

**METABOLIC SYNDROME: EFFECT OF A  
CULTURALLY APPROPRIATE DIET AND  
PHYSICAL ACTIVITY  
IN FEMALE PAKISTANI IMMIGRANTS**

Rizwana Kousar

School of Biomedical and Health Sciences

Faculty of Health, Engineering and Science

Victoria University

St Albans Victoria

Australia

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**DOCTOR OF PHILOSOPHY DECLARATION**

I, Rizwana Kousar, declare that the PhD thesis entitled, “Metabolic Syndrome: Effect of a Culturally Appropriate Diet and Physical Activity in Female Pakistani Immigrants,” is no more than 100,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work.

Signature.....

Date.....

Rizwana Kousar  
March 2010

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## **LIST OF PAPERS AND PUBLICATIONS ARISING FROM THIS STUDY**

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A culturally appropriate diet and lifestyle intervention can successfully treat the components of Metabolic Syndrome in female Pakistani immigrants residing in Melbourne, Australia.

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Changes to diet and physical activity have potential to treat Metabolic Syndrome in female Pakistani immigrants.

Proceedings of the Nutrition Society of Australia 29<sup>th</sup> Annual Scientific Meeting. 30<sup>th</sup> November-3<sup>rd</sup> December 2005. Melbourne, Victoria, Australia.

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Changes to diet and physical activity have potential to treat Metabolic Syndrome in female Pakistani immigrants.

Proceedings of The Australasia Society for the Study of Obesity 14<sup>th</sup> Annual Scientific Meeting 14<sup>th</sup> Annual Scientific Meeting: Obesity Fact and Fiction.

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The development of a culturally appropriate intervention for a migrant population at high risk of Metabolic Syndrome.

Proceedings of the 2<sup>nd</sup> Annual World Congress on the Insulin Resistance Syndrome.

18<sup>th</sup>-20<sup>th</sup> November 2004. Los Angeles, California, USA.

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Changes to diet and physical activity have potential to treat Metabolic Syndrome in female Pakistani immigrants.

Proceedings of the 2<sup>nd</sup> Annual World Congress on the Insulin Resistance Syndrome.

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## **ABSTRACT**

Metabolic Syndrome is a global health issue characterised by the clustering of cardiovascular risk factors including central/abdominal obesity, elevated blood pressure, elevated plasma glucose levels and dyslipidaemia. Diet and lifestyle modification is the key defence against Metabolic Syndrome. This study aimed to answer the following research questions: What are the metabolic characteristics of Pakistani women residing in Melbourne displaying the risk factors of Metabolic Syndrome? Do genetic markers that have been associated with Metabolic Syndrome exist in Pakistani females? Is a culturally appropriate diet and lifestyle intervention an effective mechanism for preventing the onset, or reducing the severity of Metabolic Syndrome in migrant Pakistani women?

Sixty Pakistani migrant women (aged 20-50yr) completed a 12 week culturally appropriate diet and exercise program delivered by a peer-educator. The programme involved weekly education and monitoring sessions. Results indicate that prior to intervention subjects were sedentary ( $p=0.93$ ), obese ( $p=0.959$ ), with high waist circumference ( $p=0.922$ ), hypertensive ( $p=0.284$  systolic,  $p=0.123$  diastolic), dyslipidemic (total cholesterol  $p=0.59$ , triglycerides  $p=0.259$ , High Density Lipoproteins  $p=0.349$ ), hyperglycaemic ( $p=0.613$ ) and hyperinsulinaemic ( $p=0.349$ ) (all recognised markers of Metabolic Syndrome). At the conclusion of the intervention physical activity significantly increased ( $p<0.001$ ); significant reductions were observed in BMI ( $<0.05$ ), blood pressure ( $p<0.001$  systolic and diastolic), total cholesterol ( $p<0.05$ ), triglycerides ( $p<0.05$ ), glucose ( $p<0.001$ ) and insulin levels ( $p<0.001$ ). Dietary analysis post intervention showed subjects were consuming a diet more attuned to WHO healthy diet recommendations. Results indicated on completion of the intervention the number of subjects displaying Metabolic Syndrome was

significantly reduced to 32. All participants displayed Single Nucleotide Polymorphisms (SNPs) of the candidate genes investigated (the FABP2, WNK1 and CAD genes). We can associate homozygous carriers of all investigated SNPs in our study with the presence of 5 components of Metabolic Syndrome when compared to heterozygous participants with 3 components. Overall, the genetic association with Metabolic Syndrome in our subject population was apparent but ambiguous. This study revealed that a potentially genetically susceptible population that suffers from Metabolic Syndrome can be effectively treated with a culturally competent diet and lifestyle intervention. The success of the current program raises the possibility that other high risk ethnic groups can also be treated with a culturally competent program.

## **LIST OF ABBREVIATIONS**

ABS	Australian Bureau of Statistics
Apo(a)	Apolipoprotein(a)
Apo(b)	Apolipoprotein (b)
ATP	Adenosine triphosphate
BMI	Body mass index
CAD	Coronary artery disease
DbSnp	Data base single polymorphism
DNA	Deoxyribonucleic Acid
EDTA	Ethylenediaminetetraacetate
ELISA	Enzyme-linked immunosorbent assay
HDL-C	High-density lipoprotein-cholesterol
kg	kilogram
LDL-C	Low density lipoprotein-cholesterol
mg/dl	Milligram per decilitre
mm/Hg	Millimetre of mercury
m	Metre
mmol/L	Millimole per litre
NADH/NAD <sup>+</sup>	Nicotinamide adenine dinucleotide (reduced form)/ nicotinamide adenine dinucleotide (oxidised form)
NCEP-ATP	National Cholesterol Education Program-Adult Treatment Panel
NHANES	National Health and Nutrition Examination Survey
nHDL	Nascent high density lipoprotein
NHF	National Heart Foundation
PCR	Polymerase chain reaction
RAS	Renin angiotensinogen system
RNA	Ribonucleic acid

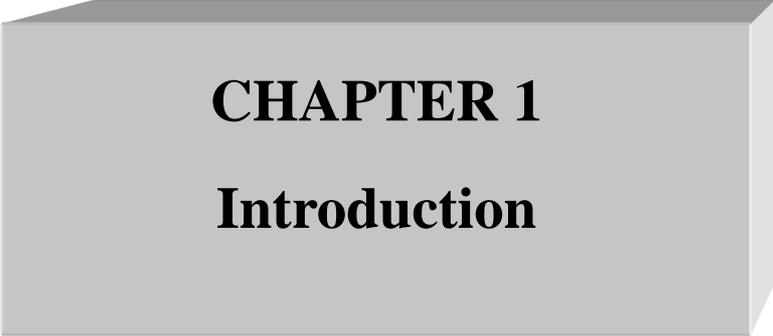
Rs	Reference number
RT-PCR	Real time-polymerase chain reaction
SD	Standard deviation
SNP	Single nucleotide polymorphism
TC	Total cholesterol
TG	Triglycerides
UK	United Kingdom
USA	United States of America
VLDL-C	Very low density lipoprotein-cholesterol
WHO	World Health Organisation

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**CHAPTER 1**

**Introduction**

# **1 INTRODUCTION**

## **1.0 An introduction to Metabolic Syndrome**

### **1.1 The definition of Metabolic Syndrome**

Metabolic Syndrome is a disorder characterised by a clustering of cardiovascular risk factors, which include central/abdominal obesity, elevated blood pressure, elevated plasma glucose levels and dyslipidaemia, according to the National Cholesterol Education Program- Adult Treatment Panel (NCEP-ATP III 2001). Metabolic Syndrome is a complex disorder, an emerging clinical challenge and important public health issue (McKeigue 1996, Token 2004). Controversy exists as to the exact definition of the Syndrome, as such many definitions currently exist (Cameron et al. 2007). The NCEP- ATP III description of Metabolic Syndrome is the most widely accepted definition and is considered to be the most practical and simple for clinical and research purposes.

#### **1.1.1 Clinical identification of Metabolic Syndrome**

The different approaches towards the clinical identification of Metabolic Syndrome elucidate the complex nature of the syndrome, as well as the difficulty associated with the formation of a definition (Stone and Saxon 2005). It was proposed by the World Health Organisation (WHO) that the Syndrome is referred to as “Metabolic Syndrome”. The NCEP-ATPIII guidelines highlighted the key features of this Syndrome and proposed a clinical definition to facilitate diagnosis and preventative interventions, (NCEP-ATPIII 2001) the definition based on having at least 3 of 5 criteria (Table 1.1)

**Table 1.1** NCEP-ATP III Clinical Definition of Metabolic Syndrome.

(Diagnosis is established when  $\geq 3$  of these risk factors are present)

<b>Risk Factor</b>	<b>Defining Level</b>
<b>1. Abdominal Obesity</b> (Waist circumference):	
Men	$>102$ cm ( $>40$ in)
Women	$>88$ cm ( $>35$ in)
<b>2. Serum Triglycerides</b>	$\geq 1.7$ mmol/L
<b>3. Serum High Density Lipoprotein Cholesterol:</b>	
Men	$\leq 1.03$ mmol/L
Women	$\leq 1.29$ mmo/L
<b>4. Arterial Blood Pressure</b>	$\geq 130$ / $\geq 85$ mm Hg
<b>5. Fasting Glucose.</b>	$\geq 5.6$ mmol/L

Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. *JAMA* (2001) 285:2486-2497.

## **1.2 Pakistanis, a unique group**

South Asian migrants are a high risk population for the development of Metabolic Syndrome.

It is generally accepted that the term 'South Asian' constitutes individuals from Pakistan, India and Bangladesh (however, some researchers have extended this definition to include Sri Lanka and Nepal) (Bhopal et al. 1999). Immigrants from Pakistan, India, and Bangladesh have strong historical and political links to one another. Indeed, under Colonial rule, the present land areas of all three formed India. In 1947, when British rule ended, the country was partitioned into the independent nations of India and Pakistan. In 1970, East Pakistan became independent from Pakistan and was renamed Bangladesh. The majority of Indians are Hindu, while Islam is the

predominant religion in Bangladesh and Pakistan. Although Pakistan, India and Bangladesh share some common ground in terms of geography, the same cannot be said about the people of each nation. In fact, great ethnic diversity exists in the South Asian region, primarily due to provincialism as well as substantial differences in languages and religious affiliations. Thus any generalised findings on South Asians as a whole need to be interpreted with extreme caution ( Kassam-Khamis et al.1999, Bhopal 2000, Raj et al.1990, Barnett et al. 2006).

Pakistanis are quite different from Indians and Bangladeshis; many Pakistanis can trace their roots to Arab nations and Iran. The Pakistani diet and culture is quite different to the Indian diet and culture, this is primarily due to religious affiliation. For example, Pakistanis generally eat increased amounts of meat and poultry and drink no alcohol, when compared to most Indians who are vegetarian and drink alcohol often (Bhopal et al. 1999). Similarly, Pakistanis consume minimal amounts of fish, when compared to Bangladeshis, as fish is a staple food in Bangladesh (Kassam-Khamis et al.1999, Sevak et al. 2004).

However, within this diversity, citizens of all three countries share broad commonalities in world views, cultural and family values, as well as traditions concerning success and education, particularly when South Asians choose to go abroad and face similar challenges of acculturation and health. As researchers generally do not categorise each South Asian group separately (that is, they assume a common background), many studies conducted in the West focus on South Asians as a single group, rather than the individual populations that the group comprises (Bhopal 2000).

### **1.2.1 Metabolic Syndrome in Pakistanis**

Research investigating the prevalence of Metabolic Syndrome in Pakistanis is very limited; researchers have not investigated Metabolic Syndrome in a female Pakistani migrant population. In this section evidence regarding cardiovascular disease and the Pakistani population will be reviewed and discussed as it is the only available data that is related to Metabolic Syndrome itself (as Metabolic Syndrome can eventually result in cardiovascular disease.) This data will be discussed in conjunction with research investigating Metabolic Syndrome in South Asian populations in general.

The risk of cardiovascular disease is not uniform among South Asians and there are important differences between Indians, Pakistanis, and Bangladeshis for many coronary risk factors. However, it has generally been established that migrant South Asians are at a higher risk of developing cardiovascular disease when compared to local host populations of various countries, such as the UK (McKeigue 1988), Singapore (Hughes et al. 1990), USA (Wild et al. 1995) Trinidad (Miller et al. 1989), New Zealand (Kolt et al. 2007), Canada (Anand et al. 2000), China (He et al. 2006). While it is clear that South Asians are generally at a higher risk of developing cardiovascular disease, it is not known if the risk is equal in both men and women, or whether there is a gender bias. As cardiovascular disease is proving to be a considerable burden on health systems worldwide it is necessary to identify high risk groups prior to the onset of the disease or, at the latest, in the early stages of the development of the disease (Williams et al.1993, Enas 1996, Enas et al. 1997 Grundy et al. 2006). Throughout 1950-1960 it was established that people with ancestral origins in the Indian subcontinent (South Asians) were highly susceptible to cardiovascular diseases after migration to Western environments (Williams et al. 1993). However, research is inconclusive as to why South Asian populations are susceptible to cardiovascular disease when compared to other ethnic groups. This higher prevalence is most likely the result of a combination of

environmental and biochemical risk factors (Balasuramanyam et al. 2008). Several studies have been conducted across the globe investigating the high prevalence of cardiovascular disease in South Asians, for example, a review by Bhopal in 2000 concluded that South Asians were indeed a high risk group for cardiovascular disease. The review revealed that in 20 years, 19 studies had been conducted and importantly all these studies suggested that South Asians were a high risk population for cardiovascular disease (Bhopal et al. 2000).

Metabolic Syndrome has received great attention recently, a statement issued in 2005 by the American Heart Association and National Heart Lung and Blood Institute emphasised that Metabolic Syndrome appears to directly promote arteriosclerosis, the major cause of cardiovascular disease (Grundy et al. 2006). It was also recognised that Metabolic Syndrome is a constellation of inter-related risk factors of metabolic origin known as “metabolic risk factors.” Furthermore, a number of “underlying risk factors” exist giving rise to primary risk factors of Metabolic Syndrome. In the past few years several expert groups have attempted to identify these underlying risk factors in the general population (Bajaj and Banerjim 2004). Underlying factors (both biochemical and environmental) such as blood lipids, blood pressure, diet, physical activity, migration to a new country and socio-economic status etc, have a great impact on the well-being and health of individuals and populations. An association seems to exist between the biochemical and environmental risk factors of Metabolic Syndrome (Ghosh 2007).

South Asian migration to western countries continues; established communities exist in the United Kingdom, however, in recent times migration to the UK has decreased and large communities have begun establishing themselves in the United States, Canada and Australia. As South Asian communities are becoming more prevalent in western countries, the investigation of South Asian health issues is also becoming more

common. In the United Kingdom it has been established that South Asian migrants are more susceptible to various non-communicable diseases, such as cardiovascular disease, type 2 diabetes and Metabolic Syndrome; furthermore, risk factors for such diseases in the second generation appear at an earlier age. It has also been shown that women are more prone to the above-mentioned diseases when compared to men, but are more likely to remain undiagnosed (Barnett et al. 2006). Researchers have been unable to conclusively establish whether biochemical or environmental factors play a greater role in the development of non-communicable diseases (Bhopal et al. 1999), thus all relevant factors will be discussed.

### **1.3 Concept of Metabolic Syndrome**

The concept of Metabolic Syndrome was proposed to highlight the simultaneous occurrence of risk factors for cardiovascular disease and type 2 diabetes (Ferrannini et al. 1987). A universally accepted criterion for the definition of Metabolic Syndrome is still in the process of being formulated.

