Reduction of risk for lifestyle diseases: Group diet and physical activity intervention in the workplace.

Michelle Brenda Cumin

Master of Applied Science

Auckland University Of Technology
Auckland

June, 2004

Acknowledgements

A huge thank you to each of the wonderful **men and women who volunteered** to participate in this study for their time, bodies, co-operation and feedback.

A special thanks to **Elaine Rush** and **Vishnu Chandu** who gave of their time and expertise to carry out the hundreds of measurements needed over the period of the study.

Thank you to **Professor Lynn Ferguson,** my cosupervisor for her help and encouragement and to **Dr Lindsay Plank** for all his help**.**

Thank you too to **Stephanie Schulz** for her help with the blood samples and the general flow of measuring days and to the **phlebotomists** from Medlab particularly **Linda** who did most of the blood tests.

Thank you to the **BCMRC team**, including **Manaia Laulu**, **Minaxi Patel**, **Naita Puniani**, **Vishnu Chandu**, **Amanda Jordan** and the many students, both undergraduate and post- graduate who taught me heaps along the way.

Thank you to all the **administrative staff** at AUT including **Heather Stonyer**, **Benneth Gilbert** and **Annette Tiaiti** for their help and advice**.**

Thank you to my husband **Mike** and children, **David and Tracey** for letting me study with them.

I would like to acknowledge the generous support of the **Auckland University of Technology, Science and Engineering Faculty** postgraduate student's contestable research grant and the **Zespri International** for the supply of the kiwifruit used in this study.

Last and most importantly, my thanks and appreciation to my supervisor

Associate Professor Elaine Rush PhD

for her encouragement, knowledge, help, advice, mentorship, friendship and sharing.
Without her this study would never have taken place.

" What the teacher is, is more important than what he teaches."
Karl Menninger

Abstract

Background

Cardiovascular disease is a major cause of death in most Westernised countries.The prevalence of obesity, type 2 diabetes mellitus and cancers is rapidly increasing. Older people with elevated blood lipids, obesity and DNA damage are at high risk of developing these diseases.

There is a plethora of research to support the claim that a healthy diet and increased physical activity can reduce the risk of increased body fatness, diabetes and generally improve health. However most interventions require intensive one to one advice. The aim of this study was to measure the effect of a group approach to advising on changes in lifestyle with particular attention to foods high in fibre. The study spanned a period of 12 weeks with a follow up session at 52 weeks to ascertain sustainability.

The study

This study was a 12 week longitudinal intervention study with a follow up after 52 weeks. Measurements of anthropometry (skinfolds, girths, weight and height), blood pressure, body fat by bioimpedance and fasting blood (lipids, glucose and insulin) were made at weeks 0, 3, 6, 9,1 2 and 52. The participants were asked to complete a food frequency questionnaire and a physical activity questionnaire at each of the 6 measuring sessions and to provide an indication of what the goals that they had set and if they had accomplished them after 9,12 and 52 weeks.

Between measurements at weeks 0 and 3 the volunteers were left to follow their usual food and activity pattern. Then as a group they were given a diet and exercise talk and provided with written material and pedometers to increase motivation. After measurement at week 6 they were randomly divided into two groups. The first group (A) were prescribed and provided with kiwifruit at a dose of 100g/30 kg body weight for three weeks while the second group (B) continued with the changes in diet and physical activity. Following measurement at week 9 group A abstained from kiwifruit while Group B added the kiwifruit to their diet and the measurements repeated. After 52 weeks, with only emails as ongoing communication, they were remeasured.

Results

For this multicultural, relatively middle aged group of 53 staff (28 women, 25 men) of mean age 46 years, measurable and statistically significant metabolic gains were made in the lipid profile over 12 weeks. Total cholesterol, LDL cholesterol, triglycerides and the ratio of total cholesterol to HDL all decreased and HDL increased significantly. Total cholesterol decreased from 5.6(±1.1) mean(±SD) mmol/L at baseline to 5.3(±1.1) mmol/L at week 12 ($p<0.001$); LDL cholesterol decreased from 3.5(±0.97) mmol/L at baseline to 3.3(±0.94) mmol/L at week 12 ($p<0.001$); and total cholesterol to HDL ratio decreased from 4.0(±1.1) to 3.7(±0.9) ($p<0.001$). In the 36 who were measured at 52-week follow- up these changes persisted. With the other outcome measures glucose showed a statistically but not biologically significant decrease over the 12 week period and body composition, blood pressure and insulin showed no significant change. The kiwifruit crossover had no apparent affect on the measures of any of the measurements reported. The participants reported that they increased fruit and vegetable and oily fish consumption and increased physical activity. These increases took place over the initial 12 week period and were maintained over 52 weeks.

Conclusion

This study has shown that changes in diet and physical activity can favourably influence blood biochemistry even without accompanying changes in percentage body fat and weight. Furthermore, small, manageable lifestyle changes can result in biochemical changes persisting over 52 weeks.

Table of Contents

List of Figures

List of Tables

Statement of originality

‘I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the qualification of any other degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made in the acknowledgements’.

Signed ……

Date…….

Abbreviations

BCMRC	Body Composition and Metabolic Research Centre.
BIA	bioelectrical impedance analysis
BMI	body mass index, $kg.m^{-2}$
BP	blood pressure
chol	cholesterol
CVD	cardiovascular disease
DALYs	disability adjusted life years
DASH	dietary approaches to stop hypertension
DEW-IT	diet, exercise and weight loss trial
DEXA	dual energy x-ray absorptiometry
DHA	docosahexaenoic acid
DNA	deoxyribonucleic acid
DPP	diabetes prevention program
DQID	Da Qing IGT and diabetes study
EPA	eicosapentaenoic acid
FDPS	Finnish diabetes prevention study
GI	glycaemic index
GL	glycaemic load
HDL	high density lipoproteins
HOMA	homeostasis model assessment
IGT	impaired glucose tolerance
IHD	ischaemic heart disease
ISH	International Society of Hypertension
JNC	Joint National Committee
LD	lifestyle diseases
LDL	low density lipoproteins
MOH	Ministry of Health
MRFIT	multiple risk factor intervention trial
MUAC	mid upper arm circumference
MUFA	monounsaturated fatty acids
NCD	non communicable diseases

OGTT	oral glucose tolerance test
PA	physical activity
RMANOVA	repeated measures analyses of variance
Scp/tcp	subscapular to triceps skinfold ratio
SD	standard deviation
SE	standard error
SFA	saturated fatty acids
SPARC	sport and recreation New Zealand
tot:hdl	total cholesterol to high density lipoprotein ratio
trgl	triglycerides
WHO	World Health Organisation

1 Introduction

"The goal of nutrition intervention", according to Franz *et al.* (2002), "is to assist and facilitate individual lifestyle and behaviour changes that will lead to improved metabolic control".

About 33 million deaths worldwide may have been prevented or delayed in 2000 if "metabolic control" had been improved. These are deaths attributed to non-communicable diseases, which have risk factors that can be ameliorated (World Health Organisation 2002a). Cardiovascular disease (CVD), type 2 diabetes, obesity, and cancers are major causes of death and disability in most countries (Zimmet *et al.* 1997; Lopez and Murray 1998; Ezzati *et al.* 2002). They are part of a group of disorders known as lifestyle diseases (LD), or non-communicable diseases (NCD). There is an explosion of these diseases in both developed and developing countries, and the World Health Organisation (WHO) predicts related mortality rates to rise from 60% in 2000 to 73% by 2020 (World Health Organisation 2002a).

In 1991, the WHO and the World Bank commissioned global estimates of morbidity and mortality, with diseases compared in terms of disability adjusted life years (DALYs). Murray and Lopez (1998) describe DALYs as "the sum of life years lost due to premature mortality and years lived with disability adjusted for severity". WHO and the World Bank used 1990 figures for ischaemic heart disease (IHD) and cerebrovascular disease, and ranked the global prevalence these diseases as 5th and 6th. They also calculated that, taking into account the higher risks of cardiovascular disease (which includes IHD, hypertension and cerebrovascular disease) and death among diabetic people, diabetes had risen from the 29th to the 14th most important cause of worldwide burden of disease. More alarmingly, projections to 2020 suggested that ischaemic heart disease and cerebrovascular disease will rise to the 1st and 4th place respectively (Lopez and Murray 1998). A recent study estimated the prevalence of diabetes for all age-groups worldwide to be 2.8% in 2000 and 4.4% in 2030. They projected that the total number of people with diabetes would rise from 171 million in 2000 to 366 million in 2030 (Wild *et al.* 2004).

There is controversy regarding the weighting of morbidity factors used in calculating DALYs (Tobias and Booth 2001), but diseases like type 2 diabetes, CVD, obesity and cancers are increasingly proving detrimental to the financial and emotional reserves of our aging world population and the DALYs is a useful tool to look at the health of populations. The New Zealand Ministry of Health (1999) in a 1996 report showed that the largest proportion of DALYs were attributable to CVD (24%). This was followed by cancer at 20%. Bowel cancer is currently the second most common cancer in New Zealand. The prevalence of both obesity and type 2 diabetes mellitus are also rapidly increasing in New Zealand (Ministry of Health 2003) and contribute to DALYs. A poor diet and a lack of physical activity have been highlighted as major causes of DALYs. Physical inactivity was cited in the 1996 report (ibid) as the second leading risk factor, behind smoking, and obesity, high blood cholesterol, and hypertension.

Scientific progress has helped researchers explain some of the mechanisms for development of lifestyle disease. It has also allowed them to examine whether or not changing dietary and physical activity patterns can reduce lifestyle disease risk factors. There is considerable evidence that the impact of smoking, obesity, and type 2 diabetes can be minimized by changes in lifestyle (Abramson 2001; Czernichow *et al.* 2001; Hu and Willett 2001; Kromhout 2001; Renaud 2001). Some risk factors, however, are related to genetic origins, and a new field of study, nutrigenomics, highlights nutrition as a major contributor to gene mutations and expression (WHO/FAO Expert Consultation 2003; Kaput and Rodriguez 2004). Genetics is known to be a factor in the development of CVD, type 2 diabetes, hypertension, obesity and some cancers, but the environment; diet, and lack of physical activity further amplify risk factors.

Deteriorating diets and activity patterns are driving the worldwide obesity epidemic (Popkin, 2001). Popkin reports that, particularly in third world countries, but also in developed areas, there has been an increase in the consumption of high-energy diets (particularly of saturated fats and refined carbohydrates) with a concurrent decrease in physical activity. Although it is not always easy, or viable, to change aspects of diet or physical activities, making some small permanent changes delivers long term benefits, and reduces a person's chance of developing lifestyle diseases. People with elevated blood lipids, obesity and DNA damage have a high risk of developing NCDs. If the public can be educated and motivated through lifestyle modification, to maintain a

moderate exercise level and a healthy diet, for example increasing fibre, risk factors can be reduced and the global increase in lifestyle disease slowed. The earlier healthy lifestyle habits are developed the better the outcome. It is, therefore, necessary to educate adults to make positive changes, so that they can influence their families' eating and exercise regimes from childhood.

The problem is that although there is large amount of research to support the claim that modifications in diet and physical activity throughout the lifecycle can reduce the risk of obesity, diabetes, and generally improve health the actual translation of these messages into behaviour changes and reduction of risk is fraught. There has been much research exploring the effects of lifestyle factors (diet, exercise, work environment, education) on disease and disease risk. Most have concentrated on individual diseases or individual risk factors. There is still some controversy over which risk factors are important, and which lifestyle changes work. The literature to support specific interventions was explored before the commencement of the study to ascertain which changes to advocate during the study. An in-depth review of intervention studies is in chapter four of this thesis but a general overview is provided in the following paragraphs that led to the formulation of the research questions for this thesis. The challenge is how to educate and effect change? The "translation into action" factors considered for the design of the proposed intervention were:

- individual or group delivery,
- tailoring,
- diet modifications,
- whole foods versus isolated nutrients
- diseases reported as responsible for mortality and morbidity and
- physical activity

Most active and effective interventions require intensive, and therefore expensive, one-on-one advice. One example of an intense one-on-one intervention random controlled trial is the Finnish Diabetes Prevention Study (Tuomilehto *et al.* 2001) which reduced the incidence of new diabetes by 58% in the intervention group. But in a study of the effectiveness of delivering an evidence based curriculum diabetes education programme to subjects with Type 2 diabetes, Rickheim *et al.* (2002) found that group education

could be just as effective in lowering glycated haemoglobin as individual educational sessions. Kreuter *et al.* (2000) looked at tailored health education material, and compared them to non-tailored material. They found that while print material tailored to meet an individuals needs was often more effective, in many cases non-tailored material, especially if it was carefully chosen, was also effective in effecting behaviour changes.

Epidemiologic studies have shown that eating patterns, (for example the Mediterranean diet), can affect the risk of disease. The diets are then analysed and individual foods associated with disease prevalence identified. These are then perused and tested in a clinical setting in an effort to isolate nutrients, which may then be used as supplements, in an effort to make disease risk reduction easier. Often these strategies do not work. A further example of this can be found in magnesium and calcium studies. Jee and colleagues (2002) have stated that including high levels of these minerals in your diet can help to reduce hypertension. But supplements of isolated nutrients such as minerals and antioxidants are not always effective at reducing blood pressure (Touyz 2003). It is not clear whether there is something else in the foods that are high in magnesium and calcium that is responsible for the effect. Perhaps there is even a synergy of elements within whole foods that improves the bioavailability and action of the minerals. This argument strengthens the case for a holistic and whole food approach.

In addition, it is questionable whether or not the sum of the individual risk factor changes is equal to the whole (Hoffmann 2003), and whether or not the measurement of individual factors gives a true picture of risk (Messina *et al.* 2001)? The statement that it is not good or bad foods that determine health but the diet as a whole can be further extrapolated to the fact that individual nutrients (or other food components, such as phytochemicals) are not active in isolation. They work synergistically and mega doses of one nutrient may affect the functioning of others. The measurement of the whole-food consumption is fraught with error (Riboli *et al.* 1997). Trying to get a clear picture of customary nutrient intake is extremely difficult. This is further complicated when factors like bioavailability and level of physical activity (which determines energy and nutrient needs) are taken into account. Food composition, particularly when looking at micronutrients and phytochemicals, is also not possible to measure accurately even if whole foods are accurately reported or measured. The vastly different reports on

associations of disease with individual foods, and their constituent nutrients, are testimony to the difficulties that this type of research presents. Reducing or increasing foods that contain individual nutrients though to be harmful or beneficial could make dietary intake 'unbalanced' in other nutrients, for example, a diet low in saturated fat could also mean low iron and zinc intakes as foods with saturated fats are also good sources of these minerals. While researchers need to broaden their understanding of food and /or nutrient actions at a micro level, a holistic dietary approach is needed to try and stem the tide of lifestyle disease. The nutritionists' mantra – variety, moderation and balance can also be applied to energy expenditure. In order to maintain body weight the quantity of food eaten (energy taken in) should not be greater than the amount of energy expended

Physical activity (PA) is not only a modifiable side of this energy equation but also improves the functional capacity and efficiency of the body. Increasing PA is probably easier and more affordable than some dietary changes but, like diet, is very much influenced by the opportunities available, the environment and the social culture. According to Sport and Recreation New Zealand (SPARC) combined sport and physical activity surveys from 1997 to 2001 showed that 68% of adult New Zealanders were active (2.5+ hours of physical activity per week) and 10% were sedentary (did no physical activity in the preceding 4 weeks). This subjective data also showed that reported sport and leisure time physical activity from 1997 to 2001 had increased by 3% among adults (van Aalst *et al.* 2003). These studies do not look at the amount of energy expended in the work environment, but it is known that the work environment has reduced opportunities for activity and increased requirements for sedentary activity e.g. sitting at a computer screen. So how and what to recommend as change?

Recommended changes should be evidence-based, but researchers should proceed cautiously, as the deeper we probe into lifestyle disease, the more puzzles are uncovered. Conflicting messages about types of fats that are preferable; types of fruits and vegetables that are best (largely due to glycaemic index); and supplementation with non- food nutrients (pills, potions and powders) need to be substantiated before they are hailed as cure- alls. At the same time, the evidence-based, whole-food approach combined with increased physical activity needs to be activated now. The lifestyle disease epidemic is snowballing and is in need of a preventative strategy. Whole foods,

whole diets, whole lifestyles and the environment need to be looked at. The insights of research often tend to be used in a reductionist manner, as simple remedies are sought. Even mortality rates from various diseases do not give a holistic picture. Most of the lifestyle diseases are linked and therefore cause of mortality is often misreported as, for example, cardiac arrest/CVD when the underlying problems may include type2 diabetes, obesity, hypertension or cancer. Besides, many suggested lifestyle changes require special foods, equipment, or modifications beyond the means and scope of the general public. Changes need to be doable, affordable, and easy to follow.

It was therefore decided that this intervention would be holistic and delivered in the workplace/group setting with the aim to impart practical and sustainable measures to reduce the risks for LDs. It was recognised that the problem with a holistic approach is that associations between outcomes and intervention tools are harder to measure accurately, and the exact reasons for change are hard to ascertain. But the important short-term goal for individuals is to reduce the lifestyle disease risk factors, and the long-term goal is reduction in disease incidence and disability.

A 12-week workplace-based study, with follow up after one year, took place at the Auckland University of Technology, Wellesley St campus. Healthy participants aged over 30 years, male and female representing the ethnic diversity of AUT staff were targeted. A number of strategies were trialled with the intention of using the findings to inform group intervention designs for at risk communities.

The lifestyle changes that were advocated in this study were:

- an increase in time spent in physical activity (see section **3.2**).
- changes to diet to increase fruit and vegetables and whole grains (see section **3.1.1**)
- alteration of dietary fat intake(see section **3.1.4**)
- increases in omega 3 fatty acids (mainly by increasing fatty fish consumption)(see section **3.1.3**)

These changes were mediated by

- education in a group setting
- the issuing of a “green” prescription that also includes dietary advice

- the issuing of pedometers to motivate participants to increase their daily number of steps
- regular emails with updated research, recipes and other relevant information
- mentorship from within the group

In addition, the prescription of the regular intake of a high fibre fruit (kiwifruit) previously shown to improve laxation (Rush *et al.* 2002) was examined, to determine if in the short term this could reduce cholesterol, oxidative stress, and cancer risk.
The rationale for each of these recommended changes is discussed in the proceeding chapters.

The aims of this study were to

- conduct a literature review of the pathogenesis and development of lifestyle diseases viz obesity, hypertension, cardiovascular disease, type 2 diabetes and some cancers
- review the literature on the relation between diet and physical activity and their influence on the development of lifestyle diseases
- review selected international literature on previous research and major, effective intervention studies
- review the literature to inform the development of intervention tools, the design of the proposed study and also how to measure effectiveness of the intervention.
- measure the effect of a group approach to advising about lifestyle changes
- examine the effect of the prescription of a nutrient-dense fruit (kiwifruit) as an addition to the diet on risk factors

Specifically measurements of body mass and relative fatness, blood glucose, insulin, insulin sensitivity and resistance, blood lipids, blood pressure, and oxidative stress before and after lifestyle changes were made.

It was hypothesised that a lifestyle intervention would result in an improvement in risk factors associated with body composition, dietary pattern, physical activity levels, and blood biochemistry risk factors.

2 Literature review –Lifestyle diseases

Chapter two reviews the pathogenesis and development of the "lifestyle diseases" - obesity, hypertension, cardiovascular disease, type 2 diabetes and cancer- in turn. Their prevalence, risk factors, dietary physical activity aspects and possible links to each other are explored.

2.1 Obesity

The World Health Organization (WHO) defines overweight as a Body Mass Index (BMI) between 25.0 kg/m^2 and 29.9 kg/m^2 and obesity as a BMI of 30.0 kg/m^2 or greater. BMI is the weight of a person divided by their height squared (kg/m^2) and is useful as an indicator of degree of body fatness in populations. Different BMI cut off values for different ethnic populations would be better action points for risk definition for overweight and obese participants (WHO Expert Consultation 2004). The WHO regional office for the Western Pacific has suggested overweight (high risk) BMI values as being 30-32 kg/m^2 for Maori and Pacific Islanders and 22-25 kg/m^2 or Asian Indians. It suggests that the BMIs for obesity (very high risk) should be over 32kg/m^2 for Maori and Pacific Islanders and greater than 25 kg/m^2 for Asian Indians (Bell *et al.* 2001; WHO 2004; WHO Expert Consultation 2004).

BMI values and cut offs are useful tools for estimates of overweight and obese populations but are less useful in determining risk of other lifestyle diseases as a result of obesity. BMI gives an idea of size and in some populations this is an indication of body fatness but measurement of body fat mass and where the fat is situated give better indications of the risk of developing diseases like type 2 diabetes, cancer and cardiovascular disease (Despres 2001).

Obesity, an excess of body fat, can be simply viewed as a consequence of having too much energy being taken in (in the way of food) compared to energy expenditure (including energy used in physical activity). Reduction in total energy intake (kilojoules) and at the same time an increase in physical activity will therefore help to reduce obesity and the risk of the other lifestyle diseases. Obesity is increasing in

prevalence in developed and developing nations and is a problem across all age groups. There are more and more children who are obese and this has psychological, social and short and long-term health implications.

Not only how much fat there is but also where the excess body fat is located on the body affects risk. Excess visceral fat (around the organs), also known as abdominal fat, places individuals at higher risk of lifestyle diseases (Despres 2001). Total body fat can inexpensively and quickly assessed in several ways including Bioelectrical Impedance Analysis (BIA)(see Measurements **4.2.2**) and using the calculations of Durnin and Womersley (1974) that use four skinfolds. These are biceps, triceps, suprailiac and subscapular (Durnin and Womersley 1974). Distribution can be assessed by waist measurements and skinfold ratios (**4.2.1**). Techniques like Dual Energy X-ray Absorptiometry (DEXA), magnetic resonance imaging (MRI) or computer axial tomography (CAT) scans are more accurate measures of both body composition and fat distribution but are more expensive and less accessible than BIA and anthropometry, and were beyond the means of this study.

The prevalence of obesity has increased by between 10 and 50 percent in the majority of European countries in the last decade and currently affects about 77 percent of urban Western Samoan males (Brown and International Obesity Task force 2003). The prevalence of obesity in New Zealand is following the trend in developed countries and needs to be addressed. In 1991 New Zealand incurred costs of approximately $135 million through obesity and obesity-related disorders. This equated to 2.5% of total health expenditure per year (Swinburn, Ashton *et al.* 1997), and in 2003 obesity related illnesses were estimated by the Ministry of Health to cost the health care system $303 million each year (Ministry of Health 2003).

The main areas for an individual to be able to modify energy balance are by changing energy intake or energy expenditure or both. It is difficult to get accurate data on energy intake and expenditure in free-living people. Study participants, particularly those who have high BMIs tend to under report dietary intake and over report physical activity (Kretsch *et al.* 1999; Goris *et al.* 2000; Washburn *et al.* 2003). Obesity needs to be prevented rather than treated and as such issues like increasing physical activity and reducing portion sizes and macronutrient density of the diet need to be looked at in

healthy populations as well as those who are already overweight. Effecting and measuring changes is more important than trying to measure absolute energy intake and output.

Energy intake can be decreased by a reduction in fat – particularly saturated fats, increase in fruit and vegetable and whole grain consumption. Portion sizes also need to be moderated to reduce energy intake. There is some debate about how much physical activity needs to be increased to be effective. In order to lose weight, the amount and intensity of physical activity needs to increase energy expenditure above intake. For optimal health, at least 30 minutes a day of moderate intensity e.g. brisk walking activity is needed. It is even more beneficial if the exercise is more vigorous e.g. running, lifting heavy loads or undertaken for a longer period of time (World Health Organisation 2003). Several studies have shown that any increase (time or intensity) in physical activity is beneficial to health (Brown *et al.* 2003; Brach *et al.* 2004). A steady increase in physical activity and a reduction in sedentary time, that can be maintained and built on, is probably more beneficial than a large increase that is not sustainable.

Evidence for the importance of reduction of sedentary time has been provided in a number of large, epidemiological studies. Over a six-year period, the Nurses Health Study showed that sedentary activities, for example watching TV, were associated with an increase in the risk of developing obesity and type 2 diabetes (Hu *et al.* 2003). In a meta-analysis of US weight loss studies, Anderson found that six studies included found that over a prolonged period, those who reduce weight by combining diet and physical activity are significantly better at maintaining their weight loss after between 2 and 3.3 years than those who exercised less. About 27 % of those who lost weight in the low exercise group maintained their weight loss compared with about 54% in the group that had a high physical activity level (Anderson *et al.* 2001).

Obesity is associated with many of the lifestyle diseases that are hugely increasing the burden on society including type 2 diabetes, cardiovascular disease, hypertension, some cancers, osteoporosis, hip and knee problems (Visscher and Seidell 2001). Manson and Spelsberg (1994) estimated that obesity contributes up to 75% to the risk of type 2 diabetes. The reason why obesity contributes to many diseases may be because of the extra strain the fat puts on the body's metabolism. It may also be partly due to the fact

that obese/overweight individuals tend to have a high fat, high sugar, hypercaloric diet and a sedentary lifestyle.

Obesity may have many origins – lifestyle, genetic, environmental, and metabolic. The environmental and lifestyle causes can be changed in the short to medium term. Genetic and metabolic factors may be able to be changed with evolution or possible genetic engineering in the future (Kaput and Rodriguez 2004).

There have been many studies done on ways to reduce body weight in the obese and overweight. The cornerstones of most of these (and the many fad diets suggested for this purpose) are hypocaloric diets and /or increases in physical activity. What foods supply the energy and how much physical activity is required are where most of the debate lies. Most dietary opinion lies in one of two camps. Some favour a diet that supplies most of its energy from carbohydrates (50-70%), less from fats (25-30%) and the rest from protein. Some favour a diet that supplies more energy from protein and less from carbohydrate. The major health agencies and public health bodies in most developed countries suggest a diet that has 45-55 % energy from carbohydrate, less than 30% from fat and the rest from protein. The diets that have a higher carbohydrate, lower protein component are less expensive, satisfying (because they have more food for the same amount of calories), and include plentiful fruits and vegetables which may have a protective effect for lifestyle diseases. The large amount of carbohydrate may, however, lead to an increase in plasma triglycerides, which may be a risk factor for CVD (Avins and Neuhaus 2000). The higher protein diets are more expensive, may be high in fats (particularly saturated fats and cholesterol which are found in foods of animal origin) and tend to be low in fruit and vegetables.

Long term prevention of obesity will be the most effective strategy but there are billions of people worldwide who are already obese or overweight. How to enable them to loose weight and, just as importantly, maintain the weight loss, is another challenge for the public health sector.

2.2 Hypertension

Blood pressure (BP) is a measure of the force exerted on blood vessels as blood is pumped from the heart around the body. It is measured in millimetres of mercury (mmHg). The higher number is known as systolic pressure and occurs as the heart contracts. The lower number (diastolic pressure) is the pressure remaining when the heart is relaxed. Blood pressure fluctuates throughout the day so it is prudent to measure it at a similar time to compare measurements. It is important that the subject is relaxed, comfortably warm, has been sitting or lying for at least 5 minutes and that the cuff is applied correctly. Chronically raised blood pressure at rest means the heart must work harder and blood flow to organs is less efficient. Systolic blood pressure is thought to be a better measure of risk than diastolic. Normal blood pressure has been defined and redefined and opinions on optimal and/or normal levels varies.
The Joint National Committee (1993) classified hypertension as follows:

	Systolic (mmHg)	Diastolic (mmHg)
Normal(including optimal)	<130	<85
High normal	130-139	85-89
Hypertension stage I	140-159	90-99
Hypertension stage II-IV	>160	>100

According to the 1999 World Health Organisation/International Society of Hypertension (WHO/ISH) guidelines optimal blood pressure is 120/80 mmHg (WHO/ISH Guidelines Subcommittee 1999). These guidelines also suggest that a 5mmHg reduction in diastolic blood pressure can result in a 35-40% reduction in stroke risk. Optimal blood pressure (at a population level) has recently been defined by Freitag and Vasan (2003) as having a systolic of 115 mmHg and a diastolic reading of 75mmmHg. In a World Health Organisation report (2002) entitled “Reducing risks, promoting healthy life”, blood pressure was defined by systolic blood pressure and a value of 115 (SD6) mmHg was given as one that would give the lowest risk of adverse health outcomes. This report also estimated that each 10mmHg reduction in systolic blood pressure resulted in a 37% lower stroke risk and a 25% lower Ischaemic Heart

Disease (IHD) risk. Risk of hypertensive disease and other cardiac diseases was also decreased with a 10mmHg drop in systolic BP. In confirmation of this recommendation the seventh report of the Joint National Committee (JNC) on prevention, evaluation and treatment of high blood pressure, stated that a systolic BP greater than 140mmHg in those over 50 years of age was more indicative of CVD risk than diastolic BP (Chobanian *et al.* 2003).

It is uncertain what associations hypertension has with diet. Primary hypertension may have genetic origins whereas secondary hypertension is thought to be linked to diet, lack of physical activity and stress. Obesity and atherosclerosis can also b e contributing factors to hypertension. Sodium is a mineral often implicated in hypertension (He and Whelton 1997) Sodium is found naturally in many foods and also in most of the pre-prepared and snack foods consumed liberally in developed countries. A recommendation to not add salt (sodium chloride) during or after cooking foods would seem sensible advice. The only advantage of added salt added to food is in populations where iodine deficiency is a potential problem and iodised salt is used. In New Zealand this could be a problem but options of adding iodised salt rather than plain salt to some foods, example bread, are currently being explored (Thompson 2003). There are issues of quantities consumed when nutrients (like iodine) are added to freely available, commercially produced foods (personal communication with Associate Professor Christine Thompson, Otago University) but these are currently being researched and addressed. The Dietary Approaches to Stop Hypertension (DASH) trial compared both the effects of two types of diets and different levels of sodium intake on hypertension. Reducing sodium reduced the systolic blood pressure but the effect was determined by the amount of sodium in the diet prior to the study, the amount it was reduced by and the type of total diet the participants followed (Sacks *et al.* 2001)

Increased dietary potassium may be a factor in preventing hypertension (Whelton *et al.* 1997). Fruit and vegetables are the major contributing foods to potassium in most diets, so, increasing fruit and vegetable consumption may help to lower risk of hypertension. This was also reported in the DASH trial (Sacks *et al.* 2001; Vollmer *et al.* 2001, see section **4.2.1**.).

2.3 Cardiovascular disease

Cardiovascular disease (CVD) in this section includes ischaemic heart disease (IHD), atherosclerosis and cerebrovascular disease. Hypertension is discussed above. Cardiovascular disease is a major killer in both developed and developing nations. It is, however, sometimes blamed for mortality where other diseases, for example type 2 diabetes, has been the underlying cause (Mann 2002).

The incidence of CVD has increased in developing countries over the last 10 years but there has been a trend, in developed countries (including New Zealand), for CVD morbidity and mortality rates to decrease over the last decade. This is thought to be due to the public health campaigns to reduce saturated fat intake, increase dietary fibre and increase physical activity. Better medication, treatment methods and resuscitation methods have also led to a decrease in mortality from CVD. With the increase, and projected explosion, of type 2 diabetes it is expected that this trend will be reversed in the near future (Mann 2002). In 1999 CVD was listed as the primary cause of about 41% of deaths in New Zealand (Ministry of Health 2003).

The major modifiable risks for CVD are thought to be smoking, hypertension, high serum cholesterol, diabetes, obesity, physical activity and diet (Franz 1998; Hooper *et al.* 2001; Hu *et al.* 2001; Kromhout 2001; Keevil *et al.* 2002; Tanasescu *et al.* 2002; Cernea *et al.* 2003; Ministry of Health 2003). Evidence that these factors influence the path of CVD is fairly conclusive but is confounded by genetic and environmental influences. How to measure the degree of change of each factor and the levels required for positive outcomes are also not clear.

The influence of factors, such as triglyceride levels and the type and intensity of dietary and physical activity interventions needed to reduce risk is also confusing (Ginsberg 1997; Avins and Neuhaus 2000; Saris *et al.* 2003) Differences between protective and harmful influences further complicate the ideal path to follow. The more reductionist the research becomes the harder it is to give clear, uncomplicated guidelines for risk reduction. For example, it is now known that some fats are protective and others increase CVD risk. This is further complicated by the fact that too much fat can result in

too high an energy intake which, even if we are eating 'protective' fats can lead to obesity, which also increases risk.

Blood lipid levels and the foods that influence these levels have also caused many debates. High serum cholesterol levels (above 5 mmol/L) are thought to increase risk of CVD but there is some question as to how low the levels should be to decrease risk. Furthermore, the quantity and proportions of serum lipid fractions such as low density lipoproteins (LDL) and high density lipoproteins (HDL) confound the picture. Are the levels of serum lipids that are regarded as' healthy 'the levels to aim for? Law *et al.* (2002) suggest that they are not. They suggest that reducing lipid levels as much as possible is more beneficial than "normalising" them. If they are right, then looking at risk factors like plasma lipid levels to decide whether changes need to be made is not adequate. They propose that "Interventions to change the risk factors should be determined by a person's level of risk, not by the level of the risk factors (Law and Wald 2002). A study done by the same authors in 1994 following the ischemic heart disease morbidity and mortality outcomes in 21 515 men who had total blood cholesterol levels measured, found that a difference in total cholesterol concentrations of 0.6 mmol/L was associated with a 24% difference in death from IHD. There was a 27% difference in death from IHD with a 0.6mmol/l drop in LDL cholesterol levels (Law *et al.* 1994).

Further questions about lowering risk of CVD need to be answered. Are foods that are high in cholesterol the ones to avoid/reduce or should we be concentrating on those high in saturated fats? Cardiovascular disease has been associated with increased saturated fatty acid consumption for about 50 years and the evidence that a diet low in saturated fat decreases CVD risk is very strong. The Mauritius experience, where in 1987 the government of Mauritius stopped the use of palm oil (high in saturated fatty acids) and made only soya bean oil (high in unsaturated fatty acids) available, is an example of this. Palm oil had been the oil predominantly used for cooking. From 1987 to 1992 plasma total cholesterol concentrations fell by 0.79 mmol/l ($P < 0.001$) in men and 0.82 mmol/l ($P < 0.001$) in women (Uusitalo *et al.* 1996). Should we be looking at increasing the protective factors (such as HDL cholesterol and omega-3 fatty acids) or at decreasing the risk factors (like LDL cholesterol and total cholesterol or both and what dietary factors influence plasma lipids and CVD risk? In a summary of the scientific conference of dietary fatty acids and cardiovascular health (2001) the nutrition

committee of the American Heart Foundation concluded that our understanding of the effects of different fatty acids on CVD risk factors is not clear. Risk factors for CVD related to fatty acid intake may include impaired platelet function, high blood pressure, endothelial dysfunction and increased blood clotting (Kris-Etherton *et al.* 2001). The role of different dietary fats is discussed further in chapter 3. Most importantly, the question is, how do we make beneficial changes in terms of the foods we eat? Current information about which fats to eat and which to reduce and the quantities which may or may not be related to total cholesterol levels is confusing and needs to be clarified in terms of whole foods and total diet.

Rather than just total cholesterol elevated plasma trigycerides (above 2.0mmol/L) and a total cholesterol to HDL ratio of above 4.5 have also been implicated as risk factors. Triglycerides have been shown to go up when fat is replaced by carbohydrate. This tends to occur when the diet becomes very low in fat (<15% energy) and so carbohydrates increase to above 70% of energy to compensate. A diet that is < 30% fat, 15-20% protein and around 50% carbohydrate should not cause plasma triglycerides to rise (Parks and Hellerstein 2000). High protein diets such as the popular Aitkin's diet have been advocated in some studies to reduce weight. This dietary regime requires a very low intake of fruit, vegetables and other carbohydrate sources like whole grains, which have been shown to be protective against obesity, CVD and some cancers (Howe GR, Benito E *et al.* 1992; World Cancer Research Fund and American Institute for Cancer Research 1997). Furthermore, it is difficult to increase the protein content of the diet without increasing the fat content and often the fat increased is saturated fat. Increased fish consumption and using legumes including soy as an alternative protein source could partially solve this dilemma.

Several studies have shown that an increase in fruit and vegetables is associated with a decrease in risk for cardiovascular disease (Rimm *et al.* 1996; Lampe 1999; John *et al.* 2002). The amount of sugary fruit and starchy vegetables (tubers, carrots, beetroot, green peas, onions, pumpkin) needs to be moderated so that the total carbohydrate consumption does not cause serum triglycerides to rise (Bantle *et al.* 2000; Kanazawa *et al.* 2003). Increasing dietary fibre, particularly soluble fibre (**3.1.3**) is thought to decrease total and LDL cholesterol levels (Brown *et al.* 1999; Story 2003).

The Framingham heart study (**4.1**) in which started in 1948 was used as a basis for developing a risk score for developing CVD in 1991. An individual's score is calculated using age, blood cholesterol (or LDL cholesterol), HDL cholesterol, blood pressure, cigarette smoking, and prevalence of diabetes mellitus and this score can then be used to estimate relative risk of developing CVD. Each sex has different cut off points for the factors used (National Heart Lung and Blood Institute 2000). A study in 2003 found that the Framingham risk formula may over estimate the risk of CVD mortality by up 47% and non-fatal coronary events by up to 57% (Brindle *et al.* 2003), but it is popular and very widely used by clinicians as the stimulus to advocate lifestyle changes to reduce these risks of CVD.

The most powerful evidence from studies looking at preventing and treating CVD supports the following modifications to diet:

- Changing saturated and trans-fatty acids to poly- and mono unsaturated fatty acids.
- Increasing intake of omega-3 fatty acids (from oily fish or plant sources)
- Eating a diet that is high in vegetables, fruits, nuts and whole grains and low in refined grains.

2.4 Type 2 Diabetes Mellitus

Type 2 diabetes mellitus is a progressive, chronic disease that, together with its complications, leaves huge economic, social and emotional costs in its wake. It develops when either the pancreas does not produce enough insulin to metabolise glucose obtained from food, or the body cells are resistant to the insulin's action or both. Initially the pancreas may produce too much insulin to try to compensate for the elevated blood glucose levels, but as the disease progresses the β cells of the pancreas begin to fail and plasma insulin levels drop.

Type 2 diabetes accounts for about 90% of the incidence of diabetes mellitus world-wide (World Health Organisation 2002). It affects a wide range of people from all ethnic, social and economic groups. Type 2 diabetes mellitus costs the American public about US$ 132 billion per year in 2002 (Hogan *et al.* 2003). Furthermore, these costs

are on the increase. The American Diabetic Association estimated the cost at US$98 billion per annum in 1997.That is an increase of about US$ 34 billion in 5 years. Type 2 diabetes mellitus is estimated to cost New Zealand in excess of NZ$240 million per year (Ministry of Health 2001). Costs will increase both with an increase in medical costs per se and an increase in the number of diabetics. The prevalence of type 2 diabetes has increased exponentially over the last few decades. In 1995 there were an estimated 135 million diabetics worldwide. It is estimated that the incidence of type 2 diabetes mellitus is will reach 220 million worldwide by 2010 (Amos *et al.* 1997). At current rates, there are expected to be 300 million in 2025 (King *et al.* 1998). Wild *et al.* (2004) estimates the number in 2030 to exceed 366 million. The World Health Organisation (2002a) estimates that there are 200 million people worldwide with impaired glucose tolerance (IGT, an inability to metabolise glucose) and that over half of these will develop type 2 diabetes within a 10 year period. IGT also increases the risk of cardiovascular disease (CVD) and those with IGT are 34% more likely to die from CVD (World Health Organisation 2002). A frightening trend in the prevalence of type 2 diabetes is the increasing incidence of the disease in younger adults, adolescents and now many children (Rosenbloom *et al.* 1999; Ehtisham *et al.* 2000; Silverstein and Rosenbloom 2001).

The complications from diabetes don't only contribute highly to mortality (death) but also to the damage or failure of most body organs. Blindness from diabetic retinopathy, renal failure from damage caused to kidneys and amputations due to peripheral vascular damage aggravated by damage to nerves are three of the possible costly results from type 2 diabetes. Type 2 diabetics are also likely to contribute to costs related to cardiovascular disease, hypertension and obesity. People who are diagnosed with type 2 diabetes mellitus often already have macro and microvascular complications and damage but have not exhibited any symptoms (Nichols *et al.* 2000). Intervention at an earlier stage (ie IGT or before) may prevent some of the damage.

It is thought that the number of diagnosed type 2 diabetics is the tip of the iceberg. There are many that either have raised plasma insulin suggesting insulin resistance or IGT or have not yet been diagnosed. This means that we either need to be doing more generalised screening or sending out more global messages as to how to reduce the risks. This would also help reduce the complications, and the societal burden of those

complications, because the lifestyle changes needed to reduce risk mirror those needed for better diabetic control in many instances. A recent protocol developed for screening the Australian population for type 2 diabetes suggested that a fasting plasma glucose of less than 5.5 mmol/L was unlikely to be indicative of a potential diabetes occurrence and that those with this level should be rescreened after 5 years. It also suggested that those with fasting plasma glucose levels between 5.5 and 6.9 mmol/L be given an oral glucose tolerance test (OGTT) to screen for diabetes. (Colagiuri *et al.* 2004). This is below the previous recommendation of 6.0 mmol/L. Deaths from diabetes are often underreported, as often the cause of death is one of the complications of the diabetes rather than the disease itself. The economic and social costs of the complications (including blindness, amputations, cardiovascular disease) are huge. The potential of developing type 2 diabetes probably has genetic origins (Rosenbloom 2003) but there is strong evidence to suggest that the expression of the disease is related to lifestyle.

There is cogent evidence that excess adiposity, particularly if it is centrally situated, is linked to type 2 diabetes (Eriksson and Lundgarde 1991; Manson and Spelsberg 1994; Edelstein *et al.* 1997; Despres *et al.* 2000; Moore *et al.* 2000; Bell *et al.* 2001; Despres 2001; 2003). Preventing overweight and obesity is therefore, important in trying to reduce risk of this disease.

Increased physical activity can contribute to decreased rates of overweight and obesity and several studies have shown that, even in the non-obese, an increase in physical activity can reduce diabetes risk (Hu *et al.* 1999; Kriska *et al.* 2001; Wang *et al.* 2002; Kriska *et al.* 2003).

A diet high in saturated fats has been strongly linked to increased risk of IGT, higher plasma glucose and insulin levels and lower insulin sensitivity. These are all indicators of an elevated risk of type 2 diabetes. Furthermore, a diet high in unsaturated fatty acids, including monounsaturated fatty acids, polyunsaturated fatty acids and/or omega-3 fatty acids, has been shown to reduce risk of type 2 diabetes and increase insulin sensitivity (Uusitupa *et al.* 1994)

Data from the Nurses Health Study (**4**.**1**) reported on by Salmeron *et al.* (2001), showed that the consumption of trans- fatty acids increased risk of type 2 diabetes while

polyunsaturated fatty acids were related to decreased risk. Total fat, monounsaturated fat and saturated fat did not have any association with risk of type 2 diabetes. This finding may have been confounded by the contributions of other risk factors. For example, participants in this study who had higher consumption of trans- fats also smoke more, consumed less alcohol and folate and were less physically active.

Dietary fibre is another factor that has been linked to type 2-diabetes prevention. The definition of dietary fibre is not universal. There are many different fibre constituents and a broad spectrum of physiological effects that have been linked to fibre intake. The results from the Nurses Health Study (4.1) (Salmeron *et al.* 1997) suggested that there was an increased risk of type 2 diabetes in women who consumed diets with a high glycaemic load (GL) and a low cereal fibre content. Glycaemic load is the product of glycaemic index (GI) and total available carbohydrate. Glycaemic index is a ranking of carbohydrates based on their immediate effect on blood glucose levels. Foods that are high in unrefined grains tend to have a low GI and may reduce risk of type 2 diabetes. This study also found that fruit and vegetable intake was not related to type 2 diabetes risk.

