The benefits of resistance training on blood lipid profile and body composition in Māori men

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

Master of Science

In

Exercise and Sport Science

Massey University, Manawatū, New Zealand

Karl William Coley

2014

Abstract

Objectives: The primary objective of this study was to determine whether 12 weeks of resistance training at time periods of three, 30 minute sessions per week would provide enough stimuli to reduce the cardiovascular disease (CVD) risk of blood lipid profile and body composition in sedentary Māori (Indigenous New Zealanders) men.

Methods: The study cohort consisted of a convenience sample of 16 Māori males aged 28 – 60y. Participants completed a resistance training intervention consisting of three 30 minute sessions per week for 12 weeks. Measures of pre- and post-BMI, waist to hip ratio (WHR), body composition and fasting lipids were made. Pre-, mid-, and post-intervention assessments of strength, aerobic fitness, body composition and blood composition were also undertaken. Exercise was controlled five days prior to the testing; whilst diet was restricted ~12 hours prior to blood tests.

Results: Percentage body fat was significantly lower after the 12 week resistance training intervention ($P<0.001$) and lean body mass (LBM) was significantly higher ($P<0.015$). A reduction in low density lipoprotein cholesterol (LDL-c) occurred ($P<0.039$), though a high density lipoprotein cholesterol (HDL-c) ($P <0.8$), body mass index (BMI) ($P<0.469$), and waist to hip ratio (WHR) ($P <0.196$) were not significantly different after completion of the intervention.

Conclusions: This was the first study to investigate the effect of half hour resistance training bouts, three times per week on male Māori as a modality to alter their CVD risk profile. These findings support the hypothesis that resistance training can improve CVD risk profile through a change in body composition; namely a reduction in percentage body fat, increase in LBM, and a reduction in LDL-c. Although in this cohort this intervention has proved effective, further studies of larger populations are required to get a stronger level of significance.

Acknowledgements

I would like to thank the following people for their help and guidance throughout the process of completing this thesis.

First and foremost, Mum, without your love, support and encouragement I would have not known my limits; you have always pushed me to go further and achieve more in life and for that I will be forever grateful and cannot express how much your words of wisdom mean to me.

Dr Isaac Warbrick, my supervisor, for your continued belief, guidance, knowledge and understanding as I bombarded you with questions and ideas about this project. You have been a constant source of knowledge and inspiration throughout this project to me.

My co-supervisors, Dr Geoff Kira and Dr Steve Stannard, for their advice and assistance throughout this project.

Acknowledgements are due to Professor Hugh Morton who helped process and organise my statistical analysis, and to Mr Bevan Erueti who taught me to understand some of the more in-depth Māori aspects of the study I conducted.

To the participants (B.R.Os), for all the laughs, good times, and hard work you all put in; without you guys this research could not have taken place.

To Jo, my partner for putting up with my hours of study, and being there for me throughout this process; and for your unwavering support and encouragement when things were overwhelming and I wanted to throw in the towel.

Special thanks are due to Dr's Matt Barnes, Darryl Cochrane, and Toby Mündel, all of whom have taught and inspired me during my time here at Massey. You all have played a massively influential role in my education and for that I am extremely grateful.

Table of Contents

List of figures

List of tables

Explanation of Māori terms

Hauora – Wellness, health.

Hinengaro – Mind, thoughts; in context of Māori health, hinengaro represents mental health and wellbeing.

Hui – Meeting.

Kai – Food.

Karakia – Prayer.

Kaumātua – Respected elders in the Māori community that have been involved with their whānau for many years.

Kaupapa Māori Research – Research methodology based on Māori ideology, values.

Mana – Authority, influence prestige, honour.

Marae – Meeting place.

Matauranga – Knowledge, comprehension or understanding of everything visible and invisible.

Mauri – Life force/spirit.

Mauriora – Access to te ao Māori (As expressed in Te Pae Mahutonga).

Patu – Traditional Māori club weapon.

Taiaha – Traditional Māori staff-like weapon used for striking and thrusting in combat.

Te Oranga – Participation in society (As expressed in Te Pae Mahutonga).

Te Pae Mahutonga – Māori model of health designed by Mason Durie, which underpins the Te Whare Tapa Whā.

Te Wheke – A Māori model of health also known as the Octopus model.

Tinana – Physical body; in context of Māori health, tinana represents physical wellbeing.

Toiora – Healthy lifestyles (As expressed in Te Pae Mahutonga).

Waiora – Environmental protection (As expressed in Te Pae Mahutonga).

Wairua – Spirit, soul, represents spiritual wellbeing in the context of Māori health.

Whakatau – Welcome used to begin a hui.

Whānau – Extended family, family group.

Whanaungatanga – Kinship, family connection. Relationship through shared experience.

Wharenui – Meeting house.

Whare Tapa Whā – A Māori model of health with four facets of health.

Abbreviations

1RM – 1 Repetition Maximum

ACSM - American College of Sports Medicine

AHA – American Heart Association

ANOVA – Analysis of Variance

BMD – Bone Mineral Density

BMI – Body Mass Index

BMR – Basal Metabolic Rate

CAD – Coronary Artery Disease

CHD – Coronary Heart Disease

CI – Confidence Interval

CO_2 – Carbon Dioxide

CVD – Cardiovascular Disease

DVT – Deep Vein Thrombosis

DXA – Dual-Energy X-Ray Absorptiometry

FFA – Free Fatty Acids

FM – Fat Mass

FT – Fasting Triglycerides

HDL – High Density Lipoprotein

HDL-c - High Density Lipoprotein Cholesterol

HPL – Human Performance Laboratory

HR – Heart Rate

IFNHH – Institute of Food Nutrition and Human Health

IHD – Ischemic Heart Disease

L - Litres

LBM – Lean Body Mass

LDL – Low Density Lipoprotein

LDL–c - Low Density Lipoprotein Cholesterol

MI - Myocardial Infarction

MLC – Med Lab Central

MPH – Miles per Hour

n – Number of Participants

O_2 – Oxygen

PARQ – Physical Activity Readiness Questionnaire

PASW – Predictive Analytics SoftWare (formerly SPSS)

QDR – Quantitative Digital Radiography

RER – Respiratory Exchange Ratio

SD – Standard Deviation

SE – Standard Error

SNZ – Sport New Zealand

SPARC – Sport and Recreation New Zealand

SRI – Sport and Rugby Institute

SST – Serum Separator Tubes

STPD – Standard Temperature Pressure Dry

TC - Total Cholesterol

VCO_2 – Carbon Dioxide Production

VO_2 – Oxygen Uptake

VO_2 max – Maximal aerobic capacity

WHO – World Health organisation

WHR – Waist to Hip Ratio

Y - Years

1. Introduction

The connection between physical activity and health was documented as early as the fifth century BC by Greek Physician Hippocrates: "All parts of the body, if used in moderation and exercised in labours to which each is accustomed, become thereby healthy and well developed and age slowly; but if they are unused and left idle, they become liable to disease, defective in growth and age quickly" (Kokkinos & Myers, 2010). As the ancient Greek civilisation declined, their values of physical activity were lost. For many centuries up to and including post World War II, physical fitness was considered purely for youth sports and military purposes (Kokkinos & Myers, 2010). Māori held similar views to that of the ancient Greeks with a high regard for physical fitness and wellbeing. As described by anthropologist Elsdon Best, Māori took part in many games and activities that required physical strength and fitness. Starting as children and continuing on through into adulthood, Māori practised many games that were viewed as valuable training for military exercises; these include boxing, wrestling, running and armed combat with weapons such as taiaha and patu (Best, 2005). Studies on cardiovascular diseases (CVDs) such as coronary heart disease (CHD), conducted by Morris and Crawford (1958) have helped change the perspective of physical activity, fitness and wellbeing. Their pilot study has since spurred a new era in association between exercise and health, and for over a half century an abundance of scientific evidence has been collected. These studies show a strong, and inverse relationship between physical activity levels, and the incidence and risk of cardiovascular disease (Myers *et al.*, 2002; Kokkinos & Myers, 2010).

Cardiovascular disease (CVD) is one of the leading causes of mortality worldwide, accounting for more than 16 million deaths in 2001 (Lopez *et al.*, 2006). During the period from 1996-1999, cardiovascular mortality for male Māori (343.8 per 1000 deaths) was three times higher than non-Māori (113.2 per 1000 deaths) (Ajwani *et al.*, 2003; Bramley *et al.*, 2004). The latest mortality statistics show that CVD accounted for 45% of female and 43% of male deaths in New Zealand in 2008 (New Zealand Ministry of Health, 2008b). This proportion increases in Māori, with 66% of Māori not meeting blood pressure or lipid management recommendations (Peiris *et al.*, 2008).

Since the 1980's there has been a drop in deaths contributed to CVD in both a Māori and non-Māori population, however there is still large scale inequality between the two groups (Tobias *et al.*, 2009). This is highlighted by the fact that Māori have a greater proportion of the population experiencing CVD (Tobias *et al.*, 2009).

Cardiovascular disease is not a single illness but a term which describes conditions affecting the heart and blood vessels. These include, coronary heart disease (CHD), stroke, rheumatic heart disease, cardiomyopathy and other diseases such as angina, arrhythmias, congenital heart disease, deep vein thrombosis (DVT), hypertensive heart disease, peripheral artery disease, pulmonary embolism and valvular disease (David *et al.*, 2004; Lopez *et al.*, 2006; Labarthe, 2011). The most common CVDs affecting Māori men are cerebrovascular disease (stroke), ischemic heart disease due to coronary heart disease, and hypertensive disease (New Zealand Ministry of Health, 2012b). Cerebrovascular disease accounted for 2488 deaths in 2009; 937 of these were males. Māori men had a higher age standardised death rate of 36.8 per 100,000; 30.4% higher than non-Māori men whose age standardised death rate was calculated to be 28.2 per 100,000. Ischemic heart disease however accounted for 5553 deaths in 2009, second only to caner. Males counted for 54.7% (3309) of these deaths. Again Māori men had a higher age standardised death rate of 166 per 100,000; 83.3% higher than non-Māori men whose age standardised death rate was calculated to be 90.5 per 100,000.

The aetiology of cerebrovascular disease, ischemic heart disease (IHD), and hypertensive disease can be linked directly to lifestyle factors of CVD risk such as a sedentary lifestyle (physical inactivity), obesity, and smoking (Danaei *et al.*, 2009).

There are a total of twelve well accepted CVD risk factors, three non-modifiable and nine modifiable. The three non-modifiable risk factors are; age, gender and family history (genetic pre-disposition) of early heart disease or stroke (Waring, 2007). However a report published by Ajwani et al. (2003) highlights that ethnicity can play a role in the development of CVD. This is confirmed in other studies; which highlight the prevalence of Māori and their increased risk of CVD development (Chan *et al.*, 2008; Sundborn *et al.*, 2008). New Zealand Māori have the highest age adjusted prevalence of CVD with 7.1%

compared to that of non-Māori 4.45% (Chan *et al.*, 2008). CVD becomes increasingly common with advancing age. As a person gets older, the heart undergoes subtle physiologic changes, even in the absence of disease. The risk of cerebrovascular disease doubles every decade after the age of 55 (Mackay & Mensah, 2004). Gender plays a role in CVD development, men being at greater risk of heart disease than pre-menopausal women; however once past menopause, a females level of risk is similar to males (Mackay & Mensah, 2004). It should be noted that there is contrasting evidence about the gender distribution of CVD. Evidence states that CVD affects as many females as it does males; however due to the later onset of CVDs, women lose less years of life in comparison to men (Mackay & Mensah, 2004). An individual whose family displays a history of CVD is at increased risk of developing CVD. If a first-degree blood relative has had coronary heart disease or stroke before the age of 55 years for a male relative or 65 years for a female relative, the risk increases 2 fold (Kate *et al.*, 1982; Hunt *et al.*, 1986; Mackay & Mensah, 2004). Three of the nine modifiable risk factors are considered biological risk factors; including diabetes, hypertension and hypercholesterolemia. The final six listed risk factors which are considered lifestyle factors include, excessive alcohol intake, poor diet, obesity, physical inactivity, psychosocial factors and smoking (Waring & Cockcroft, 2006; Thayer *et al.*, 2010).

New Zealand physical activity guidelines state that;

1) Adults should view movement as an activity not a chore.
2) Be active in as many ways as possible.
3) Do 30 minutes or more of moderate intensity on most if not all days of the week.
4) If possible adding vigorous activity for extra health benefits and fitness.

(SPARC, 2005).

The effect of aerobic exercise has long been associated with a reduction in CVD risk factors (Siscovick *et al.*, 1985). Many studies have outlined the advantages of aerobic training in those with CVDs. Benefits to aerobic exercise training include but are not limited to the following; decrease in blood pressure, reduction in low density lipoprotein cholesterol (LDL-c) and percentage body fat, and a decrease in resting heart rate (Pollock

et al., 2000; Braith & Stewart, 2006). However fewer studies highlight the interaction between resistance training and CVD risk factors.

Traditionally resistance training has been acknowledged as a means of developing muscular endurance, hypertrophy, power and strength; the beneficial relationship between resistance training and chronic health issues have only recently been documented (Pollock *et al.*, 2000). Prior to 1990 resistance training was not included as part of the recommended guidelines set out by either the American College of Sports Medicine (ACSM) or the American Heart Association (AHA) for exercise training and rehabilitation (Pollock *et al.*, 2000). Resistance training has been identified as important to overall health and wellbeing in numerous studies (Winett & Carpinelli, 2001; Baldi & Snowling, 2003; Faigenbaum & Myer, 2010). These studies outline benefits such as increases in total strength, fat free mass and bone mineral density (BMD); with reductions in fasting glucose and insulin levels, and a decrease in lipid profiles.

The majority of relevant studies have focused on using aerobic exercise to help reduce CVD risk factors and have shown that, completing three, 30 minute bouts of exercise per week, improves blood pressure, body weight and body composition (Agurs-Collins *et al.*, 1997; Nojima *et al.*, 2008). A review of studies by Marwick *et al.* (2009) show the duration of effective resistance training sessions for individuals at risk of developing CVD varies from 45 to 120 minutes, with a frequency of three to five times per week. The reviewed interventions took place over a period of between two to six months. The time frame for exercise sessions are crucial, considering that time commitment is reported to be a leading barrier to improving physical activity (Sullivan *et al.*, 2003).

It has been identified by Sport New Zealand (SNZ) (formerly Sport and Recreation New Zealand, also known as SPARC) that free time, social support and sustainability are critical barriers when it comes to New Zealand men including Māori men, being unable to commit to exercise (Sullivan *et al.*, 2003). These issues coupled with poor exercise education can lead to misconceptions about exercise, including the time commitment, and the type of exercise required to help improve their health status.

The primary point of difference within this study is the utilisation of resistance training in place of cardiovascular training with an exclusively male Māori group. The second point of difference within this study is the use of a Kaupapa Māori approach, incorporation of Māori views and values within the lab-based research setting.

The utilisation of the kaupapa Māori approach throughout this study is to ensure that the design and implementation of the exercise and testing was culturally relevant and appropriate for those involved. A Kaupapa Māori research approach is based on Māori ideology and values and the aim is to bring the participants together and feel part of the research process with the end goal of enhancing Māori well-being. Hauora, the term often used to represent a Māori view of health and wellbeing, encompasses a balance between the mental (hinengaro), physical (tinana), social (whānau), and spiritual (wairua) dimensions of health (Durie, 1985), and includes a strong sense of unity with their environment (Durie, 2004). Te Whare Tapa Whā Māori health model as developed by Mason Durie is not a rigid rule but one of many models that aid health professionals in understanding a Māori view of health and well-being (i.e. Te Wheke; developed by Rose Pere; and Te Pae Mahutonga again developed by Mason Durie). Although these other two health models are often used in conjunction with Te Whare Tapa Whā – Te Wheke, which has 8 "tentacles" representing different aspects of total well-being (Durie, 1999) with the suckers on each tentacle representing the multiple aspects within each of the dimensions, and Te Pae Mahutonga which aims to bring together all the elements of modern health promotion including Mauriora (access to te ao Māori), Waiora (environmental protection), Toiora (healthy lifestyles), and Te Oranga (participation in society) – Te Whare Tapa Whā alone provides the basis underpinning the definition of well-being in this particular Masters study.

The following text reviews the literature relating to resistance training and its effect on participants who are at risk of developing CVD. The subsequent chapters include; an explanation of the methods used during the research process, a discussion of results from this study will be undertaken which includes the three key elements of this study - body composition, biochemical markers, and training protocol. This will be concluded by closing remarks, considerations and limitations to the study design, with recommendations' and a conclusion relating to the results and validity of the project.

The primary aim of this thesis is to investigate whether participation in resistance exercise three times per week can provide enough stimuli to reduce CVD-associated risk factors of Māori men thereby improving overall health and wellbeing.

This thesis is only a small part of a much larger study examining the impact of different modes of exercise and the associated changes in Diabetes and CVD risk, and overall wellbeing. This only touches on two specific factors that relate to the onset of CVD, these include blood lipid profile, and obesity.

1.1 Hypothesis

The purpose of this study was to test the following hypothesis:

Māori men who perform 12 weeks of resistance training will have a significant reduction in the cardiovascular disease risk factors of cholesterol (blood lipids) and obesity; enhancing their overall health and wellbeing.

2. Literature review

"An overview of the major cardiovascular diseases as a public health challenge would be incomplete without recognising opportunities for prevention. It is the potential impact of effective public health action that makes the challenge more than an academic interest and a matter of urgent national and global health policy" (Labarthe, 2011). CVD is a major cause of death and disability throughout the world. The core pathology is arteriosclerosis, which develops over many years and often appears in middle age. By assessing those at risk and providing risk factor modification clinically significant cardiovascular events and premature death can be reduced worldwide (UNAIDS, 2007). Exercise prescription can help change the risk profile of those who are most susceptible to CVD.