The nature of Metabolic Syndrome was first suggested in 1923, when Kylin noted that hypertension, hyperglycaemia and gout tend to cluster together (Vitarius 2005). A more modern definition, one that includes obesity, hypertension, diabetes, and hyperlipidemia, was first suggested in the 1960's. German researchers of the 1970's are credited with the first use of the term "Metabolic Syndrome"; they also explored the syndrome's association with atherosclerosis. In the late 80s, (Ferrannini et al. 1987) and (Reaven 1988) suggested that the underlying cause of this syndrome was insulin resistance. Consequently, Ferrannini et al. favoured the term "Insulin Resistance Syndrome" whereas Reaven called it "Syndrome X" (not to be confused with another Syndrome X where patients suffer from angina despite having a normal angiogram) (Eckel et al. 2005).

Various names have been given to Metabolic Syndrome, such as 'Syndrome X', 'The Deadly Quartet' and 'Metabolic Disorder'. Metabolic Syndrome is a complex metabolic disorder and is an emerging clinical challenge. It is considered as a series of interrelated cardiovascular risk factors, in that each component of the cluster of abnormalities is a risk factor in its own right (Bosello and Zamboni 2002). It has been established that the features of Metabolic Syndrome may be present for up to 10 years before detection of a disorder (WHO 1999). One of the primary risk factors for Metabolic Syndrome is abdominal/central obesity. Weight gain exposes individuals to two major components of Metabolic Syndrome, namely, obesity and insulin resistance. Therefore the high prevalence of obesity is thought to contribute to the alarmingly increasing rates of Metabolic Syndrome across the world (Grundy et al. 2005).

The pathophysiology of Metabolic Syndromes remains a subject of continuing controversy although some researchers have suggested that a causal relationship with insulin resistance and abdominal/central obesity exists. Most researchers believe that insulin resistance may be the common aetiological factor, however abdominal/central obesity is known to be another major causative factor, as the increased production of free fatty acids (from adipose tissues) in turn may interfere with the action of insulin (Misra and Vikram 2002). Research has shown that not all components of the cluster need be present at simultaneously (Lorenzo et al. 2003). Weight gain has been shown to be the strongest predictor of Metabolic Syndrome; this aspect gives strength to the idea that weight loss is beneficial in the treatment and prevention of the disease. (Caterson et al. 1997, Despres et al. 2001, Grundy 2002, Deedwina and Gupta 2006, Misra et al. 2007).

### **1.3.1 Prevalence of Metabolic Syndrome**

#### **1.3.1.1 Prevalence in South Asians**

There have been marked differences identified in the prevalence of Metabolic Syndrome between populations classified according to ethnicity and sex. A high risk population for the development of Metabolic Syndrome are South Asian migrants (people from Pakistan, India and Bangladesh). The prevalence of Metabolic Syndrome has been shown to be much higher in South Asian migrants when compared to African-Caribbean and white European migrants (Pomerleau et al. 1991, Williams, et al.1993, McKeigue 1988, Banerji et al.1999, Jafar et al. 2004, Tillin et al. 2005, Stone and Saxon 2005, Kolt et al. 2007). It has been established that Metabolic Syndrome can increase the risk of developing cardiovascular disease four-fold in Punjabi Indians (Williams et al. 1993). According to reports primarily from the UK, the prevalence of Metabolic Syndrome ranges from as low as 29% and as high as 50% in migrant South Asians (Farooqi et al. 2000, Mellin-Olsen and Wandel 2005). It has also been shown that South Asians are twice as likely to develop cardiovascular disease when they suffer from Metabolic Syndrome, when compared to non-sufferers (Farooqi et al. 200). Although studies on migrant South Asian women are limited, the data that does exist suggests that the rate of mortality due to cardiovascular disease is higher in migrant South Asian women, when compared to South Asian men (Jafar et al. 2004). Furthermore, the prevalence of cardiovascular disease in migrant Pakistanis living in the UK, USA and South Africa is 50% higher when compared to the general population of the local host country (Tillin et al. 2005, Kolt et al. 2007). As a sub-sample of the South Asian population Pakistanis tend to have a higher rate of hyperinsulinemia and insulin resistance when compared to people of other ethnic backgrounds. insulin resistance when compared to people of other ethnic backgrounds.

Recently, the prevalence of Metabolic Syndrome has been on a sharp rise predominantly due to the ever increasing global epidemic of obesity and type II diabetes. (Eskel *et al* 2005.) The diagnosis of Metabolic Syndrome itself is difficult, as the presence of various components of the syndrome can vary greatly, furthermore, one or more components can exist for a number of years before the presence of all requisite components is established.

Studies conflict as to the overall prevalence of Metabolic Syndrome The primary reasons for this is due to the lack of a universal definition; that is many different criteria are being used in order to establish the prevalence of the syndrome. As a result of this the prevalence of the syndrome can vary extensively For example, in Australia it is estimated that the syndrome affects between 23-25% of the general population, whereas in migrant South Asians, it has been suggested that the prevalence is 50% higher, also,, the prevalence amongst different studies varies from 8% in India to 24% in individuals aged from 20-25. As above, there are conflicting views in regards to the prevalence of chronic disease in South Asians, due to differences in study design and methodologies of research in various studies, however, the majority of studies strongly suggest that South Asians in particular are a high risk population for Metabolic Syndrome, cardiovascular disease and type II diabetes, therefore, priority should be placed on prevention and treatment in South Asians.

Most available data on Metabolic Syndrome originates from western countries and is based on Caucasian populations, thus, little data is available on Metabolic Syndrome in South Asians. The number of migrant South Asians has been increasing steadily throughout the globe, for example, South Asians in the UK constitute the largest migrant population and in Canada, constitute the second largest migrant population(Gupta and Brister,2006).. One reason for the lack of data on South Asians, is due to the broad use of the word “Asian,” many researchers focus their research on

South East Asians, South Asians and even on Pacific Islanders, South Asians however are strictly from Pakistan, India, Bangladesh and Sri Lanka and should not be classified as Asians in general.

McKeigue *et al* were the first to study obesity and insulin resistance in South Asians. McKeigue *et al* have previously reported that a higher prevalence of obesity, insulin resistance and obesity related diseases in South Asians, when compared to the general population. Studies have also shown that insulin resistance can develop at an early age in South Asians, which predisposes them to Metabolic Syndrome and other chronic diseases in adulthood. (McKeigue *et al*, 1988, 1991, 1992, 1993.)

### **1.3.1.2 Global prevalence of Metabolic Syndrome**

Various reports highlight the prevalence of Metabolic Syndrome in different populations; however, comparison for the Syndrome among various populations is difficult. This is primarily due to the lack of a definitive definition of Metabolic Syndrome. As discussed earlier, researchers generally agree on what Metabolic Syndrome is, but have failed to develop a universally accepted definition. Studies that have attempted to compare various populations have further highlighted the need for a universally accepted definition for the Syndrome.

Despite controversy as to the use of definitions and criterion in the identification of Metabolic Syndrome, researchers generally agree that certain traits can be observed universally in most populations; for example, many studies have shown that the prevalence of Metabolic Syndrome increases as age increases in many different populations (Cameron *et al*. 2007). In the Iranian population, for example, the prevalence of Metabolic Syndrome is less than 10% in both sexes from ages 20-29 years. But as age group increases, so does the prevalence of Metabolic Syndrome; at age 60 the prevalence is 38%, however, by age 69, the prevalence increases

significantly to 67%. Similar conclusions have been observed in studies on French populations. The prevalence rises from 5-6% in the 30-39 age bracket to 17.5% in the 60-69 age bracket. The prevalence of Metabolic Syndrome in America, as shown in the National Health and Nutrition Examination Survey increases from 7% in the 20-29 year age bracket (Caucasian/Hispanic/African American) to 44% for those in the 60-69 year age bracket (Eckel et al. 2005, Cameron et al. 2007).

All proposed Metabolic Syndrome definitions accept the clustering of risk factors for cardiovascular disease and are also intended for clinical/research use as indicators for the risk of cardiovascular disease (Wild et al. 1995, Lakka et al. 2002). Many studies have demonstrated that there are marked similarities in certain ethnic populations pertaining to Metabolic Syndrome. The “one size fits all” approach in terms of a definition for Metabolic Syndrome is not appropriate for all populations (Wild et al. 1995, Tillin et al. 2005). Various populations are more susceptible to Metabolic Syndrome than others; South Asians are one such population. As there have been variations in the standard values and the interpretation of data, the combination of risk factors for Metabolic Syndrome results in a substantial controversy. No current definition recognises the nature and contribution of each individual component of the Metabolic Syndrome, as the prevalence of each component varies across different ethnic groups (Lakka 2002)

## **1.4 Role of obesity in Metabolic Syndrome**

### **1.4.1 The association between Metabolic Syndrome and obesity**

Obesity is defined as condition in which excess body fat may put a person at health risk. Excess body fat results from an imbalance of energy intake and energy expenditure (total energy expenditure includes energy expended at rest, in physical activity and for metabolism) (WHO 1998). Obesity is a global health issue. It has reached epidemic proportions worldwide. It can be described as excessive fat accumulation in the adipose tissues to such an extent where it can impair normal health (WHO 1998). Obesity has an association with cardiovascular disease; presumably it impacts on risk factors, which include dyslipidaemia, hypertension, insulin resistance and type 2 diabetes, all components of Metabolic Syndrome. Importantly, obesity has been recognised as one of the primary underlying causes of Metabolic Syndrome (Kolt et al. 2007).

The International Obesity Task Force of WHO proposed a system of classification based on Body Mass Index (BMI, BMI is calculated from simple measurements ( $BMI = \text{weight (kg)} / [\text{height (m)}]^2$ ). The WHO Obesity Task Force, and the National Health and Medical Research Council of Australia have accepted that BMI is a better marker of obesity. A person with a BMI of 30 or greater is defined as obese; such individuals are at an increased health risk, and further weight gain should be avoided (WHO, 2000). The BMI is less prone to errors and it also correlates with waist circumference. It has been recommended as a better index for the evaluation of obesity as it is a useful measure, regardless of frame size. There is good correlation between the classification of fatness and health risks in Asians and Australians, however, BMI is not always precisely determinative of body fat levels, as the formula is based on total weight and ignores the possibility of increased muscle mass accounting for individuals increased weight (Caterson 1997, WHO 2000, Deurenberg et al. 2002, James 2008).

Importantly, data has established that South Asians are susceptible to the development of Metabolic Syndrome at an even lower BMI when compared to other ethnic populations. Asian populations tend to accumulate intra-abdominal fat without developing generalised adiposity. (Deurenberg et al. 1998, James et al. 2002 Chang et al. 2003, Deurenberg et al. 2002, WHO 2003, Kolt et al. 2007). It has been proposed that the relationship between BMI and total body fat may be different in Asian when compared to Caucasians (WHO 1998, WHO 2000). Accordingly, the WHO established different BMI criteria for Asians, where a BMI of 23 is considered overweight, and a BMI of 25 or over is considered obese (refer to Table 1.2). While this standard cannot be directly related to South Asians, it is the preferred standard of many researchers investigating South Asian populations.

**Table 1.2** WHO Classification of BMI, A Comparison of Asian Guidelines and Standard Guidelines for the Assessment of Overweight and Obesity.

<b>WHO classification</b>	<b>BMI(kg/m<sup>2</sup>) (Standard values)</b>	<b>BMI(kg/m<sup>2</sup>) (Asian values)</b>
Underweight	<18.5	<18.5
Normal	18.5-24.9	18.5-22.9
Overweight	25.0-29.9	23.0-24.9
Obese	30.0-34.9+	25.0-27.0+

- BMI classifications as defined by a WHO expert committee (the regional office for the Western Pacific region of WHO.) Asians are classified as overweight or obese at a lower BMI when compared to the general population (WHO 2000).

Many studies have suggested that obesity may be an independent risk factor for coronary heart disease (Anand 1998). There is a direct relationship between the degree

of obesity and the development of cardiovascular disease. A moderate weight gain appears to increase the risk of cardiovascular disease (Anand et al. 2000, Bonora 2006). Upper body fat is considered a greater risk factor when compare to lower body fat. Several studies have shown that the accumulation of fat in the upper part of the body predisposes a person to important metabolic alterations which lead to an increased incidence of Metabolic Syndrome, cardiovascular disease and type 2 diabetes (Bjorntrop 1987, Despres et al. 2001, Bosello and Zamboni 2000, Carr et al. 2004)

### **1.5 South Asians and central obesity**

A typical phenotype that can be observed in South Asian populations is the general tendency to carry a higher percentage of fat and a lower percentage of lean mass at a comparatively lower BMI, when compared to European (McKeigue et al. 1992); this has been linked with the increased prevalence of insulin resistance dyslipidaemia and hypertension that can be observed in South Asian populations (McKeigue and Marmot 1988, McKeigue et al. 1993, Barnett et al. 2006, Grundy, 2000).

Many studies have shown that South Asians are more prone to the development of obesity and its related disorders at a younger age when compared to other populations. McKeigue et al. were the first to show that migrant South Asians are more prone to central obesity and cardiovascular disease when compared to the European population (McKeigue et al. 1992, WHO 2003).

However the primary reasons suggested for the increased prevalence of obesity in migrant South Asians are diet and a lack of physical activity (McKeigue et al. 1988, Bhopal 1999, Misra and Vikram 2002, Fischbacher et al. 2004, Bajaj and Benerji 2004, Vitarius 2005).

### **1.5.1 Possible pathophysiology of obesity**

Obesity predisposes an individual to several chronic diseases that are multifactorial in origin. There is rarely a one to one relationship between body fat and the disease. Studies have shown that with severe obesity, the size and number of adipocytes increases. Abdominal/central fat mass has been proposed as the major determinant of metabolic and cardiovascular abnormalities (Daskalopoulou et al. 2004).

Adipose tissue is considered to be the largest storage organ for energy in the body; it is stored in the form of triglycerides, which are mobilised through the lipolysis process to provide fuel to other organs and deliver substrates to the liver for gluconeogenesis (Banerji et al.1999). Fatty acids represent one of the most important energy sources for the human body; they circulate in the blood as triglycerides inside lipoproteins or as non-esterified free fatty acids (Abate et al. 1995).

Contrary to the former concept of fat being an “inert” tissue mass, adipocytes are increasingly being recognised as secretory entities. Recent insight on the biology of adipocytes has revealed that they secrete a wide variety of hormone and growth factors into the blood circulation. These bioactive factors are known as “adipocytokines.” The compounds can have a profound effect on fat and carbohydrates metabolism (Jafar et al. 2004). These adipocytokines include leptin, tumour necrosis factor-alpha, non esterified fatty acids, prostaglandins, adiponectin, resistin, angiotensinogen, interleukins-1 and interleukins -6, and many others. Leptin is an isolated peptide hormone that is synthesised primarily in adipocytes and binds to specific receptors found in the hypothalamus. One of the major functions of this hormone is the control of energy balance leading to reduction in food intake, elevation in temperature and energy expenditure (Wajchenberg 2000).

The adipose tissue mass within human body can be divided into three major compartments: superficial subcutaneous adipose tissue, deep subcutaneous adipose

tissue, and abdominal/central adipose tissue. Superficial subcutaneous layer is the primary compartment, the others secondary compartments (Fox et al. 2007). The superficial subcutaneous layer is present throughout the body. With energy excess, the secondary adipose tissue compartment, the deep subcutaneous (mainly upper body) and the Abdominal/central adipose tissue compartments become more prominent. Superficial subcutaneous adipose tissue is relatively inert metabolically, whereas the other two compartments are metabolically active and have the higher fatty acid flux rates and are more closely linked to Metabolic Syndrome and insulin resistance (Sniderman et al. 2007).