A report on data from the same Nurses study by Michels *et al.* (2003) found that there was a small but significant increase in the risk of breast cancer in women who had type 2 diabetes.

2.5 Cancer

Cancer is not one but many different diseases. They affect different parts of the body and have different causes. The diseases are characterised by body cells that have become genetically and metabolically abnormal and are out of control.

In 2000 globally cancer was responsible for 5.3 million deaths in men and 4.7 million deaths in women and it is estimated that the rate of new cancer cases will be 15 million by 2020 (Stewart and Kleihues 2003). In the developed world only deaths from cardiovascular disease outnumber deaths from cancer and about a third of cancers are thought to be due to lifestyle factors excluding tobacco (Key T, Allen N *et al.* 2002).

In New Zealand in 1999 the incidence of new diagnoses of cancer was about 16 700 and mortality about 7600. Cancers accounted for 27.2% of deaths from all causes and was the leading cause of death (Cancer Society of New Zealand, 2003). In women about one third of these deaths are from breast cancer (Statistics New Zealand, 2000).

In a summary of the evidence of dietary factors as a risk for cancer by Key *et al.* (2002) report that environmental rather than genetic factors are emphasized as increasing cancer risk. This interpretation is based on studies that show that people who migrate from one country to another generally suffer cancers at the same rate as citizens of their adopted country. What we eat may be important in either promoting the development of certain cancers or helping protect against them.

Major dietary influences that increase the risk of cancers are alcohol (in the case of cancers of the oral cavity, pharynx, larynx, oesophagus, liver and possibly breast) and obesity (in the case of cancers of the oesophagus, colorectum, breast, endometrium and kidney). Fruit and vegetables are determined to be protective against some cancers especially those of the gastrointestinal tract (World Cancer Research Fund and American Institute for Cancer Research 1997). Protection could be due to flavanoids and other phytochemicals and may be related to the fibre content of these foods. Cereals and wholegrain products may also be protective. The positive effects of diets high in fruit, vegetables and cereals may also be due to the fact that these diets are also usually low in fat and that the people who eat them are less likely to be obese and more likely to do adequate physical activity.

Dietary fats are thought to play a role in some cancers. In experimental animal studies, omega- 3 fatty acids have been shown to reduce the risk and progression of breast and prostate cancers (Ip 1997). In a review of intakes of fish and marine fatty acids and the risk of hormone- related cancers, including breast and prostate cancers, Terry *et al.* (2003) reported that the evidence of marine fatty acids reducing the risk of these cancers was unclear. They concluded that, despite these fatty acids inhibiting breast and prostate cancer cell proliferation in vitro, the association between consumption of these fatty acids and a reduction in cancer risk in humans was not conclusive.

Young and Leu (2002) looked at the three ways of preventing the most prolific types of cancers in Australia and summarised the most well known dietary associations with each type of cancer. They sited large quantities of red meat (>100-140g/day) and alcohol (>60g/day), as well as a diet high in total fat, saturated fat, refined carbohydrates and energy as factors that could increase the incidence of colorectal cancers. Charred red meat and processed meats may also increase colorectal cancer risk. Diets that had plentiful amounts of fruit and vegetables, dietary fibre, antioxidant vitamins and carotenoids as well as lifestyles that included regular physical activity were protective against colorectal cancers. A diet high in fat, particularly animal fat, and red meat was also implicated in increased risk of prostate and breast cancers. Milk and dairy product consumption in large quantities may increase prostate cancer risk while calcium might be protective against colorectal cancers. Alcohol probably increases the risk of breast cancer. Increased fruit and vegetable consumption may decrease prostate and breast cancer risk. This is more likely to be from their flavanoid content rather than their fibre content. Increased fibre and physical activity may be protective against breast cancer.

In New Zealand the incidence of colorectal cancer in Maori and Pacific Islanders is lower than in Europeans. This brings into question the role of weight and dietary consumption of fats and sugars in the etiology of this form of cancer as intakes of refined sugars and fats tends to be higher in these populations (Ministry of Health 2003). Maori and Pacific Islanders also tend to be more overweight and obese although they have less fat mass for the same BMIs as Europeans (Swinburn *et al.* 1996). Ferguson *et al.* (1995) found that the types of plant foods eaten by Maori and Pacific Islanders were different to those eaten by Europeans. The differences in the plant foods eaten translated to differences in the types of fibre and also differences in the potentially anti-carcinogenic chemicals found in them. They recommended that the types of fruits, vegetables and cereals consumed and associated with reduced incidence of cancer should be analysed further.

Physical activity reduces the risk of obesity and may protect against breast and colon cancers. Physically active men and women have been shown to experience about half the risk of colon cancer as those that are sedentary (Batty 2000). Friedenreich (2001) looked at 51 studies conducted on colon and colorectal cancer and found that 43 of them

demonstrated a reduction in cancer risk among the most physically active participants of both genders. Some studies have shown reduced risk for prostate, lung, testicular and endometrial cancers with regular physical activity. Physical activity is also increasingly prescribed for the treatment and rehabilitation of patients with cancer (Batty 2000).

2.6 Summary

This chapter has looked at the definitions, prevalence, future predictions, and links to each other of the major lifestyle diseases. It has also looked at some of the studies done and risk factors associated with these diseases. The definitions of the diseases and what constitutes risk varies but the fact that the incidence and prevalence of lifestyle diseases are increasing and there is an urgent need to stem this is unequivocal. The challenge is how to do this in an effective, informed way with limited funding for maximum impact. Chapter three looks at some of the dietary and physical activity studies and what can be used as evidence based information for this study.

3 Literature review – Diet and Physical activity: Relationships to the of development of lifestyle diseases

Interventions to reduce the incidence and prevalence of lifestyle disease include dietary changes, physical activity changes, medication and surgical procedures. Medication and surgical procedures have huge economic costs and also have potential side effects. They are not feasible on a large scale. On the other hand lifestyle changes are difficult to implement and sustain. Often poor dietary and physical activity habits are partly to blame for the incidence of lifestyle disease and rectifying these can reduce the risk of more than one disease with the same strategies. Deciding what changes could be most effective, communicating these to the populations at risk and making the messages understandable and practical to put into sustainable practice are the challenges researchers and practitioners face. This chapter looks at dietary and physical activity changes/messages that could possibly be used in a lifestyle intervention to reduce the risk of LDs targeted in this study. Diet, physical activity changes and their effect on health are discussed first, then the synergistic effects in the treatment of specific lifestyle diseases and finally the diet and physical activity messages that arose from this evidence base.

3.1 Diet

It is well known that dietary intake can contribute to or reduce the risk of lifestyle diseases (Abramson *et al* 2001; Hu *et al* 2001; Kromhout 2001; Renaud 2001). According to the American Dietetic Association position paper on communicating food and nutrition information, barriers to improving diet include “reluctance to give up favourite foods”(44%), “satisfaction with current diet”(39%), “lack of time to keep track of diet”(29%) and “lack of understanding of nutrition guidelines”(29%). Many surveyed (77%) believed that foods could be classified into “good foods” or “bad foods” (Freeland-Graves and Nitzke Graves, 2002). An understanding that all foods can be part of a healthy lifestyle -depending on how they contribute to a total diet may help to address some of these obstacles.

All foods have components that add to our health and excess of most nutrients, whether they are traditionally considered as “good”, for example some of the vitamins or “bad”, for example saturated fat, could be unhealthy. Moderate amounts (appropriate portion sizes) of foods that contain these nutrients could be part of a “balanced” dietary intake. Another confusing idea sometimes created by dietary guidance is the division of nutrient intakes into meals or days. Averaging nutrient intake over several days as opposed to single meals or foods is a more appropriate way to assess intake.

No one food or nutrient is a ‘magic bullet” and individuals needs differ. Variety is an important part of ensuring adequate nutrient intake. How a food or meal contributes to the total diet and the balance between foods that are thought to reduce risk of lifestyle disease and those that are convenient, popular and palatable are important factors to consider. The more difficult it is to make the changes the less likely they are to be maintained. Only long-term changes will ultimately reduce the burden of lifestyle diseases. Changes like using different fats or low fat alternatives in familiar recipes are ways of encouraging people to adapt within their comfort zone.

Clear evidence- based whole food changes that can be part of individual lifestyle choices are the messages that need to be given. This section looks at both reductionist components of the diet and the links with whole foods as reviewed recently (Messina *et al* 2001; Hoffmann 2003). One of the issues that has been looked at but has produced conflicting evidence is the micronutrient adequacy of diets that are limited in one or more macronutrients for the purpose of lowering disease risk. Of particular concern are the fat-soluble vitamins in low fat diets and some minerals, example zinc and calcium, which are found in foods (red meat and dairy) that are sources of saturated fats. Careful planning is needed to make sure that other foods that are nutrient dense in these are included in the diet (Swinburn *et al* 1999; Naglak *et al* 2000).

3.1.1 Carbohydrates

The dietary recommendation of the World Health Organisation is that an optimal diet supplies 55-75% of required energy from carbohydrates, 15- 30% of required energy

from fats and the remainder (10-15%) from protein (World Health Organisation 2003). This recommendation has been challenged in recent years particularly in relation to carbohydrates.

One aspect of carbohydrate intake that needs to be looked at is the total amount consumed in relation to other nutrients and the effect that too high a percentage energy intake from carbohydrate may have. The other aspect is the type of carbohydrate consumed. High carbohydrate diets may increase plasma triglyceride levels (Landry *et al* 2003).Low carbohydrate diets may be low in fibre and flavonoids which are only found in plants. Bravata et al, (2003) reviewed the safety of low carbohydrate diets by looking at 107 articles and concluded that there was not enough evidence to make a judgement for or against these diets. They found that weight loss associated with these diets was due to lower energy intake rather than the low carbohydrate per se and that data on sustainability of weight loss was not available.

3.1.1.1 Glycaemic Index

The Glycaemic Index (GI) is a ranking of foods on a scale from 0 to 100 according to how much they raise blood sugar levels after eating compared to a known carbohydrate, usually glucose. Foods with a high GI are those which are rapidly digested and absorbed and result in marked fluctuations in blood sugar levels.

McKeown *et al* (2004) examined the relationships between carbohydrate-related dietary factors, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham Offspring Cohort. They found that a high cereal fibre intake is inversely associated with homeostasis model assessment of insulin resistance (HOMA B%) and a lower prevalence of the metabolic syndrome and that if the carbohydrate in the diet was mainly high GI insulin resistance and metabolic syndrome increased. Practically this translates to a recommendation to increase whole-grain intake, which may reduce the risk of developing the metabolic syndrome.

Ludwig (2003) commented on the effect dietary carbohydrate on blood glucose concentration (glycaemic response). He pointed out that some studies had shown that a

low glycaemic diet may be “an optimal compromise between low fat diets, and very low carbohydrate diets.” He also noted that replacing more refined carbohydrates with whole grain foods and legumes and increasing fruit and vegetable sources of carbohydrate could produce significant reductions in GL and also advance other nutritional goals (Ludwig 2003). Adding protein to meals lowers the glycaemic index and produces a more sustained blood glucose level. GI is an interesting concept that may help manage diseases like diabetes better but practically adherence to long accepted dietary suggestions may be less confusing.

Eating balanced meals (containing protein, fat and carbohydrate) may lower LD risk, regardless of the GI value of the carbohydrate, especially if good quality protein, low saturated fats, whole grain cereals and abundant fruit and vegetables are eaten.

3.1.1.2 Dietary Fibre

Dietary fibre is a collective term for a variety of plant substances that are resistant to digestion by human gastrointestinal enzymes. They can be divided into two major groups depending on their solubility in water. Insoluble fibres (lignins, cellulose and some hemicelluloses) and soluble fibres (gel-forming fibres like pectins, guar and gums) which are found in oats, psyllum and fruits and vegetables.

The definition of dietary fibre is not consistent and, since the early 1950s, different countries and organizations have proposed various definitions of dietary fibre. Currently, a variety of definitions of dietary fibre exist worldwide. Some definitions are based solely on one or more analytical methods for isolating dietary fibre, while others are physiologically based.

A panel set up by the American National Academy of Sciences in 2001 proposed the following definitions of fibre:
“Dietary fibre consists of nondigestible carbohydrates and lignin that are intrinsic and intact in plants. Added Fibre consists of isolated, nondigestible carbohydrates that have beneficial physiological effects in humans” (National Academy of Sciences 2001).

Ha *et al* (2000) proposed a definition of fibre as "Any dietary component that reaches the colon without being absorbed in a healthy human gut."

A diet adequate in fibre-containing foods is also usually rich in micronutrients and nonnutritive ingredients that have additional health benefits. A meal that is high in fibre meal is processed more slowly which may account for its low GI. This also results in greater satiety. High fibre meals are also usually lower in energy, fat and refined sugar. These attributes make it a good option for treating and preventing obesity.

Several studies on the effects of fibre on cholesterol have shown that hypercholesterolemic participants are more responsive than normolipidemic participants (Ripsin *et al*1992; Anderson J 1999; Brown *et al* 1999). Increasing dietary fibre has been recommended as a safe and practical approach for cholesterol reduction.
High fibre foods that have been shown to lower blood cholesterol levels include apples, barley, beans and other legumes, fruits and vegetables, oatmeal, oat bran and rice hulls.(Marlett *et al* 2002). One mechanism of fibre that is thought to reduce LDL cholesterol levels is the interference of the fibre with bile acid absorption from the ileum. This causes LDL cholesterol to be removed from the blood and converted into bile acids by the liver, to replace the bile acids lost in the stool (Marlett *et al* 2002).

Diets high in fibre have been used to manage type 2 diabetes and have been looked at to lower risk of this disease. A study by Salmeron *et al* (1997) found that fibre from cereals, but not from fruits and vegetables, had an inverse independent relationship with risk of type 2 diabetes. Some studies looking at the effect of glycaemic index on blood glucose levels have suggested that diets high in fibre from whole grains and fruit and vegetables may lower type 2 diabetes risk (Mann 2001; Ludwig 2003).

Fung *et al* (2002) assessed whole grain intake in men from the Health Professionals Follow-up according to food frequency questionnaires. The 42 898 men (who had no history of cardiovascular disease or diabetes) were followed for about 12 years and completed FFQs every 4 years. Those with higher whole grain consumption tended to follow healthier lifestyles. They exercised more, ate less fat, and were less likely to smoke or have a history of elevated blood pressures than those who ate less whole grains. There was a significant decrease in the incidence of type 2 diabetes in those who

ate more whole grains. This result may be due to the lower glycaemic index of the whole grain foods or to specific nutrients found in larger quantities in whole grains as opposed to refined grains, for example, magnesium and vitamin E.

Not all studies looking at increased dietary fibre agree that it reduces cancer risk. In a study of 88 757 women, aged 34- 59 years, who were followed for 16 years no association was found between dietary fibre intake and colorectal cancers (Fuchs *et al* 1999). This is in contrast with most other studies and highlights problems associated with dietary data collection and analysis. Definitions and calculations of fibre, food mixtures and combinations, the time period needed for positive results, the negative impact of long term low fibre diets and whether increasing fibre as an adult is too late to affect the outcome are all possible reasons for the contrasting reports. It is also likely that composite factors over a lifetime contribute to raising or lowering the risk of most lifestyle diseases.

Howe *et al* (1992) examined the effect of dietary fibre on cancers of the rectum and colon. They combined the results of 13 case-control studies of colorectal cancer conducted in populations with different colorectal cancer rates and different dietary habits. The results were from 5 287 participants who had colorectal cancer and 10 470 control subjects. They concluded that there was substantive evidence that those who had fibre rich diets were less likely to have colorectal cancers than those whose diets were low in fibre. They estimated that a 13g/day increase in dietary fibre from whole foods could decrease colorectal cancer risk by about 30%.

From a whole food perspective dietary fibre can be obtained from cereals, particularly whole grains, legumes and fruit and vegetables. In epidemiologic studies, it is easier and more accurate to count servings of whole foods than analyse nutrients from data on food frequency questionnaires. It is also possible that whole foods offer more protection against chronic diseases than single nutrients like dietary fibre, antioxidants, or other biologically active components in foods, for example phytoestrogens (Craig 1997). This is possibly because of the synergic actions of the various nutrients and of the nutrients or chemicals in foods that we may not yet aware of. Some studies have found that people who eat fibre rich foods also follow other lifestyle patterns that contribute to reduced disease risk, for example, exercising more (Bazzano *et al* 2003).

3.1.1.3 Fruit and vegetables

The benefit of eating a diet rich in fruit and vegetables has been widely publicized. Many countries have this message as part if their public health guidelines and programs like the 5 plus a day campaign have helped to convey the importance of eating more fruit and vegetables to improve health. A part of a healthy lifestyle, fruit and vegetables can contribute to reducing the incidence and prevalence of obesity (Franz 1998). They are low in kilojoules, high in fibre and high in vitamins, minerals and phytochemicals.

In the Dietary Approaches to Stop Hypertension (DASH) trial, Appel *et al* (1997) examined the effects of dietary patterns on blood pressure. They reported a reduction in blood pressure in those subjects who were on diets that included 8-10 portions of fruit and vegetables per day (**4.1.2**). These changes in blood pressure could also make a difference in the risk of cardiovascular disease including stroke. Fruit and vegetables may also be protective against cardiac disease. Knekt and his colleagues, in a number of articles (Knekt *et al* 1994; Knekt *et al* 1996; Keevil *et al* 2002) have reported a protective effect of fruit and vegetable consumption in a Finnish study that followed 5 133 men and women aged between 30 and 69 years. They looked at the effect of reported intake of flavonoids and antioxidants on CVD risk and concluded that diets high in these constituents protected against CVD risk. They adjusted for age, sex, smoking, BMI, serum cholesterol and blood pressure. In a study at Harvard School of Public Health in 1995, Rimm *et al* (1995) also concluded that fruit and vegetables could have a protective effect against CVD. Several reviews (Ness and Powles 1997; Lampe 1999; Ness *et al* 1999) have looked at a variety of studies on the risk reduction and protective effects of increased fruit and vegetable consumption and found positive effects.

Fruit and vegetables have been found to help manage blood glucose in people with diabetes. It is thought that the increased fibre in these foods may delay glucose absorption from the small intestine. Recent advances in measuring glycaemic index (which enables scientists to have an indirect measure of rate of absorption) substantiates this thought. The soluble fibre found in fruit and vegetables may lower total and LDL

serum cholesterol in type 2 diabetes that would reduce complications from this disease. Whether increased fruit and vegetables can reduce the risk of type 2 diabetes as opposed to reducing complications and costs associated with the disease is less clear. The American Institute for Cancer Research (AICR) has calculated that a diet high in fruit and vegetables could reduce total cancer incidence by 20 percent or more (World Cancer Research Fund and American Institute for Cancer Research 1997). The most likely cancers to be prevented by increased fruit and vegetables are those of the lung and gastrointestinal tract.

Substances that may contribute to fruit and vegetables lowering lifestyle disease risk could be one or more of the following:

- Vitamins and minerals including the antioxidant vitamins A,C and E
- Folic acid
- Flavanoids including quercitin and phenols
- Sulphur containing compounds like dithioliones, isothiocyanates and allium
- Potassium and
- Soluble and insoluble fibre.

The wide variety of fruits and vegetables available means a wide variety of choice in type and method of preparation available to increase consumption. A variety is needed to get the benefit of all the possible protective factors available.

3.1.1.4 Kiwifruit – a high nutrient density fruit

The body composition and metabolism research centre has had some experience with looking at New Zealand kiwifruit, and its effect on laxation (Rush *et al 2002*) and also measuring its glycaemic index. Anecdotal reports from participants in the laxation study suggested that kiwifruit also reduced cholesterol and may lower blood pressure. A good relationship has been built with the New Zealand company that markets kiwifruit, Zespri, and they could supply kiwifruit as part of a further study.

Kiwifruit was the fruit of choice because it has been shown to have a high nutrient density. There are on average 1.6 grams of fibre in one kiwifruit. It is also high in phytochemicals including lutein and zeaxanthin and the antioxidants vitamin C and vitamin E. The Hayward variety (*Actinidia deliciosa*) of kiwifruit has 118 mg vitamin C and 2.5 mg vitamin E per 100g fruit (NZ Institute for Crop and Food Research). Current Australian daily recommended dietary allowances (RDAs) for adults for these nutrients are 60 mg vitamin C and 10 mg Vitamin E. Some researchers have advocated an increase in the RDA for vitamin C to 200mg per day(Levine M, Conry-Cantilena C *et al* 1996). Vitamin E is usually found in foods that are high in fats and oils. Kiwifruit is a less energy dense food from which to obtain part of the recommended daily dose. There is, however, doubt as to the bioavailability of the vitamin E as it is found in the seeds which may not be crushed sufficiently when eaten (Ferguson and Ferguson 2003). There are conflicting reports as to the value of vitamin E and vitamin C in reducing the risk of cardiovascular disease, but they are more likely to have a positive effect if taken as part of whole foods than as supplements (Brown and Goodman 1998; Paolini *et al* 2003; Roychoudhury and Schwartz 2003). Kiwifruit could be part of this source. Papaya, which is ranked slightly lower than kiwifruit in nutrient density but contains the similar phytochemicals and vitamins and has been anecdotally reported to reduce cancer risk.

Collins *et al* (1999) used comet assays to assess DNA strength before and after the consumption of kiwifruit pulp and found that the DNA strength 24 hours after ingestion of the pulp had improved by 32%. Total dietary intake was controlled over the 2 days and was nutritionally similar other than for the kiwifruit addition. He used a dose of the equivalence of five kiwifruit without their skins.

3.1.2 Dietary Fat

Different fatty acids may be protective against or contribute to lifestyle diseases. Fat is the most energy dense of the macronutrients and high intakes of any fats, even if they

are protective, may contribute to obesity which then contributes to hypertension, CVD, type 2 diabetes and some cancers.

3.1.2.1 Omega-3 Fatty Acids

Consumption of foods rich in omega-3 fatty acids (predominantly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)) are increasingly being shown to be associated with the reduction of risk of many of the lifestyle diseases. Several studies have shown that there may be a reduced risk of CVD, Type 2 diabetes, and breast, skin and colon cancers, with increased consumption of foods such as cold water fish that are high in this type of fat (Garcia-Closas *et al* 1993; de Deckere 1999; Dewailly *et al* 2001; Bonow *et al* 2002; Terry *et al* 2002; Cernea *et al* 2003; Nkondjock *et al* 2003; Rhodes *et al* 2003; Wolfram 2003). The "Mediterranean diet" is thought to promote health partly because of the relatively high proportion of seafood in the diet. Garcia-Closas *et al* (1993) compared fish consumption in some Mediterranean and Northern European diets concluded that dietary factors other than fish, such as the lower meat consumption, or other differences of lifestyle may also contribute to the healthy nature of the Mediterranean diet.

Using data from the Nurses Health Study, Hu *et al* (2002) reported that that those who ate fish one to three times per month cut their risk of a heart attack by about 20 percent. Weekly consumption dropped risk by about 37 percent. Mortality rates from cardiovascular disease were less in women who regularly ate fish. In the Health Professionals follow up study Ascherio *et al* (1995) reported that they found no significant association between fish or omega-3 intake and risk of cardiac disease. In the same study He *et al* (2002) found that there was a reduction in stroke risk in men who consumed more fish. A 2003 study by Burr *et al* in men with existing angina found that mortality increased in those eating oily fish twice a week and was even higher taking fish oil supplements. This highlights the possible difference between reducing risk of disease and treating a pre existing condition to reduce further risk. It also emphasizes the need for caution when taking nutrient supplements.

Many studies have suggested omega- 3 fatty acids as a means of preventing complications of diabetes, including cardiovascular disease, but finding studies that suggested omega-3 fatty acids could lower the risk of developing type 2 diabetes was difficult. In a study of the Inuit population of Greenland there was an indication protective effect on glucose intolerance when seal meat was eaten at least four times per, although it was not statistically significant (Berdanier C 1994).

Maillard *et al* (2001) examined biopsies from 241 patients with invasive, nonmetastatic breast carcinoma and from 88 patients with benign breast disease. They analysed the fatty acid levels in the breast adipose tissue and found lower levels of omega-3 fatty acids in those with cancer compared with those whose tumors were benign. This suggests a possible protective effect of omega-3 fatty acids against breast cancer and backs up some of the epidemiological studies.

A study in Italy and Switzerland (Tavani *et al* 2003), between 1991 and 2001, looked at 8 000 cases of cancer of oral cavity and pharynx, oesophagus, large bowel, breast and ovary and compared them to a control group of almost 23 000 control subjects who had been admitted to hospital for acute conditions unrelated to tumours or diet. Statistically significant reductions in the incidence of cancers of the oral cavity, oesophagus, colon, breast, and ovary were found in those eating the most omega-3 fats; reductions ranged from 30 to 50 percent. Information from questionnaires included socio-demographic factors, anthropometric variables, smoking, alcohol and other lifestyle habits, a problem-oriented medical history, physical activity and history of cancer in relatives. Information on diet referred to the previous 2 years and was based on a food frequency questionnaire. The researchers estimated that one gram of omega-3 fats per week, which can be obtained from one or two servings, may reduce risk of cancer by 12 to 30 percent. Fish, as a rich food source of omega -3 fatty acids, may be beneficial as it also high in protein, low in fat, and if the bones are eaten calcium. Suggestions of eating fish, particularly oily fish, at least twice a week therefore seems prudent. Fatty fishes like mackerel, salmon, sardines and tuna are good sources of omega-3 fatty acids and are readily available in New Zealand.

3.1.2.2 Saturated Fats

Obesity is a condition that arises as a result of energy intake exceeding energy expenditure. All fats contribute a high amount of energy per gram (37kJ) compared with other macronutrients protein and carbohydrate (16-17kJ) or alcohol (29kJ/g). It is therefore judicious to keep the total fat intake of the diet relatively low. Less than 30% of total energy is the general recommendation of most public health bodies.

The type of fat will not make a difference to the energy consumption but, given the high incidence of CVD, type 2 diabetes and some cancers that are promoted by saturated fat consumption, a saturated fat intake of no more than 10% of total energy is recommended. High protein, high fat (low carbohydrate) ketogenic diets have become popular to reduce weight again recently. In a study by Kwiterovich *et al* (2003) that looked at plasma lipids, lipoproteins and apolipoproteins in 141 children who were on a ketogenic diet for seizure control, there was a significant increase in atherogenic lipoproteins and a decrease in HDL cholesterol. This translates to an increased risk of atherosclerosis as the atherogenic cholesterol was increased and the anti-atherogenic cholesterol decreased. High protein diets tend to be high in saturated fats and while some studies have found no significant increase in LDL cholesterol or decreases in HDL cholesterol (Foster *et al* 2003) the long-term effect on lipoprotein profiles and associate risk of these diets is questionable.

Reducing saturated fats and or a changing them to mono- or poly – unsaturated fats has long been one of the cornerstones of CVD risk reduction strategies. Studies like the intervention in Mauritius, which changed the whole population's oil intake, have repeatedly shown that saturated fats need to be reduced to lower incidence of CVD. In a systematic review (Hooper *et al* 2001) of dietary fat intake and the prevention of CVD a meta-analysis of 27 studies that reduced or modified dietary fat intake (excluding those that concentrated on omega-3 fatty acids) showed a small (9%) reduction in cardiovascular mortality and a 16% reduction in cardiovascular events. Of particular interest in the study for this thesis is that the duration of the follow up was important. The longer the follow up (ie sustainability) of the fat changes the lower the risk of cardiovascular morbidity and mortality.

Uusitupa *et al* (1994) gave 10 healthy female students with a mean age of 23 ±2 years a diet that was either high in saturated fatty acids (SFA) or monounsaturated fatty acids (MUFA). They maintained their usual diet and physical activity regime other than the added fatty acids. They were on each of the supplemented diets for 3 weeks with a washout period of 2 weeks in between. The order of the fatty acids added was randomised and the study was conducted blind. The MUFA diet resulted in lower total cholesterol and LDL concentrations. HDL and triglycerides were not significantly changed. There was, however, a significant difference in the glucose tolerance (area under the curve) between the two diets suggesting that the type of fatty acid consumed may influence glucose tolerance in healthy women and therefore may play a role in decreasing risk of type 2 diabetes.

In a large study of 4903 Italian men and women aged between 20 and 59 years (Trevisan M, Krogh V *et al* 1990), increased reported use of butter was associated with significantly higher blood pressure and serum cholesterol and glucose levels for the men and increased plasma glucose concentrations in the women. In both sexes use of olive oil and vegetable oil was inversely associated with serum cholesterol, plasma glucose levels and systolic blood pressure. Another nutrient associated with blood pressure because it determines extracellular body fluid volume is sodium.

3.1.3 Sodium

Increased intake of sodium has long been associated with hypertension. There has been a relatively recent increase in total intake of sodium partly due to the processed and pre-cooked foods that have risen in popularity over the last few decades. Decreasing consumption of these foods and reducing the amount of sodium added to home-prepared meals during and after cooking can lower sodium intake significantly.

3.2 Physical activity

Lack of physical activity is a world –wide problem that is thought to have added greatly to the incidence of lifestyle diseases. As the world becomes more technologically advanced, the need for regular, daily expenditure of energy via physical activity has decreased and this may have had a big influence on the risk and pathogenesis of the LDs being studied. In a highly publicised article in Scientific American, Willett, an eminent Harvard researcher, suggested that the food pyramid currently used as the basis for healthy eating in many countries, is flawed. One of the changes he advocates is to make physical activity the base of the pyramid indicating its importance in a healthy lifestyle (Willett *et al* 2003).

3.2.1 Background

Cars have replaced more active means of transport (for example walking, riding bicycles and horse riding). Lifts and escalators have replaced stairs as the chosen way to access different levels in most buildings and the stairs are often difficult to find. There has been a shift toward occupations that require less energy to be expended, and new technologies allow more sedentary approaches to be taken at various occupations. Computers have allowed more sedentary ways of doing filing and communicating. Lecturers can stand at a computer and click a mouse or use an overhead projector as opposed to more energetic board writing. Time has also become a factor in sedentary work styles. Lunch and tea times have become less leisurely and are often taken in meetings. Work hours are long and many people don't have the time or motivation to spend time away from home at the gym or doing other forms of physical activity. Many arrive home after dark when safety of walking may be an issue too (Shetty 1997; Popkin 2001).

3.2.2 Strategies for increasing physical activity

As long ago as 1990, DeBusk *et al* recognised the importance of sustainable physical activity to improve and maintain fitness. They showed that three ten minute sessions of aerobic exercise was only slightly less effective in improving fitness than a single long session but that shorter more frequent activities may encourage adherence and increase weight loss. They advocated that "lifestyle activities" should be used to include physical activity into daily lives. These should fit into daily routines and not be too onerous. Examples are climbing the stairs instead of using the elevator, parking away from your destination and walking and getting off at a bus stop which is not the nearest to your destination and walking.

There is clear evidence from 38 studies conducted primarily in the United States and Europe that physically active women postpone mortality compared with inactive women. The American Heart Foundation and the WHO have long recommended that the 30 minutes of moderate intensity physical activity is needed daily to reduce obesity and LD risk. In a consensus statement at an International Association for the Study of Obesity in Bangkok in 2002, the delegates acknowledged that this was a reasonable amount to limit health risk but suggested that 60-90 minutes a day would be more appropriate to encourage weight loss and weight loss maintenance (Saris *et al* 2003).

The New Zealand government recognises the importance of physical activity and under Sport and Recreation, New Zealand (SPARC) initiatives such as the Green Prescription and the PushPlay programme to increase participation in physical activity are ongoing.

'Green Prescriptions', have been used to encourage at risk individuals to increase physical activity. These are written instructions from a health professional prescribing physical activity as a prevention and treatment of LDs (Swinburn *et al* 1998). In 2000, the Hillary Commission (now SPARC) surveyed 263 patients who had received green prescriptions from their health professionals in the last 3 months of 1999. Over half the patients were more active than before the prescription was given. Weight loss and

generally feeling better were the main health benefits after 5-8 months. This was further confirmed by a cluster randomised controlled trial (Elley *et al* 2003) to assess the long-term effectiveness of the New Zealand 'green prescription'. Participants were recruited from medical practices after a screening form identified them as doing less than half an hour of moderate to vigorous exercise on five or more days per week. After enrolment they were either placed into a group receiving the 'green prescription' or into a control group who received normal care. Measurements were taken at baseline and at a 12 month follow up and included a questionnaire about physical activities over the previous three months, questions about 'quality of life', blood pressure measurements, heights and weights and Framingham or D'Agostino CVD risk equations. The participants in the 'green prescription ' group increased their physical activity more than in the control group and reported a better quality of life (general health, vitality, bodily pain). Blood pressure improved in both groups and risk of CVD did not change significantly. Importantly, physical activity was increased and the reported changes sustained over 12 months with brief advice from a medical practitioner and ongoing telephone support.

Other initiatives by SPARC to increase physical activity in New Zealand include the Push-Play campaign to encourage people to do at least 30 minutes of physical activity a day and Hikoi 2002 which encourages families to walk together (SPARC (Sport and Recreation New Zealand) 2003).

Physical activity levels are hard to measure. One way for an individual to partially determine their physical activity level is through the use of a pedometer.

3.2.2.1 Pedometers

One of the obstacles in defining the role of physical activity in LD prevention, and the amount of physical activity needed to accomplish this, is the lack of ability to measure physical activity in free living subjects accurately. Pedometers may be an effective way of obtaining information about activities like walking that are not memorable enough to be recalled and are difficult to judge as a measure. Several pedometers, including the

Yamax DW500, have been found to be fairly accurate in calculations to measure energy expended from steps taken (Bassett *et al* 1996; Bassett 2000). There are many considerations for researchers when using pedometers to gauge pa. The amount of other physical activity, the accuracy of the instrument and what the optimal number of steps are some of these.(Tudor-Locke and Myers 2001). How many steps to aim for in order to have maximum risk reduction is also a question that needs answering.

Le Masurier and colleagues in Arizona (2003) undertook a study to see whether taking 10 000 steps in a day is equivalent to meeting the current minimum physical activity guidelines of accumulating at least 30 min of moderate physical activity each day. He found that accumulating 10 000 steps/day does not necessarily mean that the guidelines to confer the health benefits of physical activity have been met but it is more likely. The age and fitness level of the walker and intensity of the physical activity will affect the benefits gained from increased number of steps.

The number of steps is not as important as a gradual increase in daily step count over a period of time and long-term persistence. It is probably better to increase steps gradually and increase fitness levels as well than to jump to 10 000 steps from a low number or stay at
10 000 if you are already achieving this. Pedometers can be used as a tool for motivation. Having a visible record of the amount of steps walked can prompt people to increase them and also act as a reminder to do regular physical activity.

3.2.3 Physical activity, with and without diet in the treatment of lifestyle disease

Physical activity is necessary to balance the energy obtained from food. Too much food for the energy we expend leads to obesity, and this increases the risk of hypertension, CVD, type 2 diabetes and some cancers. Each of these lifestyle diseases and their treatment through lifestyle changes are considered in turn below.

3.2.3.1 Obesity - physical activity and diet effects

Ross, Janssen and Tremblay (2000) compiled a report for the Canadian Society for Exercise Physiology in 2000 that reviewed knowledge regarding physical activity and diet in relation to obesity prevention, reduction and maintenance. They looked at 5 meta-analyses (since 1980) that reviewed hundreds of published studies on exercise and weight loss. They concluded the meta- analyses revealed that although increased physical activity is not as good at reducing weight or fat mass as diet, when diet and physical activity are changed together, reduction in fat mass is better and more sustainable than when diet is used alone. They then confound this conclusion by suggesting that the studies using only a physical activity intervention mainly involved participants who were not obese and who were using physical activity as a means to attain fitness rather than weight loss. It is probable that the intensity of physical activity on its own required for the obese to lose weight would be unsustainable and also have the potential of causing injury and possible cardiovascular strain. They do finally conclude that diet and physical activity should be used together to develop a program to reduce body fat that is safe and sustainable.

Janssens *et al* (2002) looked at the effects of an energy-restricted diet with or without exercise on obese pre menopausal women. The 38 women who took part in the study each followed a 2-week maintenance diet before being divided into three groups. All participants were asked to reduce their energy intake by about 1000 calories per day. They kept daily diet records and limited fat to <30% of energy. Eleven of the women also followed an aerobic exercise program 5 days a week. Fourteen followed a resistance-training regime. Other than following the prescribed physical activity programs all thirty-eight followed their normal pre-study physical activity patterns. Total and regional fat distributions were measured by MRI scan. Plasma lipid, glucose and insulin levels were measured. There was about 10% weight loss overall and total cholesterol, LDL cholesterol and fasting insulin levels improved in all groups. This improvement was not enhanced by the addition of either aerobic or resistance exercise. Plasma triglycerides, HDL cholesterol and glucose levels did not change significantly.

The researchers concluded that exercise, without weight loss, had little or no effect on plasma lipids or glucose levels in this population.

In fifty-two obese men Ross and colleagues (2000) found that exercise and diet did improve health. They used four scenarios: a diet-induced weight loss, an exercise-induced weight loss, an exercise without weight loss, and a control group. Participants were randomly assigned to one of the groups and were observed for 3 months. There was an eight percent decrease in body weight in both weight loss groups and no change in weight in the "exercise without weight loss" and control groups. Total fat mass decreased in both weight loss groups. There was a greater reduction of fat in the exercise-induced weight loss group than in the diet-induced weight loss group. He found that those in the exercise-induced weight loss group (without dietary restrictions) substantially reduced abdominal obesity and insulin resistance. This was also the finding in a programme to reduce visceral fat in obese adolescents (Gutin *et al* 2002) There is a higher risk associated with visceral obesity than obesity per se (Despres *et al* 2001) so evidence that significant improvements in health risk can be achieved through moderate changes in dietary intake and exercise(Ross and Rissanen 1994) is very important.

A critique of many interventions is that they are only effective in the short term. Maintenance of weight loss is helped by increased physical activity levels. In a group of twenty-seven women who lost weight on a very low calorie diet Leser and colleagues (2002) evaluated the women three years after they had lost the weight and found that those who reported higher physical activity levels maintained their weight loss better than those who were more sedentary.

3.2.3.2 Cardiovascular disease -physical activity and diet effects

Regular physical activity decreases the risk of CVD mortality. Berlin and Colditz in 1990 published a meta-analyses of studies that inversely associated physical activity and CVD risk and found that the better designed studies showed a stronger link between the two. They estimated that sedentary people had almost double the risk of CVD as active

people. In life – people cannot be separated easily into sedentary and active categories – there is a continuum of activity level, frequency and intensity.

Intensity of physical activity for LD reduction has been an issue of many debates. In a twelve-week study by Goto *et al* (2003), moderate exercise, but not mild or high-intensity exercise, was recommended as being most likely to reduce CVD risk. These researchers suggested that intense exercise should be avoided, as it was potentially hazardous to the endothelial cells of blood vessels. Data from the Health Professionals' follow-up study was used to evaluate exercise type and intensity in relation to CHD in men. These researchers found that total physic al activity, walking, running and resistance training all contributed to reduced CVD risk (Tanasescu *et al* 2002).

Several large studies on cardiovascular disease risk have implicated low levels of physical activity as a contributing factor. These include:

- A study done on British civil servants reported on in 1990 that compared CVD incidence in those that played vigorous sports with those who were more sedentary;
- The Harvard Alumni Health Study, which took place from 1962 until 1972 and had physical activity data analysed for associations to CVD (Sesso *et al* 2000); The Multiple Risk Factor Intervention Trial (see 4.1.2) which tested a multiple factor intervention to lower BP and serum cholesterol and to reduce smoking. Seven years of physical activity data was analysed and compared to CVD rates;
- The Iowa Women's Health Study, which studied postmenopausal women and looked at different level of physical activity and CVD risk;
- The Finnish Twin Cohort Study, which analysed physical activity and mortality in same sex twins and found physical activity inversely related to all-cause mortality.

All of these studies found an inverse relationship between physical activity and CVD (and in some cases other LDs) even when dietary differences were taken into account (Paffenbarger and Lee 2001).

Mechanisms for physical activity to reduce CVD risk may include:

- Increasing myocardial oxygen supply.
- Decreasing myocardial oxygen demand.

- The improvement of myocardial contraction.
- Improving endothelial functioning in coronary arteries and increasing the diameter and capacity of coronary arteries.
- Promoting the growth of collateral arteries which may increase myocardial perfusion.
- Lowering blood pressure.
- Reducing platelet aggregation.
- Reducing overweight and obesity.

In a study to look at the effect of diet and exercise on blood pressure, oxidative stress and nitrous oxide availability, a combined diet and physical activity intervention resulted in a significant improvement in BP, fasting lipids and insulin levels within 3 weeks. Oxidative stress, which is thought to be a possible causative factor for hypertension, also improved. The dietary intervention consisted of 10% energy from fat, 15-20% from protein and 70-75% from mainly unrefined carbohydrates. The physical activity intervention consisted of daily walking for 45-60 minutes (Roberts *et al* 2002). This was a particularly strenuous intervention and the long-term sustainability of both the diet and physical activity regimes needs to be questioned.

Another study looking at associations between physical activity and ischemic stroke risk in middle-aged adults by Evenson *et al* (1999) found only a weak link between them that may have been due to other factors or chance.
Lee *et al* (1999), followed nearly 22 000 men aged between 30 and 83 years for 8 years. They found that unfit lean men were twice as likely to die from cardiovascular disease as fit lean men. Unfit lean men were also more likely to die from any cause including than men who were fit and obese. They concluded that “the health benefits of leanness may be limited to fit men, and that exercise is very beneficial to overall health even if no weight is lost.”

3.2.3.3 Type 2 diabetes mellitus-physical activity and diet effects

Several studies have found associations between physical activity and risk of type 2 diabetes. Most of these have been part of studies where changes to diet have been part of the equation (Lee *et al* 1999; Colditz *et al* 2003; John *et al* 2003; Lee 2003; McTiernan *et al* 2003). Physical activity has been found to be therapeutic in people who have type 2 diabetes (Pigman *et al* 2002). Does helping glycaemic control in those with type 2 diabetes translate to reducing the risk of type 2 diabetes? Harding *et al* (2001), put it succinctly when they concluded that their findings on the association between dietary fat intake, insulin resistance and physical activity, provided "further support for efforts to increase overall physical activity and modifications in the pattern of dietary fat intake in the whole population.

3.2.3.4 Cancer -physical activity and diet effects

Keeping physically active is thought to reduce the risk of many cancers including breast cancer and colorectal cancers. The studies from which this conclusion arises are mainly based on data that involves other changes (such as diet) and their is much variation in the recommended level of physical activity required (Lee *et al* 1999; Colditz *et al* 2003; John *et al* 2003; Lee 2003; McTiernan *et al* 2003). Obesity is associated with some cancers and physical activity is known to reduce body fat. Lee *et al* (1999) found that that physical activity may be associated with lower risk of lung cancer among men. They found that 6-8 hours of moderate to high intensity physical activity may significantly lower risk.

Information about physical activity levels obtained from the Nurses Health Study (**4.1.1**) was used to determine relative risk of colon cancer. The researchers (Martinez *et al* 1997) found that women who spent the most amount of time on leisure time activities and who expended the most amount of energy in activity had a lower risk of colon cancer and stroke (Hu *et al* 2000) than those who were more sedentary. They

recommended that more emphasis be put on physical activity as a way of reducing colon cancer risk.

Mechanisms for increased physical activity to reduce cancer risk may include:

- The effect of physical activity on insulin, prostaglandin and bile acid levels. These all affect colonic cell activities.
- The reduction of bowel transit time in those that do more physical activity which reduces the length of time carcinogens have contact with colon cells.
- The effect of physical activity on hormones that may be implicated in breast and endometrial cancers.
- Physical activity may contribute to reduced cancer risk by reducing overweight and obesity.