2.1 What is cardiovascular disease

CVD is a term that incorporates all diseases of the heart and circulatory system (New Zealand Ministry of Health, 2008a). Including coronary heart disease (CHD), ischemic heart disease (ISD), coronary artery disease (CAD), stroke, rheumatic heart disease, cardiomyopathy, angina, arrhythmias, congenital heart, DVT, hypertensive heart disease, peripheral artery disease, pulmonary embolism and valvular disease (David *et al.*, 2004; Lopez *et al.*, 2006; Labarthe, 2011). CVD is the leading non-communicable disease (NCD); approximately half of the 36 million deaths due to NCDs are attributed to CVDs. The world health organisation estimates that there will be 20 million deaths from CVD by the year 2015 (30% total deaths) (Abegunde & Vita-Finzi, 2005).

CVDs have featured prominently throughout the 20th century in the shift of causation of death. During this time CVDs have become the leading cause of death in the world (MacKay *et al.*, 2004). The theory of "epidemiologic transition" offers an interpretation of these shifts. Epidemiologic transition is defined as transition from acute infectious, parasitic and nutritional deficiency diseases, to predominantly non-communicable, chronic diseases as the primary cause of morbidity and mortality (Omran, 1971).

CVDs are responsible for over 17.3 million (approximately 29%) deaths per year and are the leading causes of death worldwide (Harris & Harris, 2011; Mendis *et al.*, 2011). CVD is the leading cause of premature death and disability in New Zealand (Peiris *et al.*, 2008); however the risk could be reduced by up to 80% by targeting the modifiable risk factors of hypercholesterolemia, hypertension, obesity, physical inactivity and smoking (Wald & Law, 2003; Kerr *et al.*, 2008). CVD remains the primary reason for the widening gap in life expectancy between Māori and non-Māori (Ajwani *et al.*, 2003). Age adjusted prevalence within New Zealand show that CVD affects 4.6% of New Zealanders of European decent whereas it affects 7.4% of the Māori population (Chan *et al.*, 2008). Those who suffer a CVD event such as Myocardial infarction (MI) or stroke are at high risk of suffering further events (Kerr *et al.*, 2008).

As CVD develops over a period of time it is evident that there remains no one procuring event or one cure to the disease that is multi-faceted. The underlying cause of 85% of all CVD and precursor to clinical expression of CVD is arteriosclerosis (Nieman, 2003).

There are many risk factors to the onset of CVD and can be categorised into modifiable and non - modifiable risk factors. These have also been labelled as behavioural and metabolic risk factors.

2.2 CVD risk factors

Risk factors come in two variants, modifiable and non-modifiable risk factors. Modifiable risk factors can be split into two sub- groups; biological risk factors, such as hypertension, hyperlipidaemia, and type-two diabetes; and lifestyle factors including poor diet, obesity, smoking, and physical inactivity (Thayer *et al.*, 2010). Non- modifiable risk factors include; age, family history, gender and ethnic background (Sacco, 2001; Thayer *et al.*, 2010).

As discussed further in this thesis certain CVD criteria are controlled for these include; diet, smoking and type II diabetes. Participants who were diabetic or who had not been smoke free for at least two years prior to the start of the study were excluded from participation. Diet was not altered in any way during this intervention, however

participants were encouraged to maintain the same dietary habits throughout the 12 week intervention and food diaries were kept to allow the researcher to monitor whether any 'major' dietary changes took place throughout the study. Therefore these risk factors will not be discussed within this literature review.

2.2.1 Non modifiable risk factors

Alongside modifiable risk factors there are also some which cannot be modified; age, gender, family history, and ethnicity (Sacco, 2001).

2.2.1.1 Age

Age represents a CVD risk factor primarily because of its association with hypertension, elevated blood lipid levels and glucose intolerance. From the age of 35 for men and 45 for women the risk of dying from CVD increase dramatically in comparison to those who are younger (McArdle *et al.*, 2010). It is noteworthy to mention that it is the leading cause of death amongst those aged 65 and over (Mittelmark *et al.*, 1993).

2.2.1.2 Gender

CVD was once thought to be a disease that affects only males, evidence has emerged that women show a twofold greater risk of death from CVD (CAD, stroke, hypertension) than men. It is now known that women have more lethal and severe first time myocardial infarctions and have a 70-100% greater risk of dying within months of their first myocardial infarction than men, particularly if they are younger than 50 years of age (Tunstall-Pedoe *et al.*, 1996; Marrugat *et al.*, 1998; Vaccarino *et al.*, 1999).

As stated previously gender plays a minor role in the level of risk of developing CVD. It is stated that males are more disposed to develop CVD at an earlier stage in life than females (UNAIDS, 2007). In men the highest incidence of CVD occurs between the ages of 40-60, whereas in women it occurs between the ages of 60-70 (Brannon, 1993). The key acknowledgement to this fact is that males lose more years of life as they develop CVDs earlier than their female counterparts (Mackay & Mensah, 2004).

2.2.1.3 Family history

Individuals with a first degree blood relative who has experienced coronary heart disease (CHD) or stroke before the age of 55 years for a male relative or 65 years for a female relative, the risk of developing CVD increases (Hunt *et al.*, 1986; Mackay & Mensah, 2004). This has been well documented in many studies looking at family history and possible links to early signs of CVD development (Rose, 1964; Rissanen & Nikkilä, 1979; Morrison *et al.*, 1980; Barrett-Connor & Khaw, 1984).

2.2.1.4 Ethnicity

CVD is the primary cause of early death and disability in New Zealand (Peiris *et al.*, 2008); it remains the number one reason for the widening gap in life expectancy between Māori and non-Māori (Ajwani *et al.*, 2003; Riddell *et al.*, 2007). Although ethnicity itself is not a biological or genetic category, there have been many documented disparities in relation to certain ethnic subpopulations, therefore creating a demand for CVD research on ethnic/racial minorities (Ferdinand & Armani, 2007). Disparities in CVD levels are found in individuals from differing socioeconomic backgrounds, including those who have lower levels of education, income, and occupation (Winkleby *et al.*, 1992). Ethnicity based CVD research can offer opportunities to explain vital environmental aspects of CVD development and demonstrate the importance of lifestyle, identify variations in clinical practices and has the potential to improve health care delivery (Ferdinand & Armani, 2007). Studies completed by Ajwani *et al.* (2003) highlight a link between ethnic background and prevalence of CVD development in Māori; with articles published whose data highlights that certain ethnicities namely Māori and Pacific Islanders have higher incidence of CVD compared to those of European heritage. One such study that backs up the previous statement is done by Chan *et al.* (2008) who highlighted that Māori have a higher age adjusted prevalence of CVD with 7.1% compared to that of non-Māori (4.45%). Inequalities of health status, including socioeconomic are found between not only Māori and non-Māori, but many groups within populations worldwide. It was found that the relative burden of CVD falls more heavily on those of Māori and Pacific Island descent who live in the most deprived areas in New Zealand. Deprivation is determined with the use of the NZDep2001 Index of Depravation, produced by Salmond *et al.* (2007). Level of

deprivation is determined by variables including; income, employment, communication, transport, support qualifications and living space (Salmond *et al.*, 2007). Alongside this Māori were also 10 times more likely to experience racial discrimination (4·5% [95% CI 3·2—5·8] *vs.* 0·5% [0·3—0·7]), including from health professionals, leading to health loses and inequalities in health care (Harris *et al.*, 2006).

2.2.2 Modifiable risk factors

Modifiable risk factors of CVD include; hypertension (blood pressure 140/90), obesity (body composition), type 2 diabetes, physical inactivity, hyperlipidaemia and smoking (Cyr, 2003; Mendis *et al.*, 2011). A large percentage of CVDs are preventable through the reduction of these behavioural risk factors (World Health Organization, 2008, 2011b). Modifiable risk factors can be managed through lifestyle changes to improve health. The primary objective for health professionals is to reduce the five year CVD risk to below 15% (New Zealand Guidelines Group, 2012). The WHO recommends healthy behaviours such as physical activity and smoking cessation to combat the risk of developing CVD (World Health Organization, 2011a).

2.2.2.1 Physical inactivity (sedentary lifestyle)

Physical inactivity has been identified as the fourth leading risk factor in global mortality (6% globally), alongside this it is also one of the leading risk factors for the development of CVD (World Health Organization, 2010a).

Sedentary lifestyle is an important risk factor for premature mortality especially from those whom are overweight as it contributes to an obesogenic environment; despite current health promotion efforts (Jackson *et al.*, 1995; Pate *et al.*, 1995). Sedentary lifestyle is defined by the New Zealand ministry of Health as not engaging in any strenuous physical activity for longer than 30 minutes per week as described in the New Zealand health survey (New Zealand Ministry of Health, 2008b). Sedentary lifestyle in New Zealand has been combated in a public forum via the "Push Play" campaign (launched in 1999), set up by the New Zealand Ministry of Health in an effort to increase

awareness of physical activity and to get the population to start thinking of becoming more active (Bauman *et al.*, 2003).

2.2.2.2 Obesity

It is estimated by the world health organisation (World Health Organization, 2011a), that at least 2.8 million people die each year due to being overweight or obese (World Health Organization, 2010b).

Not only is obesity a risk factor for the development of CVD but there is a strongly associated link between being obese and an increase in the risk of coronary heart disease (CAD), hypertension dyslipidaemia, and a strong association with the development of type II diabetes (Messerli, 1982; Alpert & Hashimi, 1993; Golay & Felber, 1994; Bray, 1996; Ezzati *et al.*, 2002; Dandona *et al.*, 2004; World Health Organization, 2007; Finucane *et al.*, 2011). In 2008, 34% of adults over the age of 20 were overweight (BMI of 25-29.9). Obesity is a growing problem in both developed and developing countries (World Health Organization, 2011b). Epidemiological studies have shown a relationship between overweightness or obesity and cardiovascular morbidity, CVD mortality and total mortality (Mendis *et al.*, 2011). The main form of obesity that affects males is android obesity (Després *et al.*, 2001). Android obesity is where excess adipose tissue is more predominant around the trunk (Bjorntorp, 1987).

Body composition is considered a health related component of physical fitness, however it is unlike most other health related factors as it is not a performance based measure; such as muscular strength, and cardiovascular fitness. It is because of this reason body composition has been classified as a factor of metabolic fitness (Corbin *et al.*, 2010).

Body composition was determined using Dual energy X-ray Absorptiometry (DXA) analysis. DXA is considered the gold standard of body composition analysis (Kelly *et al.*, 2009; Vanlandewijck & Thompson, 2011). DXA uses two different x-ray energy levels to distinguish between bone, fat, and other soft tissue. The x-ray will be absorbed or reflected depending on the density of the material it passes through. Minute changes in Bone mineral density (BMD) can be detected due to the very high precision of the scanning equipment. However there is discussion about possible flaws with predictions

used with DXA analysis, however it is still regarded as a highly accurate measure of limb mass and is more available (Wells & Fewtrell, 2006) than other forms of body composition measure such as magnetic resonance imaging (MRI) or densitometry.

Since a loss in body mass is related to negative energy balance, aerobic training has a greater potential to produce results than resistance training does. However studies have reported positive effects from both methods of training such as improving insulin sensitivity, LBM, and reducing FM (Dunstan *et al.*, 2002; Cuff *et al.*, 2003).

Waist to hip girth ratio (WHR) is an indicator of relative fat distribution in adults and risk of disease. The distribution of body weight and body fat is recognised as an important predictor of health risks of obesity. Individuals with a greater circumference or more weight situated around the trunk are at heightened risk of CVD, than those who have a smaller trunk circumference with more weight located at their extremities (Dwyer *et al.*, 2008). A ratio higher than 0.95 for males and 0.86 for females is classed as obese, and reflects a larger proportion of abdominal fat with a greater risk of developing hyperinsulinemia, hypertension, type II diabetes and arteriosclerosis (Whaley *et al.*, 2006; Katch *et al.*, 2010) . Studies have shown that those with an elevated WHR can have a two-three fold increase (95% CI 1.53-1.84) in CVD risk and premature death (Rexrode *et al.*, 1998; Yusuf *et al.*, 2005; Pischon *et al.*, 2008).

Body mass index (BMI) is the most widely used indicator of health, it has application values in both the public health and clinical practices. BMI is used to identify individuals at risk of comorbidities (Taylor *et al.*, 2010). Epidemiological studies have identified a direct link between BMI and increased risk of medical complications (relative risk of 2.90, 95% CI 2.37-3.56) (Klein *et al.*, 2007). BMI is used to asses' normality of individuals' body mass and to determine health risk. BMI is calculated as weight (kg)/height squared (m^2) (Initiative, 1998; Katch *et al.*, 2010) The main assumption of BMI is that body mass adjusted for body height squared closely correlates to body fatness and resulting morbidity and mortality (Bray, 1996) However one major limitation to BMI is the fact that it does not differentiate between FM and LBM, as demonstrated by Rush *et al. (2010)* Garn *et al.* (1986) have theorised that BMI may be better at predicating fatness or LBM for different age groups.

Māori men have on average a higher BMI (29.8) than those of European (26.8) descent (New Zealand Ministry of Health, 2008b). This was stated previously by Rush *et al,* (2004) where she found Māori men average BMI of 30.4 compared to that of Europeans with 25.8. However as previously stated BMI does not differentiate between FM and LBM, this is highlighted in a study completed by Rush *et al.* (2010), which demonstrates that those of Māori descent have both a higher LBM and a higher bone mineral density than those of European descent. These findings have led to the New Zealand Ministry of Health changing BMI thresholds for Māori from 25, and 30/kg/m^2 to 26 and 32kg/m^2 to be classed as overweight or class one obesity respectively (Rush *et al.*, 2010). The previous study was a follow on from one completed by Rush *et al,* (2004) where it was found that Māori men had an average body fat mass of 22.6kg and an average fat free mass 65.9kg compared to Europeans with 8.2kg of fat mass and a FFM of 62.9kg.

However since the publication of the previous study further research has been completed and has recommended the adjustment of BMI for those of Māori descent be reversed (Taylor *et al.*, 2010).

2.2.2.3 Hypercholesterolemia

Cholesterol is a sterol molecule with vital biological importance. It is an indispensable component of cell membranes, and plays a central role in lipid digestion and absorption, affecting their stability and permeability. It is also a precursor of the bile acids that play vital roles in lipid digestion and absorption (Stipanuk & Caudill, 2012). The disposal and synthesis of cholesterol must be regulated to meet cellular cholesterol needs and to prevent excess accumulation. Excess accumulation of cholesterol and cholesterol rich lipoproteins in coronary arteries increases risk of arteriosclerotic formation which is a major contributing factor to CVD (Glass & Witztum, 2001; Von Eckardstein *et al.*, 2001).

High density lipoprotein cholesterol (HDL-c) are a blood-borne transport molecule that picks up cholesterol and assists in the removal from the body by transporting it to the liver which then breaks it down (Corbin *et al.*, 2010). It is noted that there is a negative correlation between HDL-c levels and the probability of developing CVD (Berg *et al.*, 1976; Myhre *et al.*, 1981).

A vast body of epidemiological evidence indicates a direct link between dyslipidaemia and the development of CVDs (Shepherd *et al.*, 1995; Graham *et al.*, 2007; Merkler & Reiner, 2007) such as; CAD, CHD, MI, stroke, and peripheral arterial disease. Trials testing this hypothesis started taking place in the 1970s and 1980s with the view that lowering LDL-c leads to decreased risk of CHD. These trials showed generally positive results, with a reduction in LDL-c by 5-8 mg/dl, and concluded that MI could be prevented by lipid lowering therapy (Mancini *et al.*, 2011).

Dyslipidaemia is the most heavily studied risk factor for CVD (Sacks *et al.*, 1996). Results of numerous large studies including the Framingham study and the prospective Cardiovascular Munster (PROCAM) both illustrate the importance of treatment of hyperlipoproteinemias in the prevention of CVD (Gordon *et al.*, 1977; Mancini *et al.*, 2011). Both studies have also identified low level HDL-c (less than 40mg/dl) (Ashen & Blumenthal, 2005) as an independent risk factor for CHD. This is backed up by data from Third National Health and Nutrition Examination Study (Ford *et al.*, 2002), and the West of Scotland Coronary Prevention Study (WOSCOPS) (Sattar *et al.*, 2003). Taken together the data of the aforementioned studies have led to recognition of HDL-c as a major risk factor for CHD in international treatment guidelines (National Institutes of Health, 2001; Chapman *et al.*, 2004).

Data from the Framingham study (Gordon *et al.*, 1977) have shown a positive relationship between low HDL-c and an increase in cardiovascular morbidity and mortality in men and women; this is irrespective of LDL-c levels (Lopez *et al.*, 2006).

It was reported as early as 1951 by Barr *et al.* that healthy men had higher levels of HDL-c than those who had CHD including angina pectoris and myocardial infarction (Castelli *et al.*, 1977). This is backed up in subsequent cross sectional studies (Nikkila, 1953; Jencks *et al.*, 1956). Evidence shows that low levels of HDL-c are an important risk factor for developing CVD (Chapman *et al.*, 2004). Moreover, the Framingham Offspring study found an association between the premature development of CHD and lipid abnormalities including low levels of HDL-c in 18.2% of male patients (Schaefer *et al.*, 1994). For every 1mg/dL decrease in HDL-c levels there is a 2-3% increase in risk of CHD development independent of other risk factors including high levels of LDL-c (Chapman *et*

al., 2004). As identified in the New Zealand Guidelines Group the optimal level of HDL-c is > 1.0mmol/L for those who are at risk of CVD development, which the cohort being studied are (New Zealand Guidelines Group, 2012).