Fat within the superficial subcutaneous layer is organised into tightly packed lobules, whereas fat in the deeper subcutaneous layer is found in lobules that are larger, more irregular, and less well organised. Abdominal/central fat however is even more vascular and the lobules are less well defined. Abdominal/central adipose tissue is the smallest of the three adipose tissue compartments, but the one most often linked to metabolic complications. Abdominal/central fat constitutes only 6-20 % of the total body fat mass and is more metabolically active (Chandalia and Abate 1999). The smaller size abdominal/central adipocytes are more responsive to the lipolytic effects of catecholamines (catecholamines are a group of physiologically important substances including adrenaline, noradrenaline and dopamine); and less responsive to the anabolic effects of insulin (Bray 2004). Weight gain causes an increased rate of lipolysis in abdominal/central fat depots, this then results in an increased levels of non-esterified fatty acids in the bloodstream. (Caterson 1997, Bjorntorp 1999, Freedland 2004).

It has been suggested that abdominal/central adipocytes are metabolically active at a higher rate when compared to peripheral adipocytes, as a result of increased lipolysis (Eckel et al. 2005). Importantly, obese individuals have increased non esterified fatty acids from expanded adipose tissue mass in their blood circulation. Elevated non

esterified fatty acids levels in the liver results in the increased production of hepatic glucose (due to stimulation of gluconeogenesis by providing a continuous source of energy ATP and substrate and hepatic insulin resistance) and increased secretion of very low density lipoprotein (VLDL) particles (Rendell et al. 2001 ). These changes are associated with lipid/lipoprotein abnormalities such as reduction in high density lipoprotein cholesterol ( HDL-C) and hepatic steatosis (non-alcoholic fatty liver) (Matsuzawa 1999, Misra and Vikram 2003). Hepatic steatosis gives rise to an increased level of peripheral low density lipoprotein cholesterol particles (LDL-C) and triglycerides. Consequently, abnormalities such as dyslipidaemia and other components of Metabolic Syndrome begin to develop (Caterson 1997, Matsuzawa 1999, Defronzo 2006 ). Elevated non esterified fatty acids levels in the liver will also decrease hepatic insulin extraction by inhibiting insulin binding and degradation (Wajchenberg 2000) leading to systemic hyperinsulinemia and insulin resistance (Vitarius 2005, Defronzo 2006, Wajchenberg 2000).

Controversy exists in regard to the true rate of lipolysis among obese individuals. The rate of lipolysis also varies, based on gender and the type of obesity (upper or lower) (Banjeri et al. 1999). It is generally accepted that in all cases of obesity, the lipolysis rate increases (Matsuzawa et al. 1999). It has also been shown that the rate will decrease when fat mass is reduced (Large and Arner 1998). Lipolysis is intensively regulated by multiple hormonal activities (Bray 2004). In human beings, the major hormones are insulin (inhibition of lipolysis) and catecholamines (stimulation of lipolysis) ( Defronzo 2006 ). Thus, dyslipidaemia can be directly associated with elevated levels of non-esterified fatty acids released from adipose tissue (Vitarius 2005, Deedwania and Prakash 2004). Increased levels of non esterified fatty acid into the portal venous system often signal the onset of the pathophysiology of type 2 diabetes, insulin resistance, hypertension and Metabolic Syndrome (Banjeri et al. 1999). Escalating

evidence exists suggesting that obesity is the driving force behind the development of the components of Metabolic Syndrome. The relationship between non-esterified fatty acids and insulin resistance was first suggested by Randle et al. in 1963, the relationship will be discussed further in the next section.

## **1.6 Insulin resistance and Metabolic Syndrome**

### **1.6.1 Insulin resistance and South Asians**

In this section insulin resistance will be discussed in context with Metabolic Syndrome. Insulin resistance is a condition in which the normal levels of insulin are insufficient in producing an adequate insulin response in the body (Reaven 1988). A high fasting insulin level is a strong predictor of cardiovascular disease. (Ramachandran 2004, Carr et al. 2004, Lemieux et al. 2001). Insulin resistance is a critical characteristic of Metabolic Syndrome and also serves as a pre-diabetic condition to its onset (Despres et al. 1989). Environmental, metabolic and genetic factors could serve as contributory factors for the presence of insulin resistance of Metabolic Syndrome (Lewis et al. 2002). Many researchers believe that obesity and insulin resistance are the pathophysiological components underlying the clustering of cardiovascular risk factors in Metabolic Syndrome (Reaven 1988, Matsuzawa et al. 1999). The mechanistic link between obesity, insulin resistance and Metabolic Syndrome is complex because the relationship is modulated by factors, such as physical activity, body fat distribution and genetic polymorphic makeup (Reaven 2004). There are a series of potential mechanisms by which the components of Metabolic Syndrome and insulin resistance itself can exert independent effects to promote cardiovascular disease (DeFronzo 2006). It has been consistently demonstrated that in almost every case, insulin resistance is present some years prior to the onset of type 2 diabetes and Metabolic Syndrome (McKeigue et al. 1991). Although the primary factors for the development of type 2 diabetes are

unknown, it is obvious that insulin resistance plays a major role in the development of type 2 diabetes as well as Metabolic Syndrome (Reaven et al. 1993).

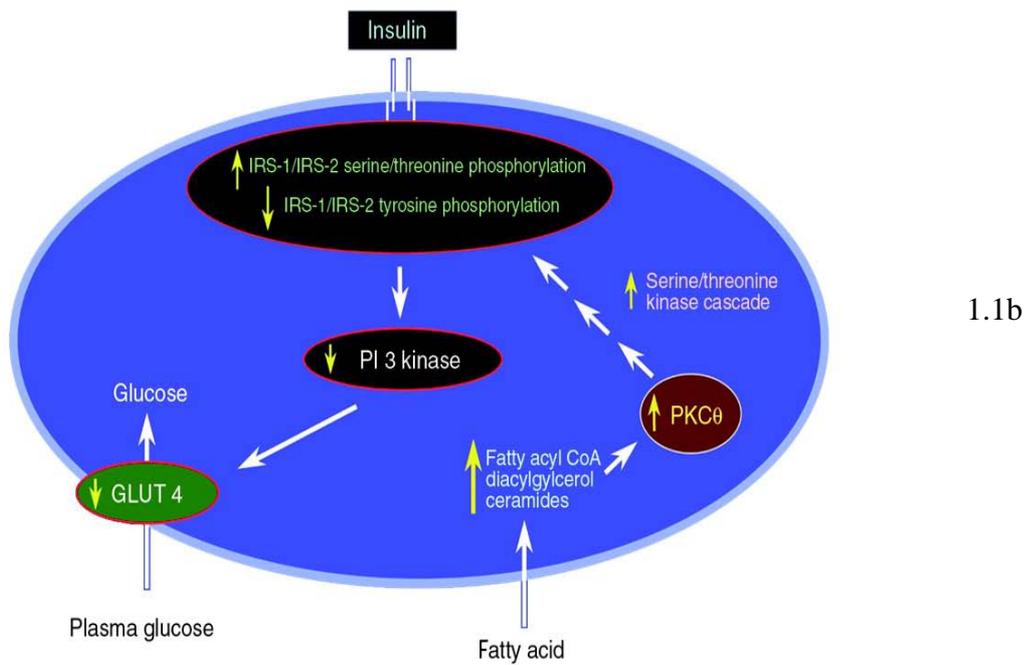
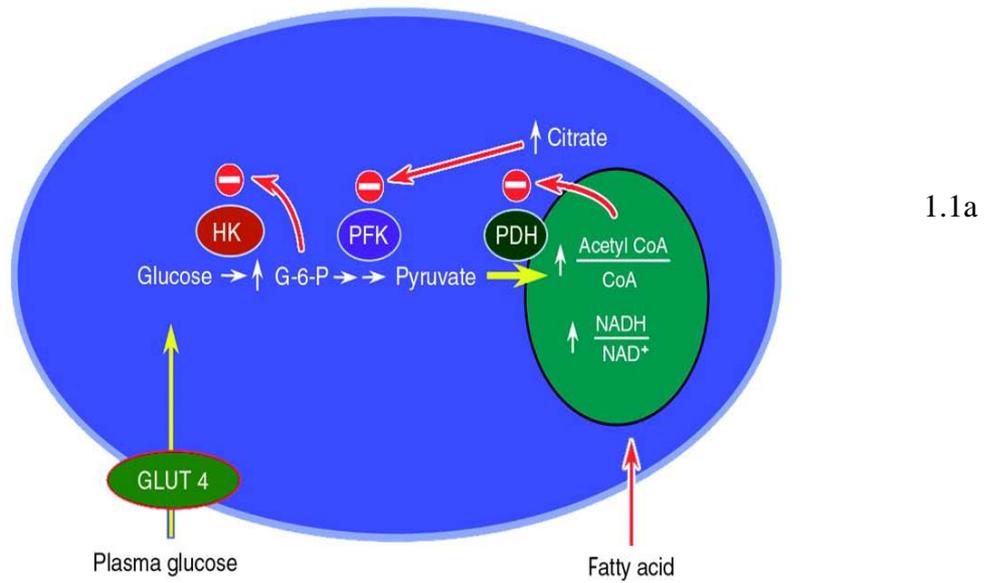
South Asians tend to have a higher rate of hyperinsulinemia and insulin resistance when compared to people of other ethnic backgrounds such as Hispanics and Europeans (McKeigue et al. 1988, McKeigue 1992, Banerji et al. 1999, Grundy 2000, Kolt et al. 2007). In fact it has been suggested that plasma insulin levels in migrant South Asians (Indians) residing in UK, USA, and Canada are two-fold higher when compared to Europeans (Ramachandran 2004). It is a characteristic feature of South Asian blood analyses; it has been suggested that the presence of high fasting insulin levels in South Asians is indicative of an underlying state of insulin resistance that could predispose the individuals to type 2 diabetes, cardiovascular disease and Metabolic Syndrome (Misra and Vikram 2004). Insulin resistance could be the best explanation for the high prevalence/pathogenesis of type 2 diabetes and the cardiovascular components of Metabolic Syndrome in South Asians (Shulman 2000).

#### **1.6.1.1 Possible pathophysiology of insulin resistance**

There is mounting evidence that insulin resistance is the fundamental defect linking each individual component of Metabolic Syndrome. The strength of the association of insulin resistance to these components is variable amongst different populations as well as within specific populations (Standl, 2005, Vitarius 2005).

Elevated levels of plasma non esterified fatty acid have been implicated in the pathogenesis of insulin resistance and other components of Metabolic Syndrome. The mobilisation of non-esterified fatty acids in visceral adipose tissue occurs at a faster rate when compared to subcutaneous tissue; this is because of the higher rate of lipolytic activity in visceral adipocytes (adipocytes from obese subjects generally show increased lipolytic response to catecholamines) (Shankar and Sundarka 2003). The higher

lipolytic activity in visceral fat in comparison with subcutaneous adipose tissue can be attributed to regional variation in the action of major lipolysis-regulating hormones, catecholamines and insulin, the lipolytic effect of catecholamines being pronounced and the anti-lipolytic effect of insulin being weaker in visceral than in subcutaneous adipose tissues (Goodpaster et al. 1997, Large and Arner 1998, Carr et al. 2004) Elevated levels of plasma non esterified fatty acids are typically associated with an insulin resistant state in the muscles and liver. Randle et al. first suggested the most widely accepted hypothesis for the free fatty acid induced mechanism of insulin resistance in the muscles and liver. It was reported that fatty acids compete (Refer to Figure 1.1) with glucose for substrate oxidation in the rat heart muscle, it was also suggested that increased fatty acid oxidation causes insulin resistance. As the intracellular concentration of free fatty acids rises, it causes an increase in the intramitochondrial acetyl CoA/CoA and NADH/NAD<sup>+</sup> ratios (DeFronzo 2006). Subsequently pyruvate dehydrogenase is inactivated. This in turn would cause intracellular citrate concentrations to increase leading to the inhibition of phosphofructokinase (a key enzyme in glycolysis) (Randle et al. 1963, Petersen and Shulman, 2002). This gives rise to the subsequent accumulation of glucose 6-phosphate which would inhibit hexokinase II activity resulting in an increase in intracellular glucose concentration and decrease glucose uptake (refer to Figure 1.1).



**Figure 1.1** Mechanism of fatty acids-induced insulin resistance in skeletal muscles as proposed by Randle (Randle et al. 1963).

Researchers generally agree that the reduced glucose transport activity could be the result of the direct effect of non-esterified fatty acids on the Glut 4 transporter, a protein present in cell membranes for the transportation of glucose. (Roden et al. 1996). Increased plasma non esterified fatty acid levels alter cell trafficking, budding, fusion and Glut 4 activity. Increased non esterified fatty acids can also induce alterations in upstream insulin signalling events resulting in decreased Glut 4 translocations to plasma membranes (Petersen and Shulman, 2002) Which in turn leads to insulin resistance and elevated levels of insulin in the bloodstream (hyperinsulinemia and its associated complications) as shown in Figure 1.1 (Randle et al. 1963, Lewis et al. 2002). It has been shown that individuals who suffer from insulin resistance are also quite likely to suffer from dyslipidaemia. The influx of non esterified fatty acids is considered as a possible mechanistic link between dyslipidaemia, obesity and insulin resistance.

### **1.7 The association between Metabolic Syndrome and atherogenic dyslipidaemia and South Asians.**

‘Atherogenic dyslipidaemia’ is an important feature of Metabolic Syndrome. It is characterized by: elevated fasting triglyceride levels, HDL-C and increased levels of LDL-C (Palaniappan et al. 2007). Irregularities in blood lipid profiles have been shown to accurately predict various risk factors for cardiovascular disease and type 2 diabetes. A 10 year follow up of the “The Quebec Cardiovascular” study revealed that the presence of atherogenic dyslipidaemia serves as a strong predictor for cardiovascular disease and type 2 diabetes (Lemieux et al. 2001, Barter 2006). Atherogenic dyslipidaemia has been shown as a characteristic feature of South Asian blood lipid profile analysis (Feroz et al. 1999). HDL-C is believed to have cardio protective benefits. (Barter 2005). Notably, a few studies have reported that South Asians display the presence of high concentration of small HDL-C particles as well small dense LDL-

C particles. Thus it can be assumed that South Asians are at an increased risk of cardiovascular disease (Misra and Vikram 2004). However it has been established that by rectifying irregularities in one's lipid profile, the likelihood of a major coronary event is significantly reduced. It has also been suggested that the determination of LDL-C particle size in a clinical routine lipid profile may improve the ability to predict cardiovascular disease (Misra and Vikram 2003).

### **1.7.1 Biochemical markers of dyslipidaemia (lipids)**

Biochemical markers of atherogenic dyslipidaemia will be discussed in this section, namely, low density lipoproteins and high density lipoproteins. Lipids are characterised by their insolubility in aqueous solutions, such as blood and their solubility in organic solvents, such as ether, chloroform and acetone. Lipids are classified either as compound lipids (phospholipids), simple lipids (including fatty acids, triglycerides, and waxes) or steroids (sterol esters).

#### **1.7.1.1 Low density lipoprotein (LDL)**

LDL particles are major carriers of cholesterol, carrying about 60% of total serum cholesterol. Their function is to transport the cholesterol to the tissues where it can be used. The LDL concentration in the plasma appears to be influenced both by the rate of production of LDL from intermediate density lipoprotein (IDL), VLDL of hepatic origin, and LDL receptors on cell surface (Ginsberg 1994). LDL interacts with LDL receptors on the cells via apo B-100, which clears the lipoprotein from the circulation. About 60 % of LDL is removed from the circulation by the liver. The rest is taken up by the peripheral tissues. Cholesterol which is delivered by LDL to the cells (Richards et al. 1989), regulates the amount of intracellular cholesterol synthesis and the number of LDL receptors expressed on the cell surface (Kolovou et al. 2005).