3.3 Summary

This chapters' literature review has emphasised that the following messages should diet and physical activity messages be given to the participants this study:

- Decrease total energy intake by attention to portion sizes and number of portions per day.
- Increase dietary fibre and other possible protective factors, like vitamins, minerals and phytochemicals, by increasing whole foods that contain dietary fibre.
- Increase fruit and vegetable consumption.
- Increase wholegrains
- Decrease foods that are high in saturated fatty acids.
- Increase foods that are high in omega-3 fatty acids
- Increase physical activity

4 Literature Review –Large Studies – Prospective and Controlled Intervention

4.1 Background

Many studies have recorded over time the potential of dietary and physical activity interventions to reduce the risk of the lifestyle diseases focused on in this research. The first part of this chapter looks at selected prospective studies and the second looks at the few large controlled intervention studies that have measured the effect of interventions on parallel groups.

4.1.1 Prospective studies

The prospective studies are the

Nurses Health Study

Framingham Heart Study

British Regional Heart Study

World Health Organisation Multinational Monitoring of Trends and Determinants in Cardiovascular Disease (WHO-MONICA)

European Prospective Investigation into Cancer and Nutrition (EPIC)

The **Nurses Health Study**, which was started in 1976 by researchers at Harvard Medical School, has implicated lifestyle as an important factor in prevention of the non-communicable diseases looked at in this study. The Nurses Health study is based on biennial questionnaires that are mailed to the approximately 121 7000 participants. Information is updated on demographic, anthropometric and lifestyle factors including diet and physical activity. Health factors including diagnoses of diseases like diabetes, cardiovascular diseases and cancers are reported. Regular exercise, healthy weight maintenance, reduction in saturated and trans- fatty acids, increasing fruit and vegetables and whole grains and abstinence from smoking were found to reduce risk of type 2 diabetes by up to 80% and reduce cardiovascular disease by 70-80%. Risk of postmenopausal breast cancer and colorectal cancer was also reduced in those with a healthier approach to diet and physical activity (Nelson 2000). Hu et al. (2001) reported that Nurses' Health Study data provides evidence that walking at an average pace for a

total of two hours a week or more were associated with a reduction of 25% for risk of stroke. Increased physical activity was associated with greater reductions in risk.

The **Framingham Heart Study** was set up in 1948 under the direction of the National Heart Institute (now known as the National Heart, Lung, and Blood Institute; NHLBI), to identify the common factors or characteristics that contribute to CVD. The NHLBI has a comprehensive website on the Framingham study that most of the following information is based on (http://www.nhlbi.nih.gov/about/framingham/).
Researchers wanted to follow the development of CVD over a long period of time in a large group of participants who had not yet developed overt symptoms of CVD or suffered a heart attack or stroke. In the original cohort, 5209 men and women aged 30 to 62 years were recruited from the town of Framingham, Massachusetts. They have since then returned biennially for a detailed medical history, physical examination, and laboratory tests.

In 1971, the study enrolled a second-generation group consisting of the original groups' adult children and their spouses. Five thousand, one hundred and twenty-four people are in this cohort. A Third Generation (the children of the Offspring Cohort) is currently being recruited and examined, seeking to further understand how genetic factors relate to cardiovascular disease. Researchers are hoping to recruit and examine 3,500 grandchildren of the original cohort.

The Framingham Study has led to the identification of the major CVD risk factors such as high blood pressure, high blood cholesterol, smoking, obesity, diabetes, and physical inactivity. Information on the effects of related factors such as blood triglyceride and HDL cholesterol levels, age, gender, and psychosocial issues have also been studied.

Major milestones in the Framingham project have been:
"1960 - Cigarette smoking found to increase the risk of heart disease
1961 - Cholesterol level, blood pressure, and electrocardiogram abnormalities found to be associated with an increase in the risk of heart disease
1967 - Physical activity found to reduce the risk of heart disease and obesity to increase the risk of heart disease

1970 - High blood pressure found to increase the risk of stroke

1987 - High blood cholesterol levels found to correlate directly with risk of death in young men

1988 - High levels of HDL cholesterol found to reduce risk of death" (National Heart Lung and Blood Institute 2002)

Framingham data has been used to prepare algorithms for estimating CVD risk in men and women. The factors used to attain a risk factor score are blood pressure, age, total cholesterol, HDL cholesterol, diabetes and smoking .The Framingham risk score formula estimates the risk of developing CHD within a 10-year time period and was based on a mainly Caucasian population (D'Agostino 2001).

The **British Regional Heart Study** in 2003 reported the relationships of systolic BP, smoking, serum total:HDL cholesterol ratio, diabetes and enlargement of the heart with CVD risk in 7700 British men after a 10 year follow up. They reported that the Framingham risk formula overestimated in this British population the risk of CVD mortality by up 47% and non-fatal coronary events by up to 57% (Brindle *et al.* 2003).

This disparity in risk may be because different populations have different risk levels, are living in different environments and CVD mortality and morbidity have changed since the Framingham data was collected (1968-1975), and the risk score may be compromised by factors not considered such as alcohol consumption, triglyceride levels and homocysteine concentrations (Hense 2003).

The **World Health Organisation Multinational Monitoring of Trends and Determinants in Cardiovascular Disease (WHO-MONICA**) project was set up in 38 populations in 21 countries from mid 1980 to mid 1990 to look at the relationship between risk factors and CVD incidence. Kuulasmaa *et al.* (2000) examined changes in well-known risk factors and found only weak association between these and changes in CVD incidence. They did find that although individual risk factors exhibited only weak associations with coronary events, most populations who lowered their risk factors also lowered the incidence of CVD.

The **European Prospective Investigation into Cancer and Nutrition (EPIC)** is the largest study of diet and health ever undertaken. It involves researchers based at 23 centres in 10 countries and has over half a million recruits. Anthropometric and biochemical measurements and lifestyle questionnaires were collected. Several reports have been published and it is expected that the project will clarify and strengthen the understanding of some of the links among nutrition, lifestyle factors and disease.

4.1.2 Intervention studies

Major findings of six large intervention studies and associated research are summarised in this section. The main studies are the

Dietary Approaches to Stop Hypertension study (DASH)
Multiple Risk Factor Intervention Trial (MRFIT)
North Karelia Project
Da Qing IGT and Diabetes Study (DQID)
Finnish Diabetes Prevention Study (FDPS)
Diabetes Prevention Program (DPP).

The **Dietary Approaches to Stop Hypertension** (DASH) study involved 459 participants. Participants had a mean systolic blood pressure of <160mmmHg and a mean diastolic blood pressure of 80-95 mmHg. The Joint National Committee (1993) classification of these blood pressures is from "normal" to "Hypertension stage I" (**2.2**) Joint National Committee (JNC-V) 1993). The study looked at changing dietary patterns but paid attention to micronutrients in the whole foods to differentiate between diets (Vogt *et al.* 1999). It took place over 8 weeks and participants either ate their meals at various centres or they took pre-packaged foods home to eat off- site. (multicentre outpatient feeding trial). Three diets were used. A control diet was based on a typical American diet pattern. This diet was used as a run in for three weeks with all the participants. They were then randomised into three groups. One group continued with the control diet. Group two followed a diet that was very high in fruit and vegetables (6-10 portions per day) and had fewer sweets and sweetened beverages than the control diet. This diet 2, also known as the fruit and vegetable diet, was high in fibre and, compared to the average American's consumption, was high in potassium and

magnesium. The third group followed a “combination diet”. This diet was high in fruit, vegetables (6-10 portions per day) and low fat dairy products (just under 3 servings per day). Diet 3, the combination diet, included whole grains, poultry, fish and nuts and was low in fats, red meats, sweets and sweetened beverages. It was slightly higher in protein than the other two diets and provided calcium, potassium and magnesium at above the average American intake. It was also low in saturated fat, total fat and cholesterol. Other possible influences on blood pressure, like sodium intake and body weight, were kept constant over the time of the study. These controlled diets were followed for a period of eight weeks (Vogt *et al.* 1999).

The fruit and vegetable diet reduced blood pressure more than the control diet but the combination diet reduced blood pressure more than the fruit and vegetable diet. Blood pressures started reducing within two weeks of starting the diets and were reduced irrespective of sex, age or ethnicity. The participants in this study who had higher blood pressures had greater reductions in blood pressure with the dietary restrictions than those that were normotensive. In the groups who increased their fruit and vegetables blood pressures dropped by 2.8-5.5 mmHg in those who were not hypertensive and by 11.4 mmHg in those whose blood pressure was high at the start of the study (Harsha *et al.* 1999).

A study that used a similar diet plan to the DASH combination diet but reduced the calories, added physical activity, and reduced the sodium was called the Diet, Exercise and Weight Loss Trial; DEW-IT study. The DEW-IT study showed that a holistic approach to treatment of hypertension including weight loss and increased physical activity as well as a diet that is high in fruit, vegetables and low fat dairy products and low in sodium can reduce blood pressure in hypertensive, overweight adults (Miller *et al.* 2002).

Another related study by Roberts *et al.* (2002) looked at the effect of diet and exercise on hypertension and other lifestyle disease risks. This was a small (n=11) three-week, intensive intervention carried out in a live-in centre. The duration of the intervention was important for application to the study reported in this thesis because it showed that the effects of a dietary and physical activity intervention could be demonstrated in a short period of time. The dietary intervention led by Roberts consisted of a low fat, high

fibre diet and daily exercise regime and resulted in dramatic and significant improvements ($P<0.01$) in blood pressure and metabolic profile e.g. systolic blood pressure on average was lowered by 13.6%.

One of the first major intervention studies was the **Multiple Risk Factor Intervention Trial** (MRFIT). MRFIT screened over 360 000 men aged between 35 and 57 years in 18 US cities for risk factors of blood pressure, total serum cholesterol and smoking and followed them for 16 years. It excluded those who had had previous cardiovascular histories and those who were on drug treatment for diabetes. Twelve thousand eight hundred and sixty six men took part in the study (Stamler *et al*. 1999). At the time of the MRFIT study several studies had shown that the risk factors screened for contributed to coronary heart disease risk, but no evidence had been found that changing the factors that led to this risk would change the outcome or incidence. The study comprised of an intervention group and a control group. The intervention group was given counselling to stop cigarette smoking, dietary advice to lower cholesterol and treatment for their hypertension. The control group was referred to their usual health care provider. The unexpected result was that the control group made more favourable changes than expected making the mortality difference between the two groups statistically not significant (Gotto 1997).

The MRFIT did not yield expected findings but the data collected has been used for other analyses. For example, the participants who completed the trial were grouped by their incidence of type 2 diabetes and non-fatal cardiovascular disease. Eighteen years after the MRFIT it was found that diabetes significantly increased cardiovascular risks and that those who had non fatal cardiovascular events were more likely to die sooner (Eberly et al. 2003).

Greenland and colleagues carried outdid a meta-analysis of data from this trial and others to see how much the major risk factors of cigarette smoking, hypertension and elevated blood cholesterol as well as overweight contributed to coronary heart disease. They found that each of the risk factors contributed to increased CVD and that they should all be given priority. It is important not to concentrate on only one factor rather to take a more holistic approach and focus on many risk factors (Greenland *et al*. 2003; Stamler *et al.*1999). This also supports trying to change the lifestyles of populations

rather than targeting just those with risk factors. This would mean more people having less exposure to possible risk factors and also support within the family and the community thereby reducing from birth exposure to long term risk factors for CVD. It also emphasizes the need for long term changes in lifestyle (Stamler *et al.* 1999).

The **North Karelia Project** was initiated in response to statistics that showed that men in Finland had the highest global mortality rates from IHD in the 1960s. It was a community-based prevention program started in 1972 that was targeted at changing the target risk factors and health behaviours (serum cholesterol, blood pressure, smoking, diet) at the population level. Health information aimed at dietary changes was disseminated to the community and a health policy statement released. The practical intervention activities were integrated into the existing service structure and social organisation. Interventions ranged from mass-media education for example a series of half-hour TV programmes, to small-group face-to-face education in skills such as healthier cooking methods. Cholesterol screening was introduced which focused the population on their diets and high risk. There has also been an anti smoking campaign.

Mortality from CVD in 1997 had decreased from that in 1972 by 73% in North Karelia and by 65% in Finland as a whole. Most of the drop seems to be related to the public awareness campaign in North Karelia that was then rolled out nation-wide. Mortality from other LDs, particularly cancers, has also decreased (Puska *et al*. 1998; Vartiainen *et al*. 2000). The North Karelia study has contributed substantially to the evidence for the use of multifactor interventions to reduce LD risk. Some researchers believe that the reduction in CVD incidence was due to the general trends of decreasing CVD incidence in similar countries at the time and not because of the interventions per se (Ebrahim and Davey Smith 2000).

There have been several studies looking at preventing or delaying type 2 diabetes mellitus including the Malmo feasibility study (Sweden), several community based studies and studies looking at different forms of drug interventions. The next part of this section looks at the three most recent large scale intervention studies aimed at reducing or delaying the risk of type 2 diabetes mellitus using lifestyle changes.

They are the Da Qing IGT and Diabetes Study in China (Pan *et al.* 1997), the Finnish Diabetes Prevention Study (Tuomilheto *et al.* 2001) and the Diabetes Prevention Program in the USA (Diabetes Prevention Program Research Group 2002). All three of these studies looked at the prevention or delaying of impaired glucose tolerance (IGT) progression to type two diabetes mellitus. Impaired glucose tolerance is an intermediate stage between normoglycaemia and diabetes mellitus.

The **Da Qing IGT and Diabetes Study (DQID)** in China was a 6 –year study (1986-1992) that randomised 577 people (over the age of 25 years) with IGT into four groups. IGT was diagnosed after a plasma glucose test two hours after a standard breakfast followed by an OGTT in those who screened positive (What "positive" was is unclear in the easily available literature). It was a controlled clinical trial in which participants identified as having IGT were either given dietary advice, physical activity advice, both dietary and physical activity advice or neither. The clinics were randomised to carry out the different treatments, rather than the participants (cluster trial). Baseline measurements (blood pressure, height, weight, and glucose levels) were recorded and questionnaires relating to family history, medical history, dietary intake and physical activity were completed. Participants were classified into lean (BMI<25 $kg.m^{-2}$) or overweight (BMI>25 $kg.m^{-2}$)(**5.2.1**).

The dietary interventions were normocaloric, 55-65% carbohydrate, 10-15 % protein and 25-30% fat for those classified as lean. These lean participants were also advised to increase their fruit and vegetable consumption, control their alcohol intake and reduce simple sugars.

Those who were classified as overweight (BMI>25$kg.m^{-2}$) were advised to reduce their energy intake to lose weight at 0.5-1.0kg per month until a BMI of 23$kg.m^{-2}$ was reached .Individual targets of consumption of foods from the five major food groups (breads and cereals, fruit and vegetables, meat, milk and oils) were set and exchange lists of foods and portion sizes in these groups were provided.

The participants in the physical activity group encouraged and shown how to increase their physical activity by 'units' of exercise. These were defined as mild to very strenuous and different amounts of time were required for each degree of exercise to

make up one unit e.g. 30 minutes of mild exercise but 10 minutes of strenuous exercise was one unit. One to two units increase per day was advised.

The diet plus exercise group combined the above two strategies individual counselling and goals were given initially to these three groups. They also had small group counselling sessions weekly for 1 month, monthly for three months and then once every three months for the remainder of the study. The control groups were given general information on diabetes, diet and physical activity with no individual or formal group sessions. Evaluations were carried out biannually.

The results showed an overall reduction in the incidence of diabetes of 33% ($p<0.03$) in the diet-only group, 47% ($p<0.0005$) in the exercise only group and 38%($p<0.005$) in the diet-plus-exercise group, compared with the control group. These findings may be affected by the randomisation of the clinics because the population attending the specific clinic may have been biased socio-economically and culturally (Pan *et al*.1997).

The Finnish Diabetes Prevention Study (FDPS) took place between 1993 and 1998 with a mean duration of follow up of 3.2 years (Tuomilheto *et al.* 2001). Those eligible for the study were diagnosed with IGT after primarily being screened as a result of other possible risk factors being present (family history, overweight - BMI>25kg.m^{-2}). They were between the ages of 40 and 65 years (mean 55 years). IGT was defined as a fasting plasma glucose of <7.8mmol/l and a 2 hour OGTT of 7.8-11.0 mmol/l. The 523 participants were randomly assigned to either an intervention or a control group. The intervention group were given detailed, individualised advice. The advice was aimed at achieving the goals of the intervention which were:

- a reduction in weight of 5% or more
- a total fat intake of < 30% energy consumed
- a saturated fat intake of < 10% energy consumed
- an increase if fibre to 15g+ per 1000 kcal
- and moderate exercise for at least 30 minutes per day.

The intervention group had seven individual sessions with a nutritionist in the first year and one session every three months thereafter. They also received individual physical activity guidance and opportunities to have individualised training sessions. The control group were given general oral and written dietary and physical activity information.

There were significantly fewer participants diagnosed with diabetes in the intervention group compared with the control group (cumulative incidence of diabetes was 58% lower). The incidence of diabetes was lower in both genders. Participants were given a compliance score (number of goals met at the one-year examination) and those who attaining a higher score were less likely to develop diabetes.

The **Diabetes Prevention Program** DPP was a clinical trial involving "at risk" individuals at 27 centres in the USA. People screened were over the age of 25 years, had a BMI of at least 24 $kg.m^{-2}$ (above 22 $kg.m^{-2}$ for Asians) and a fasting plasma glucose of 5.3-6.9 mmol/l followed by a OGTT - 2 hour glucose of between 7.8-11.0 mmol/l. Those identified as having IGT were randomly assigned to three groups – lifestyle intervention, metformin medication or a placebo/control group. The medication and placebo groups received written lifestyle information and an annual individual session that emphasised healthy lifestyle using the food pyramid and dietary guidelines from a national cholesterol education program. They were advised to increase physical activity and reduce weight.

The intensive lifestyle intervention group were assigned "lifestyle coaches". They were given goals of a weight reduction of at least 7% of initial body weight and moderate physical activity of at least 150 minutes per week. These goals had to be maintained. They were given individual tuition sessions of a 16 –lesson curriculum covering diet, exercise and behaviour modification. The lessons took place in the first 24 weeks and were flexible, culturally sensitive and individualised. Initially the dietary intervention looked at ways of reducing total fat intake. This reduced total calorie intake and after a few weeks ways of reducing total energy intake while achieving a balanced diet was emphasised (Diabetes Prevention Program, 2002). Individual and group sessions were arranged as required. Monthly contact was also made with the participants during the study and in-person contact was made at least once every two months. A 'tool box' of incentives, such as exercise classes, tapes, equipment, more structured eating plans, liquid formula diets and home visits was available for those who were having problems meeting goals (Bray *et al*, 1999)

4.2 Summary

The application of all the above diabetes lifestyle interventions showed a reduction in the development of type 2 diabetes compared to the control groups. The Da Qing study had a 31% reduction with diet; a 46% reduction with physical activity and a 42% reduction with diet and physical activity interventions. The DPP showed a 58% reduction, as did the FDPS. These studies took place in different countries with a diverse range of people. The DPP consciously recruited about 45% ethnic minorities (who are more at risk). It can, therefore, probably be assumed, that interventions to diet and physical activity using the 'healthy eating' guidelines (with cultural applications) will reduce or delay the onset of type 2 diabetes in the short term. These studies all concentrated on those already at high risk for developing type 2 diabetes mellitus, that is, those with impaired glucose tolerance.

Several large and many small intervention studies have taken place to try to reduce the rates of LDs. Many of these have used dietary and physical activity changes as the cornerstones of the intervention and many of them have shown reduction in risk and the development of new disease. Most used either individualised programs or counselling or individual counselling with group follow-ups. Most of these intervention studies have been with participants who are already known to be at risk.

There is no doubt that factors like diet and physical activity contribute hugely to "lifestyle" disease. How to encourage meaningful and sustainable change, particularly in those in whom biochemical and/or anthropometric risk factors are not yet measurable or recognised by the public, for example invulnerable youth, is the challenge.

5 Literature review –Choice of Design and Methodologies

This chapter looks at some of the ways of inspiring change and measuring their effectiveness and the rationale behind design of this study. Previously used methods of imparting knowledge and providing opportunities and choice are explored and what it is possible to measure with anthropometry, blood biochemistry and questionnaires is discussed.

5.1 Methods of imparting knowledge.

5.1.1 Group vs individual

A number of studies have looked at the outcomes of group delivered messages. Rickheim *et al.* (2002) measured the effectiveness of consistent evidence-based messages to deliver information to people with type 2 diabetes. They randomly assigned 170 participants to either a group or an individual program of four sessions over a 6-month period and assessed the changes in knowledge as well as behaviour and diabetic control (including weight and HbA1C). Participants in both groups had comparable improvements with the mean HbA1C being slightly better in those who had group instruction than those on the individual program. The group education program implemented as part of the Mediterranean Alpha-linolenic Enriched Grogigen Dietary Intervention (MARGARIN) trial (Bemelmans *et al*. 2000) successfully increased fish consumption in 124 men and 158 women who had multiple CVD risk factors. After two years the combination of supplying a margarine enriched in alpha- linolenic acid and group education resulted in an improvement in the total cholesterol to HDL ratio. Closer to home in New Zealand, Bell *et al.* (2001) used a one year diet and physical activity program in Samoan church communities. They increased reported physical activity by 10% in those involved in the program compared to a reported decrease in physical activity in the control group. Members of the intervention group lost weight while those in the control group gained weight. These studies highlight the efficacy of a dietary and physical activity intervention and the role that community group interventions can have.

In a study (Pyke *et al*. 1997) where married couples were given the same diet and physical information to lower CVD risk, 1 204 pairs of partners were followed up 1 year after their baseline screening and intervention program. Favourable changes in systolic blood pressure, BMI, cholesterol and glucose levels were greater in one partner when the other partner did well. Targeting a couple or family, rather than an individual may result in lower CVD risk through 'mutual reinforcement of lifestyle changes.'

Many nutrition health promotion programs are part of broader, multiple-risk-factor intervention programs such as the (Finnish) North Karelia project **(4.2.3).**It is often difficult to separate out the effect of the nutrition intervention from that of the other risk factor modifications. Providing choice and opportunities is important in any health promotion programme and allowing individual's to exercise self responsibility is likely to effect long term change when the active intervention stops. However, it is generally recommended that disease prevention programmes should be comprehensive, that is, encompassing more than intervention and targeting many risk factors or diseases.

But measuring change in all the above studies has been difficult. Attributing changes in blood lipid (or other) concentrations to one particular nutrient or even food is not possible in an uncontrolled environment and over a period of time when other factors may be effecting the result e.g. season change and aging. Assessment of self reported dietary intake is fraught with obstacles and therefore accurate comparison before and after interventions is not possible. At best a trend in individual and group dietary habits can be detected and associations found with risk reduction by anthropometric and biochemical measurements.

Measuring change in body proportions and composition will be considered first and then measurement of biochemistry change will follow.

5.2 Anthropometrics

Anthropometry is the measurement of the human body and usually comprises a series of non-invasive, inexpensive, and easy-to-perform methods for estimating body composition (bone, muscle and fat (adipose) tissue). However, they are operator

dependent and, to be useful clinically, must be performed in a precise, standardised, and reproducible manner. Several studies have shown linear correlations between anthropometric measures like waist measurements, weight/height^2 (BMI) and subscapular to triceps skin fold ratios with central body fat measurements (Durnin and Womersley 1974; Tran and Weltman 1989; Lean *et al*.1995; Lean and Han 2002:Zhu *et al*. 2002; Janssen *et al*. 2002a). Correlations have also been shown with mid upper arm circumference and waist measurements (Lean *et al*.1995; Manandhar *et al*.1997).

5.2.1 Body Mass Index (BMI)

Body mass index is an index of body size commonly used to classify people as being under weight, normal weight, overweight or obese. It has been shown that the BMI cut off values for the classifications varies among different ethnic groups (WHO Expert Consultation 2004).The use of BMI cut-offs is also limited by the fact that they are a measure of body size rather than body fatness. BMI does not take into account more muscular frames at different heights. Someone who lifts weights or engages in resistance exercises may, therefore, have an increased BMI due to an increase in lean body mass which weighs more than fat tissue. However, it is generally agreed that BMIs are a useful guide to body composition and that values equal to or greater than 32 kg.m^{-2} generally indicate an excess of adipose tissue and increased risk.

The current classifications (Swinburn 1998; WHO Expert Consultation 2004) are as follows and these would be used to define levels of risk by BMI in this study:

Table 5.1 BMI comparisons by ethnicity

	Ideal weight	Overweight	Obese
Europeans	<25 kg.m^{-2}	25 - 30 kg.m^{-2}	>30kg.m^{-2}
Maori/Pacific Islanders	<26kg.m^{-2}	26-32 kg.m^{-2}	>32kg.m^{-2}
Asian Indians	<22 kg.m^{-2}	22-25 kg.m^{-2}	>25kg.m^{-2}

.

5.2.2 Waist

Many researchers have confirmed that abdominal fat deposition measured anthropometrically by waist circumference is an indicator of cardiovascular risk (Despres *et al*. 2001; Doucet *et al*. 2002; Sharma 2002). One very large study was recently reported by Janssen *et al.* (2002). Fourteen thousand nine hundred and twenty four 14 924 participants in the Third National Health and Nutrition Examination, were categorised into groups by body mass index (BMI) and waist circumference (WC). The aim was to see if the prevalence of hypertension, dyslipidemia, type 2 diabetes and other metabolic risk factors was greater in those with high WC measurements compared with those of average WCs within the same BMI group. The biochemical measurements included total serum cholesterol, HDL and LDL cholesterol, triglycerides, and glucose levels. They also asked questions relating to age, ethnicity, socio economic background, and alcohol consumption, smoking and physical activity. They found that women who were normal weight (BMI 21.7±1.7 to 23.6±1.4), overweight (BMI 26.4 ±1.1 to 27.5±1.4) and obese (BMI 31.0 ± 0.7 to 32.0± 1.4) with high WC values had a greater health risk than those within the same BMI ranges with lower (normal) WC values. Men who had a high WC and were overweight (BMI 26.7±1.3 to 28.2±1.2) were at risk of CVD, whereas those who were normal weight (BMI 22.5 ± 1.7to 24.4 ± 0.7) with a high WC were at risk of type 2 diabetes and those who were obese (BMI 30.9± 98.2(sic) to 32.0 ± 1.3) with a high WC were at risk of metabolic syndrome (Janssen *et al*. 2002). This highlights the value of WC as well as BMI as an indication of LD risk.

5.2.3 Skinfolds

Skinfold thickness, measured by skinfold calliper, is a simple, reasonably accurate way of assessing body fat. Equations using four skinfolds (biceps, triceps, subscapular and suprailiac) to calculate percentage body fat were formulated by Durnin and Womersley in 1974 and have been used widely in research (Durnin and Womersley 1974). The accuracy of equipment, measurers and placement of the callipers in skinfold measurements has been a cause for concern to researchers. In 1997, Durnin and de Bruin measured four skinfolds on each of fifty-three women and forty-five men to see if

the exact sites of measurement affected validity of the estimation of percentage body fat. They measured skinfolds in the standard sites and then in sites about 2 cm away. This resulted in a 1-3% difference in body fat (Durnin *et al*. 1997). This difference is not significant for most research but it may influence results in research that looks at changes in body composition over a short period of time, such as this study.

5.3 Bioelectrical Impedance Analysis (BIA)

BIA is a fast, inexpensive, non-invasive method that is used to estimate body composition. It uses a small (800 micro amps) constant current at a fixed frequency (usually 50 kHz) to measure the opposition to the current flow through body fluids. The measurement utilises the principles relating to flow of electrical current and the concept that the essentially cylindrical human body can be divided into two compartments, lean and fat, with different electrical conducting properties. The lean compartment contains most of the body's water and is a good conductor of electricity. The fat compartment does not have much water and therefore opposes electrical current flow. Using these properties a value for impedance (opposition to alternating electric current flow) can be calculated from a fixed strength and frequency alternating current being passed through the body. In single frequency BIA measurements, surface electrodes are placed on the hand and foot and the voltage drop between electrodes provides a measure of impedance. Prediction equations based on total body resistance and using other variables like age, sex, ethnicity, standing height and weight, have been developed to predict total body water (TBW) and lean body mass (LBM) against a criterion measure of body composition such as underwater weighing or measurement of body water by deuterium dilution (Thomas *et al*. 1992; Liedtke 1997). Impedance is the vector sum of the resistance and reactance of body tissues. The phase angle is a linear way to measure of the relationship between conductive characteristics of body tissue (resistance) and the opposition due to the capacitance of cell membranes and other non ionic substances (reactance). The phase angle in body BIA measurements for a healthy person is about 3 to 10 degrees and varies with gender. Low phase angles may be indicative of cell membrane dysfunction and therefore may indicate illness or malnutrition (Thomas *et al*. 1992; Liedtke 1997; Ellis *et al*. 1999)

Placement of the electrodes for BIA measurements is very important in the accuracy and repeatability of the measurements. Other factors that can influence readings are blood viscosity, skin temperature, hydration status, recent dietary intake, pa and how long the subject has been supine

Multifrequency BIA uses several frequencies to measure impedance of extracellular fluid volume as well as total body fluid volume thus taking into consideration the imperfect capacitor attributes of the cell membranes. BIA measurements assume the relationship between the fat and fat free compartments of the body to be static. These can vary depending on age, sex, ethnicity and physical condition of the individual being measured. The hydration factor of fat free mass is reported as 73%, which is used for TBW calculations. Multi frequency impedance may be more sensitive to physiological variables like blood pressure or menstrual cycle (Chumlea and Guo 1994) but the machines are more expensive and not necessarily validated in the population of interest and will not be discussed further in this thesis.

BIA predicts TBW and fat free mass (FFM) with a 96-98% certainty in healthy adults according to Kushner *et al.* (1996). The more accurate measures of weight and height are and the more standardised factors like posture (e.g. how far apart the arms are), exercise and food intake prior to measurements and skin temperature are, the more precise the readings will be and the more they will reflect TBW and FFM. But changes in bioelectrical impedance may not accurately reflect changes in body composition in longitudinal studies where body composition changes or weight loss occurs. BIA predicts TBW and fat free mass (FFM) with a 96-98% certainty in healthy adults according to Kushner *et al.* (1996). The more accurate measures of weight and height are and the more standardised factors like posture (e.g. how far apart the arms are), exercise and food intake prior to measurements and skin temperature are, the more precise the readings will be and the more they will reflect TBW and FFM (Kushner *et al*. 1996). A statement from the National Institute of Health Technology Assessment conference said that BIA was not useful in measuring short-term changes in body composition (i.e., in response to diet or exercise) among individuals (Anonymous 1996). In the same year Houtkoper *et al.*, reported that single frequency BIA can

accurately measure acute and longer period changes in TBW in groups of subjects but not in individuals.

Not all authors agree that BIA cannot be used to assess body composition changes in an individual. A study by Powell *et al.* (2001) compared BIA measurements to underwater weighing (hydrodensitometry) in a group of overweight and obese premenopausal women who were taking part in a community-based weight loss program. They used a leg to leg BIA system and reported that BIA, skinfold measurements and underwater weighing all accurately detected decreases in fat free mass over the thirty-two week study. The ability to measure change with BIA was further validated in a study (Utter *et al*. 1999) using leg to leg BIA to assess body composition changes in obese women after a twelve-week hypocaloric diet, an exercise program or both. They concluded that underwater weighing and BIA measurements were comparable measures of body fat in all three scenarios ($r=0.78$, $p<0.001$). But another small study (Deurenberg *et al.* 1989) reported the measurement of body composition of thirteen obese premenopausal women who participated in an eight- week weight reduction study based on a hypocaloric diet. Body composition was measured before and after the study by BIA and hydrodensitometry. Their calculations of fat and fat free mass showed that BIA underestimated fat loss compared to densitometry. Dietary restrictions initially cause glycogen to be lost from the liver and skeletal muscle. Water is bound to this glycogen and is also lost. Loss of this glycogen and water may effect the density of the fat free mass explain why the BIA calculations of fat free mass after weight loss were different to those measured by densitometry. It also emphasises the fact that the bioimpedance equation used should be validated in the population and conditions that it is used in. Confirmation of this possible dependence of the accuracy of BIA on changing hydration states was reported by Fogelhom *et al.* in 1997 where in 32 obese premenopausal women overestimation of fat mass occurred more after weight loss than pre weight loss and seemed to be because of unexpected changes in the hydration of the fat free mass. In 37 men Ross *et al*. (1989) compared fat free mass and percentage body fat using BIA, skinfolds and hydrodensitometry. In an experimental group of 17 healthy men who lost weight over 12 weeks and a control group of 20 healthy men Lukaski equations were used for BIA conversions and Durnin equations for percentage body fat by skinfolds and it was concluded that in their hands BIA is a valid means of measuring changes in body composition with weight loss.

A cautionary note must be included. At the individual level Demura *et al.* (2002) concluded that percentage body fat calculated using any BIA measurement can be influenced by changes of body water caused by exercise, sweating, and drinking. It is therefore important to make sure subjects have not been exercising and have emptied their bladders before measurement of impedance to improve precision.

So, bioimpedance does seem to be a valid way of measuring fat loss especially in a group measured in the past. But, as the prevalence of obesity rises, the question of the accuracy of bioimpedance measurements of high body fatness is raised, especially when the validation was not in a similar population. This question was addressed by Deurenberg (1996) who looked at limitations of using BIA to measure changes in fat mass in obese and extremely obese people. He concluded that for very obese individuals BIA can be inaccurate for several key reasons. The very obese tend to have increased total body water. They also tend to have a different somatype - usually their cylinder shape bulges around the middle. Both the differences in hydration and irregular body shapes are contrary to the fundamental principles needed for BIA accuracy. Thus it should be considered that in very obese people loss of fatness may not be accurately measured with BIA.

Overall it seems that if bioimpedance, which measures total body fatness, is used in conjunction with other measures – weight, height, girths (particularly waist) and skinfolds, in a study designed to change body function and reduce risk, all these measurements would contribute to a better assessment of the interventions effectiveness in changing body shape, size and composition so long as the limitations of each method are also recognised.

5.4 Blood biochemistry

Chemicals present in the blood can reflect long term and short term changes in body function. Particularly if the blood is sampled when the person is fasting and preferably in the morning then chemicals such as glucose, insulin and lipid complexes can be very good assessments of risk and stability of control systems of the body related to lifestyle diseases.

A brief outline of the principles of a simple measurement of insulin resistance measurement follows:

5.4.1 Homeostasis Model Assessment (HOMA)

Body fatness and physical inactivity are related to increased resistance of the cells of the body to the action of insulin – i.e. a decrease in insulin sensitivity. Blood glucose levels usually remain exquisitely controlled within defined limits but insulin levels will initially rise as resistance increases and then as the beta cells of the pancreas become over stressed insulin will fall and glucose rise. The homeostasis model assessment (HOMA) is a non- invasive, inexpensive method of assessing insulin sensitivity using mathematical modelling (Hermans *et al*.1999). Its calculation requires the input of fasting insulin and glucose levels. HOMA relies on the interaction of basal plasma glucose and insulin concentrations i.e. the secretion of insulin in response to plasma glucose levels. HOMA accuracy has compared well with oral and intravenous glucose tolerance tests, euglycaemic and hyperglycaemic clamp in several studies (Matthews *et al*. 1985; Hermans *et al*. 1999; Bonora *et al*. 2000). Wallace and Matthews (2002) conducted an extensive assessment and literature review (1966-2001) of techniques used to obtain insulin resistance measures. They concluded that HOMA was an appropriate way to measure basal insulin resistance in large studies.

The HOMA method has the advantage for the subject, and also cost-wise, of a single fasting blood sample being required. Its strength is more for measuring group change rather than individuals. A MS-DOS-based computer program has been designed to determine beta-cell function (insulin resistance or HOMA B%) and insulin sensitivity (HOMA S%) (Levy *et al*. 1998) and Jonathan Levy (Oxford University) provided this programme to the research centre free of charge.

Insulin resistance has been implicated in the pathology of several metabolic disorders including impaired glucose tolerance (IGT), dyslipidemia and hypertension (Reaven 1995; Bonora *et al*. 2003). Insulin sensitivity in normoglycaemic individuals may provide an early detection method for those who are at risk of type 2 diabetes (Bonora *et al*. 2000). The Mexico City Diabetes Study and the San Antonio Heart Study were used

as basis to evaluate the use of HOMA in epidemiological studies. In both studies, Haffner and his team (Haffner *et al*. 1996; Haffner *et al*. 1997) found that HOMA was a useful tool to assess insulin resistance, HOMA B% and insulin sensitivity, HOMA S%, using only fasting plasma glucose and insulin levels. These two measures, decreased HOMA S% and HOMA B%, were good predictors for later development of type 2 diabetes mellitus.

5.4.2 Fasting plasma lipids, glucose and insulin.

Plasma lipids that have implications for lifestyle disease risk are total cholesterol, HDL cholesterol, LDL cholesterol, total cholesterol:HDL ratio and trigyceride levels (Despres *et al*. 2000; Kris-Etherton *et al.* 2001; Lemieux *et al*. 2001; Keevil *et al*. 2002) Total cholesterol, LDL cholesterol and HDL cholesterol are all used as predictors of CVD risk. But not all individuals with 'abnormal' levels of these biomarkers are unhealthy. Lipid levels and interactions are complex both as independent predictors of risk and in combinations. LDL cholesterol levels, particularly, have a large overlap between those with CVD and those who are healthy. The Quebec cardiovascular study followed 2 103 men, aged 56±7 years, for 5 years. They found HDL cholesterol to be an independent predictor of a first ischaemic heart event. The total cholesterol:HDL ratio was the best predictor of CVD events and the risk was lessened more by reductions in HDL than by increases in LDL cholesterol. Low HDL levels were often seen in conjunction with elevated triglyceride levels (Despres *et al*. 2000). Fasting plasma glucose and insulin levels are indications of possible risk of type 2 diabetes. They can also be used in HOMA equations to indicate insulin sensitivity and insulin resistance which are both measurers of diabetes risk

5.5 Questionnaires

In a study of free living people it is necessary and only possible to obtain information by asking questions. Interviewing can elicit good information but is time consuming, expensive and subject to interviewer bias. The type of questionnaire used needs to be determined by the type of analyses that will be used, the amount of time that the

participants have (responder burden) and the surety of the validity and repeatability of the data gained.

5.5.1 Food frequency Questionnaire (FFQ)

Methods like diet histories and diet diaries are time consuming and may lead to changes in usual dietary behaviour. They also are limited in that they only give a small snapshot of dietary intake. Twenty four hour recalls do not give enough information to indicate changes in dietary behaviour for intervention studies as daily diet varies markedly in an individual. Two weekdays and one weekend day are considered necessary to get valid information. Food frequency questionnaires are less burdensome and give a better overall picture but quantitation is difficult. However they have the advantage of giving a more clear indication of variety and specific foods consumed and not consumed over a period of time (Quatromoni *et al*. 2002) and can and should be tailored to the requirements of the study they are being used for.

Many studies have used FFQ as an indication of food intake but the reliability and validity of these questionnaires varies. In a comparison of dietary assessment methods in a low fat intervention program (Simon *et al*. 2001) the FFQ was found to be less precise than a 3-day food diary or 24 hour recall and correlation only for fat intake among methods was found and this was low (r=0.37). This has implications in the interpretation of dietary data and disease risk. Even in very large studies, such as the EPIC study Bingham *et al.* (2003), differences in dietary reporting by 7- day diary and FFQ are marked and these reflect only repeatability of the same diet story and not necessarily accuracy.

Different ways of setting out the FFQ and presenting the questions can also affect the results and accuracy. Thompson *et al*. (2002) looked at 4 design issues and concluded that the design of the FFQ could make a difference to the information obtained. The FFQ used in this study was in line with the recommendations of the Thompson study which were to group foods but to separate out different foods in the same group for example tomatoes and tomato juice and also list additions, for example salt, milk, sugar, separately. They also suggested that nutritionally different forms of a food, for example

milks with differing fat contents, should be 'nested'. This study also found that standard units were better than asking portion size.

More than one method of assessing dietary intake is recommended and the use of biomarkers is also necessary for validation. Tinker *et al.* (2001) found that different methods were susceptible to different biases and that all self-reporting was subject to some bias. Two methods of assessment would help to counteract this. A study by Kabagambe *et al.* (2001) showed that biomarkers did not indicate long-term dietary intake better than FFQs and that they should be used to complement each other.

Food frequency questionnaires have many shortcomings and have been shown to be inaccurate and unreliable in many studies. They are, however, easy to administer, inexpensive and require only a low responder burden. Carefully set out and used in a longitudinal study together with other dietary assessments and checks they can be useful in indicating a change in dietary behaviour.

For the purposes of the study reported in this thesis, a means of comparison at different points in the study rather than a comparison with recommended guidelines was desired. A food frequency questionnaire together with a questionnaire about perceived changes was used. The food frequency questionnaire was an adaptation of one developed by Metcalf *et al.* (1997) for use in New Zealand.

5.5.2 Physical activity questionnaires (PAQ)

Physical activity is difficult to assess. Questionnaires can give a good picture of patterns of perceived activity but do not indicate intensity or fitness. Intensity can really only be gauged by direct measurement e.g. with accelerometers as answers to questionnaires are very subjective.

There are many physical activity questionnaires available that have been validated for various aspects of physical activity in different studies. A study by Bonnefoy *et al.* (2001) validated ten previously developed physical activity questionnaires in older men (aged 73.4 ± 4.1years) using doubly labelled water to estimate total energy expenditure. Based on this these researchers strongly recommended the Stanford Usual Activity

Questionnaire for epidemiological studies in older people. The Stanford Usual Activity Questionnaire (Sallis *et al.* 1985) was developed to obtain physical activity information from community groups. It was designed to be practical and to be able to detect changes over time. It has been used in many epidemiological and longitudinal studies since its development in 1985.

5.6 Other health assessment methods

The Healthy Eating Index (HEI), the Alternate Healthy Eating Index (AHEI) (Kennedy *et al*. 1995; Kennedy *et al.* 1996; McCullough *et al*. 2002) and the Recommended Food Score (RFS) developed by Kant *et al.* (2000) all use a food frequency questionnaire to ascertain a score that indicates adherence to the Dietary Guidelines for Americans. These score do not consider some of the aspects that we wanted to look at in this study like oily fish, type of carbohydrate and physical activity levels.

Part of the planned intervention was to look at the prescription of a fruit, kiwifruit and to see if this had an affect on oxidation of DNA and lipids and also if antioxidants levels in the blood would change. This part of the intervention is not reported in this thesis but the principal of the comet assay for DNA damage/protection is outlined below.

5.7 Comet assays

The comet assay is "a microgel electrophoretic technique for the detection of DNA damage and repair in individual cells" (Phillips and Venitt 1995). Oxidative DNA damage is thought to play a role in many diseases including cancers. There are many nutrients that are considered to be antioxidants and levels of these, individual nutrients and synergies between them have been the subject of several studies (Crott and Fenech 1999; Lampe 1999; Pool-Zobel *et al.* 1999; Owen *et al*. 2000; Yamashita. 2000; Collins *et al*. 2001; Fleischauer *et al*. 2001; Morley and Trainor 2001; Fang *et al*. 2002; Meydani 2002).

Giovannelli *et al.* (2002) evaluated diet and lifestyle patterns on DNA oxidative damage in healthy adults in Italy. They performed comet assays on lymphocytes donated forty-eight males and 23 females aged between 35 and 64 years. They found that differences

in dietary patterns had very little effect on DNA oxidation. However Collins and his team (2001, 2003) have done several studies using comet assays to evaluate oxidative DNA damage. They have used kiwifruit as the antioxidant agent both *in vitro* and in a human intervention study with findings that show kiwifruit does stabilise DNA.