LDL's are made up of a core of cholesterol surrounded in protein and are taken up by macrophages inside arterial walls thus initiating arteriosclerotic plaque development, narrowing blood vessels (McArdle *et al.*, 2010). It is known that lowering LDL-c concentration in plasma considerably reduces cardiovascular morbidity and mortality (Pedersen, 2004). An 11% reduction in total mortality was found over a period of 15 years (Pedersen, 2004). Lowering LDL-c is presently the prime focus in lipid lowering therapy for prevention of CHD (National Institutes of Health, 2001; De Backer *et al.*, 2003), it has been suggested that by lowering LDL-c it may help reduce development of vascular disease (Yusuf, 2002). However high levels of residual risk; 70% of statin-treated patients expected further coronary events in the subsequent 5 year period. This indicates there is reason for modification of other components of the atherogenic lipid profile (Chapman *et al.*, 2004). As identified in the New Zealand Guidelines Group the optimal level of LDL-c is < 2.0mmol/L for those who are at risk of CVD development, which the cohort being studied are (New Zealand Guidelines Group, 2012).

2.3 Exercise training adaptation

Adaptations to exercise training are numerous and well documented; these include but are not limited to increase in muscular strength, increase muscle size (hypertrophy) and lean body mass (LBM), increase in basal metabolic rate (BMR), VO_2max, endothelial function and vasomotor function in blood vessels, improved glycaemic control, and an increase in bone mineral density (BMD). Exercise provides a decrease in blood pressure, reduction in percentage body fat and resting heart rate (RHR), reduction in lipid profile, insulin sensitivity and basal insulin levels (Pollock & Vincent, 1996; Baechle *et al.*, 2000; Pollock *et al.*, 2000; Banz *et al.*, 2003; Hambrecht *et al.*, 2003). However the type of training that is undertaken will determine the type of adaptation that occurs. Completing a prolonged (greater than 10 weeks) aerobic exercise regime adaptation occurs. This

includes a decrease in; blood pressure, resting heart rate, percentage body fat, total fat mass (FM), and low density lipoprotein cholesterol (LDL-c). Alongside these reductions there are also elevations in; basal metabolic rate (BMR), VO_2 max, high density lipoprotein cholesterol (HDL-c), insulin sensitivity, and bone mineral density (Pollock & Vincent, 1996; Pollock *et al.*, 2000; Banz *et al.*, 2003; Williams *et al.*, 2007).

However resistance training also elicits change that reduces CVD risk. Such changes include an increase in LBM, insulin sensitivity, HDL-c, BMR; accompanied by decreases in obesity, FM, and percentage body fat, (Albright *et al.*, 2000; Braith & Stewart, 2006; Stiegler & Cunliffe, 2006; Strasser *et al.*, 2010).

This evidence shows that aerobic exercise and resistance training are both beneficial for those aiming to reduce CVD risk. The rationale for completing this study is to determine whether three, 30 minute resistance training sessions per week will have an effect on CVD risk factors in Māori men. The primary point of difference from the aforementioned studies is the shorter training durations.

2.3.1 Exercise and CVD

Insufficient physical activity is the fourth leading risk factor of mortality for CVD (behind hypertension, smoking, and high glucose) (Mathers *et al.*, 2009). Approximately 3.2 million deaths worldwide are attributed to insufficient physical activity (Mathers *et al.*, 2009). It is found worldwide that those who are not sufficiently active physically have a 20-30% increased risk of all-cause mortality compared to those who engage in 30 minutes of physical activity most days of the week (Alwan, 2011); this includes New Zealand where physical inactivity is a major problem with a 95% CI of 44.7-47.3. Given that physical activity reduces risk of early mortality and important focus of research is the role exercise plays and the symptoms associated with disease progression.

Studies that have examined the relationship between physical activity and CVD have reported a decreased risk of death from both CHD and stroke (Berlin & Colditz, 1990; World Health Organization, 2007, 2011b). Physical activity is a modifiable element of energy expenditure, and therefore fundamental in energy balance and weight control. Regular physical activity of more than 60 minutes aerobically has been demonstrated to

improve endothelial function and vasomotor function in blood vessels (Hambrecht *et al.*, 2003). Physical activity also contributes to improved glycaemic control, a decrease in blood pressure, and a reduction in lipid profile, and insulin sensitivity (Cornelissen & Fagard, 2005; Kelley *et al.*, 2005). It is stated by the New Zealand Guidelines Group (NZGG) that individuals should participate in at least 30 minutes of moderate intensity exercise daily (New Zealand Guidelines Group, 2012). However only half the population living in New Zealand are getting the recommended 30 minutes of physical activity a day (New Zealand Ministry of Health, 2012a) Physical activity is beneficial in many ways; exercise reduces body fat, whilst preserving lean mass, increase metabolism thus increasing heart strength leading to greater weight loss (Warburton *et al.*, 2006). As a result of this the human body becomes more efficient at storing and utilising energy at rest and during exercise. In addition to the previously mentioned benefits it also decreases the tendency of plaque to accumulate around the heart thus reducing the risk of a cardiac event (Cannon, 2009).

2.3.2 Exercise modalities prescribed

A review of studies prescribing exercise for CVD patients was conducted for this thesis; with the aim of examining the effect of both aerobic, and resistance exercise on CVD risk factors. Articles examined presented findings on modifiable measures i.e. diet, physical activity, hypertension, hypercholesterolemia, with an intervention period greater than 8 weeks were examined. Six trials prescribed only aerobic training (e.g. running) (Agurs-Collins *et al.*, 1997; Dunstan *et al.*, 1997; Cuff *et al.*, 2003; Sigal *et al.*, 2007; Nojima *et al.*, 2008; Marwick *et al.*, 2009); whilst resistance training was prescribed in ten trials(Morris & Crawford, 1958; Honkola *et al.*, 1997; Pollock *et al.*, 2000; Byrne & Wilmore, 2001; Dunstan *et al.*, 2002; Baldi & Snowling, 2003; Dunstan *et al.*, 2005; Braith & Stewart, 2006; Sigal *et al.*, 2007; Williams *et al.*, 2007). Seven trials however used both aerobic and resistance training and compared the benefits of each to one another(King *et al.*, 1995; Stefanick *et al.*, 1998; Banz *et al.*, 2003; Bassuk & Manson, 2005; Snowling & Hopkins, 2006; Stiegler & Cunliffe, 2006; Sukala *et al.*, 2012). However during this review no studies were found to incorporate 30 minute resistance training sessions three times

per week over a period of 12 weeks, therefore this is the first of its' kind to use this protocol on an indigenous population.

Resistance training was added to the list of recommended exercise for CVD rehabilitation in 1990; whereas the use of aerobic exercise training has been known to be effective since the 1950's (Morris & Crawford, 1958); this was further supported by Ralph Paffenbarger, with his study on work activity and risk of coronary heart disease (CHD) (Paffenbarger Jr *et al.*, 1970). The study completed by Morris and Crawford (1958) highlighted the connection between physical labour and a reduced rate of CVD. In comparison to resistance training aerobic training has varying effects on those with CVD. The primary points of difference between the two forms of training is; resistance training stimulates more LBM growth than aerobic training thus increasing basal metabolic rate (BMR), however it provides equal benefit insulin sensitivity, basal insulin levels and cholesterol levels. Aerobic training however provides greater adaptation to percentage body fat, resting heart rate (RHR) and VO_2max (Pollock & Vincent, 1996; Pollock *et al.*, 2000).

2.3.2.1 Resistance training and CVD

Disease management approaches regarding resistance training have historically been conservative. Prior to 1990 resistance exercise training was not a method of exercise rehabilitation accepted or published by either the American college of sports medicine (ACSM) or the American heart association (AHA) (Pollock *et al.*, 2000). This has now changed with many associations and journals strongly recommending resistance exercise as a form of prevention and rehabilitation of CVD including the United States Centres for Disease Control (Banz *et al.*, 2003). Data from Health Professionals' Follow up study suggest that as little as 30 minutes of resistance exercise three times per week may reduce the risk of an initial coronary event (Tanasescu *et al.*, 2002), however the optimal length of exercise that is needed to prevent CHD was not found.

2.3.2.1.1 Acute bout

Improving blood lipid and lipoprotein levels of males in a single bout of requires considerable effort. For example trained men needed to expend 1100kCal in one exercise bout to elevate HDL-c, 1300kCal to lower LDL-c (Ferguson *et al.*, 1998). The energy expenditure is equivalent to a single exercise session will only provide transient favourable changes in lipid concentrations; for the 24 hours after exercise HDL-c concentration was elevated 15% and LDL-c concentration was reduced by up to 22% (Ferguson *et al.*, 1998). During a resistance exercise session males can expect to be utilizing 8.2 Kcal/min (70kg male) (Costill *et al.*, 2012). During an acute bout of resistance exercise training it has been noted that in both young and older adults (22, and 59 - 77y), there is an increase of ~3% in those older adults in basal metabolic rate (BMR) 48 hours after exercise has ceased; thus increasing energy expenditure (Williamson & Kirwan, 1997; Jamurtas *et al.*, 2004).

2.3.2.1.2 Chronic bout

As stated previously it requires considerable effort in a single exercise session to alter a male's blood lipid and lipoprotein profile, and the changes are only transient. However the change in lipid profiles continues to happen if the exercise persists every other day, with a 9% reduction in LDL-c concentrations after 24 weeks of resistance training (Crouse *et al.*, 1997). Through sustained resistance exercise interventions of at least three exercise sessions per week, for a duration of more than four weeks, studies have demonstrated a decrease in CVD risk and a reduction in severity of risk factors including an increase in HDL-c, and a decrease in LDL-c, percentage body fat and insulin sensitivity (Thompson, 2003; Sigal *et al.*, 2004; Cornelissen & Fagard, 2005; Braith & Stewart, 2006; Wolfe, 2006; Williams *et al.*, 2007). The studies mentioned had exercise session durations of 45 minutes or more; therefore the reasoning for doing this study is to discover whether or not 30 minutes of resistance exercise will display similar effects.

2.4 Kaupapa Māori

When the WHO was first established it gave a definition of health as a state of complete physical, mental and social wellbeing, not just as an absence of disease or illness (WHO 1946). A Māori view of health comprises of similar values and is holistic in nature as it focuses on wellbeing rather than just nonappearance of illness or disease (Durie 1985). A Māori approach to health differs greatly from those of non-Māori and cultural sensitivity is very important and can be an ethical issue if assumptions are made that all research and research interventions are suitable for Māori as it is for non-Māori. The Māori term Hauora, often used to describe wellbeing and health; includes spiritual wellbeing and is inclusive of friends and whānau (family) compared to the naturalistic and individual views of mainstream health (Durie, 1994; Durie, 2004).

Programmes and interventions have been aimed at improving Māori health and wellbeing; but the success of these is less dependent on the 'perceived' quality of the information gathered, and more on whether or not the approach of the researchers is culturally suitable. Others have suggested that Māori will continue to respond poorly to interventions if those researchers who develop and implement the research fail to recognise Māori views (Turtle, 2002; Murphy *et al.*, 2003; Mhurchu *et al.*, 2007). Previous attempts to recruit Māori into conventional programmes have not been completely successful. By employing kaupapa Māori values throughout the research, we ensure that research is culturally sensitive. As research programmes evolve it is vital to have feedback from participants, from which changes are based. This is essential to ensure Māori participation in such research in future (Murphy *et al.*, 2003). An example of this is identified by Murphy *et al.* (2003) whereby Māori recruitment into interventions were not successful due to staff and researchers being unaware of kaupapa theory and not being culturally appropriate. Also the interventions themselves were a limiting factor as they were poorly designed and did not meet the needs and desires of Māori. Reasons given for poor recruitment also included lack of Te Reo (Māori language) signage, and lack of space for collecting data.

Loosely defined, Kaupapa Māori is 'the Māori way' of doing things; a principle that describes traditional Māori ways of being, doing and thinking, captured in a Māori view

(Henry & Pene, 2001). The discussion of kaupapa Māori and research, attempts to capture the essence of Māori ideas, some of which may be unfamiliar to non-Māori audiences.

Māori researchers have provided many definitions of Kaupapa Māori research such as:

"Research which is 'culturally safe' which involves mentorship of *Kaumātua* (elders) which is culturally relevant and appropriate while satisfying the rigour of research and which is undertaken by a Māori researcher, not a researcher who happens to be Māori" (Irwin, 1994).

"A desire to recover and reinstate *matauranga* Māori the indigenous system that was in place before colonisation" (Glover, 1997).

"Research by Māori, for Māori and with Māori" (Smith, 1995).

For the purpose of our research the focus was predominantly on the first and third definitions which were the most relevant definitions in this context. However other Māori researchers have highlighted significant differences between Māori and non-Māori approaches to the gathering of knowledge. For example (cram, 1993) argues that Māori knowledge is used to uphold mana within the community, or in the research context, the mana of participants and their whānau. The use of kaupapa Māori protocol during my study will be discussed further in the discussion chapter of this thesis.

3. Methods

3.1 Participants

Sixteen sedentary Māori men [mean (SD) age 37.60 (7.83) years, BMI 34.10 (4.68), body weight 107.6kg (15.7kg)] were recruited and briefed on the details of the study and the potential risks associated with both testing and training. A completed Physical Activity Readiness Questionnaire (PARQ) form (appendix A) and voluntary consent were obtained. One participant did not complete the intervention; and withdrew halfway through the programme with a work related injury. As this participant did not complete the entire intervention his data was not included in the statistical analyses.

The recruitment of participants involved placing flyers around Massey University, emailing family, social and other networks and by word of mouth. During an initial information evening the details of the study were outlined (an information sheet was sent to potential participants prior to the information evening) (appendix A). Those who met eligibility criteria (appendix B) and health screening then proceeded to familiarisation and testing stages. Eligibility criteria included: (1) self-identified Māori descent; (2) smoke free for the previous two years; (3) having a BMI over 25; (4) led a sedentary lifestyle. All participants lived in the Manawatū region.

For the purposes of this study, “sedentary lifestyle” is defined as not engaging in any strenuous physical activity for longer than 30 minutes per week as described in the New Zealand health survey (New Zealand Ministry of Health, 2008b). Written informed consent was obtained from the participants and the study was approved by the Massey University Human Ethics Committee Southern A 12/19.

3.2 Study design

The aim of this study was to ascertain whether resistance training for 30 minutes, three times per week will affect CVD risk factors in Māori men.

All exercise sessions were completed at the Sport and Rugby Institute (SRI) located at Massey University, Turitea campus. This was a temperature controlled environment with full gym facilities. All participants were familiarised with the equipment and exercises prior to starting their training intervention. Participants were encouraged to perform only the exercise prescribed in this intervention during the 12 week period (i.e. avoid working out more than prescribed).

All participants were asked to attend three training sessions per week between Monday and Friday, inclusive; for a total of 36 sessions, providing there was 24 hours rest between exercise sessions. Health and functional measures were assessed prior to the start of the resistance training intervention (pre), then at six (mid), and 12 weeks (post).

To prevent variations in participants' testing results, diet was restricted ~12 hours prior to blood analysis; and exercise was restricted for the five days leading up to the day of testing. Diet was controlled with an overnight (12 hour) fast before blood sampling; participants were then provided with a standardised breakfast prior to exercise. A five day period of no vigorous activity before testing was instated to ensure there was no residual muscle damage, soreness, or fatigue on the day of physical testing.

Diet was not controlled during the 12 week intervention but participants were encouraged to maintain their usual dietary habits throughout the trial and dietary logs were kept for four days before and then four days after the 12 week intervention.

3.2.1 Kaupapa Māori design

As part of the investigation into the effects that resistance training has on blood lipid profiles and body composition, a kaupapa Māori research method was employed to be more culturally sensitive to all subjects. As previously mentioned kaupapa Māori research is research for Māori, by Māori with Māori. By doing research this way with the participants it place greater emphasis on the overall welling (Hauora) of the subjects and gave a more holistic overview of the study, rather than if it was based purl on western values of health. Implementation of kaupapa Māori methodology within the intervention consisted of frequent discussion (hui) and coupling this with Kai (Food) and discussing all matters relating to not only physical health (Tinana), but also mental and emotional (Hinengaro), spiritual (Wairua) and social (Whānau) wellbeing. By implementing these procedures into the intervention, as researchers we made sure that it would be a culturally relevant and appropriate as possible.

3.3 Outcomes

The outcomes measured in this study are split into primary and secondary status. Primary outcomes, related to known cardiovascular risk factors include examining body composition - this includes fat mass (FM) and lean body mass (LBM) - and change in circulating blood lipid parameters, low density lipoprotein (LDL) and high density lipoprotein (HDL cholesterol). The secondary outcome of this study was to investigate the efficacy of a structured programme with minimal time commitment (i.e. 3x30 minutes) on health status, including subjective wellbeing. To assess the secondary outcomes, participants were asked to complete a questionnaire.

The outcome assessment was conducted prior to participants undertaking the exercise intervention. The same measurements were then conducted again at six and twelve weeks.

3.3.1 Pre experimental protocol

Participants initially reported to the human performance laboratory (HPL) at Massey University one week prior to undertaking the testing procedure to complete a familiarization session. During this session all participants became acquainted with lab surroundings, essential equipment use and emergency procedures. Participants undertook unloaded bench press and leg press exercises to familiarise themselves with the exercise movement.

3.4 Experimental protocol

Prior to biochemical and exercise testing, participants were asked to refrain from exercise, apart from normal daily physical activity, in the five days leading up to the protocol; participants were also asked to fast for a period of 12 hours prior to blood sampling allowing for fasting measures to be taken.