### **1.7.1.2 High-density-lipoprotein (HDL)**

The function of HDL is to remove unesterified cholesterol and other lipoproteins from cells, where it may have accumulated, and return it to the liver for excretion in the bile. Nascent high density lipoprotein (nHDL) has the ability to bind to lipoprotein receptors on both hepatic and extrahepatic cells. Lipoprotein receptors may be specific for HDL, but they also include the LDL receptors to which HDL can bind via its apo-E components. The implication is that HDL can compete with LDL at its receptor site. (Kolovou et al. 2005).

The net effect of HDL particle's properties is that cholesterol and other lipoproteins are retrieved from peripheral cells and returned in an esterified form to the liver (Richards et al. 1989). This reverse cholesterol transport is a benefit to the cardiovascular system, as it reduces the amount of deposited cholesterol in the vascular endothelium, and reduces the risk of plaque formation.

## **1.8 Possible pathophysiology of atherogenic dyslipidaemia**

The link between obesity and dyslipidaemia is an elevated level of triglycerides. In general, an increased influx of free fatty acids into the liver induces increased hepatic triglyceride content in obese persons, which sets the stage for dyslipidaemia. An elevation of serum triglycerides is a consequence to hepatic triglyceride overload. As a result, the liver synthesises/secretates VLDL into the blood stream, and this is generally accompanied by altered lipid turnover in peripheral tissues ( Lemieux et al. 2000).

Under normal physiological conditions the liver secretes VLDL's that are rich in triglycerides and poor in cholesterol. The endothelial enzyme lipoprotein lipase (LPL) (an insulin sensitive enzyme that is partially responsible for the decreased clearance of triglycerides and decreased production of HDL particles in peripheral tissues adipose/muscle tissues), converts most of these triglycerides in VLDL's into free fatty

acids which are used by peripheral tissues as an energy source. Most extrahepatic cells (with the exception of those synthesising steroid hormone) are unable to metabolize cholesterol, which would therefore accumulate if supply exceeded demand (Misra and Vikram 2004). Insulin inhibits the secretion of VLDL from the liver into the peripheral circulation. This response is actually an effect of insulin on the degradation of triglycerides (apo-B protein) as insulin is also lipogenic, increasing the transcriptional and enzymatic activity of many genes that relate to triglyceride synthesis (Large and Arner 1998).

Insulin resistance could also reduce the activity of LPL; this alteration in lipoprotein lipase seems to contribute less to increased levels of triglycerides than does the overproduction of VLDL (Grundy 2005).

In the presence of increased VLDL in the plasma, and normal levels of activity of plasma cholesterol ester transfer protein (CETP), VLDL triglycerides can be exchanged for HDL cholesterol. This is due to VLDL particles donating one molecule of triglyceride to HDL molecules in the presence of the CETP molecule (Barter 2005). This leads to two different outcomes: a cholesterol-rich VLDL remnant particle that is atherogenic, and a triglyceride-rich cholesterol depleted HDL particle. This triglyceride-rich HDL particle can undergo further modification including hydrolysis of its triglycerides by hepatic lipase, which can disassociate the structurally important protein apo A1 (Misra and Vikram 2004). These triglyceride-enriched lipoproteins are a good substrate for the enzyme hepatic lipase. Free apo A1 molecules in the plasma are cleared more rapidly than apo A1 molecules which are associated with HDL (Palanlappan et al. 2007). The major site of apo A1 molecule clearance is the kidney where HDL are reduced, together with an amount of circulatory apo A1 molecules. Consequently, there is a reduction in the number of HDL particles (Despres 1993). A similar process leads to increased levels of small LDL in the circulation. An increased

level of VLDL in the presence of CETP can promote the transfer of triglycerides into LDL in exchange for LDL cholesterol ester. The triglyceride-rich LDL particles can undergo hydrolysis by hepatic lipase which leads to small, dense, cholesterol-depleted and in general, lipid-depleted LDL (Misra and Vikram 2004).

### **1.9 The association between hypertension and Metabolic Syndrome**

Hypertension is defined as blood pressure greater than 130/85 mm/Hg (WHO 1999). Hypertension is a major health problem affecting approximately 30% of adults in western societies; it is also a well recognised component of Metabolic Syndrome.

In the past decade the prevalence of overweight and obesity has risen dramatically and contributed to the rise in hypertension throughout the world (Sharma and Chetty 2005).

Moderate weight gain is associated with the risk of developing hypertension; however there is some individual variability in the blood pressure response to weight gain, as not all obese individuals become hypertensive (Bjorntop et al. 1987). Obesity, hypertension and insulin resistance can occur simultaneously. Obesity is suggested to predispose an individual to develop insulin resistance, hypertension, and ultimately cardiovascular disease. Weight loss is associated with a reduction in blood pressure in most cases (Juhaeri et al. 2002). The positive relationship between obesity and hypertension has been well reported (Juhaeri et al. 2002, Sharma and Chetty 2005, Tobe et al. 2006, Balasubramanyam et al. 2008). The relationship between obesity and hypertension appears to be linear, however the strength of association of obesity with hypertension varies among different racial and ethnic groups (Misra and Vikram 2004, Smith et al. 2005). Data from the Framingham heart study suggests that between 65%-75% of cases of hypertension in men and women are directly attributable to overweight and obesity (Müller-Wieland et al. 1998, Doll et al. 2002, Davy and Hall 2004). However the

mechanistic link between obesity, insulin resistance, and hypertension, Metabolic Syndrome is yet to be determined.

### **1.9.1 Hypertension in South Asians**

The relationship between blood pressure and risk of cardiovascular disease event is continuous, consistent, and independent of other risk factors. It is well established that high blood pressure predisposes an individual to hypertension. Hypertension has not been investigated in a Pakistani population. However, limited studies have been conducted on South Asians and hypertension. Most data originates from the UK and relates to hypertension as a risk factor for cardiovascular disease. Importantly, studies on South Asian populations have not been conducted on a large cohort; most studies are cross-sectional in nature. Published studies are inconsistent in their conclusions regarding the prevalence of hypertension in South Asian populations (Bhopal et al. 1999). Some studies have suggested that mean systolic blood pressure is higher in South Asian populations (when compared to Caucasians) while other studies have suggested that mean diastolic blood pressure is higher in South Asian populations (when compared to Caucasians) (Agyemang and Bhopal 2002). The Health Survey of England (Stanner 2001, Zaninotto et al. 2007) reported that Pakistani women had an elevated blood pressure when compared to other women in England. A cross-sectional study conducted in Oslo investigated 5 different ethnic groups; it was revealed that Pakistanis on average had the highest blood pressure when compared to the other ethnic groups examined (Iranians, Bangladeshis, Vietnamese and Indians) (Glenday et al. 2006). Such studies generally suggest that South Asians suffer from high blood pressure when compared to the Caucasian population. Studies have shown that South Asian migrant women generally have a higher blood pressure, when compared to South Asian men (Agyemang and Bhopal 2002, Barnett et al. 2006).

The incidence of hypertension is highest in overweight and sedentary individuals. It has been shown that individuals who are physically active are at a lower risk of hypertension when compared to their sedentary counterparts (Davy and Hall 2004). A reduction in elevated blood pressure should also confer health benefits. Prevention of weight gain should be the major priority for reducing hypertension and its related future consequences. Studies have demonstrated that regular physical activity can reduce blood pressure (systolic and diastolic) between 7-10 mm/Hg. For each decline of 1mm/Hg in diastolic blood pressure the risk of coronary heart disease will decrease by approximately 2-3% (Chobanian et al. 2003, Sharma and Chetty 2005, Grundy et al. 2005). There seems to be a general agreement among researchers that any elevation in blood pressure above the optimal level (130/85mm/Hg) will increase an individual's risk of developing cardiovascular disease. Additionally weight loss, sodium reduction and increased consumption of vegetables, fruits and low-fat dairy products can also reduce blood pressure (Levesque and Lamarche 2008). Thus, dietary modification in conjunction with increased physical activity are essential components of any intervention directed to reduce hypertension and thus cardiovascular disease and Metabolic Syndrome, accordingly, a major objective of this project was to reduce hypertension with the implementation of a diet and lifestyle modification programme.

#### **1.10 Possible mechanism of pathogenesis of hypertension in Metabolic Syndrome**

Elevated blood pressure is related with cardiovascular risk factors and increases the mortality risks in men and women. A series of endocrine, metabolic and hemodynamic mechanisms are responsible for the development of obesity-related hypertension (Zavaroni et al. 1992, Chobanian et al. 2003, Wagh and Stone 2004). To date the factors that have been most strongly associated with the development of hypertension include excess salt intake, high blood cholesterol levels, psychosocial factors (such as anxiety

and stress), as well as an unknown genetic predisposition (Goldbacher and Matthews 2007). The most widely accepted hypothesis is that visceral adipose tissues play a key role in the pathogenesis of hypertension and its various components (Nunes and Riberirio 2002). Visceral adipose tissue has been associated with insulin resistance, sodium retention, hyperinsulinemia and increased sympathetic nervous system activity, all of which have been linked to an increased likelihood of hypertension (Juhaeri et al. 2002).

The renin-angiotensinogen system (RAS) plays a central role in the regulation of blood pressure and electrolyte homeostasis (Sharma and Chetty 2005). Renin is secreted from the juxtaglomerular apparatus of the kidney in response to low blood pressure/low level of sodium chloride in the kidney. It is also released in response to stimulation from the sympathetic nervous system. Renin is responsible for converting renin substrate angiotensinogen into angiotensinogen1, a physiologically inactive substance. Angiotensinogen 1 can rapidly be converted into Angiotensinogen II in the lungs by angiotensinogen converting enzyme. Angiotensinogen II is a key effector of the activation of the renin-angiotensinogen system (RAS). Angiotensinogen II has several effects that can elevate the arterial pressure: vasoconstriction (especially of arterioles) occurs very rapidly; constriction of the arterioles increases peripheral resistance and thereby elevates blood pressure; other effects mainly relate to body fluid /volumes. Angiotensinogen II has a direct effect on the kidneys (Cheng et al. 2005): it can stimulate the release of aldosterone from adrenal gland. It causes a decreased excretion of salt and water via increased secretion of aldosterones. Both effects tend to elevate the blood volume, an important factor in the long term regulation of blood pressure (Cheng et al. 2005, Cassells and Haffner 2006).

It is hypothesised that alterations in the RAS may play an important role in the aetiology of hypertension and Metabolic Syndrome. It is a common clinical finding that

several components of the renin-angiotensin- system are elevated in obesity and hypertension.

All the risk factors for Metabolic Syndrome have the potential to be reversed/reduced/prevented in conjunction with a suitable diet and lifestyle intervention (Wagh and Stone 2004). A general increase in physical activity combined with a diet that produces an energy deficit (energy expended is greater than energy obtained from food) is ideal because it will lead to bodyweight loss and it has been shown that weight loss has the potential to significantly lower blood pressure, improve the lipid profile and blood glucose levels (NCEP-ATP III 2001, WHO 2003) and thus reduce the overall severity of Metabolic Syndrome (Goldstein 1992, Grundy et al. 2005, Scott 2003, Standl 2005, Kolt et al. 2007).

It is well documented that hypertension is a potent risk factor for Metabolic Syndrome and cardiovascular disease in South Asians (Kolt et al.2007). In order to reduce the burden of cardiovascular disease and hypertension in South Asians it is important to reduce/control high blood pressure. In this study, diet and lifestyle modification was used to reduce most risk factors for Metabolic Syndrome.

## **1.11 Genetic basis of Metabolic Syndrome**

### **1.11.1 A possible genetic link between Metabolic Syndrome and South Asians**

South Asians are known to have an increased prevalence of Metabolic Syndrome, cardiovascular disease and type 2 diabetes when compared to other ethnic populations. Common phenotypes of the Metabolic Syndrome include increased central adiposity, dyslipidaemia, hyperinsulinemia and hypertension. South Asians are known to have a higher probability of suffering from increased central adiposity, an imbalanced lipid profile and hyperinsulinemia (McKeigue and Marmot 1988, Zavaron 1999, Vimalaswaran et al. 2006).

The aetiology of Metabolic Syndrome involves environmental influence, genetic predisposition and the genetic interaction among various genes (Topol et al. 2006). The precise nature of the genetic factors that may contribute to the occurrence of Metabolic Syndrome is not clear. A proper understanding of the interplay between genetic factors and biochemical/environmental factors is required in order to prevent or reverse the different components of Metabolic Syndrome (Pollex and Hegele 2006). Certain candidate genes have been linked with various components of Metabolic Syndrome; genetic variants of these genes have been associated with the risk of developing Metabolic Syndrome. A discussion on these candidate genes follows later in this chapter.

### **1.11.2 Key approaches used in genetic investigation**

Two major approaches have been used in studies investigating genetic factors and Metabolic Syndrome, namely, the Genome Wide approach (Welcome Trust Consortium 2007), and the Candidate Gene association approaches (Phillips et al. 2006).

The Genome Wide approach involves rapidly scanning markers across complete sets of DNA or genomes. It can be used to investigate a large number of individuals and it makes use of numerous single nucleotide polymorphism (SNP) markers across the genome. Patterns of association between the SNP genotypes and disease status are identified and evaluated statistically (Lahiry et al. 2008). Genome-wide studies became more feasible after the completion of the Human Genome Project in 2005 and the International Hap Map Project in 2005. The primary objective of the Hap Map project was to establish the common pattern of DNA sequence variation in the human genome. The Hap Map is a useful tool in determining the sequence variants of genes that may contribute to various diseases (Song et al. 2006). Such research tools have become

readily available, making it much easier to collect/analyse genetic data. Computer databases with reference to maps of human genetic variation and human genome sequence make analysis of such data efficient and effective. Genome wide research does however have some drawbacks; it can incorrectly produce positive results (Song et al. 2006).

The Candidate Gene approach however involves the analysis of the association between selected genes with genetically unrelated subjects. Assumptions regarding specific genes are made based on the biological functions. The nature of the relationship that may exist between a particular disease and a candidate gene may be ascertained via either of two analytical methods, the first being the linkage analyses technique, an analysis technique that attempts to identify specific markers with a disease phenotype in a pedigree. The second technique is the association analysis technique; it measures the difference in allelic frequency of gene variants in subject and control groups. The Candidate Gene approach is a commonly used approach in investigating Metabolic Syndrome (Phillips et al. 2006).

### **1.11.3 Genetic investigation of single nucleotide polymorphism (SNP)**

One of the important applications of genetic investigation is to develop a better understanding of complex diseases. It has been established that Mendelian disease (e.g. cystic fibrosis) is the consequence of defect in one gene (monogenic). However it has been presumed that the majority of common disease (e.g. diabetes, hypertension,) are 'polygenic' disorders. This implies that the presence/absence of the disease is attributable to polymorphisms of multiple interacting genes. The variation observed in the phenotypes may also be associated with environmental variations and gene/environment interactions (Lahiry et al. 2008).

Nucleotides are the single structural units of DNA/RNA, namely Adenine (A), Thiamine (T), Guanine (G) and Cytosine (C). Single nucleotide polymorphisms or SNP (pronounced "snips") are DNA sequence variations that occur when a single nucleotide A (Adenine), T (Thiamine), G (Guanine) and C (Cytosine) in the genome sequence is altered. A SNP is a single nucleotide substitution with another; nucleotide both bases (the original and the substitute) are observed in the general population. For example, if one person has a particular DNA sequence of CCATT and another has a sequence of CCGTT, the polymorphism that exists would be A/G. Therefore, two alleles for this sequence exist. Almost all common SNP have two alleles. For a variation to be considered a SNP it must occur in at least 1% of the population (minor allelic frequency). These variations in the human DNA sequence can affect the ways in which a person can develop a disease. A single SNP may occupy a coding sequence, non coding sequence or intergenic region of a gene. SNP are the most common types of genetic variation, SNP is a single base paired mutation at a specific locus (Sale et al. 2006).