It was decided that the best way to test the hypothesis that kiwifruit would reduce possible DNA damage and oxidation was to do a cross over trial so the last six weeks of the study were devoted to this plus the continuing intervention

5.8 Summary

To summarise the design as informed by the evidence reviewed in this and the preceding chapter–

The study would be **longitudinal** over an **initial period of 12 weeks.**

A control group was ethically difficult to justify as volunteers would not be happy without any intervention and it would be very difficult and exceed the means of this study to offer an intervention to the control group at a later date – plus it is well known that just being in a study can influence behaviour.

Two sets of baseline measurements would be made three weeks apart. It was decided that 3 weeks was enough time for and effect of any intervention to stabilise.

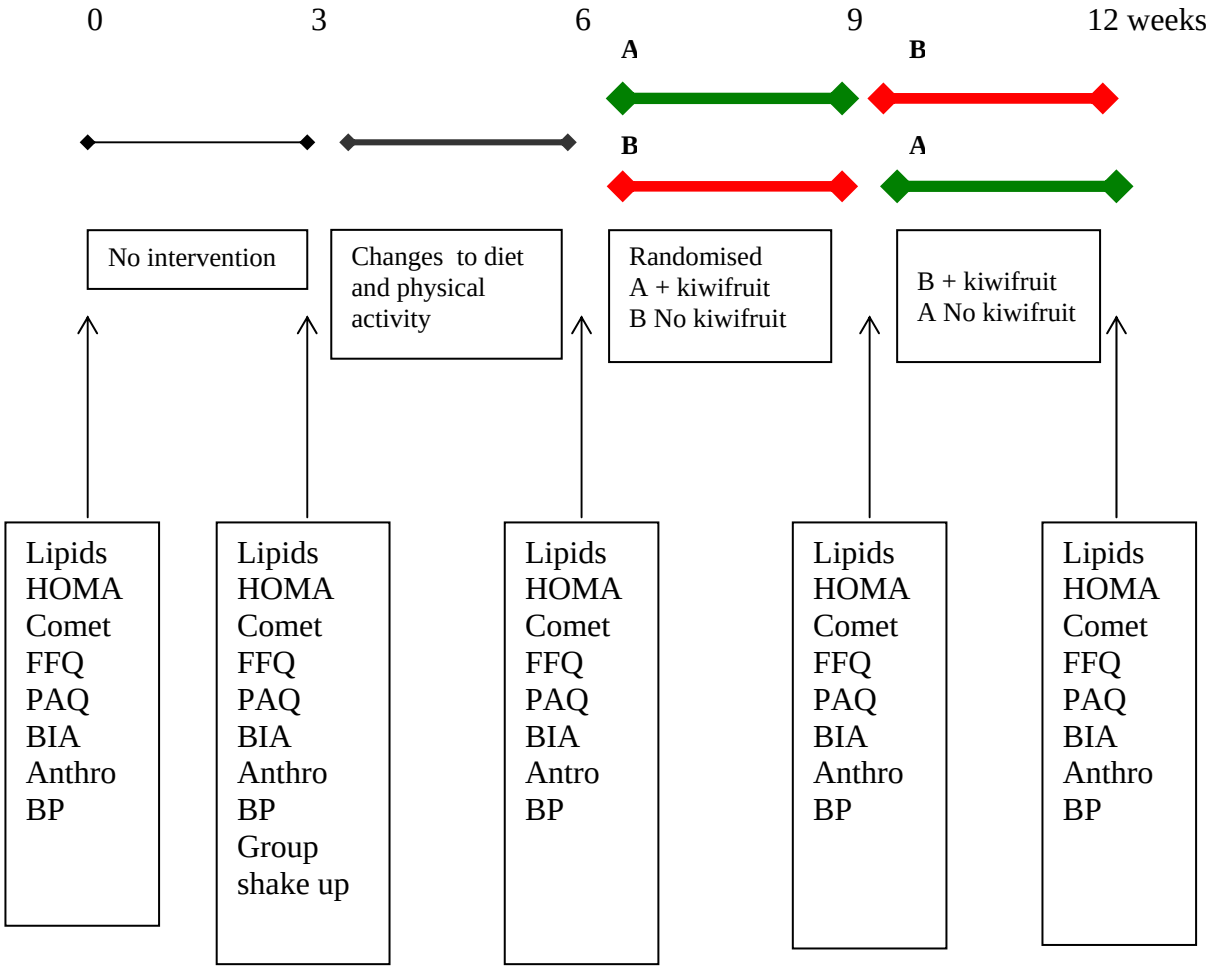
The **intervention** would be **by group** and the components of the intervention are summarised as follows (**figure 5.1**)

- A baseline set of measurements at week 0
- A 3 week period of following a usual pattern of diet and physical activity
- A set of measurements at week 3
- A diet and physical activity ‘shake-up’ and the issuing of pedometers in the week of the shake-up
- A 3 week period of following shake-up guidelines
- A further set of measurements at week 6
- Division of the group into two randomised groups- one to receive kiwifruit in a prescribed dose for 3 weeks and the other group to abstain from kiwifruit
- A set of measurements at 9 weeks

- A cross over of the two groups
- A set of measurements at week 12

Another follow-up measurement point became possible one year after the intervention which extended the study to **52 weeks**. Comet assays were not investigated at the 52 week point, otherwise all measurements were made. The figure (**Figure 5.1**) on the next page summarises the study design and measurements to be made at each point over the first 12 weeks.

Figure 5.1 Time line and design



Key
Lipids – lipid profile (blood tests)
HOMA – Fasting glucose and Insulin (measure of insulin resistance
Comet – comet assay for DNA damage
FFQ – food questionnaire
PAQ – Physical activity questionnaire
BIA – Bioimpedance Analyses
Anthro – Anthropometric measures
BP – Blood Pressure

6. Method

Ethical approval from the AUT ethics committee was sought and gained on 20 February 2002 (**Appendix 1a**). Later it was decided to extend the study to include a set of measurements and questionnaires after 12 months (to evaluate sustainability) and on 10 March 2003 ethics approval was extended to March 2005 (**Appendix 1b**).

This longitudinal study took place over a period of 12 weeks and measured healthy subjects aged 30 years or more who were recruited from AUT. The 12weeks were divided into five three-week sections (runs) and at the end of each section blood tests and measurements were done. At the end of the 12 weeks the participants were asked if they would like to be followed up after 12 months and all but one agreed. After 12 months with no follow up other than regular emails about diet and physical activity, they were recalled for a set of measurements and questionnaires.

6.1 Design

This twelve week longitudinal study was divided into four phases of three weeks each (**Figure 5.1**). During the first three weeks, participants underwent two sets (week zero and week three) of baseline measurements, at the beginning and end of the period. They were then asked to follow a diet and activity plan for the next three-week period at the end of which all measurements (week six) were repeated. The subjects were then randomised into two groups, one asked to not consume kiwifruit, the other asked to consume kiwifruit that was supplied to them weekly at the rate of one kiwi a day for every 30kg body weight. After a further three weeks (week nine) the treatments were crossed over and the subjects either consumed kiwifruit or not for another three weeks and the final measurements made (week 12). Fifty-two weeks later the measurements were repeated, week 52.

6.1.1 Recruitment

Sixty -three healthy participants, 33 male and 30 female, aged 30 years or more were recruited from staff at AUT by advertisement and personal contact. Participants who had diabetes mellitus, hypertension or were receiving treatment for high cholesterol were excluded. Fifty-eight participants came to the first week of measurements. Forty-two of these were New Zealand Europeans, six British, four Maori, three Samoan, two Tongan and one Indian. The attendance of the participants was spread over four days (Monday to Thursday) because of limitations in the number of blood tests that could be performed by available laboratory staff and limitations in equipment and trained personnel available for anthropometric measures. The participants were also restricted in the days they were available to be measured. They were divided into groups of about 15 per day but this varied due to participants changing their days because of other commitments or eating before coming for the fasting blood tests. All measurements of all participants took place in the same week.

Prior to the start of the study participants were given a list of dates they would be needed for measuring over the course of the study. At each visit they were reminded of the next measuring date. They were also sent out reminder letters and emails a week before and phoned the day before to remind them of their appointment and that they were required to come in a fasting state.

6.1.1.1 Retention

Fifty-eight (29 female and 29 male) were measured at the start of the study (week zero). This decreased to 53 for weeks three, six and nine (28 female and 25 male). Two of these were excluded because it was found that they had blood cholesterol levels that were high enough to require them to seek medical advice. One of these also had a fasting blood glucose level of 7.4mmol/L (ideal 4-5.6 mmol/L) and three dropped out for personal reasons. Over the 12 weeks of the initial part of the study seven participants did not attend all measurement points. There were 51 measured in week 12 (25 male, 26 female. The two absent participants were away overseas for an extended period. After 52 weeks with no follow up other than regular emails about diet and physical activity,

the participants were recalled for a set of measurements and questionnaires. At this recall 21 males and 15 females were remeasured. Three who dropped out were pregnant, one had moved and two declined the offer to be remeasured. One was sick. Nine said they were coming to be remeasured but despite several reminders and changing of days they declined to attend the follow up measurement session. The attendance and reasons for absence are summarised in **Tables 6.1** and **6.2**.

Table 6.1 Number of participants attending each measurement session

	Week 0	Week 3	Week 6	Week 9	Week 12	Week 52
Male	29	25	25	25	25	21
Female	29	28	28	28	26	15

Table 6.2 Reasons for non participation

(58 week 0)	Week 3	Week 6	Week 9	Week 12	Week 52
Total number measured	53	53	53	51	36
Away for an extended period/left AUT				2	3
Sick /medical advice needed	2				1
Pregnant					3
Declined offer of remeasurement	3				2
No excuse					7

6.1.2 Measurements

The measurements made were anthropometric measures (**5.2**), blood tests (**5.4**), BIA (**5.3**), blood pressure and dietary and physical activity questionnaires (**5.5**). The anthropometric measures were done by two trained and experienced measurers (ER and VC) and in most cases the participants were measured by the same person at all 6 measurement points. A trained, experienced phlebotomist from Diagnostic Medlab (Ltd) sampled blood. The questionnaires were self-administered but help was available if required. All the measurements were made at the Body Composition and Metabolic Research Centre at AUT. The blood was analysed for glucose and lipids at Diagnostic Medlab (Ltd), Ellerslie, Auckland and for insulin by A plus Laboratories (Lab+, Auckland).

Most measurements were made and blood sampled between 07h30 and 10h00 with participants in a fasting state. A few anthropometric measures were completed later in the day or on a subsequent day due to participant's time restraints. Most participants were measured by the same measurer at each measurement point.

Wherever possible BIA measurements and blood pressure and were done first; followed by weight, skinfold and girth measurements and then blood tests. Questionnaires were completed in-between wherever possible to try to keep the participants waiting time to a minimum.

Table 6.3 (page 80) is a list of equipment used and calibration methods followed. Each piece of equipment was calibrated and/or checked at the beginning of the study and the BIA machines, scales and skinfold callipers were calibrated and checked against each other before each measurement point.

6.1.2.1 Anthropometry

Duplicate measurements of height, weight, girths at waist, hip and mid upper arm circumference (MUAC) and triplicate measures of skinfolds at biceps, triceps, subscapular and suprailiac were made and averaged.

Heights were measured to the nearest mm using a wall mounted stadiometer (Mentone, Australia) using standard procedures. (Feet against the wall, head in the Frankton Plane) Weights were measured using Tanita W4357 and Tanita System 502 scales. The scales had been calibrated and checked by Wedderburn Scales Ltd (Mt Wellington, NZ) and were calibrated using pre weighed 1 litre (1000g) bottles of water up to 5kg before each measurement session. Weights were measured in kilograms (kg) taken to one decimal place.

Skinfolds were measured in mm with skinfold callipers that were either made by Harpenden (purchased from Mentone, Melbourne, Australia) or Holtain (UK). The same callipers were used on the individual participants at each measurement. Skinfolds were measured in four places (biceps, triceps, suprailiac and subscapular). The participants were asked to stand and the skinfolds were all measured on the right side of their bodies.

Figure 6.1 (below) shows the points at which skinfold measurements were made.
The biceps (1) measurement was taken at a point on the anterior surface of the biceps midway between the anterior acromion process and the antecubital fossa.
The triceps (2) was measured over the triceps muscle, halfway between the acromion process and olecranon process (bony process on elbow). The elbow was extended and the arm relaxed at the participant's side.
The subscapular (3) was measured on a diagonal fold one cm below the point of the shoulder blade and at an angle of 45º toward the arm

The suprailiac (4) was taken on a diagonal fold above the crest of the ilium at the spot where an imaginary line would come down from the anterior auxiliary line just above the hip bone and two - three cm downward.

Figure 6.1 Points at which skinfold measurements were made .Adapted from http://www.assist.co.uk/harpenden/HPSection3a.htm

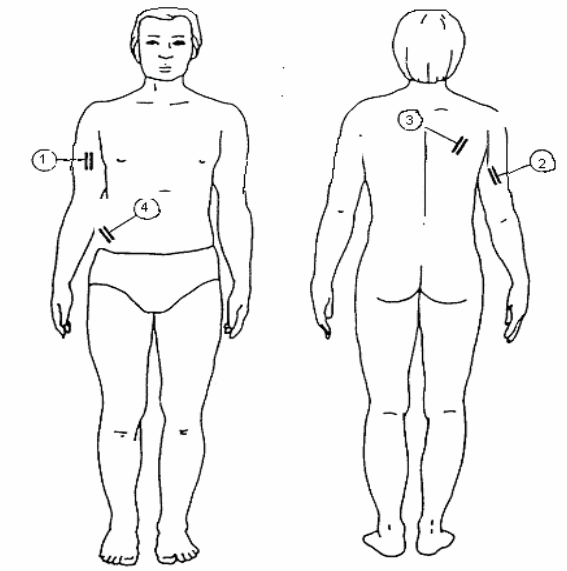


Table 6.3 Equipment used with supplier and calibration dates

Measure	Equipment	Model, manufacturer	Calibration method	Calibration dates
Weight	Scales	Tanita W4357 (Wedderburn Scales Ltd, Auckland, NZ sales@weddeburn.co.nz) Tanita System 502 (Wedderburn Scales Ltd, Auckland, NZ)	Both scales calibrated by Wedderburn Then checked with 5x 1Litre water (i.e. 1kg, 2kg, 3kg, 4kg, 5kg)	18/07/02 26/07/02, 15/08/02 06/09/02, 27/09/02, 18/10/02 26/07/02, 15/08/02 06/09/02, 27/09/02, 18/10/02
skinfolds	Callipers	Holtain (Holtain Ltd., Crosswell, Crymych, Pembs.SA41 3UF.U.K.)	Measurement of 1,2,3,4,5,10,15,20,25,30,35,40 1mm microscope slides	26/07/02, 15/08/02 06/09/02, 27/09/02 18/10/02
Waist, hips	Tape measure	Figure Finder Novel Products Rockton, IL (PE24 Mentone, Mel, Aus) http://www.mentone-educational.com.au	With tension gauge	
BIA	SEAC IMP5 Single frequency BIA	Impedimed Pty Ltd PO Box 2121 Mansfield Qld 4122, Australia enquiries@impedimed.com	Using block 409 and 1000 ohms	26/07/02, 15/08/02 06/09/02, 27/09/02 18/10/02
Blood Pressure	Mercury sphygmomanometer	Accosan mercurial AC Cosser and son (surgical)Ltd Accosan works, Vale Rd, London N4WPS		12/07/02 Calibrated by BV Medical (Bill Venendael Akl 5202962)
Height	Wall mounted stadiometer	PE27 Mentone, Melbourne, Australia http://www.mentone-educational.com.au		

80
80

Mid upper arm circumference (at the circumference joining the two midway points used for the biceps and triceps skinfold) was measured at weeks 3, 6, 9 and 12.
Waist measurements (midway between the lower rib and the iliac crest) and hip measurements (at the maximum protrusion of the gluteal muscle) were made using a Figure Finder tape measure with a tension gauge (UK) purchased from Mentone, (Melbourne, Australia) and were measured to the nearest 0.1 of a centimetre.

6.1.2.2 Blood measurements

Participants reported to the body composition and metabolic research centre (BCMRC) in the morning in a fasting state. A trained phlebotomist from Diagnostic Medlab (Ltd) took blood samples. The samples were sent to Diagnostic Medlab laboratories and measured for lipids (triglycerides, HDL, LDL and total cholesterol) and glucose. Lipids were by standard Roche-Hitachi methodology and HDL was done by direct assay. Glucose was measured by the Roche Hitachi glucose oxidase method. All assays were within target limits specified by the RCPA Quality Assurance Program. The laboratory has continuous IANZ ISO9002 Accreditation, both for laboratory and supplier status.

An EDTA tube of blood was centrifuged by a trained student and the plasma stored at -85º C. The plasma sample was taken later to Lab+ (at Auckland Hospital) to be measured for insulin levels using the Abbot IMx Insulin assay (list No2A10, Abbot Laboratories, Japan).

Insulin resistance and sensitivity was later calculated using the HOMA index algorithm (Hermans *et al*.1999) derived from the fasting glucose and the insulin levels (using a DOS algorithm supplied by Jonathan Levy, Oxford University). A team trained by the cancer research department at Auckland University prepared slides from heparin tube blood for comet assays (a measure of DNA strength and therefore cancer risk) performed as part of another study. The rest of the plasma samples were kept frozen at –85 ºC for possible further analysis (for other studies) at a later date.

6.1.2.3 Bioelectrical impedance analysis (BIA)

BIA was done using Impedimed IMP5, single frequency BIA (Impedimed Pty Ltd, Queensland Australia)

The participants were asked to have empty bladders and the measurers tried to have the participants in a supine and still position for a few minutes before doing the BIA measurements. Note was taken of menstrual cycle in the women. None of the participants were severely obese. Some of the participants had very cold extremities and needed to have them warmed so that the skin impedance was low enough to enable satisfactory measurement. Resistance, reactance, impedance and phase were recorded and percentage fat mass calculated using the equation of Kushner (Kushner *et al*. 1992). BIA measurements were made by the two trained measurers (ER and VC). Each measurement was repeated three times and an average taken for body fat calculations.

6.1.2.4 Blood pressure

Systolic and diastolic blood pressures were measured on the right arm at each of the measurement points using mercury sphygmomanometers that had been calibrated by Bill Venendael at BV Medical, Auckland, NZ two weeks prior to commencement of the study. The participants were supine for at least five minutes before measurements were taken to ensure that the blood pressure was resting.

6.1.2.5 Questionnaires

The dietary questionnaires used were a food frequency questionnaire (FFQ); questionnaires relating to the goal sheets participants were given at the 'shake-up' and a question about the effects of the kiwifruit (**Appendices 4, 6a, 6b, 7**). The FFQ used was an adaptation of the FFQ used by a study in New Zealand by Metcalf *et al*. (1997) and by the Children's Nutrition Survey in New Zealand (Metcalf 2003). The FFQ was adapted by regrouping some of the fruits and adding some low fat products not included

on the original FFQ. Each participant completed this questionnaire at each of the six measurement sessions. These were used to assess changes in diet over the period of the study.

At weeks three, six, nine and twelve a physical activity questionnaire (modified from the Stanford Usual Activity Questionnaire to include pedometer readings was filled out (**Appendix 5).**

In addition to these questionnaires a goal setting question sheet (**GSQ1, Appendix 6a**) detailing perceived changes was given at the fourth session (week nine) and a questionnaire with changes made and intention to continue changes (**GSQ2, Appendix 6b**) was given at session five (week 12). This GSQ also had questions about what prevented or motivated the participants to make changes and suggestions for changes to their workplace that may increase the opportunity for improving diet and physical activity. A question regarding the effects of the inclusion of the kiwifruit was included after the three weeks of kiwifruit eating. The questionnaire about changes made (GSQ1) was also included at session six (week 52).

6.1.3 Forms

All forms were colour coded to make it easy to see which measurement week they belonged to. Slightly different shades of the same colour were used for the protocol sheet, food frequency questionnaire and physical activity questionnaire so that they could be easily identified.

6.1.3.1 Protocol sheet (Appendix 2)

At each session a protocol sheet was filled in with participant number, run number (week), date of birth and measurements. At the first session details regarding family history, smoking history, alcohol consumption, health status and medication was recorded. Changes in health and medication were noted at each measurement point. The stage in their menstrual cycle was noted at each point for female participants.

Measurements noted on this sheet were the anthropometry, skinfold measurements, BIA readings and blood pressure readings. The person doing the measurements and the type of skinfold callipers and BIA machine number were noted on the sheet to ensure that they were the same for all measurements.

6.1.4 Diet and exercise "shake- up"

The diet and physical activity "shake-up" was the session held during week three to inform participants about the changes they could make to reduce their risk of lifestyle disease.

A group approach was used to disseminate advice on diet and physical activity. Due to time restraints for the participants three group sessions were needed. The information was presented in a PowerPoint presentation to minimise differences in each presentation. The participants were given a booklet outlining the dietary advice (**Appendix 8**) and also a booklet of recipes (**Appendix 9**) that would help with the changes suggested. The information given was taken from evidence-based sources (chapters **2, 3, 4**) and collated to suit the participants' broad background (employed, educated).

The Powerpoint™ presentation at the diet and physical activity "shake- up" told the participants what the lifestyle diseases were and the strategies used in the study to try to reduce their risk (**Appendix 11**). The booklet was explained and the participants were given information to work out their own dietary plan and tools for goal setting. This enabled non tailored information to be adapted to individual needs (Kreuter *et al*. 2000)

One aspect emphasised in the diet shake up was portion size. The quantity of food we eat has a major influence on the energy we consume and portion increases in both fast foods and home eating has played a part in the obesity epidemic. The portion sizes in the recommendations in the food pyramid for example are much smaller than the serving sizes we have become used to (Rolls *et al*. 2002; Volker *et al.* 2003).

The main messages and how to accomplish them was explained (**3.3**). Other tips like eating frequently, moderate alcohol consumption and being careful of products that may be low in fat but high in sugar or vice verse were given (**Appendix 8**)

A display of food containers and information sheets illustrating the advised changes was set out at each session. They were also issued with a pedometer (Digiwalker sw-700) to help motivate them increase their physical activity. They were shown how to set the pedometer to their step length and shown how to use it. It was recommended that they aim to do a minimum of 10 000 steps per day. (Tudor-Locke 2001; Iwane 2000) The advice given to participants was given as a group and therefore needed to be prudent and broad- based. It also needed to be doable. The dietary changes concentrated on were an increase in dietary fibre (by increasing fruit and vegetables and whole grains), an increase in omega- 3 fatty acids (mainly by increasing oily fish) and a decrease in saturated fats and dietary sodium. Portion size was discussed and the differences in portion size, as per the dietary guidelines, and serving size, as per a typical meal, were highlighted. Mentorship within the group was encouraged

6.1.4.1 Booklets

Two booklets were handed out at the shake up sessions. Copies of these are in **Appendices 8 and 9**.

The first was a booklet containing partially individualised dietary information, hints for improving diet and physical activity and goal sheets to help motivate participants to consider what changes they would make and to implement them.

An exchange group method of meeting individual dietary goals was given with the number of exchanges or portions from each food group being determined by the individuals BMI and physical activity as indicated by the FFQ and PAQ from week 0. The calculations for these diets can be found in **Appendix 10**. The dietary advice was based on 50-60% energy from carbohydrate, 15-18% energy from protein and 24-27% energy from fat. The food groups used for exchanges were based on the USDA food

pyramid and the hints and suggestions gathered from many reliable sources and the researchers' clinical experience. Potato and other root vegetables were placed in the 'bread and cereal' group reflecting their high carbohydrate content compared to other vegetables.

The second booklet was a book of recipes to help implement dietary changes like lower fat and the use of less used ingredients like legumes, beans and oily fish.

6.1.5 Kiwifruit

A cross over design was used over weeks six to nine and nine to twelve using kiwifruit as the prescription for reducing some of the risk factors. The dose, one kiwifruit per 30kg body weight, used for this study was based on the dose used in the Rush study (Rush *et al*. 2002) because it was the source of the anecdotal evidence of cholesterol lowering and was known to be a comfortable amount for even the heaviest participants to consume. Kiwifruit was delivered to AUT weekly over the period of the kiwifruit trial. Enough kiwifruit for the week (based on the dose they were having daily) were packed into bags for each individual and delivered to their work site with instructions for consumption (amount per day, preferably spread over the day).

6.1.6 Statistics

All data in the text are expressed as means ±standard deviation unless otherwise stated. Statistical comparisons of measurement points were made by repeated measure ANOVAs (divided by sex) and multivariate analysis of covariance adjusting for ethnicity, sex and BMI A combination of SigmaStat Statistical Software for Windows (2.03), Microsoft Excel 97 and SPSS 11.5.1 for Windows programmes were used to manipulate the data and perform the analyses. The first 5 measurement sessions were looked at on an intention- to –treat basis and for the last measurement session (week 52) only the thirty-six participants that returned were compared to previous results. The

cross over analysis for the kiwifruit was analysed using unpaired t tests using the methods of Hills and Armitage (1979). Associations in biochemistry measurements and the main food frequency messages were examined using Pearson product moment correlation.

7. Results

Fifty- eight participants were recruited but only 53 of these were measured at the first four measurement sessions and 51 at the fifth session. Thirty-six returned after 52 weeks. For all the statistics during the initial 12 week period (five measurement points) comparisons were made using 53 sets of values on an intention to treat basis and the last two sets of values (week 12 and week 52) were compared using only the 36 participants who returned to be measured (**Table 7.1** and **6.1.1** recruitment).

7.1 Participants

Table 7.1 Participants attendance for measurements

	Week 0†	**Week 3**	**Average wk 0, 3**	**Week 6**	**Week 9**	**Week 12**	**Week 12 ‡**	**Week 52**
Number female	28	28	28	28	28	26	15	15
Number male	25	25	25	25	25	25	21	21
Total	53	53	53	53	53	51	36	36
mean age (years)	46	46	46	46	46	47	46	46
(SD)	(± 8)	(± 8)	(± 8)	(± 8)	(± 8)	(± 9)	(± 9)	(± 9)

† Week 0 has only the 53 participants measured in weeks 3, 6, 9 and 12 because the five that were measured in week 0 but not in subsequent weeks had very high values that would skew the results. ‡ Results for 36 participants measured at week 52

The first nine weeks had three more female than male participants. At week 12 two female participants were away for an extended period. After 52 weeks 58% of the participants were males. Three of the female participants were excluded from the final measurements because they were pregnant. If these three are added back to the sample, there was a 35% loss of females and a 16% loss of males. The mean age of participants was 46 years.

The ethnicity of the 53 participants included in the statistics for weeks 0 to 12 is shown in **Table 7.2**.

Table 7.2 Ethnicities of participants by self identification weeks 0 to 12

Ethnicity	Number (n)
New Zealand European	39
British	6
Maori	3
Tongan	2
Samoan	2
Indian	1
Total	53

Thirty nine of the fifty three participants for whom results are reported in weeks 0, 3, 6, 9 and 12 were of New Zealand European origin. Six were of British extract. Three identified themselves as Maori, two as Tongan, two as Samoan and one as Indian. The fifty-three participants taking part in this study had a variety of biological risk factors as measured in weeks 0 and 3 of the study (**Table 7.3**).

Table 7.3 Pre-existing risk factors

Risk factor	Number of participants n	Percent %
Systolic BP >140 mmHg	4	8 %
Diastolic BP >90mmHg	4	8 %
Fasting plasma glucose >6.0 mmol/L	2	4 %
Insulin >7.1μU/mL	8	15 %
Total cholesterol >5.0 mmol/L	37	70 %
HDL cholesterol <1.0 mmol/L	1	2 %
LDL cholesterol >3.0 mmol/L	38	72 %
Triglyceride >2.0 mmol/L	5	9 %
Total/HDL ratio >4.5 mmol/L	21	40 %
%body fat by BIA >25% male;>30% female	39	74 %

Three of the four participants with high systolic blood pressures (> 140mmHg) also had high diastolic blood pressure (>90mmHg). One participant had a systolic BP of 142 mmHg and a diastolic of 84 mmHg and one had a systolic of 130 mmHg with a diastolic of 98mmHg.

All of those with elevated total plasma cholesterol (>5.0 mmol/L) had elevated LDL cholesterol (> 3.0 mmol/L) and one participant had a high total cholesterol, high LDL cholesterol and low HDL cholesterol. One participant had high LDL cholesterol but an acceptable total cholesterol level. All five participants with elevated triglyceride levels (> 2.0 mmol/L) also had high total cholesterol levels and high total cholesterol:HDL levels.

Five of the fifty-three participants smoked moderately (1-15 cigarettes per day). One male reported that he consumed about a bottle of whisky daily and seven either drank the odd glass of wine at social occasions or did not drink alcohol at all. Thirty-four drank wine regularly (1 glass per week to 1-2 glasses per day); ten drank between one and six bottles (330ml) beer per week and five drank spirits (1tot to1 bottle per week).

At the first set of measurements (week0), details about family history of lifestyle diseases were noted. **Table 7.4** shows the number and percentage of participants who reported a family history of one or more of the diseases looked at in the study.

Table 7.4 Family history of lifestyle diseases

Lifestyle disease	**Number**	**Percentage**
	n	**%**
None	14	26
Hypertension	8	15
Cardiovascular Disease	10	19
Diabetes Mellitus (all type 2)	16	30
Cancers	19	32

- does not total 100% as some families had more than one risk factor

Seventy- four percent of the participants had at least one lifestyle disease in their family history with nine participants having more than one lifestyle disease in their family history and one having both a maternal and paternal history of type 2 diabetes.

7.2 Anthropometric measurements

Combined anthropometric measurements are summarised in **Table 7.5**. Separate anthropometric statistics for males and females showed the following:

-The mean waist measurement of the males was significantly higher than that of the females (week 0 – male 93.7±9.8 compared to female 88.7±12.3 ($p=0.032$)) and the significant difference between waist measurements at weeks 3 and 5 was due to a significant increase in male waist measurements ($p=0.044$) but not female waist measurements ($p=0.078$).

-The mean hip measurement of females was significantly higher than that of the males ($p=0.001$) and the significant difference between mean hip measurements at weeks 2 and 5 was due to a significant increase in male hip measurements only ($p=0.003$)

-Differences in MUAC at week twelve compared with weeks three and six were also only significant in the males

-Percentage body fat by BIA was compared to percentage body fat by calculation from skinfold measurements There was a strong correlation between % body fat by skinfolds and % body fat by BIA on all 6 occasions ($r=0.87$-0.9; $p<0.05$)

Table 7.5 Anthropometry for all subjects

	Week 0 n=53	**Week 3** n=53	**Average wk 0 and 3**	**Week 6** n=53	**Week 9** n=53	**Week 12** n=53	**p* RMANOVA**	**Week12‡** n=36	**Week 52** n=36	**p† t test**
Mean weight (kg) (SD)	80.2 (± 14)	80.3 (± 14)	80.3 (± 14)	80.2 (± 13)	80.3 (± 13)	80.2 (± 13)	0.988	80.2 (±13)	81.1 (± 15)	0.866
Mean height (cm) (SD)	171.9 (± 9.2)	171.9 (± 9.2)	171.9 (± 9.2)	171.9 (± 9.5)	171.9 (± 9.2)	172.0 (± 9.1)	N/A	172.5 (± 9.5)	172.5 (± 9.5)	N/A
Mean BMI (kg.m^{-2}) (SD)	27.2 (± 4.8)	27.2 (± 4.7)	27.2 (± 4.7)	27.2 (± 4.6)	27.3 (± 4.6)	27.2 (± 4.6)	0.331	27.0 (±4.5)	27.0 (±4.8)	0.686
Mean subscapular (mm) (SD)	25.9 (± 12.0)	26. 0 (±11.7)	26.1 (±11.7)	26.9 (±11.6)	26.4 (±12.2)	25.9 (±12.5)	0.062	25.3 (±12.0)	25.6 (±13.0)	0.853
Mean Scp/tcp (SD)	1.2 (±0.43)	1.2 (±0.43)	1.2 (±0.48)	1.2 (±0.43)	1.3 (±0.45)	1.3 (±0.43)	0.324	1.4 (±0.21)	1.4 (±0.28)	0.740
Mean suprailiac (mm) (SD)	25.8^{a} (± 11.2)	25.6^{b} (±12.1)	25.7^{c} (± 11.6)	25.6^{d} (± 11.1)	25.5 (±10.8)	24.1^{abcd} (±10.8)	0.007**	22.0 (±9.6)	24.4 (± 10.04)	0.250
Mean waist (cm) (SD)	91.1 (± 11.1)	90.8 (± 11.3)	91.0 (±11.3)	90.4^{a} (± 11.3)	91.3 (± 10.9)	91.8^{a} (±11.8)	0.005 **	92.2 (±10.2)	91.4 (± 10.68)	0.892
Mean hips (cm) (SD)	105.8 (±10.1)	105.1^{a} (± 10.3	105.4 (±10.1)	105.1 (± 10.0)	105.9 (±9.6)	106.0^{a} (±9.6)	0.008**	105.0 (±9.6)	105.1 (±9.6)	0.870
Mean MUAC (cm) (SD)	Not done	32.6^{a} (± 3.9)	32.6 (± 3.9)	32.6^{b} (± 3.9)	32.6 (±3.8)	32.9^{ab} (±3.7)	0.005**	32.8 (±3.3)	34.2 (± 3.6)	0.350
Mean % body fat (BIA) (SD)	31.9^{acdef} (±8.5)	34.1^{ab} (±8.2)	33.0^{bf} ±8.3)	33.6^{d} (±8.6)	33.8^{c} (±8.4)	33.5^{e} (±8.6)	<0.001**	(±)	32.7 (±8.3)	0.709

^{a,b,c,d,e} P<0.050 for weeks in the same category with the same letters p* RMANOVA weeks 0,3,6,9,12 **p<0.05

p† t test weeks 12 and 52 (for the 36 participants measured at both weeks only)

7.3 Obesity risk factors

Table 7.6 Weight classification of participants using BMI cut off points suggested by WHO (2004) with ethnic considerations

	week 0 * n	**percent**	**week 3*** n	**percent**	**week 6*** n	**percent**	**week 9*** n	**Percent**	**week 12*** n	**percent**	**week 52*** n	**percent**
Normal	21	40 %	21	40 %	21	40 %	21	40 %	22	42 %	17	47 %
Over weight	18	34 %	18	34 %	19	36 %	18	34 %	19	36 %	9	25 %
obese	14	26 %	14	26 %	13	25 %	14	26 %	12	23 %	10	28 %
Total	53	100 %	53	100 %	53	100 %	53	100 %	53	100 %	36	100 %

* number of participants

Table 7.6 describes the classification of the participants by BMI uses ethnic specific cut-off points **(5.2.1; Table 5.1)** Over the initial 12 weeks of the study there was a small shift of BMI values from the obese range (2 participants) to the overweight (1 participant) and normal ranges(1 participant). There appears to have been an increase in the number of obese participants from weeks 12 to 52 and a decrease in the number of overweight participants. If the participants who failed to come back to the last measurement session are removed from week 12, then 28 % are obese at both measurements, 33% are overweight at week 12 compared with 25% at week 52 and 39% are a healthy weight at week 12 compared with 47% at week 52.

When adjusted for sex (average week 0 and week 3) it was found that 32 % of the males and 32% of the females were 'normal' weight, 56% of the males and 29% of the females were overweight, and 12% of the males and 39% of the females were obese

Table 7.7 Waist, percentage body fat and BMI above "healthy" levels for males and females

		Week 0* n	**Percent**
Waist	M>100	5	20 %
	F>88 cm	13	47 %
%BF**	M>25%	13	52 %
	F>30%	26	93 %
BMI***	M	17	68 %
	F	19	67 %

* number of participants

** % body fat by BIA

*** BMI for overweight and obese taking ethnic differences into account

Almost 70 percent of both genders were classified as being either overweight or obese using BMI cut off values suggested by WHO (WHO Expert Consultation 2004) and adjusted to ethnic differences. Ninety-three percent of the women and just over half the men had fat mass in excess of the amounts considered to minimize risk of the LDs reviewed. Nearly half the women and one fifth of the men had waist measurements that indicated that their abdominal fat stores were placing them at risk of CVD and type 2 diabetes.

7.4 Hypertension

Table 7.8 Blood pressure categories of participants week 0 based on JNC-V (**Figure 2.1**)

Blood pressure JNC-V hypertension categories	**systolic** mmHg	**number** n	**percent**	**diastolic** mmHg	**number** n	**percent**
Normal	<130	46	87 %	<85	42	79 %
High normal	130-139	3	6 %	85-89	7	13 %
Hypertension stage 1	140-159	4	8 %	90-99	3	6 %
Hypertension stage II-IV	>160	0	0 %	>100	1	2 %

Table 7.9 Mean Blood Pressure measurements and SD at weeks 0, 3, 6,9,12 and 52

	Week 0	**Week 3**	**Average**	**Week 6**	**Week 9**	**Week 12**	**p***	**Week12‡**	**Week 52**	**p ˝**
	n=53	n=53	**wk 0 and 3**	n=53	n=53	n=53	**RMANOVA**	n=36	n=36	**t-test**
Systolic mmHg†	117.2	120.3	118.7	117.2	118.4	117.6	0.132	117.5	121.6	0.149
(SD)	(± 1.8)	(±1.7)	(±1.6)	(±1.7)	(±1.6)	(±1.8)		(±13.1)	(±13.2)	
Diastolic mmHg	77.1	75.8	76.4	75.2	76.1	76.1	0.606	75.2	76.9	0.374
(SD)	(±1.1)	(±1.2)	(±1.1)	(±1.0)	(±1.4)	(±1.4)		(±10.1)	(±8.7)	

† Mean values

‡ for the 36 participants that returned to be measured at week 52[a,b,c,d,e] P<0.050 for weeks in the same category with the same letters *p RMANOVA weeks 0,3,6,9,12 ** p<0.05

p ˝ t test weeks 12 and 52(for the 36 participants measured at both weeks only)

Table 7.10 Reported number of times salt consumed at weeks 0, 3, 6, 9, 12 and 52

	Week 0 n=53	**Week 3** n=53	**Week 6** n=53	**Week 9** n=53	**Week 12** n=53	**p RM ANOVA**	**‡Week 12** n=36	**Week 52** n=36	**p‡ t-test**
Average number of times per week(SD)	4.76 (3.56)	4.87 (3.88)	3.69 (3.72)	3.98 (3.52)	3.91 (4.16)	0.099	4.38 (3.52)	4.10 (4.12)	0.760

‡ for the 36 participants that returned to be measured at week 52

[a,b,c,d,e] P<0.050 for weeks in the same category with the same letters *p RMANOVA weeks 0,3,6,9,12 ** P<0.05

p‡ t test weeks 12 and 52(for the 36 participants measured at both weeks only)

7.5 Cardiovascular disease

Table 7.11 Mean values and SD for cardiovascular risk factors weeks 0 to 12 and weeks 12 and 52

	Week 0 n=53	**Week 3** n=53	**Average wk 0,3**	**Week 6** n=53	**Week 9** n=53	**Week 12** n=53	**p* RMANOVA**	**Week12‡** n=53	**Week 52** n=53	**p˟ t test**
Total chol mmol/l (SD)	5.6^{abc} (±1.1)	5.4^{cde} (±1.0)	5.5 (±1.0)	5.3^{be} (±1.0)	5.5^{f} (±1.0)	5.3^{adf} (±1.0)	<0.001**	5.3 (±1.1)	5.3 (±1.0)	0.982
HDL mmol/l (SD)	1.5^{cd} (±0.31)	1.4^{bd} (±0.31)	1.5^{f} (±0.31)	1.4^{acef} (±0.30)	1.5^{e} (±0.31)	1.5^{ab} (±0.31)	<0.001**	1.4 (±0.34)	1.5 (±0.31)	0.833
LDL mmol/l (SD)	3.5^{ab} (±0.97)	3.4 (±0.90)	3.5^{c} (±0.90)	3.4 (±0.94)	3.4^{b} (±0.91)	3.3^{ac} (±0.94)	<0.001**	3.4 (±0.99)	3.3 (±0.94)	0.536
Trigl mmol/l (SD)	1.3 (±0.7)	1.3^{c} (±0.7)	1.3 (±0.7)	1.2^{b} (±0.7)	1.4^{abc} (±0.8)	1.2^{a} (±0.6)	<0.001**	1.1 (±0.4)	1.3 (±0.5)	0.023**
tot/hdl (SD)	4.0^{c} (±1.1)	4.0^{b} (±1.1)	4.0 (±1.1)	4.0^{a} (±1.1)	3.9^{d} (±1.0)	3.7^{abcd} (±0.9)	<0.001**	3.8 (±0.97)	3.8 (±0.1)	0.849

^{a,b,c,d,e} P<0.050 for weeks in the same category with the same letters

P* RMANOVA weeks 0, 3 ,6, 9, 12 ** P<0.05

‡ for the 36 participants that returned to be measured at week 52

p˟t test weeks 12 and 52(for the 36 participants measured at both weeks only

All measurements were analysed separately by sex but further changes were not detected and only combined results are reported

The majority of participants (87%, **Table 7.8**) had systolic **blood pressures** that were considered normal according to the Joint National Committee report in 1993. Seventy-nine percent had normal diastolic blood pressures. Eight percent were hypertensive and a small number (2 systolic, 4 diastolic) of participants were high normal. There were no statistically significant changes in either systolic or diastolic blood pressure measurements. (**Table 7.9**) Over the twelve-week period 6 participants lowered their systolic BP by 10mmHg or more and 2 of these maintained this decrease at 52 weeks

There was no significant decrease in salt consumption (**Table 7.10**). Reported salt added to food did decrease slightly but not enough to be statistically significant in the initial 12 weeks and the decrease was maintained over 52 weeks.

There were small but statistically significant changes in the risk factors measured as indicators of **cardiovascular risk** over the first twelve weeks of the study (**Table 7.11, Figure 7.1A)**. There was no significant change in these factors at weeks twelve and fifty-two other than an increase in the mean triglyceride level. ($p= 0.023$).

Total cholesterol decreased significantly between measurements at weeks 0 and week 3 and decreased again at week 6. It increased between weeks 6 and 9 and decreased again at weeks 9 and 12. This decrease was maintained over 52 weeks by the 36 participants who stayed in the study. Over twelve weeks fourteen participants decreased their total plasma cholesterol by 0.6-0.9 mmol/L and 4 decreased their total plasma cholesterol levels by 1.0 mmol/L or more. Six maintained the 0.6-0.99 mmol/L decrease and 2 maintained the 1.0 mmol/l or more decrease over the 52 weeks. Framingham scores calculated at week 0 and week 12 showed no significant difference in estimated percentage risk over 10 years but sustained change would have a positive effect over a long period

HDL cholesterol increased and decreased significantly over the course of the study but overall it was maintained over both 12 and 52 weeks. LDL cholesterol decreased significantly over the 12-week period and the drop persisted at 52 weeks.

Triglycerides increased significantly at week 9, decreased again at week 12 and went up significantly between weeks 12 and 52. The total cholesterol/HDL ratio decreased significantly at week 12 and was maintained between weeks 12 and 52.

.