3.4.1 Blood sampling

Blood sampling was performed by a trained phlebotomist at Palmerston North Hospital, blood samples were drawn (approx. 10 ml) via venipuncture from an antecubital vein. The drawn blood was collected into one of two different tubes; a grey top for cholesterol and an orange top for triglycerides (BD Vacutainer, Franklin Lakes, NJ, USA). The grey top contained approximately 1ml sodium fluoride to prevent clotting of the blood (anticoagulant), whereas the orange top contained approximately 1ml serum separator tubes (SST) clot activator and gel for serum separation (clotting agent). Participants' blood sampling occurred at baseline, mid (6wk) and post (12wk) intervention.

Once blood samples were in the correct tube they were then inverted; five times for the orange top, and eight for the grey top to mix blood with the additives. If this was not done immediately the tubes with anticoagulant would clot, and the tubes with SST may not completely clot. Samples were then analysed on site at Palmerston North Hospital.

3.4.1.1 Lipid profile

Analysis of HDL cholesterol was performed by Abbott Architect ci8200, chemistry "c" module (Abbott Laboratories, Abbott Park, Il, USA). LDL cholesterol levels are calculated by Med Lab Central (MLC) using the Friedewald equation. This equation works by subtracting other particles such as HDL-c, and fasting triglycerides (FT) from total cholesterol (Warnick *et al.*, 1990).

$$LDL = TC - \left[HDL + \frac{FT}{2.2}\right]$$

However the equation is inaccurate with triglyceride levels of > 4.5 mmol/l. LDL is not calculated on samples with high triglycerides. For this reason blood samples are obtained and analysed after a 12 to 14 hour fast.

Analysis of triglycerides was performed by Abbott Architect ci8200, chemistry "c" module (Abbott Laboratories, Abbott Park, Il, USA).

3.4.2 Resting measures

Prior to testing, resting measures were gathered, these consisted of heart rate, blood pressure, height, weight, and waist and hip circumference. From these measures, BMI, and WHR were calculated.

Resting blood pressure was measured three times prior to participants eating or exercising and after they had sat down for five minutes to obtain an accurate measure with an aneroid sphygmomanometer (ALPK2, Tokyo, Japan) and Littmann stethoscope (MediSave, Stratford, CT, USA).

Heart rate was monitored via heart rate sensor (Polar Wear Link®, Kempele, Finland) and received via heart rate monitor (Polar FS1, Kempele, Finland). The resting heart rate of each participant was recorded prior to exercise; and was measured again at volitional fatigue, giving a measure of maximum heart rate.

Height and weight were measured using standard protocols (Lohmann *et al.*, 1988) on a calibrated stadiometer and scale respectively, BMI was calculated from these measures. Participants had their hip and waist circumference measured using standardised sites (Dwyer *et al.*, 2008).

$$BMI = \frac{Weight\ (kg)}{Height\ (cm)^2}$$

3.4.3 Body composition

Body composition was analysed via Dual-energy X-ray absorptiometry (DXA formerly DEXA) machine Dexa - Hologic Discovery A, QDR series (Quantitative Digital Radiography) Apex System Software version 3.2, Model ID # 82414, (Hologic, Danbury, CT, USA). This provided information on fat mass, LBM, percentage fat content, bone mineral concentration (BCM) and bone mineral density (BMD). This was operated by a trained technician based at the Institute of Food Nutrition and Human Health (IFNHH) at Massey University Palmerston North.

Participants' height and weight were input to the DXA software. The participants were instructed to change out of their clothes and into a medical gown; they were also instructed to remove any jewellery. The participant was asked to breathe normally, lie supine and close their eyes for the duration of the scan.

Hip and waist circumference was measured with a retractable measuring tape (Muratech-KDS Corp, Kyoto, Japan). The waist circumference measurement was taken at the end of normal expiration midway between the lower costal margin and the most superior portion of the iliac crest of the pelvis. Hip circumference was taken from the widest point of the hips, from the buttocks and around the most distal portion of the greater trochanter(Lean *et al.*, 1995). From the two measurements waist to hip ratio can be calculated by:

$$Waist{:}\,Hip\ ratio = \frac{\text{Waist circumference(cm)}}{\text{Hip circumference(cm)}}$$

3.4.4 Strength measurements

Strength tests of 1-repetition maximum (1 RM) bench press and leg press were completed at the Sport and Rugby Institute, located at the Massey University Turitea campus in Palmerston North, under direct supervision of the lead researcher. Participants were instructed to warm up on a weight 50% of their one repetition maximum (1RM). 50% of their 1RM was determined by either their previous experience in a gym setting, or the participant started at a weight they felt would be close to their own 50%. The weight was then incrementally increased with each participant completing two repetitions then resting for four minutes before attempting a heavier weight, as recommended by ACSM (Dwyer *et al.*, 2008). When each participant could no longer increase their weight when performing the exercise this was considered their maximum weight. If two or more reps were completed at their maximum weight then the Baechle (2000) equation (Baechle *et al.*, 2000) was used to predict their maximum possible lift.

$$1RM = weight(kg)\ X\ (1 + (0.033X\ number\ of\ repetitions))$$

3.4.5 Safety precautions

Two researchers were on hand at all times during the testing phase. A defibrillator was present during the maximal testing phase of the study in the Massey HPL in case of cardiac arrest. In case of emergency the researchers always had private cell phones available if medical professionals needed to be alerted. The laboratory manager was first aid trained, if they were unavailable, a known list of first aiders within the building was also at hand.

3.5 Intervention

The resistance training intervention took place over a 12 week period with pre-, mid-, and post-testing sessions. The training sessions took place at the Sport and Rugby Institute at Massey Universities' Turitea campus using commercially available equipment. All subjects trained with a partner in keeping with the social aspect of the study; and under the direct supervision and guidance of a qualified Personal Trainer. Each subject was encouraged to perform each set and repetition with the utmost effort.

The resistance training group performed three sets of six major exercises (Appendix C) using both machine and free weights, targeting all the major muscle groups; abdominals, arms, back, chest shoulders and legs, for ten repetitions to neural fatigue. Approximately 60 seconds rest was provided between sets, and separate exercises. Tempos of exercises were maintained at a ratio of 2:2. Sessions lasted 30 to 35 minutes including a short warm up. The exercises performed in each session, including sets, repetitions and weight lifted were recorded for each participant in every session. The weight lifted (%RM) was set at 70% and was increased if participants could complete more than 10 reps for 3 sets of said exercise.

3.6 Training programmes

Details of the training programmes are supplied in the appendices (Appendix C). Briefly, the training intervention consisted of five base programs that were repeated throughout the first six weeks for neurological adaptation, and to allow the individuals to become accustomed to the stimulus. After six weeks the programmes were modified when required if the participants had an injury, started to plateau, or other extenuating circumstances arose. Training loads were increased once participants could perform 12 repetitions during all three sets of the specific exercise.

3.7 Statistical analyses

A repeated measures (for time) Analysis of Variance (ANOVA) was performed to determine a difference over time for the body composition, and biochemical measures. Post hoc analysis consisted of paired samples t tests with a Bonferronei correction to determine where significant interaction occurred during the intervention. All statistical analyses were performed using Predictive Analytics SoftWare (PASW) software (version 18, IBM Inc, Chicago, Il, USA). Statistical significance was accepted at the 95% level of confidence. All data is expressed as means ± standard deviation (SD) in the text unless stated otherwise.

3.8 Exercise views of participants

After completion of the 12 week training intervention participants were approached to complete a questionnaire to assess qualitatively their experiences of how the training programme has impacted on their overall health and wellbeing. Of the 15 participants that completed the intervention 14 filled out the questionnaire. The questionnaire was constructed in a semi structured interview style process where individuals answered in free text. They were free to ask any questions for clarification and meaning of the questions at any time from the researcher. The aim of the questionnaire was to find links between exercise and all aspects of the Whare Tapa Whā Māori health model; not just to the physical (Taha tinana) but also the mental and emotional (Taha hinengaro), social (Taha whānau), and spiritual (Taha wairua).

Qualitative analysis of the anecdotal evidence provided by the 15 participants who finished the 12 week programme was undertaken. Analysis of their responses was completed using qualitative methods namely a thematic approach. This method allows the grouping of responses of a certain theme and to draw conclusions from the group's replies. The participant responses ranged from physical gains to mental, spiritual, emotional and overall wellbeing (Hauora). The Māori health model of Te Whare Tapa Whā is used. Hauora or wellbeing in Māori is not directly measured by this questionnaire however themes are revealed throughout the participant responses, giving a clear indication where they felt change occurred.

4. Results

4.1 Compliance

Of the 16 men recruited to participate in this study, 15 completed the intervention, and 12 completed at least 75% of the training protocol. One participant withdrew at the halfway mark, due to a work related injury. The data analyses were therefore performed on 15 of the participants.

Due to family and work commitments, some participants missed a number of training sessions. Compliance to training ranged from 19 to 35 (52.8% – 97.2%) out of a total 36 possible sessions over the 12 week period with a mean average session attendance of 78.1%.

4.2 Physical characteristics

Results from post hoc analysis showed that percentage fat mass significantly decreased ($P<0.001$), lean body mass significantly increased ($P<0.05$), and LDL-C decreased ($P=0.039$).

Body fat percentage decreased significantly between pre- and post-measurements ($P<0.001$). Post hoc analysis shows there was significant interaction between exercise protocol and percentage body fat, between the pre and six week measures ($P<0.005$). An ANOVA analysis of variance found an S value of 0.631049, with an R-Sq value of 98.1%.

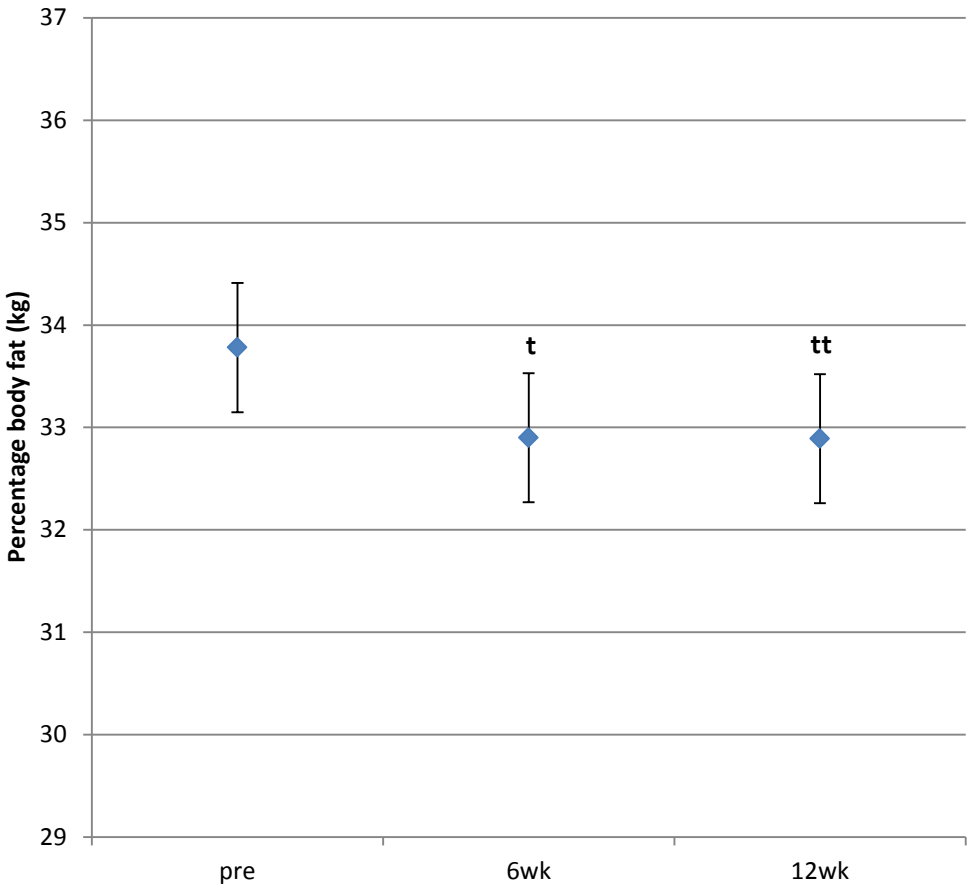

Figure 4-1. Mean (± SE) of percentage body fat pre intervention, at six weeks and at 12 weeks of the resistance training intervention (n=15).

[t] indicates a (post hoc) significant difference between pre and 6 week measures ($P<0.05$).

[tt] indicates a (post hoc) significant difference between pre and 12 week measures ($P<0.05$).

Lean body mass (LBM) increased significantly as a result of the training stimulus (P<0.015) from 67.74 ± 7.87 to 68.55 ± 7.77. ANOVA shows a significant interaction (P<*0.05*) between LBM and the exercise prescribed to participants between the pre and 6 week measures (Refer to table 4.1).

A marginally significant reduction in total body fat was observed as a result of the training intervention (P<0.054). Post-hoc analysis identified that significant interaction occurred between the pre- and six week time points and pre- and 12 week tests. Change in total body fat was measured at pre-, 6 week and 12 week (Refer to table 4.1).

Waist to hip ratio was measured; however no significant change was seen during the intervention (P>0.05).

BMI was measured, however no statistically significant change was observed (P>0.05).

4.3 Cholesterol

Both HDL and LDL cholesterol were measured during this intervention. However significant levels of change were not found within HDL-c (P>0.05), and only marginal levels of significance were found with LDL-c (P=0.081). Further statistical analysis of LDL-c was undertaken on those subjects whom completed at least 75% (27/36) total sessions, those who did not adherer strictly to the intervention had their results omitted from further analysis. Since LDL-c was determined to be marginally significant, post-hoc analysis (Paired-Samples T test) was undertaken to determine where that significance lay. In this case, there was a significance difference between baseline and the 12 week time point (P=0.039) (Table 4.1).

Table 4.1. Change in LBM, total body fat, LDL-c, HDL-c and WHR at pre, 6wk and 12wk (mean ± SD).

Measure	Pre	6wk	12wk	P value
BMI	34.1 ± 4.99	34.1 ± 4.94	33.8 ± 4.5	P=0.469
Lean Body Mass	67.74 ± 7.87 [t]	69.16 ± 8.01	68.55 ± 7.77	P=0.015
Total Body Fat	36.84 ± 8.8 [t]	35.93 ± 8.5	35.59 ± 7.6 [tt]	P=0.054
LDL-c	3.7 ± 1.3	3.5 ± 1.3	3.4 ± 1.5	P=0.081
HDL-c	1.01 ± 1.3	1.01 ± 1.8	1.03 ± 1.9	P=0.83
WHR	0.98 ± .05	0.98 ± .05	0.96 ± .04	P=0.196

[t] indicates a significant difference between pre and 6 week measures (P<0.05).

[tt] indicates a significant difference between pre and 12wk measures (P<0.05).

Table 4.2 Change in LDL-c from pre training to 12 weeks of resistance training (mean ± SD).

Measure	Pre	6wk	12wk	P value
LDL-c*	3.7 ± 1.3	-	3.4 ± 1.15[tt]	P<0.039

* indicates Paired-Samples (Pre and post) t-test of those who completed 75% of the intervention.

[tt] indicates a significant difference between pre and 12wk measures (P<0.05).

4.4 Post exercise questionnaire

The Whare Tapa Whā model of Hauora was first presented in 1984 and is widely accepted as the preferred Māori definition of health (Durie, 1994) Work done by Durie (1994) highlights the challenges that exist between the change from a Western framework to the acknowledgement of Māori knowledge. In this sense it complements Kaupapa Māori theory.

As a result, a culturally relevant approach of health called the Whare Tapa Whā model (Durie, 1994) was used as a guide for the design and implementation of this study. The Whare Tapa Whā model is a simplified explanation of Hauora that highlights the interaction of four major concepts - Taha Wairua (spiritual well-being), Taha Hinengaro (psychological well-being), Taha Tinana (physical health) and Taha Whānau (the well-being of family and relationships) (Durie, 1985). This framework of Hauora has been extensively utilised in human development, social development and health sectors to provide culturally appropriate processes to raise the profile of Māori health and identity in New Zealand (Pitama *et al.*, 2007). Te Whare Tapa whā is a figurative illustration of a wharenui (traditional meeting house) with each of the four aspects of Hauora represented as a wall of the house. This exemplifies that each dimension of Hauora must be strong and balanced in order to maintain the integrity of the house (i.e. achieve overall well-being).

Analysis of the responses to the questionnaire identified several major themes. Significant themes were observed in four questions. These themes were organized according to the four dimensions of Māori health identified in Te Whare Tapa Whā.

4.4.1 Taha Tinana

This aspect of well-being (physical activity) is perhaps the most familiar dimension to mainstream health professionals.

Firstly participants felt their overall health and wellbeing was improving, or was better than when they first started the intervention. The following response relates to participants' tinana, or physical wellbeing.

"The best thing I did was start this programme. It has boosted my motivation to exercise, thus improving my health and wellbeing."
(Participant IW-1603, age 37)

There were concerns raised about the lack of aerobic training during this intervention by some participants, however participants all agreed that they benefited from the training.

"I have more energy to play with my kids, feeling stronger but unfit".
(Participant IW-06, age 28)

4.4.2 Taha Hinengaro

Hinengaro refers to the mental and emotional dimensions of health expressed through thoughts and feelings.

Another key theme identified in the results from this questionnaire was that those who completed this training intervention found that it impacted positively on the quality of time they had with family, strengthening relationships at home. The themes discovered here relay a connection to both a feeling of increased hinengaro and tinana (mental/emotional and physical wellbeing). This is supported by the following participant statements.

"It has not only made me stronger, but has given me mental clarity. I am keen to make this a lifelong habit."
(Participant IW-0203, age 43)

"I view myself as healthy; have had a lot more energy over the previous 12 weeks, and sleeping better."
(Participant IW-1403, age 46)

4.4.3 Taha Whānau

Whānau is translated as family, and includes the maintenance and building of relationships, both in and outside of blood relatives and whakapapa (ancestry/ genealogical lines). At the centre of the whānau concept is the idea of support that incorporates the human capacity to care and nurture in all of the aforementioned concepts - physically, emotionally and spiritually.