The human genome may differ from one individual to another by up to approximately 0.1%, accordingly, 99.9% of the human genome is identical from individual to individual. The 0.1% genome variation observed in humans consists of 80% of known SNP (Lahiry et al. 2008). Thus, on average, for every 1000 bases, one base will be different from individual to individual. The National Centre for Biotechnology Information (NCBI) has produced a subset of SNP defined as a set of non-redundant markers that are used for annotation. In reference to genome sequence, NCBI plays a major role in facilitating the identification and classification of SNPs through its creation and maintenance of the public SNP database ([dbSNP](#)). 2.6 million SNP have been assigned a rs (reference number) SNP (Wellcome Trust Consortium 2007).

### **1.12 Metabolic Syndrome and possible genetic basis**

Heritability studies (both twin and family) suggest that the prevalence of the various components of Metabolic Syndrome may have a genetic basis (Groop et al. 2000). Heritability studies (studies that investigate the relationship between a phenotypic variation and the relative genotypic and environmental factors that may influence such a phenotype) have suggested that the “thrifty genotype” theory may apply to South Asians populations. The thrifty genotype hypothesis, first proposed by Neel in 1962, suggests that certain genes favour the conservation of energy; thus, an individual with a genetic makeup that favours energy conservation will more likely suffer from the components of Metabolic Syndrome when they move from an environment where food is scarce, to an environment where it is abundantly available. An increased consumption of food (and thus greater energy intake) combined with a decrease in physical activity results in a greater propensity to suffer from type 2 diabetes and obesity and Metabolic Syndrome (Song et al. 2006). The thrifty phenotype hypothesis, first suggested by Hale and Barker in 1992, associates low birth weight with an increased propensity to suffer from the components of Metabolic Syndrome later in life. Low birth weight as a possible result of intrauterine malnutrition has been associated with Metabolic Syndrome later in life; if conditions change in adulthood, for example, if a person used to limited food in a poorer rural environment, increases their energy consumption on migration to a wealthier urban environment.

The most popular approach in investigating the genetic basis for Metabolic Syndrome has been to identify candidate genes relevant to the thrifty genotype hypothesis. Accordingly, genes involved in energy balance, adipocyte function and lipolysis, lipid metabolism and glucose regulation have been the key focus of investigations (Phillips et al. 2006).

### **1.13 Candidate genes for Metabolic Syndrome**

The candidate genes that will be discussed below have all been associated with Metabolic Syndrome. However, researchers have not isolated particular candidate genes that can be associated only with the singular components of Metabolic Syndrome; rather, various genes have been associated with multiple components of Metabolic Syndrome (Pollex and Hegele 2006). Association studies have not been completely consistent in their findings. Many studies that claim to have validated a particular hypothesis have not been replicated accurately; often results differ to the extent where the original hypothesis must be amended, or refuted entirely (Pollex and Hegele 2006). Thus, although knowledge pertaining to the occurrence of Metabolic Syndrome and the role of genetic factors in its occurrence has improved, significant gaps remain (Song et al. 2006).

The key candidate genes that have been associated with Metabolic Syndrome include: i) the Fatty Acid Binding Protein (FABP2) gene, ii) the With No Lysine Kinase (WNK-1) gene, iii) the Arachidonate 5-Lipoxygenase Activating (ALOX5AP) gene, iv) Adiponectin Q (ADIPOQ) gene, v) the Angiotensinogen (AGT) gene, vi) the fat mass and obesity (FTO) gene, and vii) the Peroxisome Proliferators activated receptor (PPAR) (Lahiry et al. 2008).

In this study, only the FABP2, WNK-1 and Coronary Artery Disease,(CAD) genetic SNPs were investigated, however, a discussion on all the major candidate genes for Metabolic Syndrome follows.

i) The FABP2 gene is involved in the transportation and metabolism of saturated and unsaturated long chain fatty acids. It codes for a fatty-acid binding protein. A defect in the transportation and metabolism of fatty acid regulation may be the basis for dyslipidaemia and insulin resistance, both major components of Metabolic Syndrome (Albala et al. 2006, Lara-Castro et al. 2005). Both dyslipidaemia and insulin resistance

are often clustered in families and individuals, suggesting a genetic influence for their aetiology.

ii) The WNK gene has been associated with hypertension. The WNK gene encodes for proteins in the WNK family of serine-threonine kinases, which are involved in renal electrolyte homeostasis, thus involved in the regulation of blood pressure (Tobin et al. 2005).

iii) The ALOX5AP gene has been closely linked to atherosclerosis and the development of cardiovascular disease (Helgadottir et al. 2005). It has been suggested that the variants of the above mentioned genes may be responsible for the high prevalence of dyslipidaemia and cardiovascular disease in South Asians (Topol et al. 2006).

As South Asians (and Pakistanis in particular) have an increased propensity to suffer from hypertension and dyslipidaemia (and thus more likely to suffer from Metabolic Syndrome) it is possible that the aforementioned genes are somewhat responsible for the increased prevalence of Metabolic Syndrome in South Asians. It was therefore important to investigate the nature of the relationship that exists between the candidate gene variants and the occurrence of Metabolic Syndrome in a Pakistani population. .

iv) The ADIPO Q gene has been associated with increased body fat and type 2 diabetes. The ADIPO Q gene encodes for the adiponectin protein; this protein is exclusively secreted by adipocytes. Adiponectin acts as antidiabetic, antiinflammatory, and antiatherogenic adipokine. (Broedl et al. 2006). ADIPOQ gene is mapped to chromosome locus 3q27; this locus is closely associated with type 2 diabetes as indicated by genome-wide scans in several ethnic groups (Pemberton et al. 2008, Yang and Chung 2006). The major role of adiponectin in lipid and glucose metabolism can be explained by the activation of cAMP-activated protein kinase and the stimulation of receptor PPAR alpha which leads to increased glucose uptake and oxidation of fatty acids in skeletal muscle, while also decreasing hepatic glucose uptake; and maintains

lipid and glucose metabolism. Decreased levels of adiponectin have been observed in sufferers of Metabolic Syndrome (Matsuzawa et al. 1999). To date two adiponectin receptors have been identified: ADIPOR1 and ADIPOR2. ADIPOR1 is expressed abundantly in muscle, whereas ADIPOR2 is expressed in the liver. Adiponectin receptors mediate the antidiabetic and anti-inflammatory effects of adiponectin. Genetic variation of ADIPOQ2 may contribute to insulin resistance and type 2 diabetes. Few common SNP variants of the ADIPOR2 gene (SNP) have been identified, and they have been associated with a decreased level of adiponectin, insulin sensitivity, increased body fat and type 2 diabetes (Yang and Chuang 2006).

v) A potential candidate underlying genetic susceptibility to Metabolic Syndrome is the FTO gene. It has been associated with obesity, increased fat mass and type 2 diabetes (Gerken et al. 2007). The FTO gene has been associated with an increased BMI in various populations, including Caucasians, South Asian and Chinese Canadians, Ojibwe and the Inuit. Studies have been successfully replicated, suggesting that the FTO gene is generally related to obesity in various populations (Gerken et al. 2007, Scuteri et al. 2007). In particular, the SNP (public data base reference number, rs9939609) has been associated with obesity and type 2 diabetes in various populations. Studies have suggested that carriers of the FTO SNP (rs9939609) have an increased risk of suffering from Metabolic Syndrome, as the FTO gene can influence the levels and central adiposity (waist circumference) (Price et al. 2008).

The FTO gene is found on the 400kb chromosomal region, 16q12.2 (Loos and Bouchard 2008).

The FTO gene encodes for protein 2 oxoglutarate-dependent nucleic acid demethylase that is present in many tissues and predominantly found in the hypothalamus (the control centre for energy regulation) (Al-Attar et al. 2008). The FTO gene influences energy homeostasis, as the nucleic acid demethylation status in the hypothalamus is

regulated by feeding and fasting states of individuals (Frayling et al. 2007), and has been linked to increased fat mass (possible link may be increased energy consumption and body weight).

vi) The Angiotensinogen (AGT) gene is a candidate gene for hypertension. The AGT gene is one of the important components of the Renin-Angiotensinogen-system (RAS). This gene plays an important role in the regulation of blood pressure and electrolytes in human. An increased level of angiotensinogen has been linked to hypertension (Alvi and Hasnain 2008).

The AGT gene is located on the long arm of chromosome 1 (1q42-43), spanning 13Kb of genomic sequence. The angiotensinogen (AGT) gene encodes the angiotensinogen converting enzyme (ACE). ACE is present in plasma and released by vascular endothelial cells (Alvi and Hasnain 2008). ACE is involved in catalysing the conversion of angiotensinogen I into angiotensinogen II, a potent vasoconstrictor, and in maintaining the blood pressure (Phillips et al. 2006). Various studies have shown elevated plasma angiotensinogen levels in hypertension. SNP of the AGT gene have been identified and associated with increased blood pressure, in particular, the commonly studied variant of DNA within exon 2- with threonine (T) instead of methionine (M) at position 235 (M235T) (Alvi and Hasnain 2008).

vii) Another candidate gene for Metabolic Syndrome is the PPAR gene; this gene has been associated with the regulatory control of lipid and glucose metabolism. The human PPAR gene is located on chromosome 22 (q11.1-13.1); it encodes for the PPAR alpha receptor. The genomic locus of the PPAR gene has been associated with type 2 diabetes, obesity and insulin sensitivity. Therefore PPAR alpha gene has been suggested as a candidate gene for type 2 diabetes and dyslipidaemia (Kraegen et al. 2002): PPAR receptors have 3 isoforms: alpha, beta and gamma. PPAR gamma receptors modulate the action of many different target genes involved in both lipid and glucose metabolism

(Kraegen et al. 2002). Generally, all isoforms have been associated with Metabolic Syndrome (Robitaille et al. 2004). Genetic variants of the PPAR gene have been identified; these variants have been associated with type 2 diabetes, HDL-C and hypertension susceptibility.

The PPAR receptors are a group of nuclear proteins that function as transcriptional factors in regulating gene expression. PPAR receptors are ligand-inducible transcriptional factors for retinoid, vitamin D and thyroid hormones. PPAR receptors regulate the expression of target genes by binding to the DNA sequence; these receptors are involved in the regulatory control of lipid and glucose metabolism. (Kraegen et al. 2002). A polymorphism has been reported in a gene encoding PPAR alpha which results in the substitution of valine (V) for leucine (L) in codon 162 in the French-Canadian population. The PPAR Alpha162L → 162V polymorphism is associated with dyslipidaemia (Kraegen et al. 2002). By the use of the candidate gene approach, the association of the genetic locus to various phenotypic traits has been replicated successfully in various populations (Robitaille et al. 2004). The above-discussed candidate genes are involved in multiple regulatory pathways. For example, the ADIPOQ, PPAR and FTO genes have been associated with insulin resistance, type 2 diabetes and increased body fat mass. No single gene has been associated with a single component of Metabolic Syndrome; rather, many genes have been associated with a few key traits. Further research is required to investigate grey areas that exist in relation to particular genes and their contribution to the components of Metabolic Syndrome. This project aimed to investigate the genetic basis for Metabolic Syndrome in a female Pakistani population, via the SNP approach to genetic analysis.

## **1.14 Behavioural risk factors for Metabolic Syndrome**

It has been shown that the conventional cardiovascular risk factors such as blood cholesterol, lipid profiles and hypertension do not fully explain the increased cardiovascular mortality in South Asians. Many studies have implicated behavioural and lifestyle-related factors, but obesity and insulin resistance are thought to be the major contributors to the cardiovascular mortality rate in South Asians, however, the overall picture remains unclear (Al-Mousa 2005, Kolt et al. 2007).

### **1.14.1 Diet as a risk factor for Metabolic Syndrome/obesity**

It is well documented that diet is an important determinant of various non-communicable diseases; the WHO have suggested that diet has the potential to greatly influence overall health (WHO 2003). Diet is a particularly prominent contributory factor to the development and occurrence of various chronic diseases. Literature suggests that an atherogenic diet, high in saturated fats and low in fibre, has a strong association with chronic diseases, such as cardiovascular disease, cancers of the digestive tract and type 2 diabetes (WHO 1998, Kamath et al.1999, Khattak et al. 2002, Anderson 2005, Lip et al 1995, Williams et al 1996). Global trends suggest that many populations have gone from a largely plant-based diet, towards energy-dense, high fat animal-based diets and this is also assumed to contribute to the increased global prevalence of obesity, Metabolic Syndrome, cardiovascular disease and type 2 diabetes (WHO 1998, WHO 2003).

In order to reduce the burden of diet-related diseases, health professionals strongly promote diet and lifestyle interventions in the general public. Cost effective diet and lifestyle intervention are an important aspect of nutritional research. Many studies have portrayed the beneficial effects of the diet and lifestyle modification/interventions;

essentially, it has been proven that such interventions are an excellent means of preventing chronic diet-related diseases (Kolt et al. 2007, Vartianen et al. 1994.)

#### **1.14.2 What type of diet causes Metabolic Syndrome/obesity?**

Generally, the diet of obese individuals and Metabolic Syndrome sufferers is high in energy, saturated fat and dietary cholesterol, while it is also low in dietary fibre and fruit and vegetables (Misra and Vikram 2004, WHO 2000). Such a diet is often defined as an atherogenic diet. Excess consumption of energy dense foods, particularly fast foods is the main dietary contributor to the occurrence and development of obesity and Metabolic Syndrome (McKeigue and Marmot 1988, McKeigue et al. 1993, WHO 2003, Misra and Vikram 2004).

#### **1.14.3 What type of diet is needed to prevent/treat Metabolic Syndrome/obesity?**

In order to prevent the onset or reduce the severity of Metabolic Syndrome and its components, dietary modification is required in conjunction with increased physical activity. Change in energy consumption (to reduce obesity), reduction in dietary salt (to reduce hypertension) and reduction in consumption of fat and fatty acids (to treat dyslipidaemia) become necessary (NCEP-ATP III. 2001, Chaung et al. 2002, Bruce 2000). An optimal diet for the prevention and/or treatment of obesity and Metabolic Syndrome is one that is largely in accordance with current WHO dietary guidelines. Current WHO guidelines suggest a diet high in fruits and vegetables and dietary fibre, and low in fat, sugar and salt (WHO 2003). (WHO 2003, Stone and Saxon 2005, Grundy et al. 2005).

## **1.15 Physical activity and sedentary behaviour as a risk factor for Metabolic**

### **Syndrome/obesity**

Non-communicable diseases, especially cardiovascular disease, obesity, Metabolic Syndrome, and type 2 diabetes are becoming a major health burden worldwide (Anderson 2005, Daskalopoulou et al. 2004). The major contributors in the epidemiology of these diseases are poor diet and physical inactivity. Physical activity is defined as bodily movements that result in energy expenditure and includes daily activities, such as housekeeping, gardening, walking or shopping. In recent years, diet and physical activity has been the subject of great attention. The World Health Organisation has responded to this alarming situation by bringing attention to prevent and control the disease (WHO 2003).

Lack of physical activity has been associated with accumulation of fat mass. Increased fat mass is linked with rise in non esterified fatty acids in blood, which is thought to lead to insulin resistance, hypertension and dyslipidaemia, all risk factors for cardiovascular disease and Metabolic Syndrome (Dhawan and Bray 1997, WHO 2003, Deepa 2004, Grundy et al. 2005) Increased physical activity has been associated with beneficial physiological effects; it can improve cholesterol, lipid profiles, insulin sensitivity and reduces fat mass ( Grundy et al. 2005). The benefits of physical activity have been shown to improve the condition of various disorders and diseases, such as obesity, cardiovascular disease, and Metabolic Syndrome and type 2 diabetes across many populations. This is particularly the case when physical activity is combined with appropriate dietary modification (Williams et al. 1993, Kamath et al. 1999, Grundy et al. 2006).