7.6 Type 2 diabetes mellitus

Diabetes risk factors also tended to show a positive improvement. There was a small but significant decrease in fasting plasma glucose. There was a tendency for plasma insulin and HOMA S% to increase and HOMA B% to decrease over the first 12 weeks Plasma insulin and HOMA S% decreased at week 52 and HOMA B% increased but they were still much more positive than at the start of the study (Table **7.12**, figure **7.1B**)

Table 7.12 Mean values and SD for Type 2 diabetes risk factors weeks 0 to 12 and weeks 12 and 52

	Week 0 n=53	**Week 3** n=53	**Average week 0, 3**	**Week 6** n=53	**Week 9** n=53	**Week 12** n=53	**P* RMANOVA**	**Week 12‡** n=36	**Week 52** n=36	**P ˝ t test**
Mean glucose mmol/l (SD)	5.2[ab] (±0.1)	5.1 (±0.1)	5.1[cd] (±0.1)	5.1[c] (±0.1)	5.0[ad] (±01)	5.1 (±0.1)	0.003**	5.1 (±0.4)	5.0 (±0.4)	0.207
Mean insulin pm (SD)	58.42 (±28.93)	58.62 (±29.33)	58.52 (±29.13)	53.73 (±25.55)	57.94 (±27.55)	55.31 (±29.72)	0.369	50.54 (±21.50)	49.65 (±24.07)	0.869
Mean HOMA B% (SD)	92.3 (±28.5)	94.1 (±27.7)	93.2 (±26.5)	92.1 (±26.8)	97.9 (±27.8)	90.8 (±27.8)	0.285	86.0 (±25.8)	89.9 (±27.3)	0.779
Mean HOMA S% (SD)	109.0 (±45.5)	112.9 (±53.6)	110.9 (±49.5)	119.8 (±52.5)	116.5 (±65.0)	118.0 (±54.4)	0.063	125.7 (±56.3)	127.3 (±50.3)	0.957

[a,b,c,d,e] P<0.050 for weeks in the same category with the same letters

P* RMANOVA weeks 0,3,6,9,12 ** P<0.05

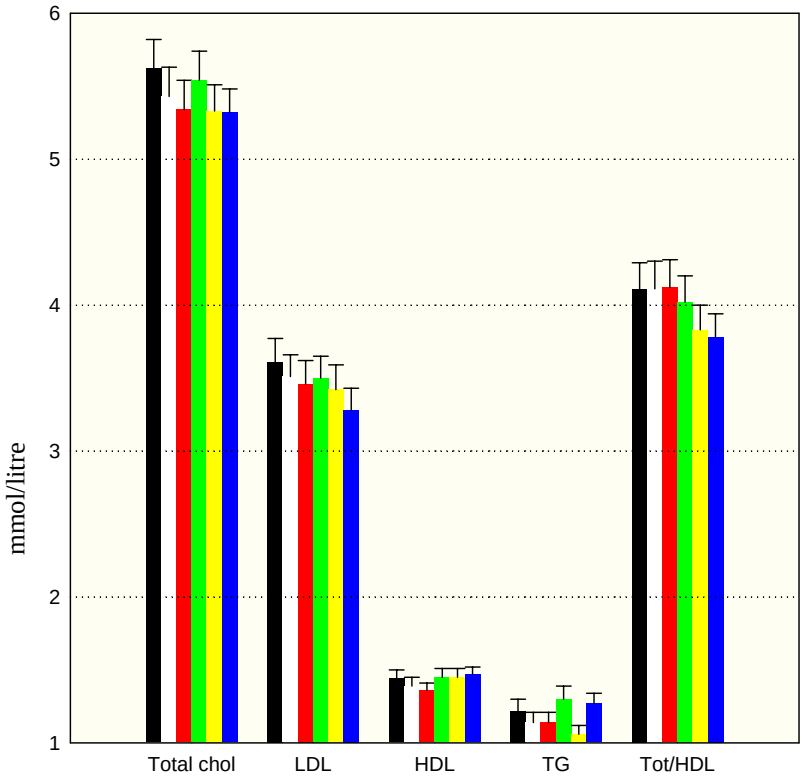
P ˝ t test weeks 12 and 52 (for the 36 participants measured at both weeks only)

‡ participants measured at week 52

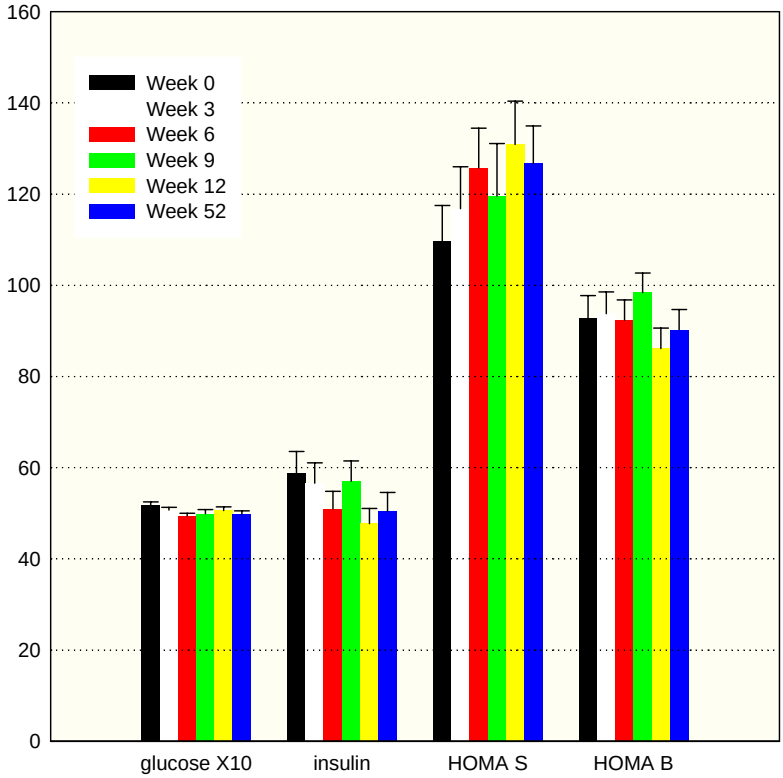
Figure 7.1 Bar charts (mean and SE) showing changes in lipid profile (upper graph A) and diabetes risk factors (lower graph, B) at each measurement point. The plasma glucose level (mmol/litre) has been multiplied 10 fold to show the decrease more clearly. Insulin units pm/litre and HOMA S and HOMA B are expressed as percentage

A

.



B.



7.7 Kiwifruit crossover.

The effects of the kiwifruit on blood lipids, blood glucose and insulin were looked at using the method of Hills and Armitage to statistically analyse cross over designs (Hills and Armitage 1979). No statistical evidence was found that kiwifruit influenced the plasma levels of the lipids, glucose or insulin. There was an indication that triglycerides may have been affected but this was confounded by a reported increase in fruit consumption between weeks six and nine, changes in fat consumption and a possible period effect. DNA fragility and antioxidants were (will be) analysed as part of other studies. Preliminary findings seem to indicate that the DNA strength (measured by comet assay) was positively affected by kiwifruit consumption. Kiwifruit crossover

7.8 Questionnaires

The data from the food frequency questionnaires were rearranged and collated to reflect the questions about fat and foods related to the goal sheets e.g. eat more fruit and vegetables related to change in fruit and vegetable consumption in the food frequency questionnaire. There was only a low correlation between these two qualitative instruments indicating that their memory / honesty regarding what they had eaten or changed was not good. The questions relating to the goal sheets and the answers given in the summary were also not very congruent. The accuracy of the Food Frequency Questionnaires (FFQ including physical activity) and Goal sheet questionnaires (GSQ) pose serious questions when they are compared to each other. These questionnaires were filled in at the same time but differ considerably in their answers as is shown by **Table 7.13**.

Table 7.13 Pearson moment correlation and significance comparing answers of FFQ and GSQ for all participants at week 12.

	FFQ vs GSQ (r)	**p value**
Less total fat	0.060	0.684
Less saturated fat	0.146	0.322
More whole grains	0.237	0.106
More beans, lentils and peas	0.225	0.152
Increase fish consumption	0.383	0.716
More servings of fruit and vegetables	0.065	0.663
Move more physically	-0.091	0.537

FFQ food frequency questionnaire, GSQ goal setting questionnaire

Associations in biochemical measurements and the main food frequency messages were examined using Pearson product moment correlation. Changes over the initial twelve-week period between fruit, vegetable, whole grain and oily fish intakes and fasting glucose, HOMA %B, HOMA %S, and the plasma lipids measured showed only the following significant correlations **Table 7.14**

Table 7.14 Significant correlations found between biochemistry and FFQ in weeks 0 and 12

Correlation between	r	p
Vegetable intake and whole grain intake	0.368	0.0067
Whole grain intake and total cholesterol	-0.323	0.0185

The participants who increased their vegetable intake over the twelve weeks also increased their whole grain consumption. An increase in whole grains resulted in a decrease in total cholesterol.

Table 7.15 summarises the intake of the average daily number of fruit, vegetable and fruit and vegetable servings, average weekly number of oily fish servings and the

average daily physical activity (excluding pedometer readings) over the period of the study. These are all possible protective factors against various cancers.

Table 7.15 Frequency of consumption of foods with protective factors (FFQ reports)

	week 0 n		week 3 n		week 6 n		week 9 n		week 12 n		week 12a‡ n		week 52 n	
Fruit 2+per day	25	47%	33	62%	36	67%	41	77%	33	62%	23	64%	27	75%
Vegetables 3+ per day	43	81%	40	75%	44	83%	44	83%	44	83%	31	86%	30	83%
Fruit+Vegetable 5+ per day	37	64 %	42	79 %	45	85 %	45	85 %	38	72 %	26	72 %	28	78 %
oily fish 3x/wk	8	14 %	6	11%	12	23 %	17	32 %	20	38 %	14	39 %	15	42 %
Physical activity	5	9 %	5	9 %	5	9 %	10	19 %	6	11 %	4	11%	4	11 %

‡ week 12 with only those 36 participants that returned for measurements in week 52
physical activity – Number of participants doing ≥ 30 minutes per day strenuous activity
No significant changes in sodium intake were detected from the FFQ.

Table 7.15 shows an increase in numbers of participants eating 2 or more fruit per day from 47% to 62% over the initial 12 weeks. At 9 weeks 77% reported eating 2 or more fruit per day and at 52 weeks 75% reported eating this amount. Those that reported eating 3 or more vegetables a day dropped from 81% to 75% in the first three weeks and then increased to 83%. This was maintained in both the 12 and 52-week FFQ. Overall fruit and vegetable consumption increased from 64% eating 5 plus a day in week 0 to 85% in week 9. This decreased to 72% in week 12 but increased slightly over the year to 78% in week 52.

Oily fish consumption decreased from 14% eating oily fish three times a week or more at baseline (week 0) to 11% in week 3. It then more than doubled to 23% in week 6 following the group education session and continued to rise reaching 38% in week 12. At week 52, forty two percent of participants reported eating oily fish 3 times a week or more.

7.9 Physical Activity

Figures 7.2 A, B and C show the physical activity changes reported at weeks 0,3,6,9 and 12 for the previous three weeks, analysed from the physical activity questionnaire (appendix …).

The physical activity questionnaire asked how many times in the previous week activities mentioned on the goal sheets had been participated in. This average also fell from the week prior to commencing the study (11.5 times) to the week prior to the diet and physical activity talk. (10.8 times). After the talk it increased to 12.7 times and was maintained at just over 12 times until the end of the initial period of the study. It dropped to 9.1 times for the week prior to the measurements done at 52 weeks (**Figure 7.2A**).

Activities reported on were: Climbing some stairs for exercise instead of taking the elevator; walking instead of driving a short distance; parking away from your destination so you have to walk more; walking on your lunch hour or after dinner; getting off at a bus stop which is not the one nearest your destination and walking; other mild/moderate physical activity'

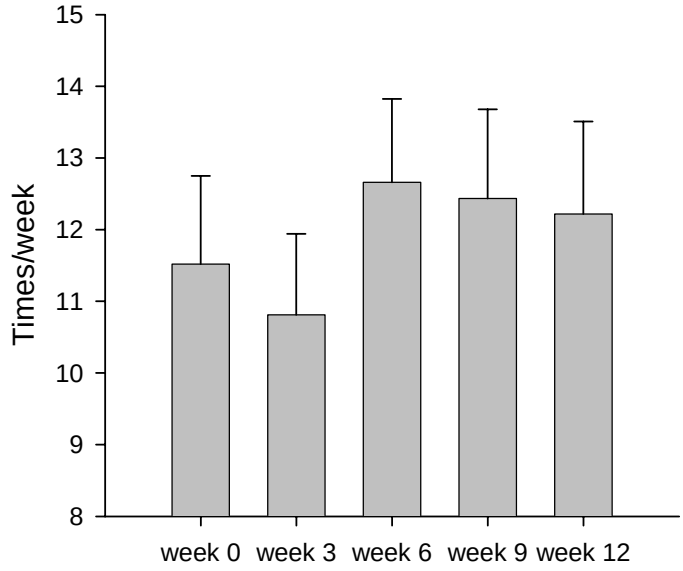
The number of minutes of more strenuous activity per week (**Figure 7.2B**) decreased from 72 at week 0 to 67 at week 3. It increased to 91 at week 6 and 130 at week 9 and decreased to 100 at week 12.

The more strenuous activities reported on were: Jogged or ran; played strenuous racquet sports (singles tennis, paddle ball etc); played other strenuous sports (rugby, netball, basketball, soccer, rowing etc); rode a bicycle; swam; went to the gym.

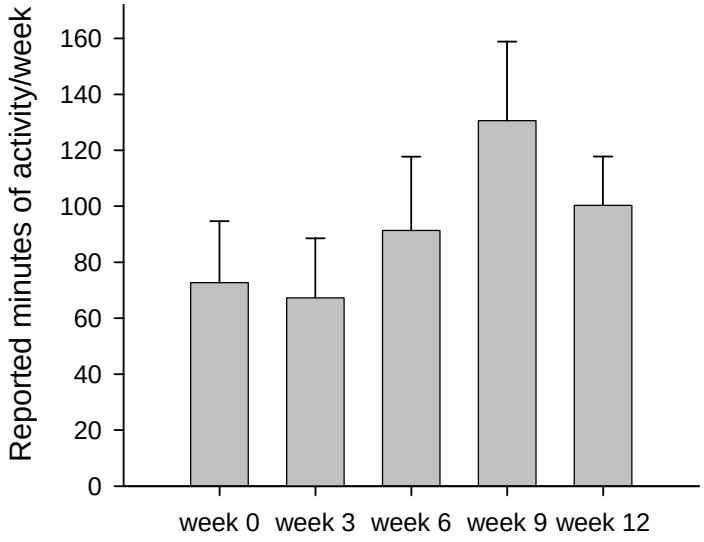
Questions about how many steps were measured by the pedometers issued at the diet and physical activity talk were asked at measurements in weeks 6, 9 and 12. Steps were reported for each of the weeks between questionnaires and the average weekly total was taken for this graph. There was an initial increase in the average number of steps per day from 6903 to 7673). This decreased to 7654 per day during the last 3 weeks of the initial study period (12 weeks) (**Figure 7.2C).**

Figure 7.2 Physical activity changes **A**. Goal sheet activity occasions **B.** Minutes of more strenuous activities. **C.** Average pedometer steps per day

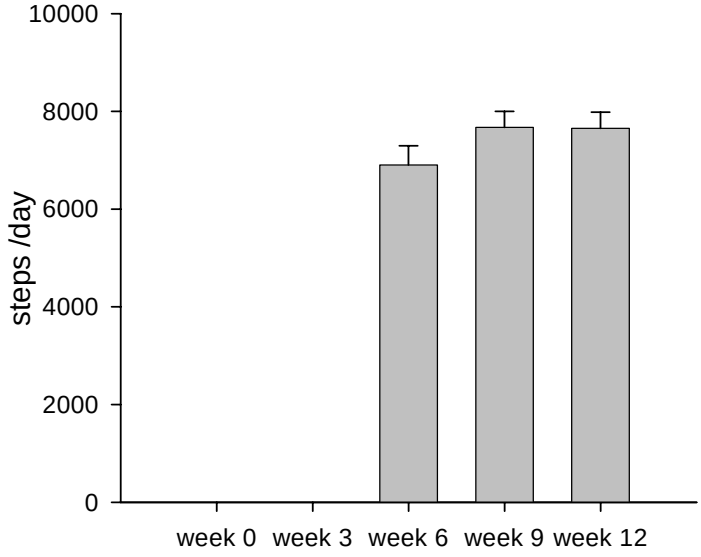
A.



B.



C.



The scoring system for the level of physical activity was calculated from the PAQs as shown in **Table 7.16**:

Table 7.16 Calculation of physical activity levels from PAQ data

	Low	Medium	High
Activity occasions per week(number per week)	0-3	3-5	5-7
Strenuous activity (minutes per day)	< 9.99	10-19.99	>19.99
Pedometer steps (average per day)	<5	6-10	>10

A score of 1 (low), 2 (medium) or 3 (high) was given to each participant in each category. These were summed and the participant's level of physical activity rated as low (1,2 total), moderate (3,4 total) or active (5 and ≥6 total) overall.

The number of participants in each category is set out in **Table 7.17**.

Table 7.17 Physical activity levels calculated from low, medium/moderate and high/active categories from PAQ analyses.

	Week 0 n		Week 3 n		Week 6 n		Week 9 n		Week 12 n	
Low 1	0	0%	0	0 %	0	0 %	0	0 %	0	0 %
Low 2	35	66 %	37	70 %	33	62 %	22	42 %	24	45 %
Total low	**35**	**66** %	**37**	**70 %**	**33**	**62 %**	**22**	**42 %**	**24**	**45** %
Moderate 3	10	21 %	8	15 %	11	21 %	11	21 %	13	25 %
Moderate 4	5	9 %	6	10 %	5	9 %	14	26 %	12	23 %
Total moderate	**15**	**30** %	**14**	**25** %	**16**	**30** %	**25**	**47** %	**25**	**48**%
Active 5	2	4 %	2	4 %	4	6 %	5	9 %	2	4 %
Active ≥ 6	1	2 %	0	0 %	0	0 %	0	0 %	0	0 %
Total active	**3**	**6** %	**2**	**4** %	**4**	**6** %	**5**	**9** %	**2**	**4** %

7.10 Goal sheets

The goal sheets were used as the basis for ascertaining what dietary and physical activity actions had been followed before, during and after the study that may influence lifestyle disease risk.

Table 7.18 Lifestyle actions pre- study and those taken as a result of the study as reported by GSQ at week 12.

Goal	* **pre** n	**Top pre** rank	* **yes** n	**Top after** rank	* **no** n	* **n/a** n
Choose low fat or no fat versions eg green top milk instead of standard	33	2	10		5	0
Cut visible fat off meat	33	2	8		3	4
Take the skin off your chicken	21		9		14	4
Use cooking methods that don't use much fat(boiling, microwaving, baking, stir fry)	34	1	11		3	0
Eat fish more often(aim for twice a week or more)	10		31	2	5	2
Increase your legume consumption (try some of our yummy recipes!!)	10		20	4	18	0
Eat less takeaways	25	4	16	5	7	0
Decrease your use of margarine and butter	27	3	14		6	1
Wait for toast to cool before you add the marg./butter	7		20	4	15	6
Use oil free salad dressings	18		12		14	4
Don't add salt to your food –use more herbs and spices for flavour	23		10		12	3
If you have high blood pressure watch out for hidden salt(sodium)	7		9		4	28
Eat more cereals(rice, pasta, grains) and legumes(dried peas and beans, baked beans, lentils)	25	4	15		8	0
Use the stairs instead of the lift	21		22	3	5	0
Park a little way away and walk	14		13		19	2
Walk rather than drive where possible	24	5	13		11	0
Walk around the field/court when watching sport	11		6		22	9
Take breaks away from your desk. Go for short walks	16		22	3	10	0
Go for a walk at lunchtime	13		12		23	0
Play actively with the kids	16		2		16	13
Have a stretch. Rotate your feet. Stand up and sit down again	16		10		22	0
Use less labor saving devices	13		6		29	0
Use a pedometer (number of steps)	0		49	1	10	0

* pre- was doing prior to the study, yes- changed since the start of the study, no- did not change, n/a- not applicable

The previous table (**Table 7.18**) shows the number of participants who were already making the changes suggested on the goal sheets to their diet and physical activity. It also gives the number who made the changes after the diet and physical activity information was given (week three) and those who did not change these specific behaviours. The behaviours already followed and those followed after the diet and physical activity shake-up are ranked to illustrate those which had penetrated and been implemented from previous knowledge and those which were most influenced by the shake-up.

Not all questions were answered by every participant. Thirty-four participants indicated that they were already using cooking methods that required less/no fat. Thirty-three were using lower fat milk and cutting visible fat off their meat before joining the study. Prior to the diet and physical activity talk 27 were trying to use less butter or margarine and 33 were trying to increase cereal and legume consumption. None were using a pedometer before the study and 10 were trying to eat fish twice a week or more.

After the diet and physical activity talk 49 participants were regularly using their pedometer and 31 tried to eat more fish. Twenty-two were trying to take breaks away from their desks and 22 reported using the stairs instead of the lift. After the talk 11 more participants changed cooking methods, 10 more changed to lower fat milk, 16 decreased take-away consumption and 14 decreased butter and margarine intake. Fifteen more increased their cereals and legume intake.

The eight "best" participants and the eight "worst" participants were identified. This was done by ranking the biochemical results form best to worst for each test taken. The eight participants that were ranked highest in the most tests were taken as the best eight and those that were ranked lowest in the most tests were taken as the worst eight.
Pearson moment correlations were done with each group to see how changes in their blood test results correlated with their FFQ and GSQ answers. Pearson correlation analysis is shown in Table **7.19.**

Table 7.19 Significant correlations between measurements and dietary and physical activity reports using FFQ and GSQ for the best and worst eight biochemistry results.

Best eight(GSQ)	**r**	**p**
Total Cholesterol : Oily fish	-0.785	0.021
HOMA B% : Whole grains	0.815	0.014
Best eight (FFQ)		
HOMA B% : Whole grains	0.744	0.055
HOMA S% : Whole grains	-0.774	0.041
Worst eight (GSQ)		
Diastolic BP: Saturated fat	-0.744	0.022
Systolic BP : Dairy(low fat)	0.708	0.033
Worst eight (FFQ)		
HDL : Dairy (low fat)	0.789	0.012
HDL : Oily fish	0.677	0.045

In those eight participants who showed the most significant biochemical changes, there was a significant negative correlation between changes in oily fish intake assessed by the GSQ and changes in total cholesterol (r=-0.785,p=0.0210). This did not extend to a significant correlation when changes in FFQ were used.

Insulin resistance (HOMA B%) showed a positive correlation with an increase in whole grain consumption with information from both the GSQ and FFQ., while whole grain intake showed a negative correlation with insulin sensitivity (HOMA S%) when compared with the FFQ but not the GSQ.

The eight participants who had the least significant biochemical changes showed no correlations between changes indicated on their GSQs and blood results. They did show correlations with changes in systolic blood pressure and low fat dairy intake(r=0.708, p=0.033) and changes in diastolic blood pressure and saturated fat intake.(r=-0.744, p=0.022).

In these participants correlations were found between changes in their HDL results and their oily fish changes (r=0.677, p=0.045) and their HDL and low fat dairy intake (r=0.789, p=0.012).

7.11 Case studies

The changes identified by data analysis have been small and it is difficult to analyse because there are so many confounding factors in the heterogeneous population studied. I have chosen 2 case studies of each sex to illustrate how, on an individual level progress was (or was not) made.

Case A

Case A is a 53 year old female. She is NZ European and is an administrator. Her mother died from breast cancer and her father from pancreatic cancer. She smoked about 10 cigarettes a day but gave up just over 4 years ago. She drinks an average of two glasses of wine a day.

On the goal sheets filled in at week 6 she ticked the goals of "cut visible fat off meat", "Eat less takeaways", "Wait for toast to cool before adding margarine /butter" and "reduce salt-use more herbs and spices for flavour". Her physical activity goals were to "park a little way away and walk" and to "go for a walk at lunchtime."

After the initial 12 weeks of the study the questionnaire indicated that she had started meeting her goals as well as eating more fish and whole grains. She had not decreased her salt intake. The comparison of her goal sheet questionnaire (GSQ) to her food frequency questionnaire (FFQ) were as follows:

Table 7.20 Comparing answers of FFQ and GSQ for case study 1 at week 12.

	FFQ	GSQ
Less total fat	yes	yes
Less saturated fat	yes	yes
More complex carbohydrates(less sugar)	yes	yes
More whole grains	yes	yes
More beans, lentils and peas	no	yes
Increase fish consumption	no	yes
More servings of fruit and vegetables	no	no
More variety /different foods	yes	yes
Move more physically	yes	yes

Her fruit and vegetable consumption remained the same over the first 12 weeks. She did, however, increase her fruit and decrease her vegetable intake. Her prescription for kiwifruit was for three a day and she enjoyed eating them. At 52 weeks she indicated that she had continued having at least one kiwifruit per day. At 52 weeks she had increased her fruit intake and her vegetable intake from week 12. She had also increased her oily fish and whole grain intake, decreased her saturated fat and salt intakes and maintained her physical activity increase.

Over the first twelve weeks she maintained her weight and decreased her waist measurement. Both of her blood pressure readings increased. Her glucose decreased slightly and her insulin resistance (HOMA B%) went up and her insulin sensitivity (HOMA S%) went down. Her total cholesterol and trigyceride levels improved; her LDL and total cholesterol: HDL was maintained and her HDL and percentage body fat deteriorated.

At 52 weeks she had decreased her weight, waist and percentage body fat from week 12.Her diastolic blood pressure had decreased but her systolic had increased. Her systolic measurement was in the high normal range. Her lipid profile had improved. Her cholesterol, LDL cholesterol and total:HDL ratio had improved. Her triglycerides had increased slightly but not to the initial reading and her HDL cholesterol was the same as her initial measurement. Her glucose and insulin sensitivity increased and her insulin resistance decreased between weeks 12 and fifty-two.

Overall there was an improvement in Case A's diet and physical activity levels, which was maintained and, in some cases, increased over 52 weeks. She lost a small amount of weight, which was maintained over the year, and her blood lipid levels were improved and maintained. Her diabetes and hypertension risk factors were not improved but they were maintained at a low (normal) level and the increases were not enough to be of concern.

Case A reported that she was feeling better and more in control and she felt her changes to her diet and physical activity levels, though small, were easy to maintain and add to. Her motivation for making changes was to 'lose some weight and achieve a level of fitness" and she felt she had gone some way to achieve these aims.

Case B

Case B is a 46-year-old New Zealand European male lecturer with normal lipid and glucose profiles. His fasting plasma glucose did tend to be above the 5.5mmol/L that the Australian protocol recommended have an OGTT as a further screening procedure (Colagiuri *et al*. 2004). He has no known family history of any of the lifestyle diseases examined in this study and has never smoked. He drinks and averages 4-5 glasses of wine or bottles of beer a week. He had been trying to keep his saturated fat intake low by cutting visible fat off meat and taking the skin off chicken but his motivation and impetus for making positive dietary changes had been decreasing. After starting the study he increased his physical activity and improved his diet. He changed from standard blue top milk to lower fat milk, decreased his use of butter, margarine and salt and increased his fish consumption. He blamed lapses in his efforts on his busy lifestyle and on his wife's cooking.

He increased his physical activity and found the pedometer a motivating tool to do this. He has a recurring knee injury that sometimes prevented him from running/walking as much as he would have liked to. He did swim on occasion though. He enjoyed the recipes in the recipe booklet and those added by email newsletters

His weight increased marginally over the 52 weeks. (0.1kg) and his waist measurement decreased slightly over this period(-0.2cm). His blood pressure dropped over the 12 week period (-13 mmHg systolic; -3 mmHg diastolic) and this decrease was maintained (± 1 mmHg) over 52 weeks. His fasting plasma glucose increased by 0.2 mmol/l after 12 weeks and decreased by 0.1 mmol/L between 12 and 52 weeks. His insulin resistance decreased by 5.2% after 12 weeks and increased by 28.5% after 52 weeks. His insulin sensitivity increased by 25.4% after 12 weeks and decreased by 137.9% after 52 weeks. His total cholesterol, LDL cholesterol, triglycerides and total:HDL cholesterol all improved slightly after 12 weeks and were maintained or improved after 52 weeks. His percentage body fat decreased after 12 weeks but increased after 52 weeks. He disliked eating the kiwifruit on a regular basis and was only eating one a week on average according to his FFQ at 52 weeks.

Overall, Case B made some positive dietary and physical activity changes and was motivated by the pedometer, recipes and regular newsletters. While he didn't achieve any biochemical or anthropometric changes, he remained within normal limits He did

make sustainable changes to his milk and oily fish consumption and was motivated to increase his physical activity in spite of the knee injury he had been using as an excuse to reduce exercise.

Case C

Case C is a 54-year-old female personal assistant. She is New Zealand European. She tends to be sedentary at work. She does not smoke and drinks a moderate amount of 2-3 tots of gin and tonic a week. She has a maternal and paternal family history of cardiovascular disease and hypertension. She was classified as high risk by BMI ($31 kg.m^{-2}$) and waist measurement (>100cm). She was normotensive (<130/85 mmHg) and her fasting plasma glucose was between 5.5 and 6.0 mmol/L.

She found making dietary changes difficult but did exclude the butter she was eating between week 0 and week 12. She maintained this and increased her oily fish consumption between 12 and 52 weeks. She did not increase her physical activity and stopped wearing the pedometer after one week as it made her feel guilty that she was doing so little.

She hated eating the kiwifruit and did not always eat all the kiwifruit prescribed in the three weeks she was supposed to be eating them. She enjoyed the emails but did not action any of the suggestions made in them. Her FFQ and GSQ did not correlate well. According to her FFQ, she maintained her fruit intake but decreased her vegetable intake during the first 12 weeks. She increased them again in the 52-week period but was still below the original consumption at 52 weeks. She made no changes to her oily fish or whole grain consumption at any stage and decreased and maintained lower saturated fat consumption. She decreased her salt consumption between weeks 0 and 12 and weeks 12 and 52. Her physical activity did not change during the initial 12 weeks and increased slightly between weeks 12 and 52.

Her glucose profile improved marginally (glucose –0.5mmol/L) after 12 weeks and increased by 0.3 mmol/L after 52 weeks. Her total cholesterol and LDL cholesterol decreased after the first 12 weeks and were maintained after 52 weeks. Her HDL cholesterol did not change in weeks 0 to 12 but increased in weeks 0 to fifty-two. The lipid changes were possibly due to her exclusion of butter, which she had been consuming liberally before the study.

Her percentage body fat, waist and weight measurements remained at high-risk values.
The small changes Case C made seemed to have lowered her total cholesterol, LDL cholesterol and triglyceride levels and increased her HDL cholesterol. It is a pity she did not put more effort into changing other aspects of her diet and her physical activity as she was responsive to change and with her family history and risk factors she could have benefited from the study more. Hopefully she is more aware of changes she could be making and she will make them as and when circumstances allow.

Case D

Case D is a 46-year-old New Zealand European lecturer. He has a maternal history of cancer and is a non-smoker. He drinks about 6 bottles of beer a week. He believed he had a healthy lifestyle and was doing all the positive things we had suggested already. He had a healthy BMI and his waist measurement was low risk. His body fat ranged from 11 to 17 %. He was normotensive (the highest measurement we had for him was 110/74 mmHg). His initial cholesterol was 6.8 mmol/L and his HDL cholesterol was a low 0.5 mmol/L. His triglycerides were 'normal'. His fasting plasma glucose was below 5 mmol/L.

His weight decreased over the period of the study but his percentage body fat and waist measurement increased. His blood pressure remained normotensive. His total cholesterol decreased by 0.5 mmol/L over the first 12 weeks and a further 0.3 mmol/L over the period from 12 to 52 weeks. However his HDL cholesterol decreased.

In spite of believing that his diet and physical activity patterns were good, he did increase his fruit and vegetable consumption over the fifty-two week period. They had decreased in the first 12 weeks. His whole grain consumption decreased slightly at 12 and 52 weeks. His oily fish consumption remained at zero His salt and saturated fat intakes increased slightly and his physical activity increased marginally.
He enjoyed the kiwifruit and continued to eat at least two a day when they were in season.

If he had known that his lipid profile was higher risk than he thought I am sure he would have made more positive changes.

8. Discussion and Conclusion

8.1 Discussion

This thesis opened with the statement

"**The goal of nutrition intervention is to assist and facilitate individual lifestyle and behaviour changes that will lead to improved metabolic control**".

Was this study able to achieve this?

Yes. For this multicultural, relatively middle aged group of 53 AUT staff with an average age of 46 years, measurable and statistically significant metabolic gains were made in the lipid profile over 12 weeks. Total cholesterol, LDL cholesterol, triglycerides and the ratio of total cholesterol to HDL all decreased and HDL increased. In the 36 who were measured at 52-week follow up these changes persisted.

With the other outcome measures for body composition, blood pressure and glucose and insulin there was no significant change, which is a measure of success as it is likely given our national statistics, for example, of NZ people gaining a gram of weight each day between 1989 and 1997 that this was not happening in this group.

8.1.1 The participants

The mean age of the participants in this study was 46 years. Which age groups interventions should target is a debatable point. Ideally healthy lifestyles should be followed from childhood and continued throughout life. The explosive rise in childhood obesity and type 2 diabetes in children is indicative of a lifestyle problem that starts early on in life. The Barker hypothesis on the foetal origins of disease emphasises the need for young women to lead healthy lifestyles before and during their pregnancies to ensure their infants have healthy birth weights and are less at risk of cardiovascular disease in later life (Fall *al*.1995). But at any age risk of lifestyle disease and associated complications can be reduced through modification of lifestyles. Adults in the thirty to sixty year age groups, as participated in this study, have the potential of influencing

their own, their children's as well as their parents lifestyle habits and were therefore a sensible age group to target in this pilot study.

Any study, unless there is national enrolment, will not be representative of the general population or generalisable. The participants in this study responded to advertisements recruiting 'healthy' men and women of all ethnicities over the age of 30 years. Of the 58 who were measured in week zero, two had risk factors that were so overt they were referred to their doctors for medical advice and further testing and were excluded from the study. Fifty-two of the 53 participants measured in weeks zero and three had one or more risk factors for lifestyle disease. There were a number of participants (75%) who were at risk of lifestyle diseases because of their family history. Some indicated that the reason why they had participated in the study was because of their family history and most thought that they had been leading healthy lifestyles for the same reason. None of the participants were at higher risk of lifestyle disease because of smoking or excessive alcohol consumption (with the exception of one who may have been less at risk from being obese if he had reduced his alcohol consumption). This highlights the need for preventative interventions to take place before the realisation of possible risk factors and that more intensive screening will detect, in this case 2/58, 3% in need of immediate treatment.

8.1.2

Body composition

It was not one of the aims of this study to reduce weight or body fat substantially as the researcher recognises that this should be a slow process in order to be maintained but body composition was analysed. Classification of participants into normal weight, overweight and obese by using BMI cut off values that had taken ethnicity into account, showed very little change during the study in risk of diseases related to obesity and overweight. Weight loss from week 12 to week 52 resulted in an 8 % (three) decrease in those who were classified overweight but the percentage who were obese did not change. The reduction in BMI over the 52-week period compared to the initial 12 weeks is probably indicative of weight loss being a slow process. The obese participants did not change BMI ranges. This could either be due to their BMI s being too high to

register a range change or indicative of obese people needing more consistent monitoring. Of the nine who did not complete measurements at 52 weeks without apologising, four were classified as obese and I suspect they did not return to be measured because of non-compliance. More of these were women than men.

Waist measurements indicate that only 20% of the males were at risk, whereas percentage body fat put just over half at risk. BMI indicated that 68% were at risk. These anomalies were found in the female risk factors as well. Ninety-three percent of the females had an excessive percentage body fat and the 47% of women participants with waist measurements of over 88cm could have visceral fat that is too high for health. The evidence that abdominal fat is a better indicator of risk than total body fat (Despres 2001) means that about a quarter of the males and half the females in this study were at risk at baseline. These risk factors did not change significantly over the period of the study but indicate that many people who consider themselves healthy are at risk

Significant changes in anthropometric means included a decrease in the mean suprailiac skinfold measurement at week 12. Skinfold measurements are difficult to measure accurately and although there as a statistically significant difference, because there was only a difference at the last measurement and none of the other skinfold measurements decreased significantly, it is probably not of biological significance. There were possibly differences in the measurement techniques of the two measurers and one measurer was more consistent with measurements than the other. The differences in the measurers were shown when the anthropometric measures were analysed according to sex. The main significant changes occurred in week 12 males when many more of the males were measured by the female measurer due to the male measurer being away on several measuring days. The interpretation of these changes is therefore compromised. In spite of this none of the anthropometric measures increased significantly which is encouraging. Anthropometric changes take time, particularly when the objective of this study was for the changes to be small and sustainable rather than to make an impact on body composition and possibly compromise sustainability. This highlights the problems with using skinfold and girth measurements as the basis for assessment of changes in body size. It also indicates a need for caution if divulging individual results back to the individuals as they misinterpret changes inappropriately as success or failure.

However, there was a strong correlation ($r=0.87-0.9$; $p<0.05$) between mean percentage body fat calculated from skinfolds and mean percentage body fat measured by BIA at all six measurement points. Skinfolds, waist measurements and percentage body fat by BIA were all useful measurements to have done. They were all inexpensive, non-invasive and add to information about body composition obtained from BMI calculations. BMI is not indicative of percentage body fat. BIA gives a good indication of body fat and does not require removal of clothing. It does, however, need more expensive, not readily available equipment. BIA also requires precise measuring techniques and compliance of the participants with regards fluid intake and physical activity prior to measurement. The skinfolds and waist measurements give an indication of the location of the body fat, which is important in risk assessment. But these measurements require removal of clothing and are less accurate and more susceptible to measurer error than BMI and BIA. Using more than one measurement of body composition, without greatly adding to the cost, gives a much clearer picture of risk.

The 1997 National Nutrition Survey found that 14% of the males of all ethnicities and 19.2 % of the females were obese by BMI cut offs (adjusted for ethnicity) (Russel *et al*. 1999). In this study 12% of the males and 39% of the females were obese by BMI. The difference in the percentage of females who were found to be obese is disturbing but may be related to the fact that they were all AUT staff and not doing much physical activity. Physical activity needs to be promoted in the workplace, particularly in those places were occupations are mainly sedentary. Suggestions of better access to stairs and posters about taking the stairs instead of the lift have been given to the employer. Walking groups were also suggested to participants but not enough participants were motivated enough to start regular group walking. The gym at AUT has recently (2004) tried to organise such groups and, given these statistics, I hope this will encourage many more staff to participate. The level of risk in this study is concerning as the participants probably volunteered because they are more health conscious than most and they thought that they were healthy.

8.1.3 Blood pressure

Most participants were normotensive and did not make changes to their sodium intake in the form of table salt. One participant was hypertensive and was referred to his doctor for medication. He continued to be involved in the study. Over the 12 week period six participants lowered their systolic BP by 10mmHg or more and two of these maintained this decrease at 52 weeks According to the World Health Report (WHO 2002a) this would translate to them reducing their risk of stroke by 37% and their IHD risk by 25% respectively.

8.1.4 Lipid profile

Cardiovascular disease risk factors measured by the lipid profile were the most significantly changes in this study. Total cholesterol and LDL cholesterol both decreased in the first three weeks, even though the participants were supposed to be following their usual diet and physical activity patterns. The answer to the questions about what they had been doing previously to reduce risk (**Table 7.18**) showed that they were aware of the importance of reducing fat, particularly saturated fat. Their participation in the study probably motivated them to further put their knowledge into practice.

Thirty-seven participants (70%) had total plasma cholesterol that was above the maximum considered healthy at baseline (>5.0mmol/L). LDL cholesterol was elevated (>3.0mmol/L) in 38 participants (72%) and one participants HDL was below the accepted healthy level (>1.0mmol/L). Mean total cholesterol at the start of the study was 5.6 (±1.1) mmol/L compared with 5.7 mmol/l for all New Zealanders in the 1997 nutrition survey. In spite of the publicity and knowledge surrounding the need to lower plasma cholesterol to lower CVD risk it is concerning that the mean level was similar to that found in 1997. Total plasma cholesterol was reduced to a mean of 5.3 (±1.0) mmol/L over the 12 weeks and maintained at that level when measured at 52 weeks. It was, however 5.5 (±1.0) mmol/L at nine weeks which is indicative of the inconsistency

of the levels. Even though the 52 week mean was the same as the mean at 12 weeks it is impossible to know how it fluctuated in between or on a daily basis. Variations in blood plasma levels may occur for various reasons. Analytical (due to equipment or methodology) and biological (due to physiological fluctuations) variations may occur. According to the Medlab/diagnostic handbook (Gill *et al*. 2000), at a level of 6.5 mmol/L, there is a biological variance in total cholesterol of ±0.5 mmol. This means that measurements need to be repeated and the trend looked at over a period of time

A meta-analysis by Hooper *et al.* (2001) looking at dietary fat changes and CVD found that the longer the changes were sustained the lower the risk of CVD morbidity and mortality. Small reductions in cholesterol maintained for a long period may be more important than big changes that can't be maintained.

Plasma triglycerides increased significantly in week nine but week nine was also where the consumption of fruit peaked (77% of group A eating more than two pieces per day and an average of 21 pieces per week). This may have increased the carbohydrate content of the participants' diets to levels that increased triglycerides. Total cholesterol and HDL also increased between weeks six and nine but the total cholesterol:HDL ratio decreased. This was confounded as this was also a period of time that covered University holidays and the FFQ indicated that both diet and physical activity varied over this time. The FFQ showed a mean increase in more strenuous physical activity and increased pedometer steps were noted. There was also an increase in fruit, saturated fats, fish and whole grains and a decrease in dairy consumption. Exactly which changes may be responsible for the difference in plasma lipids is difficult to extract, but there were changes in both dietary and physical activity factors and plasma lipids over this time.

The total cholesterol/HDL ratio was significantly decreased at measurements in week 12 and was maintained at the measurement in week 52. This ratio is a good indication of risk (Mensink *et al*. 2003) so this finding is positive for health. Over the 12 weeks 18 participants decreased their total plasma cholesterol by more than 0.6mmol/L.Eight maintained this decrease over the 52 weeks. Law *et al.* (1994) found that a difference in

total cholesterol levels of 0.6mmol/L was associated with a 24 % difference in death from IHD.

There is some debate as to the levels a risk factor should be at to consider a person at risk. Law and Wald (2002) suggest that "interventions to reduce risk factors should be determined by a person's level of risk, not by the level of risk factors". They advocate the use of drugs in anyone who is at risk and question whether the 'normal' values we use for blood lipids and blood pressure are valid indicators. Maybe we should be aiming for even lower values. Their argument further enhances this studies' argument that everyone should be trying to reduce the risk factors associated with the LDs and not just those already exhibiting high risk.

A recent study that used systolic BP, smoking, serum total:HDL cholesterol ratio, diabetes and enlargement of the heart to evaluate CVD risk in 7700 British men in a 10 year follow up of the British regional heart Study found that the Framingham risk formula overestimates the risk of CVD mortality by up 47% and non-fatal coronary events by up to 57% (Brindle *et al*. 2003). The MONICA project (**4.1.1**) and the discussions as to the validity of Framingham scores to predict CVD risk highlight the uncertainty and weakness of the individual risk factors in predicting LDs. This strengthens the notion that a holistic approach is best.
The questions around the validity of the Framingham risk score also emphasise the evolutionary nature of research and the need to be flexible and up to date in the implementation of research studies.

8.1.5 Glucose and insulin

There was a significant decrease in plasma glucose levels from baseline to weeks six and nine. The mean levels were within the range of 'normal' and even though there was a statistically significant difference, the biological and risk significance is probably not high. There were no significant changes in plasma insulin or HOMA values but HOMA B% (insulin resistance) showed a decrease and HOMA S% (insulin sensitivity) an

increase over 12 weeks, which is an encouraging trend. These values deteriorated slightly over 52 weeks but were still much improved over baseline measurements.

Most studies looking at reduction of type 2 diabetes risk have looked at changes that are aiming to improve the metabolic control of people with type 2 diabetes or who already have IGT. Given the huge increase in the prevalence of type 2 diabetes and the expected increase in type 2 diabetes incidence over the next decade it is important to implement lifestyle changes in those who appear to be normoglycaemic as well as those who have IGT.