A trend found between participants was that both they and their whānau benefited from completing this intervention by having a better support network around them, and a more positive environment. The responses from participants imply an increase in whānau (social) wellbeing; this is exemplified by the following responses:

"Yes since I have been doing this programme my son has joined a gym and two other whānau members spend time with me working out, or me with them, talking sharing and supporting."
(Participant IW-1403, age 46)

"More energy, instead of parking on the couch, I'm doing more with my whānau."
(Participant IW-1603, age 37)

"No change in the amount of time in terms of time spent with whānau, however time spent talking about personal and whānau health has increased."
(Participant IW-0403, age 28)

"I have increased energy levels to spend more proactive time with whānau."
(Participant IW-1503, age 40)

4.4.4 Taha Wairua

Taha wairua is an vital requirement for health; It infers the ability to have faith, understand the links between human situation and the environment; without a spiritual awareness and a mauri (spirit or life-force) an individual cannot be healthy, and includes religious beliefs and practices but is not synonymous with regular churchgoing, belief in God is one reflection of wairua, but it is also evident in relationships with the environment; land, lakes, mountains, and reefs have a spiritual significance (Durie, 1994).

Participants felt their view of resistance training had changed over the previous12 weeks whilst undertaking this protocol. The trend of this fourth and final theme points towards an increase of participants' spiritual wellbeing (wairua). This is represented in the following responses:

"It does not have to be long, if done correctly it can be highly beneficial to personal health. It is also more enjoyable in a group environment."
(Participant IW-0703, age 38)

"I feel stronger and happier with myself."
(Participant IW-1303, age 38)

4.4.5 Conclusion

In summation, this description and anecdotal evidence suggests that this cohort of Māori men found this 12 week resistance training intervention was beneficial for them physically (tinana) and enhanced other aspects of their wellbeing (Hauora) including Wairua (self-esteem), hinengaro (mental/emotional) and whānau (social) wellbeing.

5. Discussion

The current investigation is the first to examine the effects of prescribing short bouts of moderate frequency resistance training for 12 weeks on CVD risk in Māori men. Previous studies such as those done by Kelley *et al.* (2006), and De Faire *et al.* (1993) show that aerobic training can reduce CVD risk factors in individuals with raised CVD risk including obese males. Resistance training studies completed by Banz (2003) and Bassuk & Manson (2005) have also shown a reduction of CVD risk factors in men with android obesity. However our study was completed in a much shorter exercise duration and intervention length. The aforementioned studies had durations of up to and including 120 minutes of exercise per session for three to six months.

The findings from our research suggests that resistance training prescribed three times per week for 12 weeks in 30 minute bouts is sufficient to improve risk factors associated with CVD such as blood lipid profile and obesity. However there is no evidence of improvement in biochemical markers of CVD risk. Previously there have been no other studies to use this specific protocol i.e. 30 minute sessions at three times per week.

This investigation was developed in response to the high prevalence of CVD amongst male Māori in comparison to non-Māori. The discussion of this study will be conducted within three distinct sections to bring clarity and simplicity to the findings.

The three sections are as follows:

1) Body composition
2) Biochemical markers
3) Exercise protocol

For the purposes of this study, body composition observation records significant changes in body mass index (BMI), total fat mass, total fat percentage, lean body mass (LBM) and waist to hip ratio (WHR), produced from the 12 week intervention.

Biochemical investigation focuses upon the results and analysis of blood samples taken from the participants and determining whether or not there is a significant change in biochemical markers during this study. These markers include total cholesterol and the comparison of HDL-c and LDL-c.

Exercise protocol is the third key point of discussion, and is the crucial point of difference in this study, as participants were told they were only allowed to exercise three days per week for 30 minutes each day. This is less than previous research studies and the first to do so in an indigenous population.

5.1 Body composition

The analysis of body composition when observing change in CVD risk factors is vital; as it critical to differentiate between fat mass (FM) and lean body mass (LBM) to determine risk level (Segal *et al.*, 1987).

The most significant finding of this study was that resistance exercise training 30 minutes a day, three days a week for 12 weeks reduced percentage body fat in Māori men. These data show a significant reaction to the intervention ($P<.001$) and are consistent with previous exercise training studies that show an improvement in body fat percentage post resistance training (Pollock & Vincent, 1996; Prabhakaran *et al.*, 1999; Banz *et al.*, 2003). This result can be attributed to a significant increase in lean body mass (LBM) ($P<0.015$). The increase of LBM in participants parallels that seen in previous resistance training studies that look at CVD risk factors (Baldi & Snowling, 2003; Dunstan *et al.*, 2005). With an increase in LBM, comes an increase in basal metabolic rate (BMR). Thus leading to an increase of fuel utilization during rest, and typically reducing fat mass, and decreasing body fat percentage (Pratley *et al.*, 1994; Byrne & Wilmore, 2001; Stiegler & Cunliffe, 2006).

Basal metabolic rate (BMR) is the minimum amount of energy required by our bodies to perform basic cellular functions (Weibel, 2002). Having a higher proportion of LBM will increase BMR and fuel utilisation at rest compared to those who have lower LBM content;

thus resulting in a reduction in FM (Ravussin *et al.*, 1988). BMR typically ranges between 1100 to 2500kcal per day however when activity is added, typical daily expenditure is around 1700 to 3100kcal per day (Costill *et al.*, 2012).

A third finding of this study was a marginally significant reduction in total body fat ($P<0.054$). This indicates there was a trend to a reduction in fat mass (FM) but not to the same extent as the LBM. This marginal result probably reflects the relatively short session duration and frequency (30 minutes three times per week) not providing significant time in negative energy balance to greatly influence body fat stores. A training period greater than 12 weeks is possibly required to produce significant reductions in total fat, as was seen by Misra *et al.* (2008). Nevertheless, even a slight an increase in duration/frequency of exercise sessions may provide sufficient stimulus to produce a truly significant reduction in body fat stores.

Data published by Byrne & Wilmore (2001) describing adiposity either side of a 20 week training intervention also display an increase in LBM, with no significant reduction in fat mass. It should be noted that percentage body fat is affected by a reduction in FM, and/or an increase in LBM.

A reduction in fat mass is directly related to a reduction in body fat when assessing individuals with DXA. Body mass index, on the other hand, is essentially an indicator of population-based health status because it does not take into account individuals' body composition, only their total weight in relation to their height. As previously stated, there was no significant change in BMI throughout this intervention. For this reason is DXA analysis included in body composition measures to track changes in body compartments (LBM and FM).

Waist circumference was measured to determine if there is a reduction in android obesity and weight distribution around the trunk of participants.

WHR has been found to be more reliable predictor of both CHD and CVD than BMI in some population groups (Larsson *et al.*, 1984; Folsom *et al.*, 1993), and is a better predictor in men than women (Hu *et al.*, 2004). Waist to hip ratio (WHR) was measured in the present study to estimate the distribution of fat. No significant change in this

variable occurred as a result of the training program (P<0.196). Again, whilst this oft-used health marker showed a non-significant decrease (and thus by this measure the subjects' health status remains unchanged), DXA measurements show a decrease in adiposity and increase in lean body mass.

5.2 Biochemical

Biochemical analysis consisted only of measurement of participants' cholesterol levels. The data show that there was no change in HDL-c levels in participants, however there was a marginally significant reduction in LDL-c levels P<0.081. Further analysis of this result found a significant reduction in LDL-c in those who completed at least 75% of the intervention P<0.039.

The intervention data shows no significant change in HDL-c (P<0.83). It is presumed likely that training for 30 minutes three times per week does not provide sufficient stimuli to change the HDL-c lipid profiles of these individuals. Our data and data from previous studies suggest that aerobic training at sufficient intensity levels and appropriate duration are more effective at elevating HDL-c levels (King *et al.*, 1995; Byrne & Wilmore, 2001). Subjects in the trial by Banz *et al*. (2003) were subjected to 10 weeks of resistance training with measures of both HDL-c and LDL-c taken, with no significance found in change between pre and post measures. However in the same trial by Banz *et al*. (2003), aerobic training was found to significantly increase (P <0.05) HDL-c in those who undertook 40 minutes of aerobic activity three days per week. King *et al*. (1995) also found that aerobic training took an extended period of time to increase HDL-c levels in participants'; this study ran for two years. The above-mentioned articles also found that aerobic training was more effective than resistance training in elevating HDL-c levels (King *et al.*, 1995; Banz *et al.*, 2003).

Based on current results, there is a marginally significant change in LDL-c (P<0.081) when doing a one way analysis of variance (ANOVA) of the entire collective of individuals. However when a compliance protocol is used to remove the data of those who did not strictly adhere to the protocol (complete 75% total training sessions), it is clear there is a

significant interaction between pre and post measurements (P=0.039). This is in line with other studies that show results similar to this study (Fahlman *et al.*, 2002; Augusto *et al.*, 2012). However the study completed by Augusto *et al.* (2012) had a cohort who consisted of men and post-menopausal women, who trained three times per week for a total of 16 weeks, and carried out nine exercises per session. The study completed by Fahlman *et al*. (2002) had a cohort of elderly women age 73±3 years; they completed eight exercises per session over a 10 week period.

A positive correlation is evident between the results of this study and the hypothesis. These data show that completing 30 minutes of resistance exercise hypercholesterolemia and cardiovascular disease risk factors in male Māori can be reduced.

5.3 Exercise protocol

Numerous studies examine the effect of exercise on CVD risk factor reduction in aerobic training (Savage *et al.*, 2003; Rognmo *et al.*, 2004; Kodama *et al.*, 2007); however fewer analyse the effect of resistance training as a modality to reduce CVD risk factors. This study focused on providing resistance exercise in a shorter format than has been previously trialled. This involved setting the amount of exercise to three 30 minute resistance training sessions per week. The principal reason for specific emphasis on duration was identified by the researchers who found that many respondents stated that lack of time was the key factor which restricted their time to be able to complete physical activity. Unavailability of free time was also identified by Sport New Zealand as a barrier for Māori to commit fully to an exercise regimen (Sullivan *et al.*, 2003).

5.3.1 Training type

Numerous studies have examined the effect aerobic training has on cardiovascular disease risk factors. The vital point of difference within this study is the implementation of a resistance training protocol to determine the effect it may have on CVD risk factors in male Māori. With regard to risk factors afflicting the participants, by implementing a resistance training protocol, participants are then no longer physically inactive from their total risk of CVD.

5.3.2 Training duration

Specific emphasis was placed on the duration tempo of exercises during the training session throughout this study, as it was the first study to specifically examine both; the effect of 30 minutes of resistance training, three times per week, over a 12 week period and the effect of this protocol on a male Māori cohort.

By utilizing a training protocol that was 30 minutes in length, it was found that many respondents were able to fit this short period of exercise into their daily schedule. The key idea was to make participants stick to a controlled tempo of 2:2 when lifting and

lowering the weight. This way the participants completed six exercises for three sets and ten reps during training sessions in a controlled manner.

This type of protocol is vastly different from the majority of studies that focus on exercise and CVD risk. Primarily due to the 30 minute time frame during which the exercise must be completed; whereas many studies range between 45 (Castaneda *et al.*, 2002; Dunstan *et al.*, 2002; Dunstan *et al.*, 2005; Sigal *et al.*, 2007) and 90 minutes in length (Sigal *et al.*, 2007; Marwick *et al.*, 2009).

By implementing a resistance exercise regimen it is possible for those who suffer from CVD, or those who present with CVD risk factors to minimise the potentially harmful effects of a sedentary lifestyle.

5.4 The Application of a Kaupapa Māori approach

A kaupapa Māori approach was used throughout the entirety of this intervention. The key stages of this intervention include recruitment, testing, training and follow up (final stage is part of a larger study, not covered within the scope of this thesis). The implementation of kaupapa Māori protocol is seen as important step to breaking down barriers between Māori and research science as is described by Mason Durie (1994).

The recruitment process started for those who responded to fliers and social media interactions by accepting invitations to attend a gathering (hui). Once all together, the head researcher (Dr Isaac Warbrick) welcomed potential participants as part of a whakatau; a traditional welcoming ceremony. The research was then verbally outlined to go along with the physical information sheets previously delivered to potential participants. Once informed, kai (food) was served following a karakia (prayer) and allowed participants to eat, talk and become familiar with one another. Conducting whakatau and sharing kai is part of traditional tikanga and provides an opportunity to acknowledge research participants and their whānau, get to know one another by establish familial links and discuss matters of importance pertaining to the research. When sharing kai, it matters not where you sit as in Māoridom all are as important as one

another. (Richardson, 1990). Karakia on the other hand acknowledges the influence of the unseen and also provided a form of motivation and focus for the activities to follow (Mead & Mead, 2003).

Once eating was finished all gathered in a marae (meeting place) to discuss their feelings and thoughts and ask questions openly about participating in the study. "The marae is the focal point of Māoridom, it enables the Māori to carry out traditions and express their values fully and with dignity" (Tauroa *et al.*, 1986). The marae is a significant aspect of Māori culture and is an institution of Māori society that has survived the impact of western civilisation it is a fundamental feature of Māori cultural identity. It is a place where Māori values and philosophy are reaffirmed and is absolutely sacred. The marae itself is socially integrative in the sense that it nurtures identity, pride, and self-respect. Once all are settled in the marae both participants and researchers took turns speaking to the group who had gathered to express their own thoughts and feeling towards not only the kaupapa (purpose) of the research but why and how it was important to Māori as a people. When all discussion had finished a finial karakia was said to thank everybody present physically, and spiritually for their time and thoughts (Tauroa *et al.*, 1986).

During testing the kaupapa of whanaungatanga, a principle relating to the importance of family and relationships was expressed by conducting research in a group setting. Whanaungatanga can be defined as the building of relationships within a group through shared experiences, allowing for support during difficult experiences (Mead & Mead, 2003). By expressing whanaungatanga within the group the tone was set for a more relaxed and supportive environment for those completing the testing elements. Throughout the training phase of the intervention whānau and whanaungatanga played a critical role in keeping with the kaupapa Māori research technique. This was done by scheduling training sessions in groups of at least two, and up to five participants to keep in line with kaupapa Māori theory of providing an environment where participants are part of a collective who are being part of research that is run by Māori researchers (not just researchers who happen to be Māori), for Māori and completed with a Māori population and Māori aspirations in mind; in line with Linda Smith's definition of kaupapa Māori research (Smith, 1995), as well as Irwin's (1994) definition.

Once the final testing was completed the participants all completed a questionnaire regarding their experience and feelings to the intervention they had just completed. Participants were also invited to a follow up session where kai (food) was served and they were able to voice their opinions both openly and privately with the researchers (part of larger study being completed by Dr Isaac Warbrick). This plays to a theme identified by Murphy *et al.* (2003) where he describes research as; when interventions evolve it is vital to have feedback from participants, from which changes are based. This is essential to ensure Māori participation in such research in future.

5.5 Considerations/limitations

Having 15 participants may have been a restrictive factor in regards to the significance of the data; of that only 12 completing 75% or more of the intervention could again produce a statistically underpowered study. Having a larger group of participants completing the intervention may have increased the level of significance on some of the dependent variables. This is exemplified by comparison with the studies completed by Honkola *et al.* (1997), and Fahlman *et al*. (2002) which had greater participation rates within their studies.

The lack of control group is a major limitation in the design of this study. Without a non-exercising control group, we were unable to truly account for the effect of the intervention, as the results could also be in part be a function of involvement in the research. A more highly powered study would either recruit a second cohort of individuals as a control (Control Trial), or have the cohort act as their own control whereby they undertake the intervention once they have participated in control trials (Cross-over Trial).

Also, there is scope for a larger, more personal interview process rather than a five minute semi structured interview questionnaire regarding individuals' feelings and thoughts about the resistance training intervention and the effect they feel it has had on their overall health. Having a more comprehensive interview process would have provided in-depth detail for the psychological and social effect of the protocol.

There is scope to look at more variables within the same exercise framework to draw more significant conclusions from the exercise completed, and results seen by the participants and researchers alike.

5.6 Future research

While this study demonstrated an improvement in body composition, there was only marginally significant change in biochemical markers. With respect to both statements there are three recommendations for future studies in regards to internal and external feedback provided by both data, and participant responses.

First; the fact the protocol specifically focused on 30 minute training sessions, this was seen as the foremost limiting factor in only producing marginally significant total fat loss, LDL-c decrease and HDL-c increase. With marginally significant results in total fat loss, LDL-c reduction and HDL-c increase, it is proposed that future research investigates one of the following options; lengthen the duration of the training sessions, increase the frequency of training sessions or extending the intervention duration.

Secondly; a common theme mentioned among participants was the lack of aerobic exercise left them feeling unfit and at times wanting to embark on their own exercise regime outside of the stipulated protocol. This could be resolved in future studies with either a purely aerobic training intervention, or combined resistance and aerobic training intervention looking at how they affect cardiovascular disease risk factors including the effect of HDL-c.

Thirdly doing a qualitative arm to this study may help back up what the scientific evidence produces. However by opening up the scope of the qualitative study from a semi structured interview style, to possibly a complete more personal interview or expanding it into a focus group style, researchers may gain a greater understanding of the individuals' and or groups' thoughts and overall wellbeing pertaining to the intervention. A second note to this point is the possibility of doing a pre interview with the subjects rather than solely post protocol, providing view of change in participant perceptions.