### **1.15.1 What type of physical activity is needed to treat/prevent Metabolic Syndrome/obesity?**

The World Health Organisation has recommended that moderate physical activity (such as brisk walking) is sufficient for treating conditions such as Metabolic Syndrome and obesity (WHO 2003). Current Australian and international guidelines recommend at least 30 minutes of moderate activity on a daily basis approximately 10,000 steps a day are considered adequate. However, exercises of higher intensity would confer greater health benefits (Centre for Disease, Control and Prevention 1999, AIHW 2003, WHO 2003, Grundy et al. 2006, Deedwania and Singh 2005, WHO 1998).

### **1.16 Dietary practices of South Asians**

It is difficult to establish and compare a dietary pattern amongst the various subgroups of South Asians. Data on dietary intake in South Asians living in foreign countries is limited. Many Indian Hindus are vegetarians, however many Indian Muslims, Pakistanis and Bangladeshis consume meat and poultry freely (Bhopal et al. 1999). It is therefore difficult to establish a 'South Asian' diet, Despite this, a 'typical' South Asian diet can be considered as including a high content of carbohydrates and fats (Gupta 1996, Khattak et al. 2002, Gupta and Brister 2006). According to literature, Indian populations consume high amounts of carbohydrates, and high carbohydrate intake has been reported to induce hypertriglyceridemia and hyperinsulinemia in South Asians ( Sevak et al. 1994, Misra and Vikram 2004 ).

Rapid acculturation has resulted in significant change in the dietary habits of migrant South Asians. Possibly this may contribute to a high prevalence of obesity in migrant populations (Nazroo 1997, Yancey et al. 2004, Glenday et al. 2006 Misra and Ganda 2007, Kolt et al. 2007). The implications of these dietary changes in the development of obesity and Metabolic Syndrome remains to be ascertained.

It has been reported that as the socioeconomic status of individuals improves (as may occur moving from the country of origin to the host country) so does their capacity to spend money on different types of energy dense foods. Studies have shown that an increased consumption of fat (particularly animal fat) is a key component of dietary westernisation and nutritional transition (Satia-Abouta et al. 2000, Nicolaou et al. 2006). It has been documented that a strong shift occurs in the types of foods individuals consume on migration. For example, a study on changes in dietary practices among Pakistani migrant women in Norway showed that the consumption of fat increased on migration to Norway (Mellin-Olsen and Wandel 2005).

Typically, migrants tend to eat more energy dense foods when compared to the types of foods they would have consumed in their home countries. However many studies on the Indian population have shown that on average Indians have a high intake of dietary fat and generally purchase more dietary fats and oils when compared to other populations residing in the UK. This intake of saturated fat may cause an accumulation of excess body fat and central/abdominal adiposity (McKeigue et al. 1988, McKeigue et al. 1996) it was also shown that the uneven distribution of meals and the consumption of a large amount of energy during evening meals may be a responsible for dyslipidemia. However their effect on insulin resistance and adiposity are not known (Yagalla et al. 1996).

### **1.17 Low levels of physical activity in South Asians**

Literature has consistently shown that South Asian particularly Pakistanis and Bangladeshis are less physically active when compared with Europeans (Dhawan and Bray 1997, Chowdhury 2003 , Palaniappen et al. 2007). In the Health Survey of England (Zaninotto et al. 2007), in which the levels of physical activity in ethnic groups were investigated, it was reported that the general population was most active, Indians

were the next most active, followed by Pakistanis and the least active were Bangladeshis. Gender differences were also highlighted, women being less active when compared to men. All the above findings are consistent with previous studies (Nazroo 1992, Erens, et al. 2001, Fischbacher et al. 2004, Kolt et al. 2007 ).

There are a number of possible explanations for the relative physical inactivity in South Asians. Cultural beliefs about body image are quite different in certain ethnic groups (Farooqi et al. 2000). These beliefs were found to be stronger in South Asian populations that had migrated recently and had spent less time in the host country; such beliefs were also found to be much stronger in Pakistani populations. Cultural attitudes are largely determinative of physical activity levels in South Asian populations. Generally they do not encourage participation in sport and physical activity. For example, in Selhti (the Bangladeshi language) no phrase exists to describe physical activity, instead English or words from other languages are used (Greenhalgh et al. 1998). It has been suggested that many Bangladeshis believe that exercise is not associated with health and wellness because it might exacerbate illness, and that a large body size is considered as being in a healthier state when compared to a thinner body size (Bush et al. 2001).

There are a number of reasons for low participation in organised physical activity by South Asians. Unisex environments can pose a barrier for South Asian females who wish to participate in sport and other leisure activities. Many South Asian populations avoid unisex gymnasiums or swimming pools. Some South Asians such as Hindus and Sikhs are generally culturally prohibited, whereas Muslims are also religiously prohibited. Another possible reason for physical inactivity is the fear of racism, which may prevent the South Asian community from entering recreational facilities as well as participating in activities such as running, walking and cycling in public places.

(Farooqi et al. 2000) Additionally, migrants have shown to be of a lower socioeconomic status when compared to the general public: lack of money or transport is a very common barrier to the participation in recreational activities (Hayes et al. 2002, Hamdy 2005). There is little evidence of interventions that have been successful in promoting physical activity in South Asian groups. Health promotions in minority ethnic groups are not straight forward and to be successful require a major investigation in understanding the attitudes towards physical activity, so as to guide intervention efforts. There is strong evidence to suggest that physical activity protects against cardiovascular disease and diabetes, the lower level of physical activity reported in high-risk population (South Asians) is a concern and suggests a crucial need in the area to promote physical activity in these groups. There is little evidence for interventions that have been successful in promoting physical activity in South Asian groups. Health promotions are needed in order to gain an understanding of the attitude towards physical activity which will in turn guide intervention efforts.

## **1.18 Environmental determinants of Metabolic Syndrome/obesity**

### **1.18.1 Migration and acculturation**

#### **1.18.1.1 Migration to Australia**

Political instability, wars and economic disparity throughout the centuries have caused massive movements of population between nations. Migration refers to the movement of a population between and/or within nations. Immigrants are defined as people who come to a country of which they are not native, with the intention of settling there (Al-Issa 1997). The second half of the twentieth century saw an enormous movement of migrants and refugees from non-western regions, specifically Africa, Asia, Latin America, to western countries such as America, Canada and Australia (Al-Issa 1997, Schofield 1995).

Historically, Australia's migration has always been a process that involves a significant involvement of state action and control (Schofield 1995). Since the changes made to the immigration policy in 1973, an increase of immigrants from Asia and the Middle East has occurred and, as a result, the number of English-speaking and European immigrants arriving to Australia has decreased (ABS 2001). Australia is a nation widely renowned for its cultural diversity, and a home to people with many different backgrounds. Of the approximately 90 000 immigrants that arrived in 2001-2002, 38.7% came from Asian countries, 21.5% arrived from Oceania, and 17.3% came from African nations (ABS 2001).

#### **1.18.1.2 Women and migration**

Women have migrated to Australia from diverse countries and cultures. Most migrate voluntarily, although some are refugees. The experiences of women of non-English speaking backgrounds who have migrated to Australia are very different from those of English speaking backgrounds. Some women come from environments that facilitated their easy accommodation of Australian culture, bureaucracies, and health systems. For other women, either because of their language, their culture, or their previous experience of health care systems, they find accessing conventional health services in Australia intimidating (Allotey et al. 2000).

For most migrants, migrating to a new country is indisputably a life changing experience. For some women the burden of migration will be even greater than for their male companions. This may be particularly applicable in cases in which the women come from societies with highly specified gender roles, where women are traditionally house-bound, such as South Asians. Upon arrival in Australia the women may discover that they are required to work outside the home in order to support the family's economic well-being, and generally engage with the local community in a manner that

would traditionally be considered inappropriate. Families are disrupted when women are impeded from supplying their usual care, household conflicts occur when women define new roles for themselves in host countries ( Plunkett and Quine 1996, Shahwan-akil 2001, Bhugra 2003).

Women migrating from liberal societies, who may have been successful professionals in their own right prior to migration, may find that this is quite the contrary. They are now more house-bound than before, unable to access ample family support services, and are working in unskilled jobs that are of lower status and more poorly paid to which they had become accustomed (Carballo and Mboupm 2005). The effects of migration experiences on men and women have been shown in literature; it is evident that women appear to suffer more hardship than men (Hull 1979, Kasl and Berkman 1983, Bhugra 2004 ) because women in certain cultures have an important role in raising a family in isolation without the traditional support of the extended family. This lack of support will lead most of the women into some form of sub-clinical depression (Schofield 1995). Typically, such depression is not admitted nor recognized by these women. These women also experience difficulties with access to the health system consequent to language barriers and cultural issues in women's health ( Bhugra 2003).

Due to strict health requirements for entry into Australia, often migrant women initially have higher health status and lower mortality than non-migrants ( Shahwan-Akil 2001, Gushulak et al. 2006). Within a few years, however, their overall health status may be lower, due to various factors, such as social isolation, low socio-economic status, and sub-clinical depression. Migrant women may also experience stress, anxiety and/or guilt resulting from the fact that they have left their home country and relatives alike. Settlement issues may also lead to depression and anxiety (Bhugra 2004). Fear may continue to dominate their lives and subsequently they may lose confidence. One distressing factor many female immigrants face is the inability to sponsor remaining

relatives in their home countries, to allow them to visit and offer support. Many women report a persistent sense of isolation and helplessness. Upon arriving here they feel isolated from their own support group of relatives as they face the barrier of undertaking a new, common, lower status occupation such as low-income unpleasant factory or cleaning jobs (Mehta 1998, Shahwan-Akil 2001, Bhugra 2005). They may come home from a full-time job to face another, which is caring for children and maintaining a stable internal environment. Their stress will be exacerbated if their husbands restrict their social activity and impede their efforts to further their knowledge of English. A woman's children may reject her traditional role as the central figure in the home and teacher of homeland culture. A lack of knowledge about Australian facilities and services, or mistaken information, compounds these problems (Shahwan-Awkl 2001)

### **1.19 Acculturation**

Migration is the term used to describe the physical relocation between countries. Acculturation is defined as the social, psychological, and behavioural changes that occur following this relocation (migration) (Al-Issa 1997, Gushulak and McPherson 2006). As a person moves from one country to another, the process of acculturation occurs concurrently. Acculturation is a dynamic process and occurs at two distinct levels. At the individual level, acculturation is the changes in attitudes, beliefs, behaviours and values in the individual consequent to life in the new host society. The second level of acculturation occurs at the group or community level and is represented by changes in physical, biological, political, economic and cultural aspects in the individual (Satia-Abouta et al. 2002). Attitudes towards health are greatly influenced by the culture in which an individual resides (Allotey et al. 2002). Thus, the process of acculturation affects a person's cultural views, and therefore alters how they perceive health and their attitudes towards seeking health-helping behaviours. Acculturation

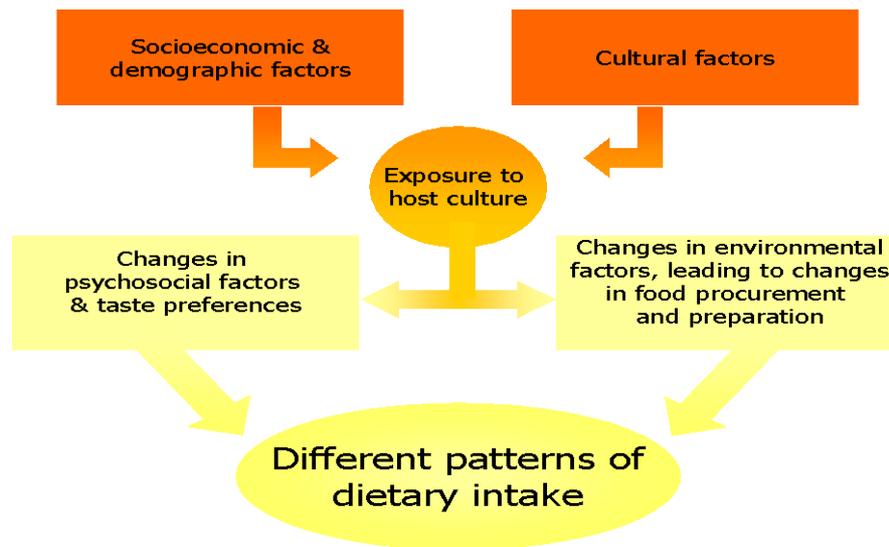
provides an opportunity to educate and define the behaviours that modify or replace those that an individual has become accustomed to in their country of origin. The behaviours formed during acculturation have two major influences, they may have the ability to promote good health, but if they are neglected or poorly managed, they may decrease general well-being and increase the risk of developing chronic and lifestyle-related diseases (Palinkas et al. 1995, Satia-Abouta et al. 2002). With regards to immigrant health, two major issues have been recognized: physical activity and nutrition (Khunti and Samani 2004). These lifestyle factors in turn predispose immigrants to obesity and chronic disease (Williams et al. 1993) such that with as their length of stay in the host country extends the patterns of obesity and chronic disease of immigrants mirrors that of their hosts (Kolt et al. 2007).

#### **1.19.1. Dietary acculturation**

One of the fundamental adjustments directly linked with acculturation is changes in the dietary pattern of ethnic minority groups. This new dietary pattern may result in a change of nutrition status, and impact their present health status. As immigrants undergo the process of acculturation the change in dietary pattern can be very dramatic, which may result in a significant increase in the risk of developing nutrition-related diseases (Satia-Abouta et al. 2002). Two major issues have been recognized as paramount to migrant health: physical activity and nutrition (Khunti and Samani 2004). Studies have shown that an increased consumption of fat (particularly animal fat) is a key component of dietary westernisation and nutritional transition (Satia-Abouta et al. 2000). It has been documented that a strong shift occurs in the types of foods individuals consume on migration. Typically, migrants tend to eat more energy dense foods when compared to the types of foods they would have consumed in their home countries. It has been noted that as the socioeconomic status of individuals improves

(moving from the country of origin to the host country), so does their capacity to spend money on different types of energy dense and unnecessary foods (Popkin 2001). Rapid acculturation has resulted in significant change in the dietary habits of migrant South Asians. The implications of these dietary changes in the development of Metabolic Syndrome and obesity remains to be ascertained. Possibly, they may contribute to a higher rate of obesity in migrant populations ( Misra and Vikram 2004, Yancey et al. 2004).

Dietary acculturation has numerous dimensions (as represented in Figure 1.2) and produces a dietary continuum starting from not at all acculturated and only using traditional foods available, to fully/complete acculturation without the use of any traditional foods or preparation/cooking techniques. Research suggests that part of the acculturation process involves finding new ways to utilise traditional foods, while selectively excluding various traditional foods and consuming new host culture foods (Satia-Abouta et al. 2002, Wahlqvist 2002). Socio-economic and demographic factors include gender, age, age at immigration, years in the host country, education, income, employment, household composition, fluency with host language, etc. Examples of cultural factors include religious beliefs, cultural beliefs, attitudes, and values. When these factors are influenced by the host culture they will influence the extent to which an immigrant will change their attitudes and beliefs about food, taste preferences, food purchasing and food preparation (Satia-Abouta et al. 2002).