8.1.6 Cancer

The participants in this study made positive changes to their diet and physical activity that should reduce their risk of cancers. Nineteen had a family history of one or more of the cancers so reducing their risk by lifestyle changes is a way they can influence the outcome of this predisposition. There was an increase in the consumption of foods that may be protective against cancers, like fruit and vegetables, whole grains and oily fish and an increase in physical activity. The kiwifruit treatment may have had an influence on DNA strength. Results from this testing (by comet assay) will be reported elsewhere.

8.1.7 Reported changes in food and activity

The first FFQ, filled in at week zero, showed that 37% of participants were eating the recommended 5+ per day of fruit and vegetables. Forty-seven percent reported eating the recommended 2+ per day of fruit and 81% the desired 3+ per day of vegetables (including potatoes). This dropped to 62% if potatoes were excluded. The National Nutrition Survey (NNS 97) carried out in New Zealand in 1997, found that about 46% of New Zealanders were eating two or more servings of fruit and about 67% were eating three or more servings of vegetables per day (Russell *et al*. 1999). The amount of fruit consumed by this sample was therefore similar to the NS97 results. The vegetable consumption was higher than the average NNS97 intake but this is probably because the sample was predominantly NZ European and middle to high income. In the NNS97 NZ European and other new Zealanders were more likely to consume recommended fruit

and vegetable intakes than Maori and Pacific Islanders and those in less deprived socio-economic groups were also more likely to be compliant with recommended amounts of fruits and vegetables.

The average number of participants eating the recommended number of portions of fruit and vegetables increased over the initial 12-week period and was maintained over the 52 weeks of the study. Recommended fruit intake increased to 77% compliance in week nine dropped to 62% in week 12 and was 75% at week 52. Vegetable consumption increased to 83% compliance with the recommendation of 3+servings per day after the group 'shake-up' and remained at 83% for the rest of the study. This is, encouragingly, substantially above the numbers recorded in the NNS97 report (Russell *et al*. 1999).

Oily fish consumption was an area that few participants had been paying attention to at the start of the study. Only 10 had been trying to eat fish regularly (**Table 7.18**) and the FFQ indicated that only eight (14%) were eating oily fish three times per week (**Table 7.15**). This increased to 23% after the diet and physical activity 'shake up' and to 39% by the end of the 12 weeks. After 52 weeks this had again increased to 42%. The shake-up and the subsequent email information on the benefits of oily fish consumption increased awareness and made a positive impact on the dietary intake of the participants.

The 1997/1998 New Zealand Health Survey found that about 40% of New Zealanders were regularly active for 30 minutes or more a day (Ministry of Health 1999). The amount and intensity of physical activity needed to reduce the risk of lifestyle diseases is still a controversial issue. Some studies have shown benefits with intensive activity only (McAuley *et al*. 2002) while others indicate that even moderate increases in regular activity will decrease the risks of non communicable diseases (*Hu et al*. 2000; Tanasescu *et al*. 2002). While it is probably better to do more intensive, regular exercise, a gradual increase in physical activity that is easier to maintain and build on is possibly more useful over the long term. Exercise also increases free radicals by oxidation and a gradual improvement in fitness is probably better than a sudden burst of oxidative energy for cell health (Goto *et a*l. 2003). This study found that only 9% (increasing to 11% over the period of the study) were reportedly vigorously active for 30 minutes or more a day. This is possibly partly because only more strenuous activities

were included in the questions and also because of the sedentary nature of many of the participants' jobs. Most of the participants were lecturers or administrative staff at AUT. Two were students. The use of computers has greatly reduced the amount of physical activity done by these groups and ways of increasing movement in these types of vocations needs to be given more thought. Pedometers were found to be a useful tool in increasing awareness and motivating activity. There was an overall increase in physical activity and pedometer readings increased over the period of the study. Increased physical activity persisted over the 52 weeks and several participants purchased pedometers so that they could continue to monitor and be motivated by their daily/weekly efforts. Several AUT staff members outside of the study were also motivated by the pedometers and either loaned or bought pedometers as a result of the study. The wearing of pedometers was noticed by people not involved in the study and often became an initiator of discussions about physical activity and LD risk. They created an awareness and opportunity for dissemination of knowledge. Over the twelve-week period more participants who initially had a very low activity level increased their level to moderate. There was a reported decrease in activity over the first three weeks when those doing only a low level of activity increased from 60% to 70%, while those doing a moderate amount decreased from 30% to 25%. After the diet and physical activity talk and the issuing of pedometers there was an increase in the percentage of those who were doing a moderate level of physical activity and decrease in those doing a low level. The increase in moderate activity continued over the twelve weeks. The pedometers were not sealed or readings checked as they were provided as motivators, not for objective measurements.

The FFQ was filled in at each of the six measurement times. To get an idea of the progress the participants thought they were making and to encourage them to a goal setting questionnaire (GSQ) was given to them at weeks 9 and twelve. The answers given in the GSQ to questions relating to changes made in fruit and vegetable consumption, fish increases, whole grain intake, increases in beans, lentils and peas, decreases in total and saturated fat and increases in physical activity were compared to computations of these factors in the FFQ at week 12. There was no significant correlation between the two. This highlights the inaccuracies of information received by self –reporting. It was also interesting to note that the participants who did not return to be measured in run six (and had not offered an excuse or apology) were particularly

confused as to whether they had made changes or not. They were either trying to fool themselves or the researchers or were not paying enough attention to what they were doing indicating a lack of interest and motivation.

The top three actions (using low fat cooking methods, choosing low fat milk, cutting the fat off meat) that were already followed indicate that public message about reducing fat intake are being noticed. (**Table 7.18**) This concurs with evidence that CVD was decreasing in developed countries over the last decade partly due to messages about fat reduction and increased physical activity reaching the public.

Increasing fish intake and increasing physical activity were the main messages taken from the group session on the importance of diet and physical activity in reduction of lifestyle disease risk. Fruit and vegetable consumption were not asked in this GSQ questionnaire but from the FFQ results it was clear that the 5+ per day message was being followed from the public message and reinforced by the information given in week 3 of the study. Thirty-seven participants reported eating five or more portions of fruit and vegetables in week zero, which would rank this message as the one that had been followed most by participants before they started the study.

The best eight individual results showed a correlation between whole grain consumption and both insulin sensitivity and insulin resistance (**Table 7.19**). This is in line with studies of Liese *et al.* (2003) and with recommendations in a report by Franz *et al*. (2002). The case studies give an indication of the differences among participants and the complexities of trying to measure changes and associations between changes.

Many of the changes made were not measurable and many participants personally described changes in attitude, awareness and motivation.

8.2 Limitations of this study and recommendations for future interventions

8.2.1 Limitations

The clinical changes were small but the changes in awareness, motivation and attitude and the expansion of the awareness of the importance of dietary and physical activity changes to other staff members at AUT and to the families of participants and beyond, whilst hard to measure, continue to be communicated at a personal level to the researcher and colleagues. Several AUT staff not involved in the study bought or borrowed pedometers and many asked for dietary advice. As a result of this study, a group at an AUT affiliated site took part in a similar study that refined some of the methods used in this study and created an awareness of ways of reducing LD risk in another group of people- a ripple effect.

The changes made and changes quantified by measurement were small and mostly not statistically significant. Small changes or even no changes in a relatively healthy population may be practically significant. In their review of health care based physical activity and dietary interventions in 2001, Wilcox *et al*. found modest effects in physical activity, dietary fat intake and weight loss associated with reductions in blood pressure and serum cholesterol. They commented that even though the results were modest, the implications of even small changes were meaningful when looked at from a Public Health perspective.

Attributing changes in risk factors to specific lifestyle changes in this study is difficult as data on dietary intake and physical activity was not consistent or reliable and was very subjective. Many factors can contribute to biochemical and anthropometric fluctuations in free living people.

In evaluations of the "green prescription" (**3.2.2**) changes with reported increased physical activity were small over 12 months but, as with this study, they were sustained (Elley *et al*. 2003). If a single group education session and ongoing email newsletters can prompt sustainable changes it may be possible to make major reductions in LD risk over the long-term.

As can be seen from **Table 7.3**, a number of healthy participants had one or more risk factors at baseline. They were not given this information unless they required medical attention and these participants were excluded from further measuring and not included in the statistics. One participant was hypertensive at weeks 3 and 6 and was referred to his GP. He received treatment but remained in the study. They may have been more compliant if they had been told their results but as the study was to measure the effect of the intervention, this would have complicated the data analysis even more. If the incidence of lifestyle diseases is decreased by diet and physical activity modification then it is important that people without measurable or overt risk factors make the necessary changes. Motivation should, therefore not be dependent on knowledge of risk.

Having a control group was considered. But, given the large amount of information in the public arena and the possibility of discovering people who needed interventions because of high risk factors it was decided to do two baseline measurements (week zero and week three) and to let the participants act as their own controls. The number of participants who made changes between the first and second measurement points prior to the discussion and booklets aimed at helping them change justified this view. The number of participants who had risk factors that they were not aware of further emphasised the problem of denying the program to the control group. A control group, may, however, have clarified some of the results, although the experience of the MRFIT (**4.1.2.2**) suggests that this was not likely.

The intervention studies discussed in **chapter 4** (and many others) have been undertaken with participants who were already at high risk of developing lifestyle diseases. This study tried to look at healthy people and minimize their risks. This is essential in preventing lifestyle disease. It is important to prevent the early risk signs and aim for a population that will not be a burden in terms of health costs (financially and/or emotionally). Empowering people who do not have obvious signs of risk and

educating them to be aware of doable changes they can make to reduce the risk of lifestyle diseases is essential in trying to reduce the social, emotional and fiscal burden of the lifestyle diseases.

But using "healthy" participants has drawbacks. They do not have the added motivation of knowing that they are unhealthy and need to do something to minimize getting sick or dying prematurely. They tend to be people who are aware of their health and are already following healthier lifestyles or who think they have been following sub-optimal lifestyles and are not exhibiting any ill effects, so why change? Their blood results are within the healthy range and as levels fluctuate naturally, positive changes are unlikely to be as noticeable. Normolipidemic participants are less likely to show marked cholesterol changes (Ripsin *et a*l. 1992; Anderson 1999; Brown 1999). In the study of premenopausal obese women by Janssens *et al.* (2002a) it was suggested that the lack of positive response of triglycerides, HDL cholesterol or glucose variables was possibly due to the relatively normal levels of these biomarkers pre study. Another example of the difficulty of showing change when risk factors are low is found in the DASH study (**4.1.2**) which reported that participants with elevated blood pressures showed a larger decrease in both systolic and diastolic blood pressure than those that had lower blood pressures at the start of the study (Harsha *et al*. 1999).

Participants volunteered to take part in the study. This means that they were interested in their health and in at least hearing about ways to stay healthy and minimise risk of lifestyle diseases. This could mean that they were more compliant than many in the community who may need to be targeted.

The question also arises of the level of the dose of intervention. The dose used in the kiwifruit crossover was based on results from a previous study of the effect of kiwifruit on laxation (Rush *et al*. 2002) but this dose might not have been optimal for lowering cholesterol.

Making dietary and physical activity changes is only one part of the equation. Maintaining those changes over a long period of time (preferably the rest of your lifetime) is the more important part. If small changes can be made and maintained it is

preferable to large changes that are made for a short while. Reverting to old habits may be more detrimental, if not physically then psychologically, than not changing at all. This can lead to negative changes over the long term. A bigger awareness of better eating practices and the importance of physical activity as well as the small changes that were made will hopefully filter down to the participants' children and up to their parents as well as horizontally to their peers. If a torch effect (more people seeing the light at various levels) can be created and the knowledge and practice of evidence based changes promoted maybe we can lessen the burden of lifestyle disease by group education.

The confusion generated by different studies having different outcomes is partly to blame for the public not following or trusting lifestyle messages. Mixed messages are often sent out by media and not balanced against information that the public already has. Balance, variety and moderation seem to be, simplistically, what is needed. We need to look at whole foods because, even though knowing the mechanisms and lowest common denominators for lowering risk is important, realistically the availability of the correct, individualised combinations of dietary and physical activity programs can only be very limited. Whole foods also have the advantage of having all the chemicals available (whether we know about them or not) and limit the availability of some chemicals which might influence the bioavailability of others. All factors work synergistically to promote health and manipulation of one or more can lead to major imbalances, which probably increase the risks.

This study showed (data not reported) a possible protective effect of kiwifruit up to twelve hours after eating them and also used a dose related to body weight. Details and statistics for the comet assays will be reported as a separate study but this finding adds to the possibility that the consumption of a specific fruit could be used as part of a sustainable, manageable way of reducing chronic disease risk.

Are interventions to lower the risk of lifestyle diseases worth the cost of implementing them? Interventions, by changing diet and physical activity, are worth the cost from an emotional, pain and suffering and quality of life, individual point of view. The economic costs to the community must be calculated taking the extra burden of an ageing population and possible other disease states and health costs (for example from

broken bones, dental problems, arthritic conditions) into account. The cost of implementing intervention programs versus the cost of education at school and community level also needs to be evaluated.

The question of ongoing compliance and how long lifestyle diseases can be delayed for, as well as the degree to which complications are avoided also needs to be discussed. Should people at risk be singled out for intervention or should the whole population benefit from improved education and other strategies to improve health by better diet and increased physical activity? One advantage of screening for those at risk is the attention it draws to the seriousness of the problem. This might result in a more motivated, compliant audience.

Interventions, like those in the described studies, need to be complimented by public health strategies that encompass the education system, recreation and transport divisions, media, industry and big business. Any intervention program requires a multi-pronged, holistic approach. Education and awareness needs to be heightened in both the young and the older members of society and 'healthy' foods and opportunities for physical activity need to be accessible. Hopefully, this would promote ongoing attention to diet and physical activity and the long-term compliance that is required for any intervention to work.

In an editorial about the future of community based cardiovascular disease intervention studies in the American Journal of Public Health, Winkleby (1994) refers to the difficulty in evaluating cardiovascular disease risk studies. She points out that many of the benefits, like raising awareness, audience exposure, and small changes that may make a difference over a longer period of time are not quantifiable or measurable by anthropometric or biochemical analysis.

8.2.2 Suggestions for improvements

Many choices were made in terms of measurements and interventions used in this study. Suggestions as to possible improvements are documented below. Some changes have

already been implemented in another AUT study and another study with older Asian Indians in New Zealand.

8.2.2.1 Improvements to the methods

- The intention of this study was to recruit and measure 60 people with equal numbers of each gender. Fifty-eight were recruited of which 53 completed the first 12 weeks and 36 the last 40 weeks. The numbers used were based on numbers used for a kiwifruit study previously undertaken in the same research centre (Rush *et al*. 2002). Given the scope of this study, the numbers could possibly have been powered to detect small changes and a larger cohort used.

- Including partners or other family members in the study may have enhanced motivation and compliance. While mentorship and group actions were encouraged, more organisation of participants in supporting each other may have been advantageous.

- Including a beep test (or similar) may have been useful in assessing changes in physical fitness.

- More visible reminders e.g. signs at lift to use the stairs, sign on fridge to encourage changes to type of milk used may have reminded and challenged participants to change.

- The group advice given was personalised on the basis of height, weight and physical activity in a table they could refer to in their own booklet. Some individual spoken personalised advice first (still based on the information in the study) would ensure that participants understood the tables better and make them feel more cared about and motivated.

- Measuring participants more frequently and giving them their results would help with motivation too

- The group information session would have been better if divided into several sessions. One session gave too much information in too short a time. Several sessions, each covering different aspects of the advice and maybe re emphasising some important messages may help to ensure that all messages were being received and understood.

- More visuals at the group sessions and measurement sessions may also help to drive home messages. Fact sheets were available but there was not enough time to read/look at them in detail.

- The regular newsletters worked well. They were sent as emails which is an easy, cost-effective way of sending messages (in populations that have email access). They served as a way of disseminating new information, stressing messages already given and motivating participants by reminding them that they were supposed to be making changes. Recipes, jokes and motivational statements/sayings helped to make the newsletters friendly.

8.2.2.2 Improvements to the advice given

- Recent research (Yancy 2004, Samaha 2003) suggests that a diet that is higher in protein and lower in carbohydrate may be better for some people. The diet suggested in this study was 50-60% energy from carbohydrate, 15-18% energy from protein and 24-27% energy from fat. It may be better to use a less carbohydrate, more protein and relative fat with an emphasis on omega-3 fatty acids.

- Advice given was to aim for 10 000 steps per day. A steady increase in steps from baseline to slowly improve fitness is probably more prudent advice. A recent article by Tudor- Locke and Bassett (2004) posed the question of “how many steps are enough?” They pointed out that different ages and sexes require different numbers of steps and that increasing increments to indicate different levels of activity may be more appropriate use of pedometers. While the

pedometer was used in this study for motivation rather than as a measure of change, goals may have been more easily attained if the number of steps to aim for was less definitive.

The challenge is to find ways of effectively influencing the behaviours of healthy individuals to reduce lifestyle diseases and the burden of ill health (financial and emotional) within their own environment and using readily available resources.

8.2.3 What was achieved?

- A small reduction in measures of lifestyle disease risk was achieved.
- Awareness was raised in the AUT workplace and beyond – most did not actively realise at first that they were at risk.
- Recommendations for changes in the workplace, suggested by participants were sent to management.
- Pedometers were promoted as motivation tools and a 'club' that records steps walked and provides destinations to walk to as groups was instigated to further motivate walking.
- Other interventions using much of the same messages and methods were undertaken.
- A book (Move more, eat less. Elaine Rush, 2004) was written using some of the material from the study and sales are being used to fund further research. A website was also started as part of the second AUT worksite intervention study.
- As a result of some participants being recruited from the Arts faculty, posters were designed by graphic design students, using some of the messages from the study. These were used in the book, as posters and as magnets to further create awareness of lifestyle factors in disease.

8.2.4 Subsequent studies and future research

Studies and searching for evidence for interventions to reduce the risk of lifestyle disease are ongoing and increasing in importance and interest as the burden of lifestyle diseases increases.

Two of the larger reports on LD are the World Health Organization report (WHO 2002a) and a study looking at the burden of disease in New Zealand by Auckland University and the Ministry of Health (Ministry of Health 2003).Many smaller studies and many more reductionist studies have been done since work on this study started in 2002. Some of the results have further confused the evidence and need further study. Other studies have strengthened the evidence.

At AUT a study in another off site workplace and another involving the Asian Indian population in Auckland have adapted and refined methods used in this study with good results.

Also at AUT studies associated with this intervention viz. the effect of kiwifruit on the stability of DNA assessed by comet assay and antioxidant changes in plasma are looking promising and will be reported in the near future.

It is essential to find ways of lowering the continually growing burden of lifestyle diseases. While some of the explosion is related to genetics and some to the increased lifespan of humans, much of the increase of chronic disease is related to the changes in dietary and physical activity factors over the last few decades. Technological, environmental and medical advances have increased the possibilities of humans suffering from one of the diseases researched in this study. Making changes that can reduce the risks of these diseases is feasible and what changes to make and the way to accomplish such changes needs to be widely disseminated.

Group ‘shake- ups’ with regular follow up emails to broadcast evidence-based advances could also be used to facilitate change. There is no magic bullet and small positive changes that become part of a usual lifestyle are needed to effect a reduction in overall risk. These changes need to be aimed at everyone- not just those at high risk. They need to become part of the norm rather than a prescription for only those identified as being at risk

"An ounce of prevention is better than a pound of cure" - Desiderius Erasmus

8.2.5 Suggestions from participants to facilitate change.

The following are suggestions from the participants and discussions with the participants when they were advised of the findings in the first 12 weeks of the study. By actively participating in the research and suggesting changes themselves improved compliance and created interest. E.g. one participant who logged thousands of steps by riding in the back of a bumpy bus!

The last words in this thesis are a report of what the participants said that they wanted to happen.

Ideas on ways to improve staff healthy lifestyle options for staff

Support networks set up to:

- go walking
- play sport
- compare pedometer readings
- promote attendance at exercise programs

Fruit more available in

- offices
- cafeterias and
- the fruit to be subsidized by management?

Increase availability of healthy snacks – particularly outside of cafeteria hours

Cafeteria food – healthy options labelled and maybe subsidized (e.g. no margarine; low fat dressings; less protein, more salad; grainier breads)

Notices to encourage

- use of stairs as opposed to lift
- green milk vs light blue vs dark blue e.g. Use the healthier option with less fat
- Lower fat milk more available – would need to be monitored to prevent wastage.

Proper, designated, mandatory lunch breaks. NO meetings, seminars etc scheduled over lunch breaks. Maybe they could be scheduled at an alternate time.

Better facilities for

- cooking
- showering – many volunteers mentioned shower availability for staff who want to walk or exercise during lunch. Maybe awareness of where there are showers available for a start

Better management of good measures already in place

- Keeping water coolers well stocked
- Milk as mentioned
- Stairs as already mentioned
- showers as mentioned
- Free Gym membership – I think only if used- maybe a sliding scale dependant on how many times they actually go!!
- Pedometers available
- Remove snack bars and vending machines

8.3 Conclusion

This study has shown that changes in diet and physical activity can favourably influence blood biochemistry even without accompanying changes in percentage body fat and weight. Furthermore, small, manageable lifestyle changes can result in biochemical changes persisting over 52 weeks.

References

Abramson B (2001), Risk factors and primary prevention of ischemic heart disease in women. Can J Cardiol; **17**(Suppl D): 24D-31D.

Amos AF, McCarty DJ, Zimmet P. (1997), The rising global burden of diabetes and its complications: estimates and projections to the year 2010. Diabet Med; **14** (Suppl 5): S1-85.

Anderson JW, Konz EC, Frederich RC, Wood CL. (2001), Long-term weight-loss maintenance: a meta-analysis of US studies. Am J Clin Nutr; **74**(5): 579-84.

Anderson JW and Hanna TJ. (1999), Impact of nondigestible carbohydrates on serum lipoproteins and risk for cardiovascular disease. J Nutr; **129**(7 Suppl): 1457S-66S.

Anonymous. (1996), Bioelectrical impedance analysis in body composition measurement: National Institutes of Health Technology Assessment Conference Statement. Am J Clin Nutr; **64**(3 Suppl): 524S-533S.

Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al. (1997), A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med; **336** (16): 1117-24.

Ascherio A, Rimm EB, Stampfer MJ, Giovannucci EL, Willett WC. (1995), Dietary intake of marine n-3 fatty acids, fish intake, and the risk of coronary disease among men. N Engl J Med; **332**(15): 977-82.

Avins AL and Neuhaus JM. (2000), Do triglycerides provide meaningful information about heart disease risk? Arch Intern Med;**160**(13): 1937-44.

Bantle JP, Raatz SK, et al. (2000), Effects of dietary fructose on plasma lipids in healthy subjects. Am J Clin Nutr**; 72**(5): 1128-34.

Bassett DR, Jr., Ainsworth BE, Leggett SR, Mathien CA, Main JA, Hunter D, Duncan GE. (1996), Accuracy of five electronic pedometers for measuring distance walked. Med Sci Sports Exerc; **28**(8): 1071-7.

Bassett DR Jr. (2000), Validity and reliability issues in objective monitoring of physical activity. Res Q Exerc Sport; **71**(2 Suppl): S30-6.

Batty D. (2000), Does physical activity prevent cancer? BMJ; **321**(7274): 1424.

Bazzano LA, Serdula MK, Liu S. (2003), Dietary intake of fruits and vegetables and risk of cardiovascular disease. Curr Atheroscler Rep; **5**(6): 492-9.

Bell AC, Swinburn BA, Amosa H, Scragg RK. (2001), A nutrition and exercise intervention program for controlling weight in Samoan communities in New Zealand. Int J Obes Relat Metab Disord; **25**(6): 920-7.

Bell AC, Swinburn BA, Simmons D, Wang W, Amosa H, Gatland B. (2001a), Heart disease and diabetes risk factors in Pacific Islands communities and associations with measures of body fat. N Z Med J; **114**(1131): 208-13.

Bemelmans WJ, Muskiet FA, Feskens EJ, de Vries JH, Broer J, May JF, Jong BM. (2000), Associations of alpha-linolenic acid and linoleic acid with risk factors for coronary heart disease. Eur J Clin Nutr; **54**(12): 865-71.

Berdanier C. (1994), Omega-3 fatty acids: a panacea? Nutrition Today; **29**(4): 28-33.

Berlin JA and Colditz GA. (1990), A meta-analysis of physical activity in the prevention of coronary heart disease. Am J Epidemiol; **132**(4): 612-28.

Bingham SA and Cassidy A. (1995), Validation of weighed records and other methods of dietary assessment using the 24 h urine technique and other biological markers. Br. J. Nutr; **73**: 531–550.

Bonnefoy M, Normand S, Pachiaudi C, Lacour JR, Laville M, Kostka T. (2001), Simultaneous validation of ten physical activity questionnaires in older men: a doubly labeled water study. J Am Geriatr Soc; **49**(1): 28-35.

Bonora E, Kiechl S, Kiechl S, Willeit J, Oberhollenzer F, Egger G, Bonadonna RC, Muggeo M. (2003), Metabolic syndrome: epidemiology and more extensive phenotypic description. Cross-sectional data from the Bruneck Study. Int J Obes Relat Metab Disord; **27**(10): 1283-9.

Bonora E, Targher G, Alberiche M, Bonadonna RC, Saggiani F, Zenere MB, Monauni T, Muggeo M. (2000), Homeostasis model assessment closely mirrors the glucose clamp technique in the assessment of insulin sensitivity: studies in subjects with various degrees of glucose tolerance and insulin sensitivity. Diabetes Care; **23**(1): 57-63.

Bonow RO, Smaha LA , Smith SC Jr, Mensah GA, Lenfant C. (2002), World Heart Day 2002: the international burden of cardiovascular disease: responding to the emerging global epidemic. Circulation; **106**(13): 1602-5.

Brach JS, Simonsick EM, Kritchevsky S, Yaffe K, Newman AB. (2004), The association between physical function and lifestyle activity and exercise in the health, aging and body composition study. J Am Geriatr Soc; **52**(4): 502-9.

Bravata DM, Sanders L, Huang J, Krumholz HM, Olkin I, Gardner CD. (2003), Efficacy and safety of low-carbohydrate diets: a systematic review. JAMA; **289**(14): 1837-50.

Bray GA, Culbert JW, Champagne CM, Dawson L et al. (1999), The Diabetes Prevention Program: Design and methods for a clinical trial in the prevention of type 2 diabetes Diabetes Care; **22**, 623.

Brindle P, Emberson J, Lampe F, Walker M, Whincup W, Fahey T, Ebrahim, S. (2003), Predictive accuracy of the Framingham coronary risk score in British men: prospective cohort study. BMJ; **327**(7426): 1267-1271.

Brown D and International Obesity Task Force. (2003), About Obesity. International Association for the study of Obesity. http://www.iotf.org Accessed 10/02/04.

Brown DJ and Goodman J. (1998), A review of vitamins A, C, and E and their relationship to cardiovascular disease. Clin Excell Nurse Pract; **2**(1): 10-22.

Brown DW, Balluz LS, Heath GW, Moriarty DG, Ford ES, Giles WH, Mokdad AH. (2003), Associations between recommended levels of physical activity and health-related quality of life. Findings from the 2001 Behavioral Risk Factor Surveillance System (BRFSS) survey. Prev Med; **37**(5): 520-8.

Brown L, Rosner B, Willett W, Sacks FM. (1999), Cholesterol-lowering effects of dietary fiber: a meta-analysis. Am Clin Nutr; **69**(1): 30-42.

Burr ML, Ashfield-Watt PA, Dunstan FD, Fehily AM, Breay P, Ashton T, Zotos PC, Haboubi NA, Elwood PC.(2003), Lack of benefit of dietary advice to men with angina: results of a controlled trial. Eur J Clin Nutr; **57**(2): 193-200.

Buzzard IM, Asp EH, Chlebowski RT, Boyar AP, Jeffery RW, Nixon DW, Blackburn GL, Jochimsen PR, Scanlon EF, Insull W Jr, et al. (1990), Diet intervention methods to reduce fat intake: nutrient and food group composition of self-selected low-fat diets. J Am Diet Assoc; **90**(1): 42-50, 53.

Cancer Society of New Zealand (2003). New Zealand Cancer Statistics. http://www.cancernz.org.nz/INFO/PDF/misc.pdf/NewZealandCancerStatistics1.pdf Accessed 18/06/04.

Cernea, S, Hancu N, Raz I. (2003), Diet and coronary heart disease in diabetes. Acta Diabetol; **40** Suppl 2: S389-400.

Chobanian, AV., Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, Jones DW, Materson BJ, Oparil S, Wright JT Jr, Roccella EJ. (2003), Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension; **42**(6): 1206-52.

Chumlea WC and Guo SS. (1994), Bioelectrical impedance and body composition: present status and future directions. Nutr Rev; **52**(4): 123-31.

Colagiuri S, Hussain Z, Zimmet P, Cameron A, Shaw J. (2004), Screening for Type 2 Diabetes and Impaired Glucose Metabolism: The Australian experience. Diabetes Care; **27**(2): 367-371.

Colditz GA, Feskanich D, Chen WY, Hunter D, Willett WC. (2003), Physical activity and risk of breast cancer in premenopausal women. Br J Cancer; **89**(5): 847-51.

Collins A, Harrington V, Drew J, Melvin R. (2003),Nutritional modulation of DNA repair in a human intervention study. Carcinogenesis; **24**(3): 511-515.

Collins B, Horska A, Hotten P, Riddoch C, Collins A. (2001), Kiwifruit protects against oxidative DNA damage in human cells and in vitro. Nutr Cancer; **39**(1): 148-153.

Craig WJ. (1997), Phytochemicals: guardians of our health. J Am Diet Assoc; **97**(10 Suppl 2): S199-204.

Crott JW and Fenech M. (1999), Effect of vitamin C supplementation on chromosome damage, apoptosis and necrosis ex vivo. Carcinogenesis; **20**(6): 1035-41.

Czernichow S and Hercberg S. (2001), Interventional studies concerning the role of antioxidant vitamins in cardiovascular disease: a review. JNHA; **5**(3).

D'Agostino RB, Grundy S, Lisa M Sullivan, Peter Wilson. (2001), Validation of the Framingham coronary heart disease prediction scores: Results of a multiple ethnic groups investigation. JAMA; **286**(2):180 –188.

de Deckere EA. (1999), Possible beneficial effect of fish and fish n-3 polyunsaturated fatty acids in breast and colorectal cancer. Eur J Cancer Prev; **8**(3): 213-21.

DeBusk RF, Stenestrand U, Sheehan M, Haskell WL. (1990), Training effects of long versus short bouts of exercise in healthy subjects. Am J Cardiol; **65**(15): 1010-3.

Demura S, Yamaji S, Goshi F, Nagasawa Y. (2002), The influence of transient change of total body water on relative body fats based on three bioelectrical impedance analyses methods. Comparison between before and after exercise with sweat loss, and after drinking. J Sports Med Phys Fitness; **42**(1): 38-44.

Despres JP. (2001), Health consequences of visceral obesity. Ann Med; **33**(8): 534-41.

Despres JP, Lemieux I, Dagenais GR, Cantin B, Lamarche B. (2000), HDL-cholesterol as a marker of coronary heart disease risk: the Quebec cardiovascular study. Atherosclerosis; **153**(2): 263-72.

Despres JP, Pascot A, Lemieux I. (2000a), Risk factors associated with obesity: a metabolic perspective. Ann Endocrinol (Paris); **61** Suppl 6: 31-38.

Despres JP, Lemieux I, Prud'homme D. (2001), Treatment of obesity: need to focus on high risk abdominally obese patients. BMJ; **322**(7288): 716-20.

Despres JP, Lemieux I, Tchernof A, Couillard C, Pascot A, Lemieux S. (2001), Fat distribution and metabolism. Diabetes Metab; **27**(2 Pt 2): 209-14.

Desroches S, Paradis M-E, Archer WR, Corneau L, Couture P, Bergeron N, Lamarche B. (2004), Low fat/high carbohydrate eating plan is not bad for good cholesterol, The American Heart Association's 5th Annual Conference on Arteriosclerosis, Thrombosis and Vascular Biology. http://www.americanheart.org/presenter.jhtml?identifier=3021874 Accessed 12/05/04.

Deurenberg P. (1996), Limitations of the bioelectrical impedance method for the assessment of body fat in severe obesity. Am J Clin Nutr; **64**(3 Suppl): 449S-452S.

Deurenberg P, Westrate JA, Hautvast JG. (1989), Changes in fat-free mass during weight loss measured by bioelectrical impedance and by densitometry. Am J ClinNutr; **49**(1): 33-36.

Dewailly EC, Blanchet C, Lemieux S, Sauve L, Gingras S, Ayotte P, Holub BJ. (2001), n-3 Fatty acids and cardiovascular disease risk factors among the Inuit of Nunavik. Am J Clin Nutr; **74**(4): 464-73.

Diabetes Prevention Programme. (2002), Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med, **346,**393-403

Doucet E S, St-Pierre S, Almeras N, Imbeault P, Mauriege P, Pascot A, Despres J P, Tremblay A. (2002), Reduction of visceral adipose tissue during weight loss. Eur J Clin Nutr; **56**(4): 297-304.

Durnin JVGA, de Bruin H, Feunekes GI. (1997), Skinfold thicknesses: is there a need to be very precise in their location? Br J Nutr; **77**(1): 3-7.

Durnin JVGA and Womersley J. (1974), Body fat assessed from total body density and its estimation from skinfold thickness:measurements on 481 men and women aged 16 to 72 years. Br J Nutr; **32**: 77-92.

Eberly LE, Cohen JD, Prineas R, Yang L. (2003), Impact of incident diabetes and incident nonfatal cardiovascular disease on 18- year old mortality : The Multiple Risk Factor Intervention Trial experience. Diabetes Care; **26**(3): 848-856.

Ebrahim S and Davey Smith G. (2000), Multiple risk factor interventions for primary prevention of coronary heart disease. Cochrane Database Syst Rev(2): CD001561.

Edelstein SL, Knowler WC, Bain RP, Andres R, Barrett-Connor EL, Dowse GK, Haffner SM, Pettitt DJ, Sorkin JD, Muller DC, Collins VR, Hamman RF. (1997), Predictors of progression from impaired glucose tolerance to NIDDM: an analysis of six prospective studies. Diabetes; **46**: 701-710.

Ehtisham STG, Barrett TG, Shaw NJ. (2000), Type 2 diabetes mellitus in UK children-an emerging problem. Diabet Med; **17**(12): 867-71.

Elley CR, Kerse N, Arroll B, Robinson E. (2003), Effectiveness of counselling patients on physical activity in general practice: cluster randomised controlled trial. BMJ; **326**(7393): 793.

Ellis KJ, Bell SJ, Chertow GM, Chumlea WC, Knox TA, Kotler DP, Lukaski HC, Schoeller DA. (1999), Bioelectrical impedance methods in clinical research: a follow-up to the NIH Technology Assessment Conference. Nutrition; **15**(11-12): 874-80.

Eriksson KF and Lundgarde F. (1991), Prevention of type 2 (non-insulin dependent) diabetes mellitus by diet and physical exercise. Diabetologia; **34**: 891-898.

Evenson KR, Rosamond WD, Cai J, Toole JF, Hutchinson RG, Shahar E, Folsom AR (1999), Physical activity and ischemic stroke risk. The atherosclerosis risk in communities study. Stroke; **30**(7): 1333-9.

Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. (2002), Selected major risk factors and global and regional burden of disease. Lancet; **360**(9343): 1347-60

Fall CHD, Vijaykumar M, Barker DJP, Osmond C, Duggleby S. (1995), Weight in infancy and prevalence of coronary heart disease in adult life. BMJ; **310**: 17-20.

Fang YZ, Yang S, Wu G. (2002), Free radicals, antioxidants, and nutrition. Nutrition; **18**(10): 872-9.

Ferguson AR and Ferguson LR. (2003), Are kiwifruit really good for you? Acta Horticulturae; **610**: 131-135.

Ferguson LR, Yee RL, Scragg R, Metcalf PA, Harris PJ. (1995), Differences in intake of specific food plants by Polynesians may explain their lower incidence of colorectal cancer compared with Europeans in New Zealand. Nutr Cancer; **23**(1): 33-42.

Fleischauer AT, Olson SH, Mignone L, Simonsen N, Caputo TA, Harlap SW. (2001), Dietary antioxidants, supplements, and risk of epithelial ovarian cancer. Nutr Cancer; **40**(2): 92-8.

Fogelholm GM, Sievanen HT, van Marken Lichtenbelt WD, Westerterp KR. (1997), Assessment of fat-mass loss during weight reduction in obese women. Metabolism; **46**(8): 968-75.

Foster GD,Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein SW. (2003), A randomized trial of a low-carbohydrate diet for obesity. N Engl J Med; **348**(21): 2082-90.

Franz MJ. (1998), Managing obesity in patients with comorbidities. J Am Diet Assoc; **98**(10 Suppl 2): S39-43.

Franz MJ, Bantle JP, Beebe CA, Brunzell JD, Chiasson JL, Garg A, Holzmeister LA, Hoogwerf B, Mayer-Davis E, Mooradian AD, Purnell JQ, Wheeler M,. (2002), Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. Diabetes Care; **25**(1): 148-98.

Freeland-Graves J, and Nitzke S. (2002), Position of the American Dietetic Association: total diet approach to communicating food and nutrition information. J Am Diet Assoc; **102**(1): 100-8.

Freitag, M. H. and R. S. Vasan. (2003), What is normal blood pressure? Curr Opin Nephrol Hypertens; **12**(3): 285-92.

Friedenreich CM. (2001), Physical activity and cancer: lessons learned from nutritional epidemiology. Nutr Rev; **59**(11): 349-57.

Fuchs CS, E. L. Giovannucci, Colditz GA, Hunter DJ, Stampfer MJ, Rosner B, Speizer FE, Willett WC. (1999), Dietary fiber and the risk of colorectal cancer and adenoma in women. N Engl J Med; **340**(3): 169-76.

Fung TT, Hu FB, Pereira MA, Liu S, Stampfer MJ, Colditz GA, Willett WC. (2002), Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. Am J Clin Nutr; **76**(3): 535-40.

Garcia-Closas RL, Serra-Majem L, Segura, R. (1993), Fish consumption, omega-3 fatty acids and the Mediterranean diet. Eur J Clin Nutr; **47** Suppl 1: S85-90.

Gill M, Ockelford P, Morris A, Bierre T, Kyle C. (2000), The Interpretation of Laboratory Tests Diagnostic Medlab http://www.diagnostic.co.nz/hbook/index.htm Accessed 16/01/03

Ginsberg HN. (1997), Is hypertriglyceridemia a risk factor for atherosclerotic cardiovascular disease? A simple question with a complicated answer. Ann Intern Med; **126**(11): 912-4.

Giovannelli L, Saieva C, Masala G, Testa G, Salvini S, Pitozzi V, Riboli E, Dolara P, Palli D. (2002), Nutritional and lifestyle determinants of DNA oxidative damage: a study in a Mediterranean population. Carcinogenesis; **23**(9): 1483-9.

Goris AHMS, Westerterp-Plantenga MS, Westerterp, K. R. (2000), Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake. Am J Clin Nutr; **71**(1): 130-4.

Goto C, Higashi Y, Kimura M, Noma K, Hara K, Nakagawa K, Kawamura M, Chayama K, Yoshizumi M Nara I. (2003), Effect of different intensities of exercise on endothelium-dependent vasodilation in humans: role of endothelium-dependent nitric oxide and oxidative stress. Circulation; **108**(5): 530-5.

Gotto AM Jr. (1997), The Multiple Risk Factor Intervention Trial (MRFIT): A return to a landmark trial. JAMA; **277**(7): 595-598.

Greenland P, Knoll MD, Stamler J, Neaton JD, et al. (2003), Major risk factors as antecedents of fatal and nonfatal coronary heart disease events. JAMA; **290**(7): 891.

Gutin B, Barbeau P, Owens S, Lemmon CR, Bauman M, Allison J, Kang HS, Litaker MS. (2002), Effects of exercise intensity on cardiovascular fitness, total body composition, and visceral adiposity of obese adolescents. Am J Clin Nutr; **75**(5): 818-26.

Ha MA, Jarvis MC, Mann JI. (2000), A definition for dietary fibre. Eur J Clin Nutr; **54**(12): 861-4.

Haffner SM, Kennedy E, Gonzalez C, Stern MP, Miettinen H. (1996), A prospective analysis of the HOMA model. The Mexico City Diabetes Study. Diabetes Care; **19**(10): 1138-41.

Haffner SM, Miettinen H, Stern MP. (1997), The homeostasis model in the San Antonio Heart Study. Diabetes Care; **20**(7): 1087-92.

Hardiman P, Pillay OS, Atioma W. (2003), Polycystic ovary syndrome and endometrial carcinoma. Lancet; **361**(9371): 1810.

Harding AH, Williams DE, Hennings SH, Mitchell J, Wareham NJ. (2001), Is the association between dietary fat intake and insulin resistance modified by physical activity? Metabolism; **50**(10): 1186-92.

Harsha DW, LinPH, Obarzanek E, Karanja NM, Moore TJ, Caballero B. (1999), Dietary Approaches to Stop Hypertension: a summary of study results. DASH Collaborative Research Group. J Am Diet Assoc; **99**(8 Suppl): S35-9.

He J and Whelton PK. (1997), Role of sodium restriction in treatment and prevention of hypertension. Curr Opinion Cardiol; **12**(2): 202-207.

He K, Rimm EB, Merchant A, Rosner BA, Stampfer MJ, Willett WC, Ascherio A. (2002), Fish consumption and risk of stroke in men. JAMA; **288**(24): 3130-6.

Hense HW. (2003), Risk factor scoring for coronary heart disease. BMJ; **327(**7426): 1238-9.

Hermans M, Levy J, Morris R, Turner R. (1999), Comparison of insulin sensitivity tests across a range of glucose tolerance from normal to diabetes. Diabetologia; **42**: 678-687.

Hills M and Armitage P. (1979), The two period cross-over clinical trial. Brit J Clin Pharmaco; **8**: 7-20.

Hoffmann I. (2003), Transcending reductioism in nutrition research. Am J Clin Nutr; **78**(3): 514S-516S.

Hogan P, Dall T, Nikolov P. (2003), Economic costs of diabetes in the U.S. in 2002. Diabetes Care; **26**: 917-932.

Hooper L, Summerbell CD, Higgins JP, Thompson RL, Capps NE, Smith GD, Riemersma RA, Shah E. (2001), Dietary fat intake and prevention of cardiovascular disease: systematic review. BMJ; **322**(7289): 757.

Houtkooper LB, Lohman TG, Going SB, Howell WH. (1996), Why bioelectrical impedance analysis should be used for estimating adiposity. Am J Clin Nutr; **64(**3 Suppl): 436S-448S.

Howe GR, Benito E, Castelleto R, Cornee J, Esteve J, Gallagher RP, Iscovich JM, Deng-ao J, Kaaks R, Kune GA, et al. (1992), Dietary intake of fiber and decreased risk of cancers of the colon and rectum: evidence from the combined analysis of 13 case-control studies. J Natl Cancer Inst; **84**(24): 1887-96.

Hu FB, Bronner L, Willett WC, Stampfer MJ, Rexrode KM, Albert CM, Hunter D, Manson J. (2002), Fish and omega-3 fatty acid intake and risk of coronary heart disease in women. JAMA; **287**(14): 1815-21.

Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. (2003), Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. JAMA; **289**(14): 1785-91.

Hu FB, Manson J, Willett W. (2001), Types of dietary fat and risk of coronary heart disease: a critical review. J Am Coll Nutr; **20**(1): 5-19.