6. Conclusions

This was the first study to incorporate a protocol of 30 minute resistance training sessions, three times per week on Māori men; and analysing the effect it has on CVD risk factors. It is also the first to utilize a kaupapa Māori influence in the exercise physiology setting; bringing all aspects of Māori wellbeing together. With a high completion rate and low attrition this form of exercise was well tolerated by this group of participants. The findings produced from this study support the use of this protocol as an effective form of exercise to reduce CVD risk in both body composition and blood lipid profiles in this cohort. It provides a platform for further research into what form and duration of exercise is best to help further reduce CVD prevalence in the Māori population.

7. References

Abegunde D & Vita-Finzi L. (2005). *Preventing chronic diseases: a vital investment*.

Agurs-Collins TD, Kumanyika SK, Ten Have TR & Adams-Campbell LL. (1997). A randomized controlled trial of weight reduction and exercise for diabetes management in older African-American subjects. *Diabetes Care* **20,** 1503-1511.

Ajwani S, Blakely T, Robson B, Tobias M & Bonne M. (2003). *Decades of disparity: ethnic mortality trends in New Zealand 1980-1999*. Ministry of Health Wellington.

Albright A, Franz M, Hornsby G, Kriska A, Marrero D, Ullrich I & Verity LS. (2000). American College of Sports Medicine position stand. Exercise and type 2 diabetes. *Medicine and Science in Sports and Exercise* **32,** 1345-1360.

Alpert MA & Hashimi MW. (1993). Obesity and the heart. *The American Journal of the Medical Sciences* **306,** 117-123.

Alwan A. (2011). *Global status report on noncommunicable diseases 2010*. World Health Organization.

Ashen MD & Blumenthal RS. (2005). Low HDL cholesterol levels. *New England Journal of Medicine* **353,** 1252-1260.

Augusto LC, Bonganha V, Soares CM, Vergínia DSG, Fernandes BC, Secolin R, Aparecida MV & Traina C-MM. (2012). The periodized resistance training promotes similar changes in lipid profile in middle-aged men and women. *The Journal of Sports Medicine and Physical Fitness* **52,** 286.

Baechle TR, Earle RW, Strength N & Association C. (2000). *Essentials of Strength Training and Conditioning*. Human Kinetics.

Baldi J & Snowling N. (2003). Resistance training improves glycaemic control in obese type 2 diabetic men. *International journal of sports medicine* **24,** 419-423.

Banz WJ, Maher MA, Thompson WG, Bassett DR, Moore W, Ashraf M, Keefer DJ & Zemel MB. (2003). Effects of resistance versus aerobic training on coronary artery disease risk factors. *Experimental Biology and Medicine* **228,** 434-440.

Barrett-Connor E & Khaw K. (1984). Family history of heart attack as an independent predictor of death due to cardiovascular disease. *Circulation* **69,** 1065-1069.

Bassuk SS & Manson JE. (2005). Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *Journal of Applied Physiology* **99,** 1193-1204.

Bauman A, McLean G, Hurdle D, Walker S, Boyd J, van Aalst I & Carr H. (2003). Evaluation of the national 'Push Play'campaign in New Zealand–creating population awareness of physical activity. *Journal of the New Zealand Medical Association* **116,** 1-11.

Berg K, Bo/resen A-L & Dahlén G. (1976). Serum-high-density-lipoprotein and atherosclerotic heart-disease. *The Lancet* **307,** 499-501.

Berlin JA & Colditz GA. (1990). A meta-analysis of physical activity in the prevention of coronary heart disease. *American Journal of Epidemiology* **132,** 612-628.

Best E. (2005). *Games and Pastimes of the Maori*. Te Papa Press.

Bjorntorp P. (1987). Classification of obese patients and complications related to the distribution of surplus fat. *American Journal of Clinical Nutrition* **45**.

Braith RW & Stewart KJ. (2006). Resistance exercise training. *Circulation* **113,** 2642-2650.

Bramley D, Riddell T, Crengle S, Curtis E, Harwood M, Nehua D & Reid P. (2004). A call to action on Maori cardiovascular health. *New Zealand Medical Journal* **117**.

Brannon FJ. (1993). *Cardiopulmonary rehabilitation: basic theory and application*. F.A. Davis.

Bray GA. (1996). Health hazards of obesity. *Endocrinology & Metabolism Clinics of North America* **25,** 907-919.

Byrne H & Wilmore JH. (2001). The effects of a 20-week exercise training program on resting metabolic rate in previously sedentary, moderately obese women. *The International Journal of Sport Nutrition and Exercise Metabolism*.

Cannon CPV, E. (2009). *The New Heart Disease Handbook: Everything You Need to Know to Effectively Reverse and Manage Heart Disease*. Fair Winds Press.

Castaneda C, Layne JE, Munoz-Orians L, Gordon PL, Walsmith J, Foldvari M, Roubenoff R, Tucker KL & Nelson ME. (2002). A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. *Diabetes Care* **25,** 2335-2341.

Castelli WP, Doyle JT, Gordon T, Hames CG, Hjortland MC, Hulley SB, Kagan A & Zukel WJ. (1977). HDL cholesterol and other lipids in coronary heart disease. The cooperative lipoprotein phenotyping study. *Circulation* **55,** 767-772.

Chan W, Wright C, Riddell T, Wells S, Kerr AJ, Gala G & Jackson R. (2008). Ethnic and socioeconomic disparities in the prevalence of cardiovascular disease in New Zealand. *New Zealand Medical Journal*.

Chapman MJ, Assmann G, Fruchart J-C, Shepherd J & Sirtori C. (2004). Raising high-density lipoprotein cholesterol with reduction of cardiovascular risk: the role of nicotinic acid-a position paper developed by the European Consensus Panel on HDL-C*. *Current Medical Research and Opinion®* **20,** 1253-1268.

Corbin C, Welk G, Corbin W & Welk K. (2010). *Concepts of Fitness And Wellness: A Comprehensive Lifestyle Approach*. McGraw-Hill Education.

Cornelissen VA & Fagard RH. (2005). Effect of resistance training on resting blood pressure: a meta-analysis of randomized controlled trials. *Journal of Hypertension* **23,** 251-259.

Costill DL, Wilmore JH & Kenney WL. (2012). Physiology of sport and exercise. *Physiology Of Sport And Exercise-9780736094092-66, 78*.

Crouse SF, O'Brien BC, Grandjean PW, Lowe RC, Rohack JJ & Green JS. (1997). Effects of training and a single session of exercise on lipids and apolipoproteins in hypercholesterolemic men. *Journal of Applied Physiology* **83,** 2019-2028.

Cuff DJ, Meneilly GS, Martin A, Ignaszewski A, Tildesley HD & Frohlich JJ. (2003). Effective exercise modality to reduce insulin resistance in women with type 2 diabetes. *Diabetes Care* **26,** 2977-2982.

Cyr NM. (2003). *Health Promotion, Disease Prevention, and Exercise Epidemiology*. University Press of America, Incorporated.

Danaei G, Ding EL, Mozaffarian D, Taylor B, Rehm J, Murray CJ & Ezzati M. (2009). The preventable causes of death in the United States: comparative risk assessment of dietary, lifestyle, and metabolic risk factors. *Public Library of Medicne ONE* **6,** e1000058.

Dandona P, Aljada A & Bandyopadhyay A. (2004). Inflammation: the link between insulin resistance, obesity and diabetes. *Trends in Immunology* **25,** 4-7.

David AK, Fields SA, Phillips DM, Scherger JE & Taylor RB. (2004). *Taylor's Cardiovascular Diseases: A Handbook*, vol. 79. Springer.

De Backer G, Ambrosionie E, Borch-Johnsen K, Brotons C, Cifkova R, Dallongeville J, Ebrahim S, Faergeman O, Graham I & Mancia G. (2003). European guidelines on cardiovascular disease prevention in clinical practice: third joint task force of European and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of eight societies and by invited experts). *European Journal of Cardiovascular Prevention & Rehabilitation* **10,** S1-S78.

De Faire U, Berglund B, Hamsten A & Krakau I. (1993). Diet and exercise are equally effective in reducing risk for cardiovascular disease. Results of a randomized controlled study in men with slightly to moderately raised cardiovascular risk factors. *Atherosclerosis* **103,** 81-91.

Després J-P, Lemieux I & Prud'homme D. (2001). Treatment of obesity: need to focus on high risk abdominally obese patients. *British Medical Journal* **322,** 716.

Dunstan DW, Daly RM, Owen N, Jolley D, De Courten M, Shaw J & Zimmet P. (2002). High-intensity resistance training improves glycemic control in older patients with type 2 diabetes. *Diabetes Care* **25,** 1729-1736.

Dunstan DW, Daly RM, Owen N, Jolley D, Vulikh E, Shaw J & Zimmet P. (2005). Home-based resistance training is not sufficient to maintain improved glycemic control following supervised training in older individuals with type 2 diabetes. *Diabetes Care* **28,** 3-9.

Dunstan DW, Mori TA, Puddey IB, Beilin LJ, Burke V, Morton AR & Stanton KG. (1997). The independent and combined effects of aerobic exercise and dietary fish intake on serum lipids and glycemic control in NIDDM: a randomized controlled study. *Diabetes Care* **20,** 913-921.

Durie M. (1999). Te Pae Mahutonga: A model for Maori health promotion. In *Health Promotion Forum of New Zealand Newsletter*.

Durie M. (2004). Understanding health and illness: research at the interface between science and indigenous knowledge. *International Journal of Epidemiology* **33,** 1138-1143.

Durie MH. (1985). A Maori perspective of health. *Social Science & Medicine* **20,** 483-486.

Durie MH. (1994). Whaiora: Māori health development. Auckland: Oxford University Press.

Dwyer GB, Davis SE & Medicine ACoS. (2008). *ACSM's Health-related Physical Fitness Assessment Manual*. Lippincott Williams & Wilkins.

Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S & Murray C. (2002). Selected major risk factors and global and regional burden of disease. *The Lancet* **360,** 1347.

Fahlman MM, Boardley D, Lambert CP & Flynn MG. (2002). Effects of endurance training and resistance training on plasma lipoprotein profiles in elderly women. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* **57,** B54-B60.

Faigenbaum AD & Myer GD. (2010). Pediatric resistance training: benefits, concerns, and program design considerations. *Current sports medicine reports* **9,** 161-168.

Ferdinand KC & Armani A. (2007). Cardiovascular Disease in Racial and Ethnic Minorities. *Fundamental and Clinical Pharmacology* **57,** 721.

Ferguson MA, Alderson NL, Trost SG, Essig DA, Burke JR & Durstine JL. (1998). Effects of four different single exercise sessions on lipids, lipoproteins, and lipoprotein lipase. *Journal of Applied Physiology* **85,** 1169-1174.

Finucane M, Stevens G, Cowan M, Danaei G, Lin J, Paciorek C, Singh G, Gutierrez H, Lu Y & Bahalim A. (2011). Global Burden of Metabolic Risk Factors of Chronic Diseases Collaborating Group (Body Mass Index). National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. *The Lancet* **377,** 557-567.

Folsom AR, Kaye SA, Sellers TA, Hong C-P, Cerhan JR, Potter JD & Prineas RJ. (1993). Body fat distribution and 5-year risk of death in older women. *JAMA: the Journal of the American Medical Association* **269,** 483-487.

Ford ES, Giles WH & Dietz WH. (2002). Prevalence of the metabolic syndrome among US adults. *JAMA: the Journal of the American Medical Association* **287,** 356-359.

Garn SM, Leonard WR & Hawthorne VM. (1986). Three limitations of the body mass index. *The American Journal of Clinical Nutrition* **44,** 996-997.

Glass CK & Witztum JL. (2001). Atherosclerosis: The Road Ahead Review. *Cell* **104,** 503-516.

Glover M. (1997). Kaupapa Maori health research: A developing discipline. *Paper presented at the Hui Whakapiripiri, Whaiora Marae, Otara, Auckland*.

Golay A & Felber J. (1994). Evolution from obesity to diabetes. *Diabète et Métabolisme* **20,** 3-14.

Gordon T, Castelli WP, Hjortland MC, Kannel WB & Dawber TR. (1977). High density lipoprotein as a protective factor against coronary heart disease: the Framingham Study. *The American Journal of Medicine* **62,** 707-714.

Graham I, Atar D, Borch-Johnsen K, Boysen G, Burell G, Cifkova R, Dallongeville J, De Backer G, Ebrahim S & Gjelsvik B. (2007). European guidelines on cardiovascular disease prevention

in clinical practice: executive summary Fourth 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). *European Heart Journal* **28,** 2375-2414.

Hambrecht R, Adams V, Erbs S, Linke A, Kränkel N, Shu Y, Baither Y, Gielen S, Thiele H & Gummert J. (2003). Regular physical activity improves endothelial function in patients with coronary artery disease by increasing phosphorylation of endothelial nitric oxide synthase. *Circulation* **107,** 3152-3158.

Harris MH & Harris RE. (2011). *Epidemiology of Chronic Disease*. Jones & Bartlett Learning.

Harris R, Tobias M, Jeffreys M, Waldegrave K, Karlsen S & Nazroo J. (2006). Effects of self-reported racial discrimination and deprivation on Māori health and inequalities in New Zealand: cross-sectional study. *The Lancet* **367,** 2005-2009.

Henry E & Pene H. (2001). Kaupapa Maori: Locating indigenous ontology, epistemology and methodology in the academy. *Organization* **8,** 234-242.

Honkola A, Forsen T & Eriksson J. (1997). Resistance training improves the metabolic profile in individuals with type 2 diabetes. *Acta Diabetologica* **34,** 245-248.

Hu G, Tuomilehto J, Silventoinen K, Barengo N & Jousilahti P. (2004). Joint effects of physical activity, body mass index, waist circumference and waist-to-hip ratio with the risk of cardiovascular disease among middle-aged Finnish men and women. *European Heart Journal* **25,** 2212-2219.

Hunt SC, Williams RR & Barlow GK. (1986). A comparison of positive family history definitions for defining risk of future disease. *Journal of Chronic Diseases* **39,** 809-821.

Initiative NOE. (1998). *Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults*. National Heart, Lung, and Blood Institute.

Irwin K. (1994). Maori research methods and processes: An exploration. *Sites* **28,** 25-43.

Jackson R, Yee RL, Priest P, Shaw L & Beaglehole R. (1995). Trends in coronary heart disease risk factors in Auckland 1982-94. *The New Zealand Medical Journal* **108,** 451-454.

Jamurtas AZ, Koutedakis Y, Paschalis V, Tofas T, Yfanti C, Tsiokanos A, Koukoulis G, Kouretas D & Loupos D. (2004). The effects of a single bout of exercise on resting energy expenditure and respiratory exchange ratio. *European Journal of Applied Physiology* **92,** 393-398.

Jencks WP, Hyatt MR, Jetton MR, Mattingly TW & Durrum E. (1956). A study of serum lipoproteins in normal and atherosclerotic patients by paper electrophoretic techniques. *Journal of Clinical Investigation* **35,** 980.

Katch VLD, McArdle WD & Katch FI. (2010). *Essentials of Exercise Physiology [With Access Code]*. Wolters Kluwer/Lippincott Williams & Wilkins Health.

Kate LP, Boman H, Daiger SP & Motulsky AG. (1982). Familial aggregation of coronary heart disease and its relation to known genetic risk factors. *The American journal of cardiology* **50,** 945-953.

Kelley G, Kelley K & Tran ZV. (2005). Aerobic exercise, lipids and lipoproteins in overweight and obese adults: a meta-analysis of randomized controlled trials. *International Journal of Obesity* **29,** 881-893.

Kelley GA, Kelley KS & Franklin B. (2006). Aerobic exercise and lipids and lipoproteins in patients with cardiovascular disease: a meta-analysis of randomized controlled trials. *Journal of Cardiopulmonary Rehabilitation* **26,** 131.

Kelly TL, Wilson KE & Heymsfield SB. (2009). Dual energy X-Ray absorptiometry body composition reference values from NHANES. *Public Library of Science ONE* **4,** e7038.

Kerr AJ, McLachlan A, Furness S, Broad J, Riddell T, Jackson R & Wells S. (2008). The burden of modifiable cardiovascular risk factors in the coronary care unit by age, ethnicity, and socioeconomic status-PREDICT CVD-9.

King AC, Haskell WL, Young DR, Oka RK & Stefanick ML. (1995). Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50 to 65 years. *Circulation* **91,** 2596-2604.

Klein S, Allison DB, Heymsfield SB, Kelley DE, Leibel RL, Nonas C & Kahn R. (2007). Waist circumference and cardiometabolic risk: a consensus statement from shaping America's health: Association for Weight Management and Obesity Prevention; NAASO, the Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Obesity* **15,** 1061-1067.

Kodama S, Tanaka S, Saito K, Shu M, Sone Y, Onitake F, Suzuki E, Shimano H, Yamamoto S & Kondo K. (2007). Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol: a meta-analysis. *Archives of Internal Medicine* **167,** 999.

Kokkinos P & Myers J. (2010). Exercise and physical activity. *Circulation* **122,** 1637-1648.

Labarthe D. (2011). *Epidemiology and prevention of cardiovascular diseases : a global challenge*. Jones and Bartlett Publishers, Sudbury, Mass.

Larsson B, Svärdsudd K, Welin L, Wilhelmsen L, Björntorp P & Tibblin G. (1984). Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *British Medical Journal* **288,** 1401.

Lean M, Han T & Morrison C. (1995). Waist circumference as a measure for indicating need for weight management. *British Medical Journal* **311,** 158.

Lohmann TG, Roche AF & Martorell R. (1988). *Anthropometric Standardization Reference Manual*. Human Kinetics Books, Champaign, Ill.

Lopez AD, Mathers CD, Ezzati M, Jamison DT & Murray CJL. (2006). *Global burden of disease and risk factors*. Oxford University Press, USA.

Mackay J & Mensah GA. (2004). *The atlas of heart disease and stroke*. World Health Organization, Geneva.