**Figure 1.2** Proposed model depicting the process of dietary acculturation (Satia-Abouta et al. 2002).

Following exposure to the host culture, some changes may arise in psychosocial factors including diet and disease-related knowledge, the perceived value of traditional eating patterns and taste preferences. Differences between environmental factors lead to altered food procurement and preparation, as does the availability, accessibility, and affordability of traditional foods when shopping or eating out. Also, the influence of advertising, convenience, and time constraints often result in excess consumption of packaged foods and eating at fast food restaurants (Satia-Abouta et al. 2002). These factors result in different patterns of dietary intake through influencing food choices and food preparation. Three different patterns of dietary intake that occur as a result of dietary acculturation have been suggested: (i) maintenance of traditional eating patterns, (ii) complete adoption of host country eating patterns, and (iii) bicultural eating style, incorporating host country eating patterns at some meals, while maintaining traditional eating patterns at other meals (Satia-Abouta et al. 2002). Many studies have shown that it is bicultural eating that occurs most regularly e.g. Asian immigrants will retain rice as a staple and will cook traditional Asian meals when possible, but often replace

traditional foods with cereals, sandwiches, and milk when there is insufficient time (Wahlqvist 2002, Satia-Abouta et al. 2002). Members of an emigrating group take time to adjust during acculturation, and this in turn creates stress. Younger individuals will normally acculturate more quickly than older individuals. This can lead to a sense of isolation in older members as the young become socialised into the new culture much more quickly (Palinkas and Pickwell 1995). Acculturation may take more than one generation to occur. In a cross-cultural study, it was observed that culture influenced what was included and excluded as part of daily life across two and three generations (Green and Kreuter 1999).

#### **1.19.1.1 Dietary acculturation in South Asians**

A major part of research throughout the last decade in the UK has been based on the health of ethnic minorities. Some of the ethnic minorities that have been studied include Afro-Caribbean, West-African and populations from South Asia, most of this research has focused on cardiovascular disease and mental health (Donavan 1984). Very few studies have focused on South Asians (particularly Pakistanis) and their transition into the host culture (Donavan 1984, Zaininotto et al. 2007, Wahlqvist 2002).

The most recent study (2000-2001) that recruited Pakistanis for research purposes in this aspect was The National Public Health Survey of Oslo. Data from this survey showed that more than 70% of adult Pakistanis were obese (with a BMI greater than 25). The presence of diabetes was found to be 21% in men and 36% in women (Mellin-Olsen and Wandel 2005). According to Jafar et al. 2004 in the report on The National Health Survey of Pakistan 2000, the prevalence of type 2 diabetes in men and women residing in Punjab (a province in Pakistan) was 5% and 7% respectively, suggesting that migrated Pakistani population in Oslo, has considerably increased its prevalence type 2 diabetes. Key changes include an increased consumption of energy dense foods and

decreased physical activity, which results in an increased body fat percentage and obesity related complications. Such changes have been observed in migrant South Asian populations (Williams et al. 1993, Mohan et al. 2003, Zaninotto et al. 2007 ).

### **1.20 Changes in patterns of physical activity with acculturation**

A systematic review of studies investigating physical activity level in South Asian migrants residing in the UK revealed that all South Asian populations investigated could be classified as physically dormant or inactive (Dhawan 1997, Fischbacher et al. 2004, Barnett et al. 2006). It has been suggested that migration to more affluent societies will often result in a reduction in physical activity, combined with an increase in energy intake (Mellin-Olsen and Wandel 2005, Zaninotto 2007 ). There are many suggested reasons for the reduction of physical activity levels in migrant populations, key contributory factors include (but are not limited to): shift in socio-economic status (primarily income), education levels (limited education) and cultural barriers (Fischbacher et al. 2004). For example, most participants would have travelled short distances on foot, for the purposes of obtaining groceries and performing daily chores, on migration however, most participants gained access to vehicles (due to an increase in income) and thus physical activity was significantly reduced.

### **1.21 Physical and social impact of migration and acculturation**

A recent study was performed by Bhattacharyay and Schoppelrey 2004 in the USA to explore the links between pre- and post- immigration beliefs and notions of life success. (Bhattacharyay and Schoppelrey 2004). How these beliefs influence the acculturated stress among these immigrants was also explored. All subjects emigrated from non-English speaking countries such as India, Pakistan, Bangladesh to the USA. Discrepancies between the parents' anticipated life success in USA and actual

experiences after immigration were associated with expectation of their children fulfilling parents' 'own' dreams of success. Research suggests that this characteristic is typical of Asian culture where fulfilling parents' expectations were the responsibility of children in enhancing family pride (Bhattacharyay and Schoppelrey 2004). The role of education as a way to advance through social class and caste system was investigated. It was also observed that potential negative aspects of unrealistic expectations from children proved that parents were passing on the acculturation stress to the next generation as well (Bhattacharyay and Schoppelrey 2004).

Immigrants and ethnic minorities experience stressors common to all members of society i.e., daily stressful life, etc; they also experience stressors unique to them because of their migration. All these stressors are related to their cultural background and their experiences within their new host country. Minority groups also experience low socio-economic status within the general population, as they tend to be over represented among poor (Al-Issa 1997). Acculturation brings about issues related to identity and intergenerational conflicts. In addition to this immigrants go through racial abuse and discrimination from a minority of residents in the host country (Bhugra 2004, Hull 1979, Kasl and Berkman 1983).

Socio-economic status is determined by occupation. More migrants from non-English speaking backgrounds work in lower status occupations than other migrants from English speaking countries. Many of them are in lowest salary (earning minimum wage) and working in the hardest and most dangerous area of the labour market: this exposes them to health risks, such as industrial accidents that can cause long term disability and illness. These migrants have the lowest income and the highest incidence of poverty and work-related injuries (Shahwan-Akil 2001, Lin et al. 1990). According to William et al. 1993, socioeconomic circumstances such as low income and long working hours have a great impact on the health of migrants in the UK. In this study it was suggested that

British Punjabis lived in low socioeconomic circumstances and thus suffered more stress-associated problems than the general population. It was also suggested that stress and socioeconomic deprivation seem to be major contributors of the high incidence of cardiovascular disease (Williams et al.1993). It is submitted that improving the English skills of migrants is an essential public health policy and framework consideration, as ultimately, the language barrier has a strong impact on migrant health understanding.

### **1.22 Encouraging a healthy lifestyle in migrant populations**

It is well documented that migrant South Asians residing in Western countries such as the UK, USA, Australia, New Zealand and Canada constitute a high risk population, in that they are more likely to suffer from various chronic diseases (Metabolic Syndrome, cardiovascular disease, Type 2 diabetes when compared to the host population ( Kolt et al.2007).

Migrant health issues give strength to the idea that tailor-made diet and lifestyle approaches are required in order to reduce the severity of chronic diseases. Weight gain has been shown to be the strongest predictor of Metabolic Syndrome; therefore, weight control is important in preventing the onset or reducing the severity of the syndrome (Gupta and Brister 2006). It has been shown that even a minimal weight reduction of 5-10% of an individual's body weight via an improved diet and increased physical activity may reduce the overall risk of the disorder (Bosello and Zamboni 2000, Despres et al. 2001 , Case et al. 2002, Caterson et al. 1997, Tillin et al. 2005). The treatment of the disorder must be a multi-faceted process, that is, a process with a primary focus on therapeutic lifestyle change (Scott 2003, Stone and Saxon 2005 , Grundy et al. 2006).

In order to design such an approach, it is crucial to understand the cultural and social aspects in the target population, it is also important to pay attention to the gradual changes in the behaviour of the population, due to acculturation. Investigations on diet

and acculturation reveals a co-relation in the level of acculturation and changes in dietary practises, that is, as migrants are more exposed to the host environment, they tend to adapt the new culture and diet (albeit slowly) (Satia-Abouta et al. 2002, Raj et al. 1999).

### **1.23 Conclusion**

Metabolic Syndrome is defined as the clustering of various risk factors for cardiovascular disease and type 2 diabetes. Metabolic Syndrome is a potent prognostic factor for cardiovascular disease, as it has been shown that sufferers of Metabolic Syndrome have a higher risk of developing cardiovascular disease when compared to non-sufferers. The components of Metabolic Syndrome may be reversed, reduced or prevented via an appropriate diet and lifestyle intervention strategy. Possible treatment strategies need to focus and target the key abnormalities that are associated with the Metabolic Syndrome. It has been documented that ethnicity is a major determinant of the presence and severity of Metabolic Syndrome. There is a general lack of data on Pakistani populations; however it seems that the major contributing factors to the high prevalence of Metabolic Syndrome and cardiovascular disease in South Asians are abdominal/central obesity and insulin resistance. Metabolic Syndrome contributes substantially to the high prevalence of cardiovascular disease in South Asians. To date, no intervention has specifically targeted Metabolic Syndrome in Pakistani migrants. This study aims to provide information on the prevalence of Metabolic Syndrome and its treatment in female immigrants from Pakistan.

This study will thus apply the principles of cultural competence to overcome the various cultural barriers identified in literature and sustain the behavioural change needed to treat Metabolic Syndrome in Pakistani women. Please refer to Section 1.24 for details on the broad objectives of this study.

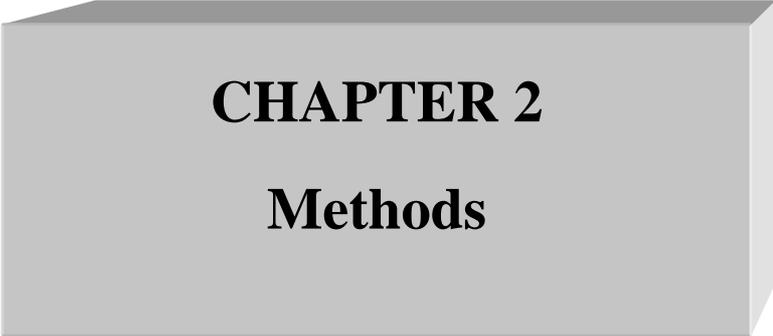
## **1.24 Broad Objectives of the Study**

The aims of the study are as follows:

1. What are the metabolic characteristics of Pakistani women residing in Melbourne displaying the risk factors of Metabolic Syndrome?
2. Do genetic markers that have been associated with Metabolic Syndrome exist in migrant Pakistani females?
3. What are predisposing, reinforcing and engaging determinants of changes in diet and physical activity in migrant Pakistani females?
4. Is a culturally appropriate diet and lifestyle intervention an effective mechanism for preventing the onset, or reducing the severity of Metabolic Syndrome in migrant Pakistani females?

## **1.25 Research questions of the Study**

- 1: What are the metabolic characteristics of Pakistani women residing in Melbourne displaying the risk factors of Metabolic Syndrome?
- 2: Do genetic markers that have been associated with Metabolic Syndrome exist in migrant Pakistani females?
- 3: Is a culturally appropriate diet and lifestyle intervention an effective mechanism for preventing the onset, or reducing the severity of Metabolic Syndrome in migrant Pakistani females?



**CHAPTER 2**

**Methods**

## **2. METHODS**

### **2.0 Human ethics**

Victoria University Human Ethics research approval was obtained for the following projects used in this study:

- (i) A culturally appropriate diet and lifestyle intervention can successfully treat the components of Metabolic Syndrome in female Pakistani immigrants residing in Melbourne, Australia. This project was approved by the Human Ethics Committee of Victoria University (HRETH.SET 04/03) (Appendix A-1, page 208)
- (ii) Genetic investigation on the association between genetic variation and risk factors for Metabolic Syndrome in a sample of Pakistani female migrants residing in Melbourne was approved by the Human Ethics Committee of Victoria University (HRETH.SET 07/222) (Appendix A-2 , page209)

### **2.1 Sampling strategy**

Sampling subjects from ethnic communities is notoriously difficult. Importantly, accessing South Asian communities has proven to be difficult and thus is a research barrier (Rankin and Bhopal 2001). It has been difficult to access these communities in the UK and Canada and achieve a proper sample size, to determine appropriate sampling and recruitment strategies (Chaturverdi and McKeigue 1994, Glenday et al 2006). Due to the aforementioned difficulties, standard random sampling techniques are not feasible, as they are time consuming, expensive, have no statistically advantageous aspect when dealing with small numbers and therefore cannot be employed with greater success than more inexpensive, efficient techniques (Small et al. 1999). Many researchers have therefore utilised non-random sampling strategies whilst studying ethnic communities. One method is the snowball sampling technique, which involved contacting individuals from within the ethnic community, including its members,

leaders and organisations. The snow balling technique is particularly effective in geographical areas where high concentrations of particular ethnic groups are easily identifiable (Plunket and Quine 1996).

## **2.2 Recruitment of subjects**

In the pilot study 17 participants were selected for participation, of which 4 participants dropped out, thus data in the pilot study was derived from 13 participants. In the major study, 75 participants were originally selected, of which 15 dropped out, thus data presented in the major study was derived from 60 participants. Note, for the purposes of our published article in the *Metabolism Journal*, data for only 40 subjects is presented as 20 further subjects were recruited after the submission the manuscript (received 10 October 2007) A convenience rather than a representative sample was obtained. It is well documented that recruitment of research subjects from ethnic minorities is difficult. Difficulties include achieving inadequate sample size, obtaining relevant population denominators, and determining appropriate sampling and recruitment strategies. Because of these difficulties, standard random sampling techniques are time consuming and too expensive to be used successfully. Therefore, in this study, a convenience sample of women meeting the inclusion criteria was used. Subjects were recruited using non-random strategies; the study was advertised through local and community papers, and snowballing techniques were specifically targeted in geographic areas known to have high concentrations of Pakistani migrants[

Participants must have met the inclusion criteria (refer to section 2.2.2). The attrition rate prior to the commencement of the study was 20% (reasons for attrition included family commitments, relocating overseas and health issues such as pregnancies.)

In this study several recruitment strategies were utilised; this study was advertised through both local and community newspapers. Snow balling techniques were

specifically targeted at geographic areas consisting of high concentrations of the Pakistani community. These suburbs were Coburg, Dandenong, Doncaster and St Albans (ABS, Australia 2001-b).

Potential participants were contacted and invited to participate in the proposed study. All necessary information was translated and conveyed to the participants in their own native language (Urdu). Participants were also given a participant explanatory statement. This ensured that participants had the opportunity to read through and be made aware of the procedures involved in the study. Volunteers were informed that this study had been approved by the Ethics Committee, and as a requirement of approval, each volunteer was required to sign the consent form (which is available in their own language). After signing the consent form, they were able to participate in the study.

A directory consisting of the names and contact numbers of Pakistani community members was obtained with the help of Pakistani community leaders, community members and religious leaders. More than two hundred families were contacted via telephone and the respondent was invited to participate in the study. The researcher herself, who is of Pakistani origin, briefly explained in Urdu the aims, participation protocol and the required tests involved in the study. All the potential participants were invited to attend an information session, which lasted just under four hours. During this information session the potential participants were given an opportunity to ask questions. They were also given an information pack which included all relevant documentation on the research project, screening tool (Appendix A-3, page 210) detailed questionnaire (Appendix A-4, 214) food frequency questionnaire (Appendix A-5, page 221) an explanatory statement (Appendix A-6, page 224), consent form (Appendix A-7, page 225), revocation form (Appendix A-8, page 226), and dietary modification modules (Appendix B1-B12, page 250-319).

### **2.2.1 Confidentiality**

All information obtained by participants was coded, each participant was randomly assigned with a unique identity number, after which point, the only association between themselves and their data was the allocated unique number. The confidentiality of the data collected had been highlighted to each of the subjects. It had also been guaranteed that during the process of data collection all information would be safe with the researcher and all the results would be reported as group data, and that no individual could in any way be identified.

### **2.2.2 Inclusion criteria**

The inclusion criteria were as follows: all candidates must have been females who were born in Pakistan, were aged between 20-60 years old, were permanently residing in Melbourne and had migrated to Australia no less than five years ago. Additionally all subjects must have presented with at least one component of the Metabolic Syndrome criteria (according to NCEP-ATP-III 2001), namely, elevated blood pressure, elevated blood glucose levels, obesity and increased waist circumference. After screening and selection participants were required to complete a questionnaire (Appendix A-4, page 214) which assessed clinical history, dietary habits and physical activity.

### **2.2.3 Exclusion Criteria**

Volunteers who had arrived in Australia within the last 3-9 months were excluded. Pregnant volunteers were also excluded. Volunteers who did not reside in Melbourne permanently were also excluded (for example, volunteers who lived in both Melbourne and Sydney intermittently.)