Hu FB, Sigal RJ, Rich-Edwards JW, Colditz GA, Solomon CG, Willett W, Speizer FE, Manson JE. (1999), Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a prospective study. JAMA; **282**(15): 1433-9.

Hu FB, Stampfer MJ, Colditz GA, Ascherio A, Rexrode KM, Willett WC,

Manson JE. (2000), Physical activity and risk of stroke in women. JAMA; **283**(22): 2961-7.

Hu FB, van Dam RM, Liu S. (2001), Diet and risk of Type II diabetes: the role of types of fat and carbohydrate. Diabetologia; **44**(7): 805-17.

Hu FB and Willett WC (2001), Diet and coronary heart disease: findings from the Nurses' Health Study and Health Professionals' Follow-up study. JNHA; **5**(3): 132-138.

Ip C. (1997), Review of the effects of trans fatty acids, oleic acid, n-3 polyunsaturated fatty acids, and conjugated linoleic acid on mammary carcinogenesis in animals. Am J Clin Nutr; **66**(6 Suppl): 1523S-1529S.

Iwane M, Arita M, Tomimoto S, SataniO, Matsumoto M, Miyashita K, Nishio I.

(2000), Walking 10,000 steps/day or more reduces blood pressure and sympathetic nerve activity in mild essential hypertension. Hypertens Res**; 23**(6): 573-80.

Janssen I, Fortier A, Hudson R, Ross R. (2002), Effects of an energy-restrictive diet with or without exercise on abdominal fat, intermuscular fat, and metabolic risk factors in obese women. Diabetes Care; **25**(3): 431-8.

Janssen I, Katzmarzyk PT, Ross R. (2002a), Body mass index, waist circumference, and health risk: evidence in support of current National Institutes of Health guidelines. Arch Intern Med; **162**(18): 2074-9.

Jee SH, Miller ER 3rd, Guallar E, Singh VK, Appel LJ, Klag MJ. (2002), The effect of magnesium supplementation on blood pressure: a meta-analysis of randomized clinical trials. Am J Hypertens; **15**(8): 691-6.

John EM, Horn-Ross PL, Koo J. (2003), Lifetime physical activity and breast cancer risk in a multiethnic population: the San Francisco Bay area breast cancer study. Cancer Epidemiol Biomarkers Prev; **12**(11 Pt 1): 1143-52.

John JH, Ziebland S, Yudkin P, Roe S, Neil AW. (2002), Effects of fruit and vegetables consumption on plasma antioxidant concentrations and blood pressure: A randomised controlled trial. Lancet; **359**(9322): 1969-1974.

Joint National Committee (JNC-V). (1993), The fifth report of the Joint National Committee on detection, evaluation and treatment of high blood pressure (JNC-V). Arch Intl Med; **153**: 154-183.

Kabagambe EK, Baylin A, Allan DA, Siles X, Spiegelman D, Campos H. (2001), Application of the method of triads to evaluate the performance of food frequency questionnaires and biomarkers as indicators of long-term dietary intake.Am J Epidemiol; **154**(12): 1126-35.

Kaput J and Rodriguez RL. (2004), Nutritional genomics: the next frontier in the postgenomic era. Physiol Genomics; **16**(2): 166-77.

Kanazawa M, Xue CY, Kageyama H, Suzuki E, Ito R, Namba Y, Osaka T, Kimura S, Inoue S. (2003), Effects of a high-sucrose diet on body weight, plasma triglycerides, and stress tolerance. Nutr Rev; **61**(5 Pt 2): S27-33.

Kant, A. K., A. Schatzkin, Graubard BI, Schairer C. (2000), A prospective study of diet quality and mortality in women. JAMA; **283**(16): 2109-15.

Keevil JG, Stein JH, McBride PE. (2002), Cardiovascular disease prevention. Prim Care; **29**(3): 667-96.

Kennedy EL, Meyers, Layden, W. (1996), The 1995 dietary guidelines for Americans: an overview. J Am Diet Assoc; **96**(3): 234-7.

Kennedy ET, Ohls J, Carlson S, Fleming K. (1995), The Healthy Eating Index: design and applications. J Am Diet Assoc; **95**(10): 1103-8.

Key T, Allen N, Spencer EA, Travis RC. (2002), The effect of diet on risk of cancer. Lancet; **360**(9336): 861.

Key TJ, Verkasalo PK, Banks E. (2001), Epidemiology of breast cancer. Lancet Oncology; **2**: 133-140.

King H, Aubert RE, Herman WH. (1998), Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. Diabetes Care; **21**: 1414-1431.

Knekt P, Jarvinen R, Reunanen A, Maatela J. (1996), Flavonoid intake and coronary mortality in Finland: a cohort study. BMJ; **312**(7029): 478-81.

Knekt P, Reunanen A, Jarvinen R, Seppanen R, Heliovaara M, Aromaa A. (1994), Antioxidant vitamin intake and coronary mortality in a longitudinal population study. Am J Epidemiol; **139**(12): 1180-9.

Kretsch MJ, Fong AK, Green MW. (1999), Behavioural and body size correlates of energy intake underreporting by obese and normal weight women. J Am Dietet Assoc; **99**: 300-306.

Kreuter MW, Oswald DL, Bull F C, Clark E M. (2000), Are tailored health education materials always more effective than non-tailored materials? Health Educ Res; **15**(3): 305-315.

Kris-Etherton PM, Daniels SR, Eckel RH, Engler M, Howard BV, Krauss RM, Lichtenstein AH, Sacks FM, St Jeor S, Stampfer MJ, Grundy SM, Appel LJ, Byers T, Campos H, Cooney G, Denke MA, Kennedy E, Marckmann P, Pearson TA, Riccardi G, Rudel LL, Rudrum M, Stein DT , Tracy RP et al. (2001), AHA scientific statement: summary of the Scientific Conference on Dietary Fatty Acids and Cardiovascular Health. Conference summary from the Nutrition Committee of the American Heart Association. J Nutr; **131**(4): 1322-6.

Kriska AM, Pereira MA, Hanson RL, de Courten MP, Zimmet PZ, Alberti KG, Chitson P, Bennett PH, Narayan KM, Knowler WC. (2001), Association of physical activity and serum insulin concentrations in two populations at high risk for type 2 diabetes but differing by BMI. Diabetes Care; **24**(7): 1175-80

Kriska AM, Saremi A, Hanson RL, Bennett PH, Kobes S, Williams D, Knowler WC. (2003), Physical activity, obesity, and the incidence of type 2 diabetes in a high-risk population. Am J Epidemiol; **158**(7): 669-75.

Kromhout D. (2001), Diet and cardiovascular diseases. JNHA; **5**(3): 144-149.

Kushner RF, Gudivaka R, Schoeller DA. (1996), Clinical characteristics influencing bioelectrical impedance analysis measurements. Am J Clin Nutr; **64**(3 Suppl): 423S-427S.

KushnerRF, Schoeller DA, Fjeld CR, Danford L. (1992), Is the impedance index (ht2/R) significant in predicting total body water? Am J Clin Nutr; **56**(5): 835-9.

Kuulasmaa K, Tunstall-Pedoe H, Dobson A, Fortmann S, Sans S, Tolonen H, Evans A, Ferrario M, Tuomilehto J. (2000), Estimation of contribution of changes in classic risk factors to trends in coronary-event rates across the WHO MONICA Project populations. Lancet; **355**(9205): 675-87.

Kwiterovich PO Jr., Vining EP, Pyzik P,Skolasky R Jr., Freeman JM. (2003), Effect of a high-fat ketogenic diet on plasma levels of lipids, lipoproteins, and apolipoproteins in children. JAMA; **290**(7): 912-20.

Lampe JW. (1999), Health effects of vegetables and fruit: assessing mechanisms of action in human experimental studies. Am J Clin Nutr; **70**(3 Suppl): 475S-490S.

Landry N, Bergeron N, Archer R, Samson P, Corneau L, Bergeron J, Deriaz O.

(2003), Whole-body fat oxidation rate and plasma triacylglycerol concentrations in men consuming an ad libitum high- carbohydrate or low- carbohydrate diet. Am J ClinNutr; **77**: 580-586.

Law MR and Wald NJ. (2002), Risk factor thresholds: their existence under scrutiny. BMJ; **324**: 1570-1576.

Law MR, Wald NJ, Wu T, Hackshaw A, Bailey A. (1994), Systematic underestimation of association between serum cholesterol concentration and ischaemic heart disease in observational studies: data from BUPA study. BMJ; **308**(6295): 363-367.

Lean MJ. (1995), Waist circumference as a measure for indicating need for weight management. BMJ; **311**(6998): 158.

Lean MJ and Han TS. (2002), Waist worries. Am J Clin Nutr; **76**(4): 699-700.

Le Masurier GC, Sidman CL, Corbin CB. (2003), Accumulating 10,000 steps: does this meet current physical activity guidelines? Res Q Exerc Sport; **74**(4): 389-94.

Lee CD, Blair SN, Jackson AS. (1999), Cardiorespiratory fitness, body composition, and all-cause and cardiovascular disease mortality in men. Am J Clin Nutr; **69**(3): 373-80.

Lee IM. (2003), Physical activity and cancer prevention--data from epidemiologic studies. Med Sci Sports Exerc; **35**(11): 1823-7.

Lee IM, Sesso HD, Paffenbarger RS Jr. (1999), Physical activity and risk of lung cancer. Int J Epidemiol; **28**(4): 620-5.

Lemieux I, Lamarche B, Couillard C, Pascot A, Cantin B, Bergeron J, Dagenais GR, Despres JP. (2001), Total cholesterol/HDL cholesterol ratio vs LDL cholesterol/HDL cholesterol ratio as indices of ischemic heart disease risk in men: the Quebec Cardiovascular Study. Arch Intern Med; **161**(22): 2685-92.

Leser M, Yanovski SZ, Yanovski JA. (2002), A low fat intake and greater activity level are associated with lower weight regain 3 years after completing a very-low-calorie diet. J Am Dietet Assoc;**102**(9): 1252-1256.

Levine M, Conry-Cantilena C, Wang Y, Welch RW, Washko PW, Dhariwal KR, Park JB, Lazarev A, Graumlich JF, King J, Cantilena LR. (1996), Vitamin C pharmacokinetics in healthy volunteers: evidence for a recommended dietary allowance. Proc Natl Acad Sci U S A; **93**(8): 3704-9.

Levy JC, Matthews DR, Hermans MP. (1998), Correct homeostasis model assessment (HOMA) evaluation uses the computer program. Diabetes Care; **21**(12): 2191-2192.

Liedtke RJ. (1997), Principles of Bioelectrical Impedance Analysis. 2003. http://rjlsystems.com/research/bia-principles.html Accessed 27/12/03.

Liese D, Roach AK, Sparks KC, Marquart L, D'Agostino RB, Mayer-Davis EJ. (2003), Whole-grain intake and insulin sensitivity: the Insulin Resistance Atherosclerosis Study. Am J Clin Nutr; **78**(5): 965-71.

Lopez AD and Murray CC. (1998), The global burden of disease, 1990-2020. Nat Med; **4**(11): 1241-3.

Ludwig DS. (2003),Glycemic load comes of age. J Nutr; **133**(9): 2695-6.

Maillard V, Bougnoux P, Ferrari P, Jourdan M-L, Pinault M, Lavillonnière F, Body G, Le Floch O, Chajès V. (2001), Predictive markers and cancer prevention. Int J Cancer; **98**(1): 78-83.

Manandhar MC and Anklesaria PS. (1997), Weight, skinfolds and circumference characteristics of poor elderly people in Mumbai, India. Asia Pacific J Clin Nutr; **6**(3): 191-199

Mann J. (2001), Dietary fibre and diabetes revisited. Eur J Clin Nutr; **55**(11): 919-21.

Mann JI. (2002), Diet and the risk of coronary heart disease and type 2 diabetes. Lancet; **360**(9335): 783-789.

Manson JE and Spelsberg A (1994), Primary prevention of non- insulin dependent diabetes mellitus. Am J Prev Med; **10**: 172-184.

Marlett JA, McBurney MI, Slavin JL. (2002), Position of the American Dietetic Association: health implications of dietary fiber. J Am Diet Assoc; **102**(7): 993-1000.

Martinez JA, Kearney JM, Kafatos A, Paquet S, Martinez-Gonzalez MA. (1999), Variables independently associated with self-reported obesity in the European Union. Public Health Nutr; **2**(1A): 125-33.

Martinez ME, Giovannucci E, Spiegelman D, Hunter DJ, Willett WC, Colditz GA. (1997), Leisure-time physical activity, body size, and colon cancer in women. Nurses' Health Study Research Group. J Natl Cancer Inst; **89**(13): 948-55.

Matthews DR, Hosker JP, Rudenski AS, Naylor BA,m Turner RC, Treacher DF. (1985), Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia; **28**(7): 412-419.

McAuley KA, Williams SM, Mann JI, Goulding A, Chrisholm A, Wilson N, Story G, McLay RT, Harper MJ, Jones IE. (2002), Intensive lifestyle changes are necessary

to improve Iinsulin sensitivity: A randomised controlled study. Diabetes Care; **25**(3): 445-453.

McCullough ML, Feskanich D, Stampfer MJ, Giovannucci EL, Rimm EB, Hu FB, Spiegelman D, Hunter DJ, Colditz GA, Willett WC. (2002), Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance. Am J Clin Nutr; **76**(6): 1261-71.

McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PW, Jacques PF. (2004), Carbohydrate nutrition, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham Offspring Cohort. Diabetes Care; **27**(2): 538-547.

McTiernan A, Kooperberg C, White E, Wilcox S, Coates R,Adams-Campbell LL, Woods N, Ockene J. (2003), Recreational physical activity and the risk of breast cancer in postmenopausal women: the Women's Health Initiative Cohort Study. JAMA; **290**(10): 1331-6.

Mensink RP, ZockPL, Kester ADM, Katan MB. (2003), Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clinl Nutr; **77**(5): 1146-1155.

Messina MJ, Lampe W, Birt DF, Appel LJ, Pivonka E, Berry B, Jacobs DR Jr. (2001), Reductionism and the narrowing nutrition perspective: time for reevaluation and emphasis on food synergy. J Am Diet Assoc; **101**(12): 1416-9.

Metcalf P, Swinburn B, Scragg R, Dryson E. (1997), Reproducibility and validity of a food frequency questionnaire in European and Polynesian New Zealanders. Ethn Health; **2**(4): 297-308.

Meydani M. (2002), Antioxidants in the prevention of chronic diseases. Nutr Clin Care; **5**(2): 47-9.

Michels KB, Solomon CG, et al. (2003), Type 2 diabetes and subsequent incidence of breast cancer in the Nurses Health Study. Diabetes Care; **26**(6): 1752.

Miller ER, Erlinger T, Young DR,Jehn M, Charleston J, Rhodes D, Wasan S, Appel LJ. (2002). Results of the Diet, Exercise, and Weight Loss Intervention Trial (DEW-IT). Hypertension; **40**(5): 612-8.

Ministry of Health, N. Z. (1999), Our Health, Our Future:Hauora pakari, koiora roa. Ministry of Health, Wellington, New Zealand.

Ministry of Health, N. Z. (2001), From Strategy to Reality - The WAVE Project - 4.Data Architecture. Ministry of Health, Wellington, New Zealand.

Ministry of Health, N.Z. (2003), Nutrition and the Burden of Disease: New Zealand 1997-2011. Ministry of Health, Wellington, New Zealand.

Ministry of Health, N. Z. (2003a), Cardiovascular disease toolkit. Ministry of Health, Wellington, New Zealand. 2003.

Ministry of Health, N. Z. (2003b), Healthy eating- Healthy action, Oranga Kai- Oranga Pumau: A background. Ministry of Health, Wellington, New Zealand.

Moore LL, Visioni AJ, Wilson PW, D'Agostino RB, Pinkie WD, Ellison RC. (2000), Can sustained weight loss in overweight individuals reduce the risk of diabetes mellitus? Epidemiology; **3**: 269-273.

Morley AA and Trainor KJ. (2001), Lack of an effect of vitamin E on lifespan of mice. Biogerontology; **2**(2): 109-12.

Naglak MC, Mitchell DC, Shannon BM, Pearson TA, Harkness WL, Kris-Etherton PM. (2000), Nutrient adequacy of diets of adults with hypercholesterolemia after a cholesterol-lowering intervention: long-term assessment. J Am Diet Assoc; **100**(11): 1385-8.

National Academy of Sciences. (2001), Panel on the Definitions of Dietary Fiber and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Proposed Definition of dietary Fiber. 2001. http://books.nap.edu/books/0309075645/html/1.html Accessed 15/12/01

National Heart Lung and Blood Institute. (2002), Framingham Heart Study. http://www.nhlbi.nih.gov/about/framingham/ Accessed 14/12/03

Nelson NJ. (2000), Nurses health study: nurses helping science and themselves. J Natl Cancer Inst; **92**(8): 597-9.

Ness A, Egger M, Powles J. (1999), Fruit and vegetables and ischaemic heart disease: systematic review or misleading meta-analysis? Eur J Clin Nutr; **53**(11): 900-4.

Ness A, and Powles JW. (1997), Fruit and vegetables, and cardiovascular disease: a review. Int J Epidemiol; **26**(1): 1-13.

Nichols GA, Glauber HS, Brown JB. (2000), Type 2 diabetes: Incremental medical care costs during the 8 years preceding diagnosis. Diabetes Care; **23**: 1654.

Nkondjock A, Shatenstein B, Maisonneuve P, Ghadirian P. (2003), Assessment of risk associated with specific fatty acids and colorectal cancer among French-Canadians in Montreal: a case-control study. Int J Epidemiol; **32**(2): 200-9.

Owen RW, Giacosa A, Hull WE, Haubner R, Wurtele G, Spiegelhalder B, Bartsch H. (2000), Olive-oil consumption and health: the possible role of antioxidants. Lancet Oncol; **1**: 107-12.

Paffenbarger RS and Lee IM. (2001), Preventing coronary heart disease: The role of physical activity. Physician Sportsmed; **29**(2): 37-47.

Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang,YY Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV. (1997), Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. Diabetes Care; **20**: 537-544.

Paolini M, Sapone A, Canistro D, Chieco, Valgimigli L. (2003), Antioxidant vitamins for prevention of cardiovascular disease. Lancet; **362**(9387): 920; author reply 921.

Parks EJ and Hellerstein MK. (2000), Carbohydrate- induced hypertriglycerolemia: historical perspective and review of biological mechanisms. Am J ClinNutr; **71**(2): 412-433.

Peters, J. and S. J. Ulijaszek (1992), Population and sex differences in arm circumference and skinfold thicknesses among Indo-Pakistani children living in the East Midlands of Britain. Ann Hum Biol**; 19**(1): 17-22.

Phillips DH and Venitt S, Eds. (1995), Environmental mutagenesis. Human Molecular Genetics, Bios scientific.

Pigman HT, Gan DX, Krousel-Wood MA. (2002), Role of exercise for type 2 diabetic patient management. South Med J; **95**(1): 72-7.

Pool-Zobel BL, Bub A, Schroder N, Rechkemmer G. (1999), Anthocyanins are potent antioxidants in model systems but do not reduce endogenous oxidative DNA damage in human colon cells. Eur J Nutr; **38**(5): 227-34.

Popkin BM. (2001), The nutrition transition and obesity in the developing world. J Nutr; **131**(3): 871S-873S.

Powell LA, Nieman DC, Melby C, Cureton K, Schmidt D, Howley ET Hill JO, Mault JR, Alexander H, Stewart DJ. (2001), Assessment of body composition change in a community-based weight management program. J Am Coll Nutr; **20**(1): 26-31.

Puska P, Vartiainen E, Tuomilehto J, Salomaa V, Nissinen A. (1998), Changes in premature deaths in Finland: successful long-term prevention of cardiovascular diseases. Bull World Health Organ; **76**(4): 419-25.

Pyke SD, Wood DA, Kinmonth AL, Thompson SG. (1997), Change in coronary risk and coronary risk factor levels in couples following lifestyle intervention. The British Family Heart Study. Arch Fam Med; **6**(4): 354-60.

Quatromoni PA, Copenhafer DL, Demissie S, D'Agostino RB, O'Horo CE, Nam BH, Millen BE. (2002), The internal validity of a dietary pattern analysis. The Framingham Nutrition Studies. J Epidemiol Community Health**; 56**(5): 381-8.

Reaven G. (1995), Pathophysiology of insulin resistance in human disease. Physiol Rev; **75**(3): 473-86.

Reaven G. (2000), Diet and Syndrome X. Curr Atheroscler Rep; **2**(6): 503-7.

Reaven, G. (2002), Metabolic syndrome: pathophysiology and implications for management of cardiovascular disease. Circulation; **106**(3): 286-8.

Renaud SC. (2001), Diet and stroke. JNHA; **5**(3): 167-172.

Rhodes LE, Shahbakhti H, Azurdia RM, Moison RM, Steenwinkel MJ, Homburg MI, Dean MP, McArdle F, Beijersbergen van Henegouwen GM, Epe B, Vink AA. (2003), Effect of eicosapentaenoic acid, an omega-3 polyunsaturated fatty acid, on UVR-related cancer risk in humans. An assessment of early genotoxic markers. Carcinogenesis; **24**(5): 919-25.

Riboli E, Elmstahl S, Saracci R, Gullberg B, Lindgarde F. (1997), The Malmo Food Study: validity of two dietary assessment methods for measuring nutrient intake. Int J Epidemiol; **26** Suppl 1: S161-73.

Rickheim PL, Weaver TW, Flader JL, Kendall DM. (2002), Assessment of group versus individual diabetes education: a randomized study. Diabetes Care; **25**(2): 269-74.

Rimm EB, Ascherio A, Giovannucci E, Spiegelman D,Stampfer MJ, Willett WC. (1996), Vegetables, fruit and cereal fiber intake and risk of coronary heart disease among men. JAMA; **275**: 447-451.

Ripsin CM, Keenan JM, Jacobs DR Jr., Elmer PJ, Welch RR, Van Horn L, Liu K, Turnbull WH,Thye FW, Kestin M. et al. (1992), Oat products and lipid lowering. JAMA; **267**(24): 3317.

Roberts CK, Vaziri ND, Barnard RJ. (2002), Effect of diet and exercise intervention on blood pressure, insulin,oxidative stress and nitric oxide availability. Circulation; **106**(20): 2530-2532.

Rolls BJ, Morris EL, Roe LS. (2002), Portion size of food affects energy intake in normal-weight and overweight men and women. Am J Clin Nutr; **76**(6): 1207-13.

Rosenbloom AL. (2003), Obesity, Insulin Resistance, beta-Cell Autoimmunity, and the Changing Clinical Epidemiology of Childhood Diabetes. Diabetes Care; **26**(10): 2954-6.

Rosenbloom AL, Joe JR, Young RS, Winter WE. (1999), Emerging epidemic of type 2 diabetes in youth. Diabetes Care; **22**(2): 345-54.

Ross R, Dagnone D, Jones PJ, Smith H, Paddags A, Hudson R Janssen I. (2000), Reduction in obesity and related comorbid conditions after diet-induced weight loss or exercise-induced weight loss in men. A randomized, controlled trial. Ann Intern Med; **133**(2): 92-103.

Ross R, Janssen I, Tremblay A. (2000), Obesity reduction through lifestyle modification. Can J Appl Physiol; **25**(1): 1-18.

Ross R, Leger L, Martin P, Roy R. (1989), Sensitivity of bioelectrical impedance to detect changes in human body composition. J Appl Physiol; **67**(4): 1643-8.

Ross R and Rissanen J. (1994), Mobilization of visceral and subcutaneous adipose tissue in response to energy restriction and exercise. Am J Clin Nutr; **60**(5): 695-703.

Roychoudhury P and Schwartz K. (2003), Antioxidant vitamins do not prevent cardiovascular disease. J Fam Pract; **52**(10): 751-2.

Rush EC, Patel M, Plank LD, Ferguson LR. (2002), Kiwifruit promotes laxation in the elderly. Asia Pac J Clin Nutr; **11**(2): 164-8.

Russell D, Parnell W, Wilson N and the principal investigators of the 1997 National Nutrition Survey. (1999), NZ Food:NZ People - Key results of the 1997 National Nutrition Survey. Ministry of Health, Wellington, New Zealand.

Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, Obarzanek E, Conlin PR. Miller ER 3rd, Simons-Morton DG, Karanja N, Lin PH. (2001), Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. N Engl J Med; **344**(1): 3-10.

Sallis JF, Haskell WL, Wood PD, Rogers T, Blair SN, Paffenbarger RS Jr. (1985), Physical activity assessment methodology in the Five-City Project. Am J Epidemiol; **121**(1): 91-106.

Salmeron J, Hu FB, Manson JE, Stamler J, Colditz GA. (2001), Dietary fat intake and risk of type 2 diabetes in women. Am J ClinNutr; **73**: 1019-1026.

Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL,Willett W. (1997), Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA; **227**: 472-477.

Samaha FF, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams T, Williams M, Gracely EJ, Stern L. (2003), A low-carbohydrate as compared with a low-fat diet in severe obesity. N Engl J Med; **348**(21): 2074-81.

Saris WH, Blair SN, van Baak MA, Eaton SB, Davies PS,Di Pietro L, Fogelholm M, Rissanen A, Schoeller D, Swinburn BA, Tremblay A, Westerterp K, Wyatt H,.. (2003), How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obes Rev; **4**(2): 101-14.

Sesso HD, Paffenbarger RS Jr., Lee IM. (2000), Physical activity and coronary heart disease in men: The Harvard Alumni Health Study. Circulation; **102**(9): 975-80.

Sharma AM. (2002), Adipose tissue:a mediator of cardiovascular risk. Int J Obes Relat Metab Disord; **26**(Suppl 4): S5-7.

Shetty PS. (1997), Obesity and Physical activity, ANF. http://www.nutritionfoundationfindia.org?ARCHIVES/APR97A.HTM Accessed 26/04/02.

Silverstein JH and Rosenbloom AL. (2001), Type 2 diabetes in children. Curr Diab Rep; **1**(1): 19-27.

Simon MS, Lababidi S, Djuric Z, Uhley V, Depper J, Kresge C, Klurfeld DM, Heilbrun LK. (2001), Comparison of dietary assessment methods in a low-fat dietary intervention program. Nutr Cancer**; 40**(2): 108-17.

SPARC (Sport and Recreation New Zealand) (2003). Green prescription/Push Play, SPARC. http://www.pushplay.org.nz. Accessed 12/12/03.

Stamler J, Stamler R, Neaton JD, Wentworth D, Daviglus ML, Garside D, Dyer AR, Liu K, Greenland P. (1999), Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: findings for 5 large cohorts of young adult and middle-aged men and women. JAMA; **282**(21): 2012-8.

Statistics New Zealand (2003). Cancer-leading cause of death among females, Statistics New Zealand. Wellington, New Zealand. www.stats.govt.nz/domino/external/web/nzstories.nsf/1167b2c70ca821cb4c256 Accessed 15/11/03.

Stewart BW and Kleihues P. (2003),World Cancer Report, World Health Organisation (IARC).

Story JA. (2003), New insights into dietary fibre. Can J Diet Pract Res; **64**(4): 194A.

Swinburn B. (1998), Using the body mass index: weigh then weigh up. N Z Med J; **111**(1075): 377-9.

Swinburn B, Ashton T, Gillespie J, Cox B, Menon A, Simmons D, Birkbeck J. (1997), Health care costs of obesity in New Zealand. Int J Obes Relat Metab Disord; **21**(10): 891-6.

Swinburn BA, Craig PL, Daniel R, Dent DP, Strauss BJ. (1996), Body composition differences between Polynesians and Caucasians assessed by bioelectrical impedance. Int J Obes Relat Metab Disord; **20**(10): 889-94.

Swinburn BA, Walter LG, Arroll B, Tilyard MW, Russell DG. (1998), The green prescription study: a randomized controlled trial of written exercise advice provided by general practitioners. Am J Public Health; **88**(2): 288-91.

Swinburn BA, Woollard GA, Chang EC, Wilson MR. (1999), Effects of reduced-fat diets consumed ad libitum on intake of nutrients, particularly antioxidant vitamins. J Am Diet Assoc; **99**(11): 1400-5.

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; **288**(16): 1994-2000.

Tavani A, Pelucchi C, Parpinel M, Negri E, Franceschi S, Levi F, La Vecchia C. (2003), n-3 polyunsaturated fatty acid intake and cancer risk in Italy and Switzerland. Int J Cancer; **105**(1): 113-116.

Terry P, Rohan TE, Wolk A, Maehle-Schmidt M, Magnusson C. (2002), Fish consumption and breast cancer risk. Nutr Cancer; **44**(1): 1-6.

Terry PD, Rohan TE, Wolk A. (2003). Intakes of fish and marine fatty acids and the risks of cancers of the breast and prostate and of other hormone-related cancers: a review of the epidemiologic evidence. Am J Clin Nutr; **77**(3): 532-43.

Thomas BJ, Cornish BH, Ward LC. (1992), Bioelectrical Impedance Analysis for measurement of body fluid volumes:A review. J Clin Eng; **17**(6): 505.

Thompson CD. (2003), The declining iodine status of new Zealanders: Reasons, consequences and possible solutions. NZNS conference: Too Little, Too Much, Dunedin, New Zealand.

Thompson FE, Subar A, Brown CC, Smith AF, Sharbaugh CO, Jobe JB, Mittl B, Gibson JT, Ziegler RG. (2002), Cognitive research enhances accuracy of food frequency questionnaire reports: Results of an experimental validation study. J Am Dietet Assoc; **102**(2):212-225.

Tinker LF, Patterson RE, Kristal AR, Bowen DJ, Kuniyuki A, Henry H, Shattuck A (2001), Measurement characteristics of 2 different self-monitoring tools used in a dietary intervention study. J Am Dietet Assoc; **101**(9):1031

Tran ZV and Weltman A. (1989), Generalized equation for predicting body density of women from girth measurements. Med Sci Sports Exerc**; 21**(1): 101-4.

Tobias M and Booth M. (2001), Evidence -based Health Objectives for the New Zealand Health Strategy. Ministry of Health, Wellington, New Zealand

Touyz RM. (2003), Role of magnesium in the pathogenesis of hypertension. Mol Aspects Med; **24**(1-3): 107-36.

Trevisan M, Krogh V, Freudenheim J, Blake A, Muti P, Panico S, Farinaro E, Mancini M, Menotti A, Ricci G. (1990), Consumption of olive oil, butter, and vegetable oils and coronary heart disease risk factors. The Research Group ATS-RF2 of the Italian National Research Council. JAMA; **263**(5): 688-92.

Tudor-Locke C and Bassett DR Jr. (2004), How many steps/day are enough? Preliminary pedometer indices for public health. Sports Med; **34**(1): 1-8.

Tudor-Locke CE and Myers AM. (2001),Methodological considerations for researchers and practitioners using pedometers to measure physical (ambulatory) activity. Res Q Exerc Sport; **72**(1): 1-12.

Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukaanniemi S, Louheranta A, Rastas M, Salminen V, Uusitupa M. (2001), Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med; **344**(18): 1343-50.

Utter AC, Nieman DC, Ward AN, Butterworth DE. (1999), Use of the leg-to-leg bioelectrical impedance method in assessing body-composition change in obese women. Am J Clin Nutr; **69**(4): 603-7.

Uusitalo U, Feskens EJ, Feskens EJ, Tuomilehto J, Dowse G, Haw U, Fareed D, Hemraj F, Gareeboo H, Alberti KG, Zimmet P. (1996), Fall in total cholesterol concentration over five years in association with changes in fatty acid composition of cooking oil in Mauritius: cross sectional survey. BMJ; **313**(7064): 1044-6.

Uusitupa M, Schwab U, Makimattila S, Karhapaa P, Sarkkinen E, Maliranta H, Agren J, Penttila I. (1994), Effects of two high-fat diets with different fatty acid compositions on glucose and lipid metabolism in healthy young women. Am J Clin Nutr; **59**(6): 1310-6.

van Aalst I, Kazakov D, McLean G. (2003), SPARC Facts Series (1997-2001), Sport and Recreation New Zealand (SPARC). www.sparc.org.nz/research/sparcfacts_3.php Accessed 05/03/04.

Vartiainen E, Jousilahti P, Alfthan G, Sundvall J, Pietinen P, Puska P. (2000), Cardiovascular risk factor changes in Finland, 1972-1997. Int J Epidemiol; **29**(1): 49-56.

Verlato G, Zoppini G, Bonora E, Muggeo M. (2003), Mortality from site-specific malignancies in type 2 diabetic patients from Verona. Diabetes Care; **26**(4): 1047.

Visscher TLS and Seidell JC. (2001), The public health impact of obesity. Annu Rev Publ Health; **22**: 355-375.

Vogt TM, Appel LJ, Obarzanek E, Moore TJ, Vollmer WM, Svetkey LP, Sacks FM, Bray GA, Cutler JA, Windhauser MM, Lin PH, Karanja NM. (1999), Dietary Approaches to Stop Hypertension: rationale, design, and methods. DASH Collaborative Research Group. J Am Diet Assoc; **99**(8 Suppl): S12-8.

Volker DH, Leary C, Gill TP. (2003), Portion size of campus takeaway foods and serving size estimates of common foods. Asia Pac J Clin Nutr; **12** (Suppl): S25.

Vollmer WM, Sacks FM, Svetkey LP. (2001), New insights into the effects on blood pressure of diets low in salt and high in fruits and vegetables and low-fat dairy products. Curr Control Trials Cardiovasc Med; **2**(2): 71-74.

Wallace TM and Matthews DR. (2002), The assessment of insulin resistance in man. Diabet Med; **19**(7): 527-534.

Wang L, Yamaguchi T, Yoshimine T, Katagiri A, Shirogane K, Ohashi Y. (2002), A case-control study of risk factors for development of type 2 diabetes: emphasis on physical activity. J Epidemiol; **12**(6): 424-30.

Washburn RA, Jacobsen DJ, Sonko BJ, Hill JO, Donnelly JE. (2003), The validity of the Stanford Seven-Day Physical Activity Recall in young adults. Med Sci Sports Exerc; **35**(8): 1374-80.

Whelton PK, He J, Cutler JA, Brancati FL, Appel LJ, Follmann D, Klag MJ. (1997), Effects of oral potassium on blood pressure. Meta-analysis of randomized controlled clinical trials. JAMA; **277**(20): 1624-32.

WHO/ISH Guidelines Subcommittee. (1999),Guidelines for the management of hypertension. J Hypertension; **15**: 151-183.

WHO. (2002), Diabetes mellitus, WHO Fact Sheet No. 138. http://www.who.int/inf-fs/en/fact138.html Accessed 15/12/03

WHO. (2002),World Health Report 2002 - Reducing Risks, Promoting Healthy Life Methods Summaries

for Risk Factors assessed in Chapter 4, World Health Organisation. www.who.int/entity/whr/2002/en/summary_riskfactors_chp4.pdf Accessed 10/10/03

WHO/FAO Expert Consultation. (2003), Diet, Nutrition and the prevention of Chronic Diseases. Technical report series 916. Geneva, WHO/FAO.

WHO Expert Consultation. (2004), Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet; **363**(9403): 157-63.

Wilcox S, Parra-Medina D, Thompson-Robinson M, Will J. (2001), Nutrition and physical activity interventions to reduce cardiovascular disease risk in health care settings: a quantitative review with a focus on women. Nutr Rev; **59**(7): 197-214.

Wild S, Roglic G, Green A, Sicree R, King H. (2004), Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care; **27**(5): 1047-53.

Willett W and Stampfer MJ. (2003), Rebuilding the food pyramid. Sci Am; **288**(1): 64-71.

Winkleby MA. (1994), The future of community-based cardiovascular disease intervention studies. Am J Publ Health; **84**(9): 1369-1372.

Wolfram G. (2003), Dietary fatty acids and coronary heart disease. Eur J Med Res; **8**(8): 321-4.

World Cancer Research Fund and American Institute for Cancer Research (1997). Food, Nutrition and the prevention of cancer: a global perspective. Washington DC, WCRF, AICR.

Yamashita N and K. S. (2000). Distinct mechanisms of DNA damage in apoptosis induced by quercetin and luteolin. Free Radic Res; **33**(5): 623-33.

Yancy WS Jr., Olsen MK, Guyton JR, Bakst RP, Westman EC. (2004), A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia: a randomized, controlled trial. Ann Intern Med; **140**(10): 769-77.

Young, G. P. and R. K. Le Leu (2002). Preventing cancer: dietary lifestyle or clinical intervention? Asia Pac J Clin Nutr 11 Suppl 3: S618-31. Zimmet P, McCarty D et al. (1997). The global epidemiology of non-insulin- dependent diabetes mellitus and the metabolic syndrome. Journal of Diabetes and its Complications **11**: 60-68.

Zhu S, Wang Z, Heshka S, Heo M, Faith MS, Heymsfield SB. (2002), Waist circumference and obesity associated risk factors among whites in the third National Health and nutrition Examination Survey: clinical action thresholds. Am J Clin Nutr; **76**(4) 743-749.

Zimmet PZ, McCarty DJ, de Courten MP. (1997), The global epidemiology of non-insulin-dependent diabetes mellitus and the metabolic syndrome. J Diabetes Complications;**11**(2): 60-8.

Appendices

Appendix 1a

M E M O R A N D U M

Academic Registry – Academic Services

To: Elaine Rush
From: **Madeline Banda**
Date: 20 February 2002
Subject: 02/08 Reduction of risk for lifestyle diseases

Dear Elaine

Your application for ethics approval was considered by AUTEC at their meeting on 11 February 2002.

Your application was approved for a period of two years until February 2004.

You are required to submit the following to AUTEC:

- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

The Committee wishes you well with your research.

Please include the application number and study title in all correspondence and telephone queries.

Yours sincerely

Madeline Banda
Executive Secretary
AUTEC

From the desk of …
Madeline Banda
Academic Services
Student Services Group

Private Bag 92006, Auckland 1020
New Zealand
E-mail: madeline.banda@aut.ac.nz

Tel: 64 9 917 9999
ext 8044
Fax:64 9 917 9812

Appendix 1b

M E M O R A N D U M

Academic Registry - Academic Services

To: Elaine Rush
From: **Madeline Banda**
Date: 14 March 2003
Subject: 02/08 Reduction of risk for lifestyle diseases

Dear Elaine

Your application for ethics approval was considered by AUTEC at their meeting on 10/03/03.

Your application was approved for a period of two years until March 2005.

You are required to submit the following to AUTEC:

- A brief annual progress report indicating compliance with the ethical approval given.
- A brief statement on the status of the project at the end of the period of approval or on completion of the project, whichever comes sooner.
- A request for renewal of approval if the project has not been completed by the end of the period of approval.

Please note that the Committee grants ethical approval only. If management approval from an institution/organisation is required, it is your responsibility to obtain this.

The Committee wishes you well with your research.

Please include the application number and study title in all correspondence and telephone queries.

Yours sincerely

Madeline Banda
Executive Secretary
AUTEC
cc

From the desk of …
Madeline Banda
Academic Services
Student Services Group

Private Bag 92006, Auckland 1020
New Zealand
E-mail: madeline.banda@aut.ac.nz

Tel: 64 9 917 9999
ext 8044
Fax: 64 9 917 9812

Appendix 2

Consent to Participation in Research

This form is to be completed in conjunction with, and after reference to, the AUTEC Guidelines Version 3 (Revised September 2000).

ONLY type where indicated by instructions eg <Click here and type>

Title of Project: **Reduction of risk for lifestyle diseases.**

Project Supervisors: **Associate Professor Elaine Rush and Professor Lynn Ferguson**

Researcher: **Michelle Cumin**

- I have read and understood the information provided about this research project.
- I have had an opportunity to ask questions and to have them answered.
- I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way. If I withdraw, I understand that all relevant tapes and transcripts, or parts thereof, will be destroyed
- I agree to take part in this research.

Participant signature: .....
Participant name:

Date:

Project Supervisor Contact Details:
Associate Professor Elaine Rush 97 9999 x 8091
elaine.rush@aut.ac.nz

Approved by the Auckland University of Technology Ethics Committee on 20 February 2002 **AUTEC Reference number** 02/08

Appendix 3

INTERVENTION STUDY PROTOCOL SHEET

Subject No:

Date: DOB: Age:

Gender: Ethnic gp: Occupation:

Medication:

Weight: Height: BMI:

Biood pressure: Smoking history

Skinfold thickness:

					Average	
Biceps:				mm		mm
Triceps:				mm		mm
Subscapular:				mm		mm
Suprailiac:				mm		mm

Girth measures:

Chest(Male):		cm
Abdomen:		cm
Iliac:		cm
Hips(buttocks):		cm
Thigh:		cm

BIA:

	Impedance	Resistance	Reactance	Phase
Read 1				
Read 2				
Read 3				
Average:				

Admin dates:

Medlab form given:	1	2	3	4	5
results:					
Variety checklist:	1	2	3	4	5
returned:					
Physical activity :	1	2	3	4	5
returned:					

Appendix 4

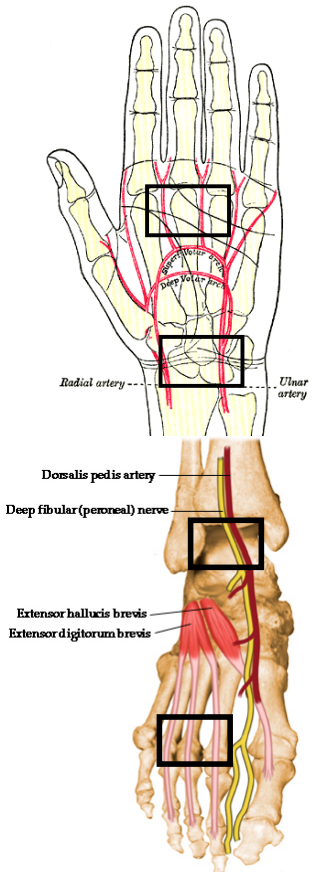
BCMRC Policy Statement

Electrode placement standardisation

The placement used must be the same that was used when the algorithm was developed. For the BCMRC this is:
Electrodes are placed on the back of the **right hand** and front (dorsum) of the **right foot**. The midline of the electrode is placed on the crease made when the wrist or ankle is flexed i.e. bend the foot or ankle to find this – anatomically this is expressed for the hand as at the level of the pisiform prominence and for the foot as between the lateral and medial malleoli. See diagrams below.

Proximal placements – the sensing electrodes
The placement that we use is the **mid-line of the electrode along the crease**. Voltage is measured over the surface area of the electrode so the mid-point is the most logical point of reference. The placement of these electrodes is the most critical as the conductive path length is a major factor in the determination of the algorithm.

Distal placements – the drive electrodes
Placement is with the lower edge of the electrode just above the middle finger or toe. Five centimetres between drive and sense electrodes is advocated but this requirement is less critical than the placement of the sense electrodes. The aim is achieve an even current density by the time the current is passing the sense electrode.