MacKay J, Mensah GA, Mendis S, Greenlund K & Organization WH. (2004). *The Atlas of Heart Disease and Stroke*. World Health Organization.

Mancini M, Ordovas J, Riccardi G, Rubba P & Strazzullo P. (2011). *Nutritional and Metabolic Bases of Cardiovascular Disease*. Wiley.

Marrugat J, Sala J, Masiá R, Pavesi M, Sanz G, Valle V, Molina L, Serés L & Elosua R. (1998). Mortality differences between men and women following first myocardial infarction. *JAMA: the Journal of the American Medical Association* **280,** 1405-1409.

Marwick TH, Hordern MD, Miller T, Chyun DA, Bertoni AG, Blumenthal RS, Philippides G & Rocchini A. (2009). Exercise training for type 2 diabetes mellitus impact on cardiovascular risk: a scientific statement from the American Heart Association. *Circulation* **119,** 3244-3262.

Mathers C, Stevens G & Mascarenhas M. (2009). *Global health risks: mortality and burden of disease attributable to selected major risks*. World Health Organization.

McArdle WD, Katch FI & Katch VL. (2010). *Exercise Physiology: Nutrition, Energy, and Human Performance*. Wolters Kluwer Health/Lippincott Williams & Wilkins.

Mead HM & Mead SM. (2003). *Tikanga Māori: Living by Māori Values*. Huia.

Mendis S, Puska P & Norrving B. (2011). *Global atlas on cardiovascular disease prevention and control*. World Health Organization.

Merkler M & Reiner Ž. (2007). The burden of hyperlipidaemia and diabetes in cardiovascular diseases. *Fundamental & Clinical Pharmacology* **21,** 1-3.

Messerli F. (1982). Cardiovascular effects of obesity and hypertension. *The Lancet* **319,** 1165-1168.

Mhurchu CN, Blakely T, Wall J, Rodgers A, Jiang Y & Wilton J. (2007). Strategies to promote healthier food purchases: a pilot supermarket intervention study. *Public health nutrition* **10,** 608-615.

Misra A, Alappan NK, Vikram NK, Goel K, Gupta N, Mittal K, Bhatt S & Luthra K. (2008). Effect of supervised progressive resistance-exercise training protocol on insulin sensitivity, glycemia, lipids, and body composition in Asian Indians with type 2 diabetes. *Diabetes Care* **31,** 1282-1287.

Mittelmark M, Psaty BM, Rautaharju PM, Fried LP, Borhani NO, Tracy RP, Gardin JM, O'Leary DH & Kronmal R. (1993). Prevalence of Cardiovascular Diseases among Older Adults The Cardiovascular Health Study. *American Journal of Epidemiology* **137,** 311-317.

Morris J & Crawford MD. (1958). Coronary heart disease and physical activity of work. *British Medical Journal* **2,** 1485-1496.

Morrison JA, Horvitz R, Khoury P, Laskarzewski P, Gartside PS, Kelly K, Mellies M & Glueck CJ. (1980). Parental history of coronary heart disease, hypertension, diabetes, and stroke: relationship to coronary heart disease risk factor variables in their adult children. *Preventive Medicine* **9,** 773-786.

Murphy E, McAuley KA, Bell D, McLay RT, Chisholm A, Hurley R, Story G, Mann JI, Thomson R & Williams SM. (2003). A new approach to design and implement a lifestyle intervention programme to prevent type 2 diabetes in New Zealand Maori. *Asia Pacific journal of clinical nutrition* **12**.

Myers J, Prakash M, Froelicher V, Do D, Partington S & Atwood JE. (2002). Exercise capacity and mortality among men referred for exercise testing. *New England Journal of Medicine* **346,** 793-801.

Myhre K, Mjøs O, Bjørsvik G & Strømme S. (1981). Relationship of high density lipoprotein cholesterol concentration to the duration and intensity of endurance training. *Scandinavian Journal of Clinical and Laboratory Investigation* **41,** 303-309.

National Institutes of Health. (2001). Third Report of the National Cholesterol Education Program Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). *NIH publication* **1,** 3670.

New Zealand Guidelines Group. (2012). New Zealand primary care handbook 2012. New Zealand Guidelines Group, Wellington.

New Zealand Ministry of Health. (2008a). *Diabetes and Cardiovascular Disease: Quality Improvement Plan*. Ministry of Health, Wellington.

New Zealand Ministry of Health. (2008b). A Portrait of Health: Key results of the 2006/07 New Zealand Health Survey. Ministry of Health, Wellington.

New Zealand Ministry of Health. (2012a). Implementing the New Zealand hetlth strategy 2011. New Zealand Ministry of Health, Wellington.

New Zealand Ministry of Health. (2012b). Mortality and Demographic Data 2009. Ministry of Health, Wellington.

Nieman DC. (2003). *Exercise testing & prescription*. McGraw-Hill.

Nikkila E. (1953). Studies on the lipid-protein relationship in normal and pathological sera and the effect of heparin on serum lipoproteins. *Scandinavian Journal of Clinical and Laboratory Investigation* **5,** 9.

Nojima H, Watanabe H, Yamane K, Kitahara Y, Sekikawa K, Yamamoto H, Yokoyama A, Inamizu T, Asahara T & Kohno N. (2008). Effect of aerobic exercise training on oxidative stress in patients with type 2 diabetes mellitus. *Metabolism* **57,** 170-176.

Omran AR. (1971). The epidemiologic transition: a theory of the epidemiology of population change. *The Milbank Memorial Fund Quarterly* **49,** 509-538.

Paffenbarger Jr RS, Laughlin ME, Gima AS & Black RA. (1970). Work activity of longshoremen as related to death from coronary heart disease and stroke. *New England Journal of Medicine* **282,** 1109-1114.

Pate RR, Pratt M, Blair SN, Haskell WL, Macera CA, Bouchard C, Buchner D, Ettinger W, Heath GW & King AC. (1995). Physical activity and public health: a recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *Jama* **273,** 402-407.

Pedersen T. (2004). Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Atherosclerosis Supplements* **5,** 81-87.

Peiris D, Murray J, Scully D, Tilakawardene V, Hetaraka-Stevens L, Stewart T & Patel A. (2008). Cardiovascular risk management at a Māori-led Primary Health Organisation—findings from a cross-sectional audit. *Clinical Correspondence*.

Pischon T, Boeing H, Hoffmann K, Bergmann M, Schulze M, Overvad K, Van Der Schouw Y, Spencer E, Moons K & Tjønneland A. (2008). General and abdominal adiposity and risk of death in Europe. *New England Journal of Medicine* **359,** 2105-2120.

Pitama S, Robertson P, Cram F, Gillies M, Huria T & Dallas-Katoa W. (2007). Meihana model: A clinical assessment framework. *New Zealand Journal of Psychology* **36,** 118.

Pollock ML, Franklin BA, Balady GJ, Chaitman BL, Fleg JL, Fletcher B, Limacher M, Piña IL, Stein RA & Williams M. (2000). Resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescriptionan advisory from the committee on exercise, rehabilitation, and prevention, council on clinical cardiology, american heart association. *Circulation* **101,** 828-833.

Pollock ML & Vincent KR. (1996). *Resistance training for health*. President's Council on Physical Fitness and Sports.

Prabhakaran B, Dowling EA, Branch JD, Swain DP & Leutholtz BC. (1999). Effect of 14 weeks of resistance training on lipid profile and body fat percentage in premenopausal women. *British Journal of Sports Medicine* **33,** 190-195.

Pratley R, Nicklas B, Rubin M, Miller J, Smith A, Smith M, Hurley B & Goldberg A. (1994). Strength training increases resting metabolic rate and norepinephrine levels in healthy 50-to 65-yr-old men. *Journal of Applied Physiology* **76,** 133-137.

Ravussin E, Lillioja S, Knowler WC, Christin L, Freymond D, Abbott WG, Boyce V, Howard BV & Bogardus C. (1988). Reduced rate of energy expenditure as a risk factor for body-weight gain. *New England Journal of Medicine* **318,** 467-472.

Rexrode KM, Carey VJ, Hennekens CH, Walters EE, Colditz GA, Stampfer MJ, Willett WC & Manson JE. (1998). Abdominal adiposity and coronary heart disease in women. *JAMA: the Journal of the American Medical Association* **280,** 1843-1848.

Richardson P. (1990). *He Tikanga Marae*. Department Maori Studies, Massey University.

Riddell T, Jackson R, Wells L, Broad J & Bannink L. (2007). Assessing Maori/non-Maori differences in cardiovascular disease risk and risk management in routine primary care practice using web-based clinical decision support:(PREDICT CVD-2). *The New Zealand Medical Journal* **120**.

Rissanen AM & Nikkilä E. (1979). Aggregation of coronary risk factors in families of men with fatal and non-fatal coronary heart disease. *British Heart Journal* **42,** 373-380.

Rognmo Ø, Hetland E, Helgerud J, Hoff J & Slørdahl SA. (2004). High intensity aerobic interval exercise is superior to moderate intensity exercise for increasing aerobic capacity in patients with coronary artery disease. *European Journal of Cardiovascular Prevention & Rehabilitation* **11,** 216-222.

Rose G. (1964). Familial patterns in ischaemic heart disease. *British Journal of Preventive & Social Medicine* **18,** 75.

Rush E, Plank L, Chandu V, Laulu M, Simmons D, Swinburn B & Yajnik C. (2004). Body size, body composition, and fat distribution: a comparison of young New Zealand men of European, Pacific Island, and Asian Indian ethnicities.

Rush EC, Freitas I & Plank LD. (2010). Body size, body composition and fat distribution: comparative analysis of European, Maori, Pacific Island and Asian Indian adults. *British Journal of Nutrition* **102,** 632.

Sacco RL. (2001). Newer risk factors for stroke. *Neurology* **57,** S31-S34.

Sacks FM, Pfeffer MA, Moye LA, Rouleau JL, Rutherford JD, Cole TG, Brown L, Warnica JW, Arnold JMO & Wun C-C. (1996). The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. *New England Journal of Medicine* **335,** 1001-1009.

Salmond C, Crampton P & Atkinson J. (2007). *NZDep2006 index of deprivation*. Department of Public Health, University of Otago Wellington, New Zealand.

Sattar N, Gaw A, Scherbakova O, Ford I, O'Reilly DSJ, Haffner SM, Isles C, Macfarlane PW, Packard CJ & Cobbe SM. (2003). Metabolic syndrome with and without C-reactive protein as a predictor of coronary heart disease and diabetes in the West of Scotland Coronary Prevention Study. *Circulation* **108,** 414-419.

Savage PD, Brochu M, Poehlman ET & Ades PA. (2003). Reduction in obesity and coronary risk factors after high caloric exercise training in overweight coronary patients. *American Heart Journal* **146,** 317-323.

Schaefer EJ, Lamon-Fava S, Ordovas JM, Cohn S, Schaefer M, Castelli W & Wilson P. (1994). Factors associated with low and elevated plasma high density lipoprotein cholesterol and apolipoprotein AI levels in the Framingham Offspring Study. *Journal of Lipid Research* **35,** 871-882.

Segal KR, Dunaif A, Gutin B, Albu J, Nyman A & Pi-Sunyer FX. (1987). Body composition, not body weight, is related to cardiovascular disease risk factors and sex hormone levels in men. *Journal of Clinical Investigation* **80,** 1050.

Shepherd J, Cobbe S, Ford I, Isles C, Lorimer A, MacFarlane P, McKillop J & Packard C. (1995). for the West of Scotland Coronary Prevention Study Group: Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia. *New England Journal of Medicine* **333,** 1301-1307.

Sigal RJ, Kenny GP, Boulé NG, Wells GA, Prud'homme D, Fortier M, Reid RD, Tulloch H, Coyle D & Phillips P. (2007). Effects of Aerobic Training, Resistance Training, or Both on Glycemic Control in Type 2 DiabetesA Randomized Trial. *Annals of Internal Medicine* **147,** 357-369.

Sigal RJ, Kenny GP, Wasserman DH & Castaneda-Sceppa C. (2004). Physical activity/exercise and type 2 diabetes. *Diabetes Care* **27,** 2518-2539.

Siscovick DS, Laporte RE & Newman JM. (1985). The disease-specific benefits and risks of physical activity and exercise. *Public Health Reports* **100,** 180.

Smith L. (1995). Toward kaupapa Maori research. In *Mattawhanui conference (Maori teachers' conference), Massey University, New Zealand*.

Snowling NJ & Hopkins WG. (2006). Effects of Different Modes of Exercise Training on Glucose Control and Risk Factors for Complications in Type 2 Diabetic Patients A meta-analysis. *Diabetes Care* **29,** 2518-2527.

SPARC. (2005). Guidelines for promoting physical activity to adults: Movement = Health. Sport and Recreation New Zealand, Wellington.

Stefanick ML, Mackey S, Sheehan M, Ellsworth N, Haskell WL & Wood PD. (1998). Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *New England Journal of Medicine* **339,** 12-20.

Stiegler P & Cunliffe A. (2006). The role of diet and exercise for the maintenance of fat-free mass and resting metabolic rate during weight loss. *Sports Medicine* **36,** 239-262.

Stipanuk MH & Caudill MA. (2012). *Biochemical, Physiological, and Molecular Aspects of Human Nutrition*. Elsevier/Saunders.

Strasser B, Siebert U & Schobersberger W. (2010). Resistance training in the treatment of the metabolic syndrome. *Sports Medicine* **40,** 397-415.

Sukala WR, Page R, Rowlands DS, Krebs J, Lys I, Leikis M, Pearce J & Cheema BS. (2012). South Pacific Islanders resist type 2 diabetes: comparison of aerobic and resistance training. *European Journal of Applied Physiology* **112,** 317-325.

Sullivan C, Oakden J, Young J, Butcher H & Lawson R. (2003). Obstacles to Action: a study of New Zealander's physical activity and nutrition. *Profiling others oriented Wellington, SPARC*.

Sundborn G, Metcalf PA, Gentles D, Scragg RK, Schaaf D, Dyall L, Black P & Jackson R. (2008). Ethnic differences in cardiovascular disease risk factors and diabetes status for Pacific ethnic groups and Europeans in the Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand. *Clinical Correspondence*.

Tanasescu M, Leitzmann MF, Rimm EB, Willett WC, Stampfer MJ & Hu FB. (2002). Exercise type and intensity in relation to coronary heart disease in men. *JAMA: the Journal of the American Medical Association* **288,** 1994-2000.

Tauroa H, Tauroa P & Hanly G. (1986). *Te Marae: A Guide to Customs & Protocol*. Reed Methuen.

Taylor RW, Brooking L, Williams SM, Manning PJ, Sutherland WH, Coppell KJ, Tipene-Leach D, Dale KS, McAuley KA & Mann JI. (2010). Body mass index and waist circumference cutoffs to define obesity in indigenous New Zealanders. *The American Journal of Clinical Nutrition* **92,** 390-397.

Thayer JF, Yamamoto SS & Brosschot JF. (2010). The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology* **141,** 122-131.

Thompson PD. (2003). Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease. *Arteriosclerosis, Thrombosis, and Vascular Biology* **23,** 1319-1321.

Tobias M, Blakely T, Matheson D, Rasanathan K & Atkinson J. (2009). Changing trends in indigenous inequalities in mortality: lessons from New Zealand. *International Journal of Epidemiology* **38,** 1711-1722.

Tunstall-Pedoe H, Morrison C, Woodward M, Fitzpatrick B & Watt G. (1996). Sex differences in myocardial infarction and coronary deaths in the Scottish MONICA population of Glasgow 1985 to 1991 Presentation, diagnosis, treatment, and 28-day case fatality of 3991 events in men and 1551 events in women. *Circulation* **93,** 1981-1992.

Turtle CR. (2002). Lessons Learned from Failed Research Among the New Zealand Maori. *Nutritional Anthropology* **25,** 13-19.

UNAIDS. (2007). *Prevention of cardiovascular disease*. World Health Organization.

Vaccarino V, Parsons L, Every NR, Barron HV & Krumholz HM. (1999). Sex-based differences in early mortality after myocardial infarction. *New England Journal of Medicine* **341,** 217-225.

Vanlandewijck Y & Thompson W. (2011). *Handbook of Sports Medicine and Science, The Paralympic Athlete*. Wiley.

Von Eckardstein A, Nofer J-R & Assmann G. (2001). High density lipoproteins and arteriosclerosis role of cholesterol efflux and reverse cholesterol transport. *Arteriosclerosis, Thrombosis, and Vascular Biology* **21,** 13-27.

Wald NJ & Law MR. (2003). A strategy to reduce cardiovascular disease by more than 80%. *BMJ: British Medical Journal* **326,** 1419.

Warburton DE, Nicol CW & Bredin SS. (2006). Health benefits of physical activity: the evidence. *Canadian medical association journal* **174,** 801-809.

Waring SW. (2007). *Cardiovascular Risk Managemment*. Elseveir, Philadelphia.

Waring WS & Cockcroft J. (2006). *Cardiovascular Risk Management*. Elsevier Science Health Science Division.

Warnick GR, Knopp RH, Fitzpatrick V & Branson L. (1990). Estimating low-density lipoprotein cholesterol by the Friedewald equation is adequate for classifying patients on the basis of nationally recommended cutpoints. *Clinical Chemistry* **36,** 15-19.

Weibel ER. (2002). Physiology: the pitfalls of power laws. *Nature* **417,** 131-132.

Wells J & Fewtrell M. (2006). Measuring body composition. *Archives of Disease in Childhood* **91,** 612-617.

Whaley MH, Brubaker PH & Otto RM. (2006). *ACSM's Guidelines for Exercise Testing and Prescription*. Lippincott Williams & Wilkins.

