tofu
VP7 Baked beans (salt versions applied)	F23 Baked beans
VP1-6 Relating to sources of vegetable proteins	F24 Other beans, lentils

Appendix 34. Sample data relating CVD-AES database construction

Sources of nutrient data and portion sizes applied to the vegetable protein (VP) within the CHEQ UP food frequency questionnaire

Food	Adult portion size (in weight)	Food entered from AUSNUT 2013 (% weighting)	Source of data or rationale
VP1 Lentils	87g	• 34% (SOURCE REMOVED) Lentils, canned • 33% Lentil, green or brown, cooked • 33% Lentil, red, cooked	Equal weighting of products Portion size: 87g. Obtained from average of 3 canned measures from 2 people (TS and JP), total 6 measures (1/2 C as per FFQ question). NO canned option for lentils available in AUSNUT 2013. To overcome this, confidential analytical results were obtained from (SOURCE REMOVED). Values were overwritten onto **Lentil, green or brown, cooked**. Starch values were not available, so it was overwritten to the same proportion of starch / CHO in the **Lentil, green or brown, cooked.** No vitamins or minerals were available except for K and Na. The values used were those WITHOUT the liquid component (IE drained)
VP1 NAS	**Serve size=87g**	• 50% (SOURCE REMOVED) Lentils No Added Salt, canned • 50% Lentil, green or brown, cooked	A mix of dried and the OVERWRITTEN (REMOVED SOURCE) canned version are used here as there is no NAS canned lentils available in either 2007 or 2013 AUSNUT. The NAS values were based on the (SOURCE REMOVED) canned version in VP1 and overwritten with SODIUM values only from the (SOURCE REMOVED) Lentils No Added Salt as the back of pack includes water in the per 100g calculation (as evidenced by the energy value per 100g of 378kJ). Equal weighting Portion size: 87g. Obtained from average of 3 canned measures from 2 people (TS and JP), total 6 measures (1/2 C as per FFQ question).

Sources of nutrient data and portion sizes applied to the vegetable protein (VP) within the CHEQ UP food frequency questionnaire

Food	Adult portion size (in weight)	Food entered from AUSNUT 2013 (% weighting)	Source of data or rationale
VP2. Kidney beans	½ Cup (listed on FFQ) **Combined serve size=87g**	• 50% Bean, red, kidney, canned, drained • 50% Bean, red kidney, dried, boiled, microwaved or steamed, drained	Both options available in AUSNUT 2013 No kidney beans available for portion size measure. Same density as lentils as given in AUSNUT 2011-13 NNPAS Food Measures Database. Therefore, same portion size as lentils applied. Equal weighting of options applied. No kidney beans available for portion size measure. Same density as lentils as given in AUSNUT 2011-13 NNPAS Food Measures Database. Therefore, same portion size as lentils applied.
NAS	½ Cup (listed on FFQ) **Serve size=87g**	• 50% (SOURCE REMOVED) Bean, red, kidney, canned, drained No Added Salt • 50% Bean, red kidney, dried, boiled, microwaved or steamed, drained	No NAS canned kidney bean available in AUSNUT 2013, and confidential information was supplied by (SOURCE REMOVED). Canned kidney beans were overwritten by analysis supplied. Where two test results were supplied, an average of values obtained was entered. The same proportion of starch to CHO was entered as no starch value was given. Sodium given as <6mg/100g was entered as 5mg/100g. No other vitamins or minerals given, so underwritten with **Bean, red, kidney, canned, drained** Dried kidney beans reconstituted make up 50% of this value to match that above. No kidney beans available for portion size measure. Same density as lentils as given in AUSNUT 2011-13 NNPAS Food Measures Database. Therefore, same portion size as lentils applied. Equal weighting of options applied.