The National Institutes of Health guide lines 1994 state: "Proper detector electrode placement is crucial for accurate and reproducible BIA measurements. The present convention is to place two distal current-introducing electrodes on the dorsal surfaces of the hand and foot **proximal to the metacarpal phalangeal and metatarsal phalangeal joints**, respectively. In addition, two voltage-sensing electrodes are applied at the **pisiform prominence of the wrist** and **between the medial and lateral malleoli of the ankle**. A 1-cm displacement of electrodes can result in a 2-percent change in resistance."

http://consensus.nih.gov/ta/015/015_statement.htm

http://www.biosci.uq.edu.au/BIA/BIA.html
Going down body
Yellow wrist
Red knuckle
Blue - ankle
Black above toes

Appendix 5 (9 pages)

Lifestyle Intervention Study

Food Frequency Questionnaire

- Please put a ✓ in the most appropriate box to indicate how often in the **last week** you have eaten each food.
- Please put only one ✓ per row

example

	None	1-2 times a week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Apples peach,apricot, cherries, nectarines, plum Berries(eg raspberry, strawberry, blackberries Pears, nashi		✓					
Citrus(eg orange, tangelo, grapefruit, mandarin, lemon)			✓				

Please put a √ [n the most appropriate box to indicate how often in the last week you have eaten this food							
	None	1-2 times a week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Apples peach,apricot, cherries, nectarines, plum Berries(eg raspberry, strawberry, blackberries Pears, nashi							
Citrus(eg orange, tangelo, grapefruit, mandarin, lemon)							
Citrus juice							
Bananas							
Melon(eg Honeydew, watermelon, rock melon)							
Pineapple							
Mango							
Fejoa							
grapes							
pawpaw							
breadfruit							
Kiwifruit							
Dried fruit							
Non-citrus fruit juice							
Other fruit (specify)							

Please put a √ [n the most appropriate box to indicate how often in the last week you have eaten this food							
	None	1-2 times week	3-4 times a week	5-6 times a week	once a day	More than once a day	comments
Avocado							
Potato(all cooking methods except fried)							
French fries or hot chips							
Taro							
yam							
Broccoli							
Silverbeet, puha or spinach,beetroot							
Cabbage, cauliflower or brussel sprouts							
Green beans							
Carrots							
Corn							
Green peas							
Pumpkin, squash or butternut, cucumber , courgette,marrow							
Kumara							
Red, green or yellow peppers							
Tomato Tomato juice							
Lettuce or green salad							
onion							
Garlic							
Mixed vegetables (specify)							
Other vegetables (specify)							

Please put a √ [n the most appropriate box to indicate how often in the last week you have eaten this food							
	None	1-2 times week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Standard/whole milk							
Trim milk							
Calci-trim							
Super trim milk							
Soya milk							
Other milk(specify)							
Yogurt							
Low fat yogurt							
Cheese							
Low fat cheese							
Cottage cheese							
Ice cream							
Ice cream substitute/low fat eg lite licks							

Please put a √ [n the most appropriate box to indicate how often in the last week you have eaten this food							
	None	1-2 times week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Butter							
Margarine(specify brand)							
Oil (specify types)							
Cream							
Fat reduced cream							
Lard or other hard cooking fat							
Coconut cream							
Coconut milk							
Mayonnaise or salad dressing							
White sauce, cream soup							

Please put a √ [n the most appropriate box to indicate how often in the last week you have eaten this food							
	None	1-2 times week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Lamb, beef or pork Roast, stew, grill							
Ham, bacon							
Boiled corned beef, brisket or mutton flap							
Tinned corned beef, brisket or mutton flap							
Meat pie, pastie, Sausage rolls or savouries							
Liver							
Other organ meats							
Chicken, with skin(including takeaways)							
Chicken without skin, cooked without added fat							
Sausages, Frankfurters							
Processed meats, luncheon or salami							
Fish, cooked without added fat, baked, steamed or microwaved							
Fish, fried, grilled with added oil or fat(includes battered fish)							
Tinned fish, tuna, salmon or sardines							
Shellfish- mussels, oysters, pipi							
Prawns, lobster, crab, shrimp							
Baked beans, lentils, other dried cooked beans							

	None	1-2 times week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Nuts and Nuts and seeds (almond, brazil nuts, cashew nut, chestnut, hazelnut, peanuts, peanut butter, pecan nut pine nut, pistachio nut, pumpkin seed, sesame seed, tahini, hommus, sunflower seed, walnut)							
coconut							
Pizza 1 slice or macaroni cheese							
Chinese takeaways eg chop suey, chow mein, sweet &sour pork							
Take away burgers beef							
Take away chicken,fried							
Take away fish, fried							
Other take aways Specify							
Restaurant meal							
Eggs-poached, fried, scrambled, boiled etc							

Please put a √ [n the most appropriate box to indicate how often in the last week you have eaten this food							
	None	1-2 times week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Rice							
Pasta, macaroni, noodles							
White bread or bread roll							
Wholemeal or mixed grain bread or bread roll							
Wholewheat or wheatbran cereal							
Toasted muesli							
Porridge or cooked cereals							
Other cereals(specify)							
Scone or muffin							
Pastries, sweet or savoury							
Cake							
Biscuits or muesli bars							
Pudding, crumble, trifle							

Please put a √ [n the most appropriate box to indicate how often in the last week you have eaten this food							
	None	1-2 times week	3-4 times a week	5-6 times a week	once day	More than once a day	comments
Chocolate or chocolate bar							
Miso, tempeh, soya sauce							
Tofu, soyabean curd							
Sauerkraut							
Vegemite, marmite							
Tomato sauce/ketchup							
Softdrinks (diet)							
Soft drinks (sweet)							
Water							
Non alcoholic Beverages(tea. Coffee, cocoa, Milo)							
Alcoholic beverages							
Salt added to your food during cooking							
Salt added to your food after cooking							
Vitamin/mineral supplement (Specify)							

Appendix 6 **Physical activity questionnaire(PAQ)** Week ID #

How often in the **last week** have you participated in the following activities?	**Never**	**1-2 times**	**3-4 times**	**5-6 times**	**7 times**	**8+ times**
a.Climbing some stairs for exercise instead of taking the elevator						
b.Walking instead of driving a short distance						
c.Parking away from your destination so you have to walk more						
d.Walking on your lunch hour or after dinner						
e.Getting off at a bus stop which is not the one nearest your destination and walking						
f.Other mild/moderate physical activity (specify)						

Have you done any of the following in the **last three weeks**?	**No**	**Yes**	**How often?**	**For how long each time?**
a. Jogged or run				
b. Played strenuous racquet sports (singles tennis, paddle ball etc)				
c. Played other strenuous sports (rugby, netballbasketball, soccer, rowing etc)				
d. Rode a bicycle				
e. Swum				
f. Went to the gym				
g. Climbed a hill				
h. Other strenuous physical activity? (specify)				

Has your level of activity changed over the last three weeks? If so please explain.

Have you used the pedometer in the last three weeks? **Yes** **No**		**Number of steps?**					
If yes - what was the average number of steps each day?		**<2000**	**2000-4000**	**4000-6000**	**6000-8000**	**8000-10000**	**>10000**
	last 7 days?						
	the week before that						
	the first week?						

Appendix 7a Goal sheet questionnaire 1(GSQ 1)
(3 pages)

Food changes	Haven't done at all	Have done for a long time	Started doing during the study	Am still doing	Length of time done for after study started
Choose low fat or no fat versions eg green top milk instead of standard					
Cut visible fat off meat					
Take the skin off your chicken					
Use cooking methods that don't use much fat (boiling, microwaving, baking, stir frying…)					
Eat fish more often(aim for twice a week or more)					
Increase your legume consumption (try some of our yummy recipes!!)					
Eat less takeaways					
Decrease your use of margarine and butter					
Wait for toast to cool before you add the marg./butter					
Use oil free salad dressings					
Don't add salt to your food –use more herbs and spices for flavour					
If you have high blood pressure watch out for hidden salt(sodium) in preprepared foods, Asian meals, spreads(like peanut butter and vegemite)					
Eat more cereals(rice, pasta, grains) and legumes(dried peas and beans, baked beans, lentils)					

Appendix 7a Goal sheet questionnaire 1(GSQ 1)
(3 pages)

Physical activity changes	Haven't done at all	Have done for a long time	Started doing during the study	Am still doing	Length of time done for after study started
Use the stairs instead of the lift					
Park a little way away and walk					
Walk rather than drive where possible					
Walk around the field/court when watching sport					
Take breaks away from your desk. Go for short walks					
Go for a walk at lunchtime					
Play actively with the kids					
Have a stretch. Rotate your feet. Stand up and sit down again					
Use less labor saving devices					
Use a pedometer (number of steps)					
Any other changes to your diet, physical activity or stress levels Positive and negative					

Appendix 7a Goal sheet questionnaire 1(GSQ 1)
(3 pages)

Summary

I believe that	**No**	**Yes**	**Still** √	**How long did you make this change for?**
I ate **less total fat**	**No**	**Yes**		
I ate **less saturated (solid) fat**	**No**	**Yes**		
I ate **more complex carbohydrate** ie less sugar	**No**	**Yes**		
I ate **more whole grains**(less white bread, rice etc)	**No**	**Yes**		
I ate **more beans, lentils and peas**	**No**	**Yes**		
I ate **more oily fish**	**No**	**Yes**		
I ate **more servings of fruit and vegetables** per day	**No**	**Yes**		
I eat **more variety/different foods** than before the study	**No**	**Yes**		
I **move more physically**	**No**	**Yes**		

If you made changes and kept them up what was it that motivated you?

If you didn't keep up changes what was it that prevented you?

Appendix 7b **Goal sheet questionnaire 2**

(3 pages)	Have been doing for a long time	Started doing after starting the study	Started doing after the study talk	Still doing	Will continue doing
Choose low fat or no fat versions eg green top milk instead of standard					
Cut visible fat off meat					
Take the skin off your chicken					
Use cooking methods that don't use much fat (boiling, microwaving, baking, stir frying…)					
Eat fish more often(aim for twice a week or more)					
Increase your legume consumption (try some of our yummy recipes!!)					
Eat less takeaways					
Decrease your use of margarine and butter					
Wait for toast to cool before you add the marg./butter					
Use oil free salad dressings					

Appendix 7b **Goal sheet questionnaire 2**

Don't add salt to your food –use more herbs and spices for flavour					
If you have high blood pressure watch out for hidden salt(sodium) in pre-prepared foods, Asian meals, spreads(like peanut butter and vegemite)					
Eat more cereals(rice, pasta, grains) and legumes(dried peas and beans, baked beans, lentils)					

Use the stairs instead of the lift	Have been doing for a long time	Started doing after starting the study	Started doing after the study talk	Still doing	Will continue doing
Park a little way away and walk					
Walk rather than drive where possible					
Walk around the field/court when watching sport					
Take breaks away from your desk. Go for short walks					
Go for a walk at lunchtime					

Appendix 7b **Goal sheet questionnaire 2**

Play actively with the kids					
Have a stretch. Rotate your feet. Stand up and sit down again					
Use less labor saving devices					
Use the pedometer- see if you can increase your steps to over 10 000					
Any other changes to your diet or physical activity??					

I would like to continue receiving emails containing information that may help me reduce the risk of lifestyle disease. ☐

would be prepared to come back in about a year (July-October 2003) to be measured, to have blood tests and to answer questionnaires to see how I have maintained or made other changes that may reduce my risk of lifestyle disease. ☐

Full name: .....

Signature: .....

Date: .....

Appendix 8

When (day and time) did you last eat a kiwifruit?	
How did you find eating the kiwifruit? Was there any noticeable effect?	

When (day and time) did you last eat a kiwifruit	
How did you find eating the kiwifruit? Was there any noticeable effect?	

When (day and time) did you last eat a kiwifruit	
How did you find eating the kiwifruit? Was there any noticeable effect?	

Appendix 9
(10 pages)

AUT
AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

healthy living

changing your future – NOW!

Things that you can change!

Eat oily fish; tuna, sardines, mackerel, herring or salmon at least once a week

Canola and olive oil are good sources of monounsaturated fatty acids that as part of a balanced diet are associated with decreased risk of diabetes and cardiovascular disease

To reduce blood pressure and increase calcium in bones **reduce salt intake**

Increased dietary fibre is associated with less body weight, lower blood pressure and reduced fats in the blood

Increased dietary fibre can help reduce appetite and improve diabetes risk factors independent of weight loss

Increased dietary fibre reduces constipation, haemorrhoids, diverticular disease and the risk of cancer of **the colon**

Increased physical activity and a sensible diet decreases the risk of obesity and diabetes

Any physical activity reduces risk; even minutes of walking each day – if you don't move it you'll lose it!

If you are overweight loss of weight improves blood pressure, blood lipids and signs of diabetes

Increased bean intake is associated with less heart disease

Breakfast improves brain function

Eat more often – people who eat more often have lower blood cholesterol and a more healthy lifestyle.

How many servings should you be having per day?

Group	Bread Cereal	Veges	Fruits	Dairy	Meat, fish, chicken, legumes	Fats and extras
A	6	3 or	2-4	2	2	2
B	8	3 or	3-4	2	2	2
C	10	3 or	3-4	2	2	2
D	10	3 or	4-5	2.5	2.5	3
E	12	3 or	4-5	2.5	3	3

Serving size examples

Breads and cereals
(preferably whole meal/grainy)

1 medium slice bread (26g)
1 roll (50g)
1 muffin (80g)
½ cup cooked cereal/porridge
½ cup muesli (preferably untoasted) (55g)
1 cup cooked pasta
1 cup cooked rice
2 plain sweet biscuits
1 small pita
3 crackers
1 cup popcorn
1 medium potato
1 medium sweet potato/kumara

Fats and extras
2 teaspoons margarine, oil or butter
1 tablespoon mayonnaise
3 tablespoons coconut cream
½ cup coconut milk

Vegetables
½ cup cooked vegetables
½ cup salad (mayonnaise or ordinary dressing belongs to fat group!)
1 tomato
1 medium carrot

Meat, chicken, seafood, pulses
2 slices cooked meat (90g)
½ cup mince or casserole meat
1 medium steak (90g)
1 medium fish fillet
½ can fish (preferably in water/brine)
1 chicken leg
¾ cup cooked dried beans or lentils
½ cup hummus

Dairy Products
250 ml milk (Super trim, Trim Calci trim or Sun Latte)
150g yoghurt
2 slices cheese (40g)
2 scoops ice cream (140g)

Fruit
1 medium apple, pear, banana, orange kiwifruit
2 small apricots or plums
½ cup fruit salad½ cup stewed fruit
½ cup fruit juice
1 medium slice melon
½ can fruit

Vegetarians need to plan more carefully
For iron whole grain cereals, dried peas, beans and lentils, dried fruits and dark green leafy vegetables. Eat foods rich in Vitamin C with these meals to help absorb iron.
If cheese, milk or eggs are not eaten a mixed daily diet of cereals and beans, tofu, peas and lentils will also give protein.

Planning

It is easier to follow the pyramid if you plan your day.

This can be used as a guide if you want to plan

I belong to group ____

I should have ____ servings of breads/cereals

____ servings of vegetables

____ servings of fruit

____ servings of dairy

____ servings of meat/fish/chicken/legumes

____ fats/extras (salad dressing, oil, margarine, chocolate)

	Bread/ cereal	Veg	Fruit	Dairy	Meat/fish legume	Fats/ Extra
Breakfast						
Mid morning						
Lunch						
Mid afternoon						
Dinner						
Evening						
Total						

Reduce your salt intake:

- Watch out for ‘hidden’ salt in pre-prepared foods, spreads (like peanut butter and vegemite) and sauces (soya sauce, tomato sauce). Keep portions of these small or use low/no added salt varieties.

For your **bread and cereal** choices:

- Choose wholegrain breads /cereals/crackers more often
- Eat more cereals and legumes (rice, pasta, dried peas and beans)
- Increase unprocessed cereals (bran cereals, brown rice, whole meal pasta)

Fruit and vegetables

- Eat as many different colours in a day as possible eg orange (carrots); purple (beetroot); red (tomato); green (spinach); yellow (capsicum) etc

Change your oil

Not all fats are the same – the good oil is unsaturated (canola and olive have good reports) and some contains omega 3 which is very good. You do need more than 30% of your energy from fat – for an average person this is 70 g a day – less than a third of a cup total!

Reduce saturated fat:

- **Choose** low fat or no fat versions eg light blue or green milk
- **Choose** lower fat cheeses
- **Cut** visible fat off meat. Have small portions of lean meats.
- **Remove** the skin from chicken
- **Eat fish at least twice a week**. Oily fish is best eg tuna, salmon, sardines, pilchards, sardines (if tinned fish is used, use fish in water or brine)
- Cooking methods – **use as little fat/oil** as possible: avoid regularly eating charred, smoked or preserved meats.
- **Eat less take-aways**
- **Use small amounts** of margarine or oils. Olive oil, Canola oil, soy oil, sunflower oil and other seed or nut oils are better than coconut oil, palm oil, butter or hard fats.
- **Wait** for toast to cool before adding margarine or butter
- Try using a thin scraping of low fat cream cheese instead of marg or butter on bread, toast or crackers
- **Watch out** for hidden fats eg in salad dressings
- Use non stick bake ware and frying pans.

General:

- Eat everything in moderation – **there are no good foods or bad foods**. It is more important to learn to control your portion sizes than to eliminate a food and then binge when you succumb.
- **Don't go shopping** when you are hungry
- Divide your food into three meals and three snacks a day.
- Limit alcohol to one or two drinks a day and do not binge drink.
- Be careful of reduced fat foods – they may be lower in fat than their full fat counterparts but are not necessarily low in fat!
- If you eat out up to once a week, choose one item from the menu and **enjoy it!**
- If you eat out more frequently, choose one item from the menu but be careful with your choice ie low in fat, not too saucy, preferably more vegetables, legumes, lentils and less meat, chicken, cheese.
- If you tend to be overweight, watch out for hidden fats ie in cooking, salad dressings
- **VARIETY** - less boring, more nutritious
- **BALANCE** - less boring, more nutritious **– you can have treats!**
- **MODERATION** – less boring, more nutritious

More Tips

- Don’t weigh yourself more than once a week
- Don’t skip meals
- When eating out: -Choose smaller portions
    - If main portions are large either order an appetiser or share with a friend
    - Resign from the ‘clean your plate ‘ club – eat a sensible portion and ask for a ‘doggie bag’ or throw it out!
    - Ask for salad dressing to be served on the side so you can choose how much to add
    - Order from a menu rather than from an ‘all you can eat’ buffet
- Measure your ‘usual’ size portions at home once or twice to get an idea of how they fit into the pyramid serving sizes
- Try using a smaller plate

Cooking tips

- Roast or grill – with a rack so the meat or poultry doesn't sit in it's own fat drippings. Baste with fat-free liquids like wine, tomato juice or lemon juice
- Refrigerate cooked dishes and remove the chilled fat before reheating
- Stir fry in a non stick wok with a tiny amount of oil
- Microwave without added fat. Place food between two paper towels when cooking to absorb extra fat
- Make scrambled eggs or omelets using one whole egg with two or three added egg whites
- Remove oils from canned tuna, salmon or sardines by draining and then rinsing in water
- Mix small quantities of creamy salad dressing with low fat yoghurt
- Use chopped vegetables or pulses, chickpeas or TVP to stretch mince dishes
- Make good use of herbs and spices for flavour

Ways to increase your exercise:

Whether it is a structured exercise program or just part of your daily routine, **all exercise** adds up to a healthier heart.

- Choose activities that are **fun**, not exhausting.
- Try to have a **variety** of several activities that you can enjoy. That way, exercise will never seem boring or routine.
- **Don't overdo it.** Do low- to moderate-level activities, especially at first. You can slowly increase the duration and intensity of your activities as you become more fit
- Use the **stairs** instead of the lift
- Think of movement as an **opportunity** (for improving health) rather than as an inconvenience
- Be **active** in as many ways as you can. (Gardening, washing the car)
- **Walk** to the shops rather than drive
- Park a little way away from your destination and walk briskly
- **Play** actively with the kids
- Walk around the field/court when you are watching the kids play sport
- Go for a swim or a brisk walk **Every little bit helps**

- Keep a **comfortable pair of shoes** at work
- Do things you enjoy that require movement eg dancing
- Minimize using the remote control –**get up** and change it
- **Surround yourself** with supportive people.

  Decide what kind of support you need. Do you want them to remind you to exercise?

  Ask about your progress?

  Participate with you regularly or occasionally?

  Allow you time to exercise by yourself?

  Go with you to a special event, such as a 10km walk/run?

  Share your activity time with others.

Intervention study goal sheet for_____Date:_____

Put a ✓ next to the changes you will try to make over the next three weeks.

Food (aim for at least **six**)

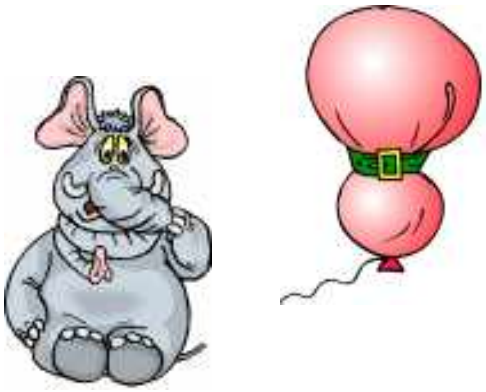
Eat more often – plan your snacks	
Increase the number and variety of fruits and vegetables eaten	
Choose low fat or no fat versions eg green top milk instead of standard	
Cut visible fat off meat	
Take the skin off your chicken	
Use cooking methods that don't use much fat (boiling, microwaving, baking, stir frying)	
Eat fish more often (aim for twice a week or more)	
Increase your legume consumption (try some of our yummy recipes!!)	
Eat less takeaways	
Decrease your use of margarine and butter – use canola and olive oil	
Wait for toast to cool before you add the marg./butter	
Use oil free salad dressings	
Don't add salt to your food –use more herbs and spices for flavour	
If you have high blood pressure watch out for hidden salt (sodium) in pre-prepared foods, Asian meals, spreads (like peanut butter and vegemite)	
Eat more cereals(rice, pasta, grains) and legumes(dried peas and beans, baked beans, lentils)	

Exercise (aim for at least **three** **plus** 20 minutes of planned exercise daily)
Think of movement as an opportunity (for improving health) rather than as an inconvenience

Keep a comfortable pair of shoes at work so that you are always prepared.

Use the stairs instead of the lift	
Park a little way away and walk	
Walk rather than drive where possible	
Walk around the field/court when watching sport	
Take breaks away from your desk. Go for short walks	
Go for a walk at lunchtime	
Play actively with the kids	
Have a stretch. Rotate your feet. Stand up and sit down again	
Use less labor saving devices	
Use the pedometer- see if you can increase your steps – long term goal 10000 /day	

Move whenever you can - every little bit helps



Putting the squeeze on lifestyle disease

Make changes that you can see yourself still doing in a years time!

Appendix 10
(14 pages)

AUT
AUCKLAND UNIVERSITY OF TECHNOLOGY
TE WĀNANGA ARONUI O TAMAKI MAKAU RAU

healthy recipes

lower fat, increase fibre, add more fruits, vegetables and whole grains, lower salt and lower sugar and they taste good and are fast to prepare

changing your future – NOW!

Things that you can change!

Eat oily fish; tuna, sardines, mackerel, herring or salmon at least once a week

Canola and olive oil are good sources of monounsaturated fatty acids that as part of a balanced diet are associated with decreased risk of diabetes and cardiovascular disease

To reduce blood pressure and increase calcium in bones **reduce salt intake**

Increased dietary fibre is associated with less body weight, lower blood pressure and reduced fats in the blood

Increased dietary fibre can help reduce appetite and improve diabetes risk factors independent of weight loss

Increased dietary fibre reduces constipation, haemorrhoids, diverticular disease and the risk of cancer of **the colon**

Increased physical activity and a sensible diet decreases the risk of obesity and diabetes

Any physical activity reduces risk; even minutes of walking each day – if you don't move it you'll lose it!

If you are overweight loss of weight improves blood pressure, blood lipids and signs of diabetes

Increased bean intake is associated with less heart disease

Breakfast improves brain function

Eat more often – people who eat more often have lower blood cholesterol and a more healthy lifestyle.

What else you can change!

Ingredient	**Use instead**
Lower fats/cholesterol	
full cream milk	• trim /super trim milk
full cream evaporated milk	• low fat evaporated milk
yoghurt	• low fat yoghurt
cream	• low fat evaporated milk/lite cream
sour cream	• low fat yoghurt/lite sour cream
coconut cream	• low fat evaporated milk + coconut essence
cream cheese	• ricotta/cottage cheese or lite cream cheese
cheese	• low fat cheese
butter	• mono/polyunsaturated margarine
lard	• mono/polyunsaturated margarine or oil
cooked chicken	• remove skin, breast best
fatty meats	• lean meats, remove visible fat

fried food	• grill or microwave
luncheon meat	• low fat ham/turkey products
tuna/salmon in oil	• tuna/salmon in brine or spring water
puff pastry	• filo pastry sprayed with oil
salad dressing	• no oil or homemade no oil

Increase fibre

white flour	• wholegrain or wholemeal flour
refined cereals(eg cornflakes)	• wholegrain or bran types
white rice	• Basmati, brown or long grain rice
plain pasta	• wholemeal pasta
peeled or boiled vegetables	• unpeeled or raw vegetables
white bread crumbs	• wholemeal bread crumbs

Ingredient	**Use instead**
Lower sugar	
sugar	• reduce quantity • use honey (less as it is sweeter) • use equivalent substitute • add fruit juice instead of sugar • use fresh or dried fruit instead of sugar

Lower salt/sodium

salt in cooking	• lemon juice, curry powder, spices
canned sauces	• home made sauces
canned vegetables	• no added salt, fresh, frozen
canned or smoked fish	• fresh fish or canned in spring water
commercial salad dressings	• home made salad dressings
corned meats	• fresh meats
bacon and ham	• mushrooms and herbs
tomato sauce	• lite tomato sauce
canned vegetables or juices	• fresh or frozen vegetables or juices low salt canned food

Spaghetti with vegetable sauce

Ingredients

500g uncooked spaghetti

1 cup broccoli florets

1 (425g) can whole kernel corn, drained

1 cup fresh sliced mushrooms

1 cup sliced carrots

1 cup tomato sauce

Method

Bring a large pot of salted water to boil, add spaghetti and return water to a boil. Cook until spaghetti is al dente; drain well.

Combine broccoli, corn, mushrooms, carrots and tomato sauce in large sauce pot. Cook on medium heat for 15 to 20 minutes or until vegetables are tender. Stir occasionally to keep sauce from sticking.

Serve sauce over spaghetti.

Lentil and Rice Pilaf

Ingredients

2 cups vegetable stock

1 cup water

½ cup long grain white rice

½ cup lentils

1 medium onion -- sliced

2 medium tomatoes -- diced

1 teaspoon dried thyme

Method

Combine the stock and water. Bring 2 cups liquid to a boil.

Rinse lentils and slowly add to boiling stock so that liquid continues to boil. Cover and cook 5 minutes.

Rinse rice and add to lentils with onion, tomatoes, thyme and remaining 1 cup liquid. Cover and simmer 20 minutes.

Mexican Baked Bean Loaf

Ingredients

2 cups baked beans

1 green pepper -- chopped

3 onion -- chopped

2 tablespoons cooking oil

4 drops Tabasco sauce

1 cup tomato sauce

1 cup bread crumbs

Method

Saute the pepper and onions in oil until lightly browned. Stir in Tabasco.

Add beans and tomato sauce. Cook 5 minutes. Turn into a greased baking dish. Top with crumbs. Spray lightly with Canola cooking spray.

Bake at 350F until crumbs are brown (about 15 minutes.)

Tabouli

Ingredients

1 cup Bulgar wheat

2 cups boiling water

2 tomatoes -- finely diced

1 bunch spring onions -- sliced

3 tablespoons fresh mint -- chopped

2 cups fresh parsley -- chopped

½ cup fresh lemon juice

2 teasp. olive oil

Black pepper to taste

Method

Place uncooked bulgar in a bowl; pour boiling water over it and let it soak one hour (stir occasionally).

Drain well in a fine strainer. Return bulgar to the bowl and add all other ingredients; mix well.

Chill for 2 hours.

Bean Burritos

Ingredients

can (or equiv fresh) pinto beans

1 can (or equiv fresh) black beans

1 can garbanzo beans (chickpeas)

2+ cloves of garlic

Approx. a cup of roasted veggies (eg. red and green peppers, zucchini, and yellow squash)

Cayenne pepper to taste

Method

Combine all beans in a pot (if using fresh beans add about 2 cups water and cook for an hour or until soft.)

Cut up roasted veggies, and add to beans.

Use mixer and mix everything together until it looks like refried beans.

Add spices and simmer for 20 minutes or so.

Serve with fillings in tortillas

Suggested fillings: lettuce, tomato, low fat grated cheese, low fat yoghurt, salsa.

Mexican Lentils and Rice

Ingredients

1 cup chopped red bell pepper

1 cup chopped onion

2 cups of veggie stock

½ tsp. cumin

¾ cup raisins (optional).

1 cup chopped carrots

½ cup lentils

½ tsp. chilli powder

1 cup peas

Method

Saute the red pepper , carrots and onion in a medium pot (sprayed with Canola cooking spray) for 2 minutes.

Add lentils and rice and saute for 2 minutes more.

Add veggie stock, chilli powder and cumin

Bring to a boil, and cover, simmering for 12 minutes

Add peas and raisins (optional). Cover and continue to simmer until water is absorbed and rice is tender.

Lazy Enchiladas

Ingredients

1 med onion chopped
½ med red/green pepper chopped
2 cloves garlic minced
9-10 med mushrooms sliced thinly
1 can (410g) tomatoes, with juice
½ cup frozen corn kernels
1 can (410g) black beans rinsed
½ tsp. cinnamon
½-1 tsp. oregano
1 Tbsp. chilli powder
1-2 tsp. cumin
pinch or more of cayenne
4 flour tortillas

Method

Water saute onion, pepper garlic, until onion translucent.
Add spices and let them coat the onion mixture.
Add mushrooms and let cook briefly for 1-2 minutes
Add can of tomatoes and bring to simmer.
Reduce heat and simmer for 10 minutes.
Add corn and simmer for 10 more minutes
Add beans and simmer for 5 minutes.
Warm tortillas so they are pliable, and with a slotted spoon scoop mixture in tortilla, roll, and place on dinner plate.
With tablespoon, take liquid and pour over tortillas

Garden Couscous and Black Bean Salad

Ingredients

1 cup uncooked couscous
2 cups cooked black beans
1 stalk celery
1 small red pepper
2 medium tomatoes, diced
½ cup chopped green olives
½ cup chopped fresh parsley
2 green onions, finely sliced
½ tbsp lemon juice

Method

Pour couscous in medium heat-proof bowl.

Bring 2 cups water to a boil and pour over couscous.

Cover and let stand 15 minutes, then fluff with a fork and allow to cool to room temperature.

Transfer couscous to large bowl. Add remaining ingredients and toss to mix.

Serve at room temperature, or chilled.

Five Bean Salad

Mix in a large bowl:

2 x 425g cans mixed beans, rinsed and drained
½ carrot, grated
½ celery stalk, finely chopped
2 green onions, finely chopped
2 cloves garlic, finely chopped
2 Tbsp. red wine vinegar
Juice from ½ a lemon (about 2 Tbsp.)
1 tsp. Dijon mustard
Chill

Spinach-mushroom-quiche

Ingredients

2x large Pita breads(wholemeal are preferable)

600 g frozen spinach

400 g fresh mushrooms

chilli powder paprika powder

pepper garlic powder

curry powder

1 Tbsp.lemon juice (fresh or concentrate)

1 Tbsp.soy sauce

Method

Preheat oven to 450F and pre-bake the pita for about 4-5 minutes on a baking sheet.

Thaw spinach and drain thoroughly.

Wash mushrooms and slice.

Cook spinach and mushrooms with a bit water, soy sauce and lemon juice until mushrooms are soft. Drain it a bit and puree.

Add, chilli powder, paprika powder, pepper, curry powder, garlic powder to taste and put this mixture over the pitas.

Bake for about 20-30 minutes

Mediterranean-Style Vegetables

Ingredients

2 cups zucchini sliced

2 cups red, yellow, or green sweet peppers sliced

2 cups broccoli flowerets ¼ cup vegetable broth

2 garlic cloves minced ¼ tsp pepper

1 Tbsp. fresh basil or oregano or 1 tsp.dried basil or oregano

3 tomatoes, medium chopped

In a Dutch oven or large pot combine zucchini, sweet peppers, broccoli, broth, garlic, basil or oregano, and pepper.

Bring to the boil then reduce heat and simmer, covered, for 6 to 8 minutes or till crisp-tender, stirring occasionally. Stir in tomatoes.

Chickpea Vindaloo

Ingredients

Canola cooking spray

1 medium onion, thinly sliced

2 teaspoons minced fresh ginger 2 cloves garlic

1 teaspoon ground cumin

1 teaspoon ground turmeric

1 cinnamon stick

2 green cardamom pods

1 bay leaf

¼ to ½ teaspoon cayenne pepper (or to taste)

2 cups thinly sliced mushrooms

2 ripe tomatoes chopped (~ 1 1/2 cups)

3 to 4 tablespoons wine vinegar or cider vinegar

2 cups cooked chickpeas

2 potatoes, peeled and diced [I don't usually peel them]

2 to 3 cups vegetable broth

1 tablespoon tomato paste

1/2 cup low fat yoghurt

freshly ground pepper

Method

Heat cooking spray in a large pan.

Cook the onion over medium heat for 2 minutes.

Stir in the ginger, garlic, cumin, turmeric, cinnamon stick,cardamom pods, bay leaf, and cayenne and cook for 2 to 3 minutes, or until the onion is very soft.

Increase the heat to high and stir in the mushrooms and tomatoes.

Cook for 2 to 3 minutes, or until most of the mushroom liquid has evaporated. Stir in the vinegar and bring to a boil.

Stir in the chickpeas, potatoes, 2 cups vegetable stock, tomato paste, yoghurt, pepper to taste.

Simmer for 10 minutes, or until the potatoes are tender.

Add stock as necessary to keep the stew moist.

Curried Brown Rice Salad

Ingredients

300ml water

240g long grain brown rice

1 onion chopped finely

1 tsp ground cumin

3 tsp curry powder

100ml orange juice

90g dried apricots chopped

60g raisins or sultanas

Method

Bring water and salt to the boil Add rice and cover pan, simmer for about 40 mins, until water is absorbed and rice is tender

Cook onion for about 3/4 min in your chosen method of "frying" (Canola cooking spray or broth)

Add cumin and curry powder, cook for further 2 mins

Pour in orange juice and simmer for 1 minute

Remove from heat

Place rice in a large bowl and stir in the warm sauce, mix well

Add apricots and raisins or sultanas .

Cover and allow to stand for 2 hours to allow the flavours to develop

Salmon Carbonaro

Ingredients

1 can salmon, drained

2 (410g) cans diced tomatoes

3 tablespoons red wine vinegar

2 teaspoons dried basil

½ teaspoon coarse ground black pepper

240g pasta, uncooked

1 cup chopped onion

3 large cloves garlic, minced

½ teaspoon dried oregano

¼ teaspoon crushed red pepper

⅓ cup lite cream or low fat yoghurt

2 tablespoons finely grated Parmesan cheese

Method

Break salmon into bite-sized pieces; set aside.

Cook pasta according to package directions; drain and keep warm.

Spray a large pan with cooking spray. Add tomatoes, onion, vinegar, garlic, basil, oregano and peppers. Cook 5 minutes or until slightly thickened.

Stir in salmon pieces and cream. Heat through.

Serve over hot cooked pasta.

Salmon with mustard

Ingredients

4 fillets salmon (about 120g each)

3 tablespoons prepared Dijon-style mustard

¼ cup dry bread crumbs

cooking spray

Method

Preheat oven to 400 degrees F (200 degrees C). Line a shallow baking pan with aluminium foil.

Place salmon skin-side down on foil.

Spread a thin layer of mustard on the top of each fillet

Top with bread crumbs, then spray with oil

Bake in a preheated oven for 15 minutes or until salmon flakes easily with a fork.

Variation: Add 1 Tablespoon honey to the mustard and ½ cup chopped walnuts to the breadcrumbs

Italian Salmon

Ingredients

6 fillets salmon (about 120g each)

1 packet dry Italian-style salad dressing mix(about 20g)

½ cup water

2 tablespoons lemon juice

1 cup fresh sliced mushrooms

Method

Preheat oven to 180 degrees C. Spray one 22 x 33cm baking dish with oil.

In a cup, combine salad dressing mix, water and lemon juice.

Arrange salmon fillets in a single layer in the prepared baking dish.

Pour the water mixture over the top and place the sliced mushrooms over the salmon.

Bake, covered, for 15 minutes. Remove cover and bake for an additional 15 minutes, basting with cooking liquids.

Tuna Patties

Ingredients

2 x 180g cans tuna, drained and flaked

¾ cup dry bread crumbs

1 clove garlic, peeled and minced

1 tablespoon teriyaki sauce

1 teaspoon black pepper

2 tablespoons oil for frying

1 egg, beaten

3 green onions, minced

1 tablespoon soy sauce

1 tablespoon ketchup

½ cup cornmeal

Method

In a large bowl, mix tuna, egg, bread crumbs, green onions, and garlic.

Blend soy sauce, teriyaki sauce, ketchup, and pepper into the mixture.

Form the mixture into about 6 patties approximately 1 inch thick.

Lightly sprinkle each patty on all sides with cornmeal.

Heat oil in a medium skillet over medium heat. Fry each patty about 5 minutes on each side, until golden brown. Serve hot or cold.

Vege Pasta

Ingredients

240g pasta (shells, wheels or spirals)

1 head broccoli, cut into florets

3 cloves garlic, finely chopped

1 (4.5 ounce) can sliced mushrooms, drained

¼ cup grated Parmesan cheese

Method

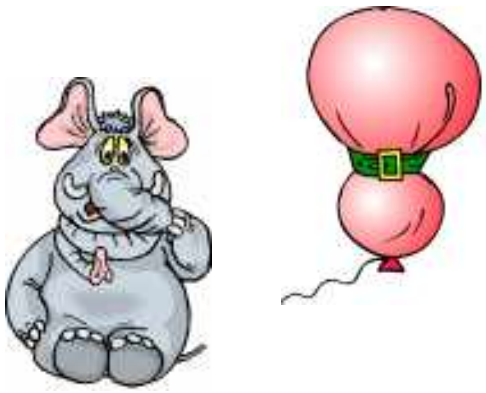
Preheat oven to 175 degrees C.

Bring a large pot of lightly salted water to a boil. Add pasta and cook for 8 to 10 minutes or until al dente; drain.

Place broccoli in a microwave safe dish with about 3 tablespoons of water. Microwave for 3 minutes, or until tender.

Spray the bottom of a medium pan with oil. Saute garlic and mushrooms until garlic becomes aromatic. Combine with pasta, broccoli and Parmesan cheese; transfer to a 1 litre baking dish.

Cover and bake in preheated oven for about 20 minutes, or until heated throughout.



Putting the squeeze on lifestyle disease

Make changes that you can see yourself still doing in a years time!

Appendix 11 Diet calculations									
	bread	veg	fruit	dairy	meat	fats	Total	xenergy	%energy
A	**5**	**2**	**2**	**2**	**3**	**2**	**16**		
cho	75	14	20	24	0	0	133	532	51.40097
Prot	10	0	0	16	21	0	47	188	18.16425
Fat	0	0	0	10	15	10	35	315	30.43478
Energy	350	60	80	160	225	90	965	1035	
B	**5**	**2**	**2**	**2**	**4**	**2**	**17**		
cho	75	14	20	24	0	0	133	532	48.01444
Prot	10	0	0	16	28	0	54	216	19.49458
Fat	0	0	0	10	20	10	40	360	32.49097
Energy	350	60	80	160	300	90	1040	1108	
C	**8**	**2**	**3**	**2**	**5**	**2**	**22**		
cho	120	14	30	24	0	0	188	752	52.77193
Prot	16	0	0	16	35	0	67	268	18.80702
Fat	0	0	0	10	25	10	45	405	28.42105
Energy	560	60	120	160	375	90	1365	1425	
D	**10**	**3**	**4**	**2.5**	**5**	**3**	**27.5**		
cho	150	21	40	30	0	0	241	964	
Prot	20	0	0	20	35	0	75	300	
Fat	0	0	0	12.5	25	15	52.5	472.5	
Energy	700	90	160	200	375	135	1660	1736.5	
E	**12**	**3**	**4**	**2.5**	**3**	**3**	**27.5**		
cho	180	21	40	30	0	0	271	1084	
Prot	20	0	0	20	21	0	61	244	
Fat	0	0	0	12.5	15	15	42.5	382.5	
Energy	840	90	160	200	225	135	1650	1710.5	
	Cho= carbohydrate								
	Prot= protein								

Appendix 12 Powerpoint presentation - group shakeup

Reducing the risk of lifestyle diseases

Obesity
cardiovascular disease
Hypertension
Type 2 diabetes mellitus
Cancer

The healthy food pyramid

Portions vs Pyramid servings

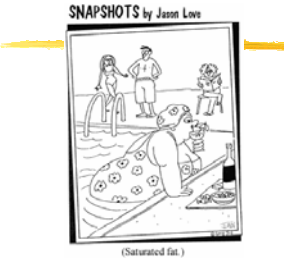
Food	Serving size	Portion	X larger than Rec serving
Popcorn	2 cups	10 cups (movie med)	5
Rice	½ cup	1 cup	2
French fries	½ cup (30g)	120g (medium)	4
Muffin	30g	120g	4

Planning

	Bread/ Cereal	Veg	Fruit	Dairy	Meat/fish legumes	Fats/ extras
Breakfast	1			0.5		
Mid morning			1			
Lunch	2	1			1	1
Mid afternoon	1					1
Dinner	2	2			1	
Evening			1	1		
Other				0.5		
Total	6	3	2	2	2	2

What should I do?

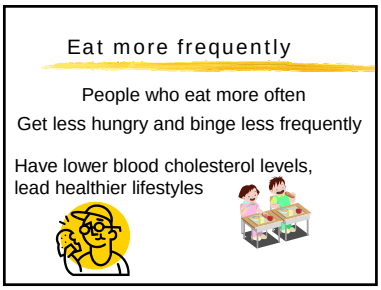
- Reduce saturated fat
  - Eat oily fish at least once a week
  - Use Canola or Olive oil
  - Choose low fat options BUT remember portion sizes
- Reduce salt intake
  - Watch out for hidden salt
  - Add salt only after tasting

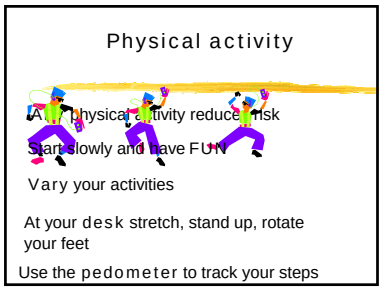


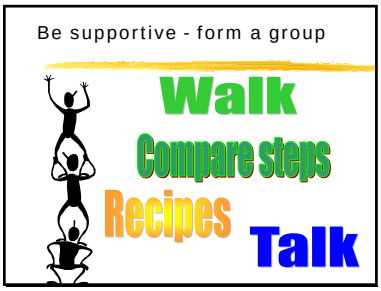
Increase dietary fibre

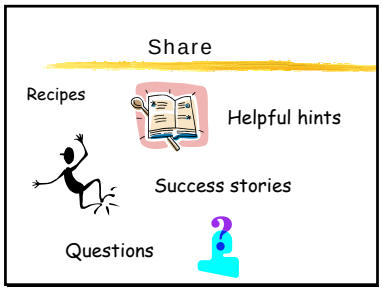
.....2 pages 216, 217

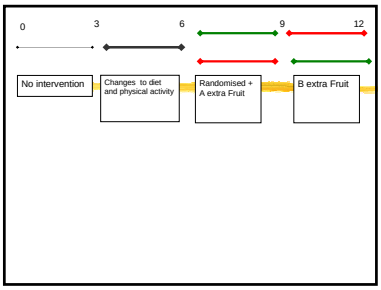
Appendix 12 Powerpoint presentation - group shakeup

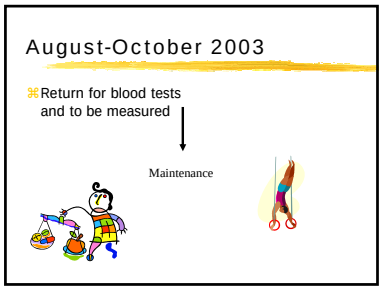












.....2 pages 216, 217