Williams MA, Haskell WL, Ades PA, Amsterdam EA, Bittner V, Franklin BA, Gulanick M, Laing ST & Stewart KJ. (2007). Resistance exercise in individuals with and without cardiovascular disease: 2007 update. *Circulation* **116,** 572-584.

Williamson DL & Kirwan JP. (1997). A single bout of concentric resistance exercise increases basal metabolic rate 48 hours after exercise in healthy 59–77-year-old men. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* **52,** M352-M355.

Winett RA & Carpinelli RN. (2001). Potential health-related benefits of resistance training. *Preventive medicine* **33,** 503-513.

Winkleby MA, Jatulis DE, Frank E & Fortmann SP. (1992). Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *American Journal of Public Health* **82,** 816-820.

Wolfe RR. (2006). The underappreciated role of muscle in health and disease. *The American Journal of Clinical Nutrition* **84,** 475-482.

World Health Organization. (2007). *Prevention of Cardiovascular Disease: Pocket Guidelines for Assessment and Management of Cardiovascular Risk:(WHO/ISH Cardiovascular Risk Prediction Charts for WHO Epidemiological Sub-regions WPR A, WPR B)*. World Health Organization.

World Health Organization. (2008). 2008-2013 action plan for the global strategy for the prevention and control of noncommunicable diseases. *Geneva: WHO* **36**.

World Health Organization. (2010a). Global Recomendations on Physical Activity for Health. *Online document at: http://whqlibdocwhoint/publications/2010/9789241599979_engpdfl*.

World Health Organization. (2010b). Obesity and overweight: fact sheet no. 311. September 2006. *Online document at: www who int/mediacentre/factsheets/fs311/en/index html Accessed September* **13**.

World Health Organization. (2011a). Cardiovascular diseases: Key messages to protect heart health.

World Health Organization. (2011b). *Global status report on noncommunicable diseases 2010*. World Health Organization.

Yusuf S. (2002). MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20536 high-risk individuals: a randomised placebo-controlled trial. Commentary. *The Lancet* **360,** 7-22.

Yusuf S, Hawken S, Ôunpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL & Lisheng L. (2005). Obesity and the risk of myocardial infarction in 27 000 participants from 52 countries: a case-control study. *The Lancet* **366,** 1640-1649.

8. Appendices

Appendix A – Training Programmes

Training programme 1

Warm up – 5 minutes cycle level 4 hill setting

	Sets	Reps	Rest
Squats	3	10	
Thursters	3	10	
High pull	3	10	
Lat pull down	3	10	
Crunches	3	30s	
Oblique crunches	3	30s	

Cool down – 5 minutes on row ergometer, level 7, 50% perceived max

Training programme 2

Warm up – 5 minutes cycle level 4 hill setting

	Sets	Reps	Rest
Bench press	3	10	
Lat pull down	3	10	
Bosu ball squat	3	10	
Seated row	3	10	
Prone bridge hold	4	15s	
Bicycle crunch	4	10	

Cool down – 5 minutes on cycle ergometer, level 5 manual setting

Training programme 3

Warm up – 5 minutes cycle level 4 hill setting

	Sets	Reps	Rest
Shoulder press	3	10	
Cable bicep curl	3	10	
Deadlift	3	10	
Dead bug	3	15	
Side Bridge holds	3	30s/side	

Cool down – 5 minutes on cycle ergometer, level 5 manual setting

Training programme 4

Warm up – 5 minutes

	Sets	Reps	Rest
Bosu ball squat	3	10	
Tricep extension	3	10	
Hamstring curls	3	10	
Knee extension	3	10	
Bridge hold	3	30s	30s

Cool down – 5 minutes on row ergometer, level 7, 50% perceived max

Training programme 5

Warm up – 5 minutes cycle level 4 hill setting

	Sets	Reps	Rest
Incline bench press	3	10	
EZ bar bicep curl	3	10	
Clean and jerk	3	10	
Bench (Tricep) dips	3	10	
Lunges	3	10/leg	
Leg up crunch	3	10	

Cool down – 5 minutes on cycle ergometer, level 5 manual setting

Appendix B – Post Exercise Questionnaire

Exercise and your views

This questionnaire is part of Karl Coley's study on what exercise is best for Māori men. Your participation in this questionnaire is voluntary however it would be greatly beneficial to us if you did participate. It consists of questions regarding your views of exercise and how it does/could affect you.

Name _____

Age ___

1) How do you currently view your overall health and wellbeing?

2) In regards to the amount of time with your whānau do you find exercise has affected it?

3) Does this differ from prior to starting the resistance training intervention?

4) If yes, how so?

5) Has resistance training been beneficial to you and your whānau?

6) What is your view on resistance training? What does it mean to you and how do you think it has affected you?

7) Have you previously partaken in resistance training?

8) If you have has this time been different?

9) If yes please explain how.

Appendix C - PARQ

Human Performance Laboratory

School of Sport & Exercise
Corner University Ave & Albany Drive,
Massey University,
Palmerston North,
New Zealand

Pre-Exercise Questionnaire and Informed Consent

Name: _____ Age: _____ Sex: _____

Address: _____

1.	Have you ever had any injury, illness, back or joint injury, muscular pain that may be aggravated by vigorous exercise?	Yes/No
2.	Have you ever had: Arthritis, Asthma, Diabetes, Epilepsy, Hernia, Ulcer or Dizziness?	Yes/No
3.	Have you ever had a Heart Condition, High Blood Pressure, Stroke, High Cholesterol, Pain in the chest?	Yes/No
4.	Have any immediate family had heart problems prior to age 60?	Yes/No
5.	Are you now or have you recently been pregnant?	Yes/No
6.	Are you taking any prescribed medication?	Yes/No
7.	Have you been hospitalised recently?	Yes/No
8.	Is there any reason not mentioned above that may prevent or affect your ability to perform this test?	Yes/No

IF YOU ANSWERED YES, PLEASE PROVIDE MORE INFORMATION (TYPE, COMMENT)

_____

_____

_____

1. **I have read the information sheet on the appropriate test protocol and have had the details of the test explained to me in full. My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.**
2. **I understand that I have the right to withdraw my consent at any time and to decline to answer any particular questions.**
3. **I understand that a maximal test may be potentially hazardous to persons with cardiovascular anomalies (heart problems).**
4. **I have completed a pre-exercise safety questionnaire and have been approved as being suitably fit and healthy to take part in the fitness test.**
5. **I have read this form and I agree to participate in this test under the conditions set out in the information sheet.**

Signed: Date: / /

Witness: Date: / /

This project has been reviewed and approved by the Massey University Human Ethics Committee, PN Protocol 01/45

Appendix D – Recruitment Flyer

The Best Exercise for Māori Men

Although studies have shown that regular exercise is an effective way of reducing the risk of diabetes, little is known about the impact of exercise on the health of Māori men, or which type of exercise is most effective.

This study will look at the impact of 12 weeks of supervised exercise training on the **overall wellbeing of Māori men**. We will also investigate whether one type of exercise is more effective at **reducing the risk of diabetes in Māori men,** than another type of exercise (i.e. lifting weights versus jogging).

We are inviting 40 Māori men:

- **Aged 25 to 45 years old**
- **With a body mass index (BMI) above 25**
- **Who are not currently doing any exercise training but are interested in beginning a supervised program**

They should be:

Non-smokers

- **Not have diabetes**

If you would like to take part, or find out more about the study, please contact:

Isaac Warbrick (Te Arawa, Tainui, Ngā Puhi)
School of Sport and Exercise - Massey University
Tel: (06) 356 9099 or 0221301208
Email: I.Warbrick@massey.ac.nz

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 12/19. If you have any concerns about the conduct of this research, please contact A/Prof Hugh Morton, Chair, Massey University Human Ethics Committee: Southern A telephone 06 350 5799 x 4265, email humanethicsoutha@massey.ac.nz.

Appendix E – Information Sheet

Kia ora,

Ko Te Arawa raua ko Tainui ngā Iwi. Ko Isaac Warbrick tōku ingoa.
My name is Isaac Warbrick and I am a Māori health researcher conducting a study of how different types of exercise training (i.e. lifting weights compared to low-intensity exercise such as jogging) impact on the health of Māori men. A description of the project follows. You are invited to take part in this study but please read this information sheet carefully before deciding whether or not to participate.

What is this study about?
This study is part of a research project comparing the impact of different types of exercise programs on the health and overall wellbeing of Māori men. Specifically we will measure particular blood markers, such as insulin concentration in the blood, and participants will be randomly assigned to do a 12 week exercise program, to observe how different types of exercise training impacts on the risk of diabetes. Another part of this study includes interviews and group discussions (focus groups) where questions will be asked to understand the thoughts of Māori men toward exercise and gauge the impact of exercise on other aspects of overall wellbeing, such as time with family, outlook on life etc.
This research will contribute to the development of Māori knowledge and development, particularly when it comes to enhancing the health and wellbeing of Māori men. Participation in this study will also provide you with an opportunity to participate in a supervised exercise program and learn how exercise benefits your personal health and wellbeing.

Your Rights as a Participant in this Research
You are under no obligation to accept this invitation to participate. If you decide to participate you have the right to:

- Decline to answer any particular question
- Withdraw from the study
- Ask any questions about the study at any time during the study
- Provide information on the understanding that your name will not be used unless you give permission to the researcher;
- Invite whānau and/or other nominated support people to the interviews, testing sessions
- Be given access to a summary of the project findings when it is concluded
- Ask for the recorder to be turned off at any time during interviews.

If you have any questions about our project, please feel free to contact:

Dr Isaac Warbrick
The School of Sport and Exercise
Massey University, Palmerston North
0221301208
I.Warbrick@massey.ac.nz

The best exercise for the health of Māori men

Participant Full Information Sheet

Participation

Participants in this study will be Māori men between the ages of 25 and 45 with a body mass index (BMI) of 25 and above. Those who have diabetes (or any other metabolic disorder) will be excluded from the study. You will be fully informed of your involvement in this study and will be invited to complete a Medical History Questionnaire and a Physical Activity Readiness Questionnaire (PAR-Q) to assess your readiness to participate in the study. Following these, if you choose to participate and are clear on what is being asked of you as a participant you will be invited to sign a consent form.

During the physiological testing session, you will be asked to undergo one incremental exercise test, a measure of weight, height and waist circumference, an oral glucose tolerance test, and give two venous blood samples. This session will be conducted three times in total over a 15 week period. All testing will be performed at either the Human Performance Lab (Massey University, Palmerston North) or in a consultation room at Ngā Purapura (Ōtaki) (depending on your location as a participant).

You may be asked to take part in an interview with the researcher who will ask questions about your thoughts and feelings regarding exercise; or you may be asked to be a part of a group discussion (focus group) where similar questions will be asked (in regard to exercise). **It is possible that you may not be asked to take part in the interview or focus group as only a few participants are *randomly* selected to take part in this aspect of the study.** If you are asked to take part in this part of the study, there will be two separate sessions (once before and once after the 12 week exercise training period), neither of which should take longer than an hour. Te Kupenga o Matauranga marae (Hokowhitu, Palmerston North) will be used as the primary location for interviews and focus group discussions. However, if you prefer that interviews take place in your own home or another location of your choice, then we will accommodate these requests.

Once interviews and focus group discussions are completed, I will transcribe the interviews/group discussions and you will be sent your transcripts to check if you agree with what was recorded. If you would like to have whānau members (or any other support person) present during interviews that it is fine, and this is strictly up to you. Please be aware that you may decide not to take part or you may withdraw from participation in the project at any time and without any disadvantage to yourself of any kind.

After baseline testing, you will be randomly assigned into one of three groups and participate a 12 week training program consisting of either i) resistance exercise training, ii) endurance-type exercise training, or iii) mixed exercise training (resistance and aerobic exercise).

1. **Testing Procedures**

Body Mass/Composition: Your height and weight will be measured as well as your waist and hip circumference.

Incremental Exercise Test: The incremental exercise test will take place on a specially designed treadmill at the Human Performance Lab in Palmerston North. You will begin this test by walking on the treadmill at a slow speed. Every two minutes the gradient and speed of the treadmill will increase until you are unable to continue and choose to stop the test.
During exercise, your oxygen consumption will be measured by collected expired gases from your

mouth. This piece of equipment measures volumes of air and oxygen passing into and out of your lungs, enabling us to determine the rate at which you use oxygen (a measurement associated with your aerobic fitness). A mouthpiece will be in your mouth during the exercise test, which is similar to having a diving snorkel in your mouth.

Strength Test: The strength test will consist of a maximum lift on the bench press and a maximum lift on the leg press machine. These tests are performed to identify any changes in strength which may take place as a result of exercise training. Spotters will supervise these tests closely to ensure safety when lifting, and will stop the test if you feel uncomfortable at any time during the test.

Oral Glucose Tolerance Test and venous blood sample: Prior to this test, a resting 10ml blood sample will be taken from a vein in your arm by an experienced staff member. The sample will be frozen and later analyzed for blood lipids, glucose, C-reactive protein and insulin. The glucose tolerance test involves drinking a sugary (glucose) solution, which is then followed up with another venous blood sample 2 hours later. While it is likely that all blood will be used in the analysis, any blood that happens to remain after analysis, will be returned to the earth by our Kaumātua following due tikanga.

2. 12-week exercise training program

You will be randomly assigned to one of three different training programs consisting of either: i) resistance exercise (weight lifting etc.), ii) low-intensity cardiovascular (cardio) exercise, or iii) a combined resistance and cardiovascular exercise program. Each program will go for 12 weeks and is made up of 3 weekly exercise sessions lasting no longer than 35 minutes. All training programs will be supervised by an experienced exercise instructor/trainer who will design individual exercise sessions under the direction of Dr Warbrick. These trainers will also assist in ensuring correct technique is used and safety maintained during each exercise session. Depending on your location as a participant, exercise sessions will take place at either the Massey Recreation Center (Palmerston North) or Ngā Purapura (Ōtaki). Depending on weather, some sessions may take place outdoors.

Research Team

My research team in this project consists of an experienced group of researchers from a variety of backgrounds. Associate Professor Stephen Stannard, the head of the School of Sport and Exercise at Massey University is an expert in exercise physiology and has worked with me on a previous study where we observed the fitness and health of another group of Māori men. Dr Geoff Kira from Massey University has been involved with research looking at the health of our tamariki and taiohi. Dr Annemarie Gillies, from the School of Management at Massey University, is experienced in research aimed at the advancement of Māori lifestyles through business and entrepeneurialship. We as a research team are dedicated to establishing and maintaining kaupapa Māori views and values in the design and implementation of our research.

If you choose to participate, you will be advised of the results of the research before the overall findings are shared among Māori health providers, communities and the general academic community. Your name will not be known to others who view the results as the results will be presented without any identification of individual participants.

Data/Information Collection and Use

For confidentiality and anonymity all personal information including people's names will be removed from published material. Each participant will receive a detailed report listing their own personal results as well as the compiled results of the entire study. If you choose to participate, you may request your personal results at any time.

Potential Risks and/or Discomforts

As with any research of this nature, there are some potential risks and discomforts which you should be aware of. The researchers will attempt to minimise these through careful, consistent monitoring of your responses to the procedures employed. Every effort will be made by the researchers to ensure your safety, comfort and familiarity with the testing procedures.

1. ***Blood test and oral glucose tolerance test:*** Sampling of venous blood may cause a degree of transient discomfort and some bruising may occur at the point of insertion up to 48 hours afterwards. This discomfort is of no lasting consequence. There is also a slight risk of fainting during blood sampling. The risks and discomforts will be minimalized as the procedure will be performed under sterile conditions by highly experienced staff.

2. ***Exercise Testing:*** The incremental exercise test, though relatively easy to begin with, will be difficult as the test progresses. However, the time of exercise at high intensity is short (less than 2 minutes) and you are able to terminate the test at any time if you feel fatigued or uncomfortable. As in any physical activity, there is a very small possibility of injuries. However, the exercise protocols we'll use are commonly performed in exercise physiology labs and potential risks to participants have been minimised.

3. ***Body Size and Composition:*** There are no physical risks associated with these techniques. However, some people may feel uncomfortable having another person measure there waist and hip circumference. All possible measures will be taken to maintain an individual's privacy and you have the right to withdraw from the study if you are not comfortable with certain procedures.

Because blood will be analysed for glucose and insulin levels, it is possible that our testing may indicate that a participant already has diabetes (or another illness) which was previously unknown. If this does occur, you will be notified in person as soon as possible by Dr Warbrick, who will discuss the results and refer you to a medical professional for further consultation.

Compensation
Should you be asked to travel out of town for particular testing sessions, we will compensate your travel costs with petrol vouchers.

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

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

Summary of your Participation in this Research

Should you agree to take part in this project, you will be invited to participate in the following activities:

1) Meet with an exercise trainer/exercise assistant for three separate 30 minute sessions of moderate-level exercise per week, for 12 weeks. These sessions will be designed by a trainer who you will meet with you for each of these exercise sessions for the entire duration of the study (12 weeks).
2) Undertake three separate fitness testing sessions (twice before the twelve weeks of training and once after the 12 weeks is over). These sessions will include measurement of height, weight and waist circumference; extraction of small blood sample; a maximal fitness test which requires walking/jogging on a treadmill; and a test of strength using specially designed equipment. The entire testing procedure will take no longer than 3 hours (which can be spread over a number of days)
3) You may be invited to take part in two interviews (before and after the 12 week program) with the researcher who will ask questions to gauge your thoughts toward exercise. These will be no longer than 1 hour in duration.
4) You may be invited to take part in group discussions (focus groups) which will be geared toward exercise. These will be no longer than 1 hour in duration.

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 12/19. If you have any concerns about the conduct of this research, please contact A/Prof Hugh Morton, Chair, Massey University Human Ethics Committee: Southern A telephone 06 350 5799 x 4265, email humanethicsoutha@massey.ac.nz.