### **2.2.3 Data collection tools**

In this study several data collection tools were used namely: general diet and lifestyle questionnaire, physical measurements (Appendix A-3/A-4, page 210-214) and food frequency questionnaire. (Appendix A-5, page 221). All materials were translated from English into the participants' native language, Urdu.

Information regarding participants' diet, health, lifestyle and demographics was obtained by a questionnaire completed by the participants. The questionnaire used in this study was adopted from the National Heart Foundation Risk Factor Prevalence study which was conducted in 1989 in collaboration with the Commonwealth Department of Community Services and Health (NHFRFPS 1989). The questionnaire has been used several times by the National Heart Foundation in order to assess cardiovascular risk factors in Australian participants.

Physical measurements (anthropometric) were taken pre- and post-intervention for the purposes of calculating BMI and establishing waist circumference values. Biochemical measurements and blood pressure values were also taken. The protocols for all these parameters are explained in the next section. Participants were also required to complete food frequency questionnaires for the purposes of analysing participant dietary habits pre- and post-intervention.

#### **2.2.3.1 Validity of data collection tools**

The questionnaire used in this study had previously been validated for its reliability on a Greek population in cardiovascular risk factors study. This questionnaire was also used on the Iranian population residing in Melbourne in a cardiovascular disease study (Shahwan-Akil 2001).

In order to test the validity of the questionnaire, a pilot study was first conducted to ascertain whether the concepts contained in the questionnaire were interpreted correctly

by a non-English speaking population; it was established that the translated documents were indeed suitable for the target population, as all participants informed us that they had no difficulty in understanding the questionnaire.

### **2.3 Screening tool**

All potential participants were given a screening tool (brief questionnaire, Appendix A-3, page 210). This questionnaire was used to obtain personal details and information on health conditions present in the target group. Thus questions were primarily based on medical history and demographics. After the potential subjects were screened and selected for participation, they were required to complete a detailed questionnaire that addressed behavioural aspects, such as physical activity, clinical history (Appendix A-4, page 214). If assistance was required the researcher was available to provide help and clarification of any questions/concerns.

### **2.4 Food frequency questionnaire analysis**

Food frequency questionnaires (FFQ, Appendix A-5,221), are widely accepted as an appropriate tool in epidemiological studies where diet is assessed as a risk factor in the aetiology of chronic diseases (Kapil et al. 2004). A few studies have developed and implemented food frequency questionnaires in South Asian populations as a reliable assessor of diet. No 'typical' South Asian diet exists, however, foods were selected and categorised based on the findings of other studies on migrant South Asian dietary pattern (Kassam-Khamis et al. 2000, Kapil et al. 2004). The majority of these studies generally stated that South Asians consume more energy dense foods on migration, as well as traditional energy dense foods such as breads (parathas, naan etc) and fried snacks (samosas and pakoras).

For the purposes of this study, the FFQ was developed based on FFQs developed for migrant South Asian women in the UK (Kassam-Khamis et al. 1999). Subjects were provided with an extensive list of food items, including foods that are commonly consumed by South Asians and were required to stipulate how often they consumed each food specified therein (Sevak 2004, Kapil et al. 2004). Data obtained via the FFQ was categorical and was collected at the commencement and completion of the intervention for comparative purposes. The FFQ aimed to establish the eating habits of subjects, by analysing the frequency at which various foods were consumed. Frequency was measured according to a scale (-3 to +3) where a rating of -3 constituted “Never, or hardly ever”, whereas the opposite rating of +3 was representative of “Often.” Category codes were allocated to various food items. Data collected at the completion of the intervention was then compared to the data collected prior to the intervention in order to establish if any notable changes in dietary habits had occurred. The chi-square statistical test was utilised for the comparison of both sets of data, as this test is useful in determining differences in percentages or ratio for categorical data (Kapil et al. 2004).

## **2.5 Physical activity measurement**

In this study data pertaining to physical activity was collected in the form of a daily ‘step count’; there was no other estimation of our subjects’ physical activity. Pedometers are widely accepted as a valid means of assessing physical activity levels in research studies (Tudor-Locke 2004). Importantly, pedometers have previously been used successfully in the measurement of physical activity levels in female populations in various studies (Lofgren et al. 2004, Kolt et al. 2007, Rosenson and Reasner 2004). The physical activity of all the subjects was measured by the use of a pedometer (OMRON HJ-005). All subjects were given a pedometer to keep for use during the

study and afterwards. All pedometers were calibrated by the researcher, by taking 100 test steps to check that the pedometer registers the correct number of steps.

In order to collect baseline data of the volunteer's physical activity, they had been asked to note down their daily step count. All the subjects were instructed to place the pedometer on to the front of their waist in line with the hip joint, so that the pedometer can record movement. The participants were instructed to attach the pedometer unit to either their belts or to the top of their pants. The unit was required to be horizontal to the ground in order to function properly.

Baseline data on the subjects' physical activity levels was collected every day (subjects only removed the pedometers for water activities, such as having a shower) for five consecutive days and an average of the steps per day were calculated. At the commencement of the intervention program, the subjects were requested to take a minimum of 10,000 steps daily (which equates to approximately 30-40 minutes of brisk walking) six days a week, as part of the diet and lifestyle intervention. As recommended by the World Health Organisation 1999, all steps were taken at one allocated exercise time, i.e. they were not accumulative steps achieved throughout the course of the day (WHO 1998, WHO 1999).

## **2.6 Study design**

The basic study design was a 12 week control period no intervention, followed by 12 week diet and exercise intervention.

Baseline data for this study was collected at week 0. Anthropometric, blood pressure measurements and blood samples were obtained. Information on the physical activity levels and medical history of the participants was also obtained through a questionnaire in Urdu. The above parameters were also remeasured at week 12, before the commencement of the planned intervention. The diet and lifestyle intervention program

was implemented for 12 weeks, after which all the above parameters were remeasured (week 24) refer to Figure 2.1 (time-line).

### **2.6.1 Parameters measured**

The following parameters were measured prior to the commencement and at the conclusion of the study, anthropometric measurements, medical history, physical activity, food frequency, blood pressure and blood biochemical analysis.

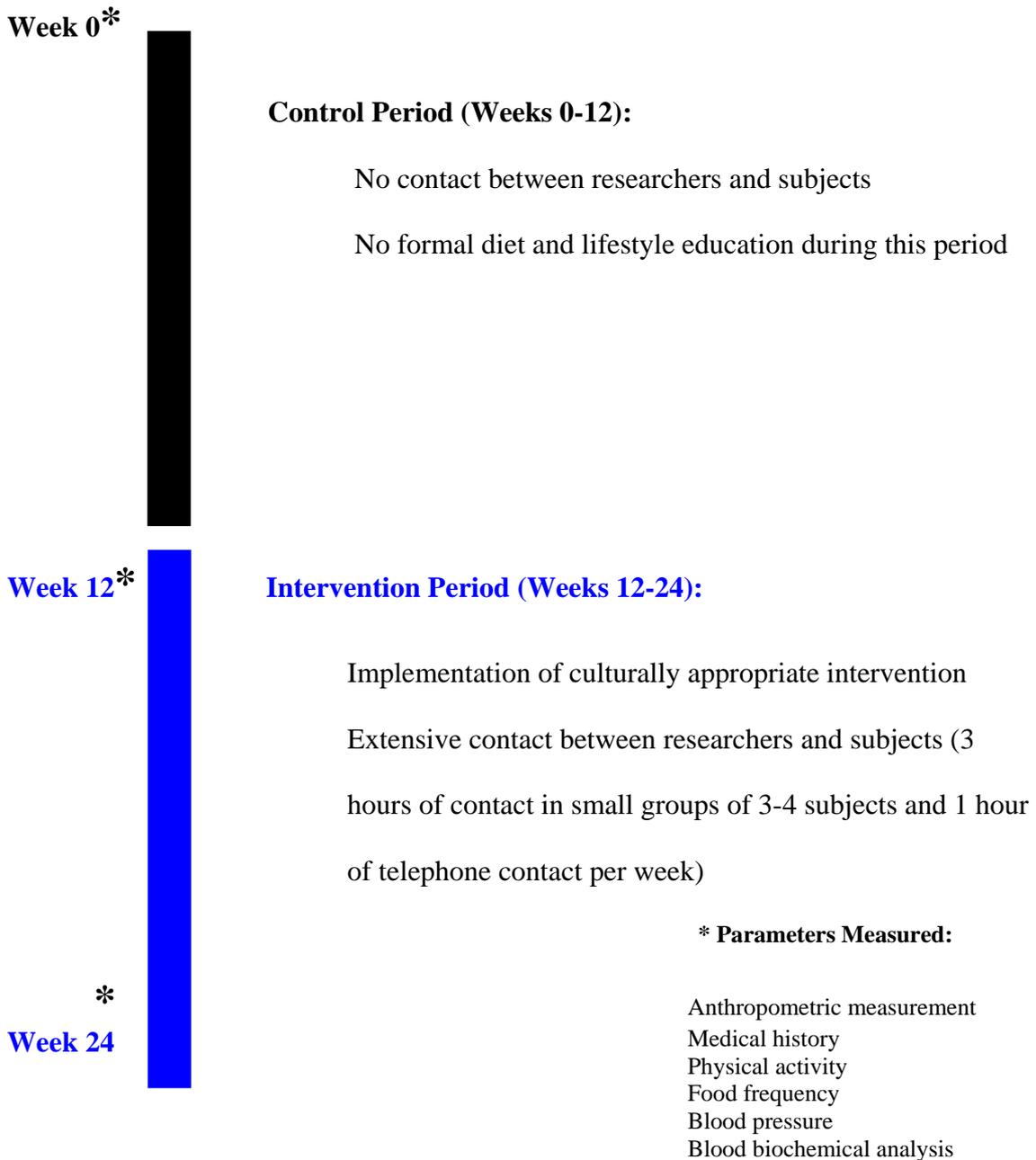
#### **2.6.1.1 Control period (Weeks 0-12)**

The study design included pre intervention (control period Weeks 0-12), and post intervention (Week 24) measurement of outcomes. Therefore baseline data was collected at week 0. The duration of the control period was 12 weeks. No formal contact occurred between the researchers or participants (i.e. no diet or lifestyle education was provided, however participants could contact the researchers with questions or enquiries) during the control period. All parameters were remeasured at week 12, prior to the commencement of the planned intervention.

### 2.6.1.2 Intervention period (Weeks 12-24)

The diet and lifestyle intervention program was implemented for 12 weeks, after which all the above parameters were remeasured (week 24).

#### TIMELINE FOR DIET AND LIFESTYLE INTERVENTION



**Figure 2.1** All subjects (n=60) commenced at week 0 and progressed to week 12 (the control period.) From weeks 12 to 24 (the end of the intervention) subjects participated in the culturally appropriate diet and lifestyle intervention.

## **2.7 Anthropometric measurements**

Anthropometric measurements were taken according to standard techniques and equipment (International Study of Obesity WHO 1998). Measurements were taken with the subjects in light weight clothing and without shoes. Measurements were taken twice and the average was used as the final reading.

BMI was calculated by the use of the formula:

$$\text{BMI} = \text{weight (kg)} / [\text{height (m)}]^2$$

A BMI of 25 or greater was considered as an obese BMI; BMI values were classified based on World Health Organisations Asian specific guidelines. Asian guidelines classify a person as obese or overweight at a lower BMI when compared to the general BMI guidelines as Asians tend to carry more body fat at a lower weight when compared to most other populations (refer to section 1.4) (WHO/IASO/IOTF 2000).

### **2.7.1 Height measurement**

Height was measured in centimetres to the nearest 0.5cm on a portable stadiometer, in accordance with WHO standards (WHO 2000). Subjects stood with their back against the wall, looking straight ahead, so that the ear passage formed a horizontal line with the lower eyelid. A wooden ruler was used as a horizontal bar on the head to compress hair.

### **2.7.2 Weight measurement**

A single portable digital scale (Omron Digital Body Weight Scale-HN-283, maximum capacity 150kg) was used to measure the weight. The scale was placed on a level surface, such as a kitchen floor. Weight was determined to the nearest 0.1kg. Care was taken to ensure the scale was calibrated at the beginning of each session (WHO 2000).

### **2.7.3 Waist measurement**

The waists of all subjects were measured with non-stretchable measuring tape. The waist was measured as the circumference between the iliac crest and the lowest rib. Waist measurement was taken in cm, to the nearest 0.5 cm (WHO 2000).

### **2.7.4 Blood pressure measurement (BP)**

The blood pressure (BP) of all subjects was measured using an automated digital BP monitor (Omron T5, Omron Japan). All subjects were seated comfortably with one of their arms (preferably the right) resting on their kitchen table. The subject's arm was adjusted in a way that the cubital fossa of the forearm was aligned with the heart. Once the position was finalised the subject was rested for five minutes prior to the taking of blood pressure measurements. Readings were taken twice (ten minutes apart) and the average of the two measurements was recorded. Hypertension was defined as "the presence of a systolic blood pressure of 135mm/Hg or greater and a diastolic blood pressure of 85mm/Hg or above, or the use of hypertensive medication" (NCEP-ATP-III 2001).

### **2.7.5 Blood collection**

Blood collection took place at approximately 9-9:30 am on planned days. All subjects were requested to fast 12 hours prior to the collection of blood. All subjects were reminded about the requisite fasting period one day before the scheduled blood collection date. The normal venipuncture technique was used, and the antecubital vein was punctured to draw blood. Subjects were instructed to sit in a comfortable position. Subject's forearms were supported by a cushion, in order to ensure that the arm was in a downward position. A tourniquet was applied above the elbow, in order to locate the antecubital vein. 10 mls of blood was drawn via the needle and syringe method and

transferred into 2 different tubes through a blood transfer device. Blood was collected in serum separator (for serum determinations) and fluoride oxalate (for plasma determinations) tubes (Becton. Dickson). These tubes were stored on ice until centrifugation.

At the end of blood collection sessions, samples were transported to research laboratories at the St Albans campus of Victoria University. All samples were transported within two hours of collection. Upon arrival at the research laboratory, blood samples were centrifuged at 3000 revolutions per minute (Beckman Avanti TM 30), for ten minutes and plasma, buffy coat, and serum were separated. Serum was then distributed into aliquots and frozen at -80°C, for determination of total cholesterol (HDL-C, LDL-C, and triglycerides). Similarly, plasma was distributed into aliquots and kept frozen at -80°C for determination of plasma insulin and glucose. Prior to use all serum and plasma samples were thawed at room temperature and appropriate aliquots were collected into Eppendorf tubes which were then mixed using a Vortex and then stored on ice.

## **2.8 Biochemical analysis, plasma lipid profile: total serum cholesterol, HDL cholesterol and plasma triglyceride determination**

The in vitro determinations of total serum cholesterol levels were quantified using a colorimetric cholesterol reagent assay kit (Thermo Electron Corporation). The in vitro determination of triglycerides was performed on serum by the use of a liquid stable reagent kit (Thermo Electron Corporation). The in vitro determination of HDL-cholesterol was performed on serum by the use of HDL-cholesterol automated reagent assay kit (Thermo Electron Corporation). All the above assays were performed according to manufacturer's instructions.

The LDL-C levels were determined by the Friedewald, Levy and Fredrickson formula: (Friedwald et al. 1972):

$$\text{LDL-C} = \frac{\text{Total Cholesterol} - \text{HDL-C} - \text{Triglycerides}}{2.2}$$

2.2

The estimation of LDL-C using this formula provides a reliable fast and inexpensive method of measuring the major lipoprotein in the blood. This is widely used in clinical practice and epidemiological studies (Al-Mousa 2005). Since LDL cholesterol is expressed in mg/dL cholesterol values were converted in mmol/L using the following conversion factor:

$$\text{Unit Conversion} - \text{mmol/L} \times 38.7 = \text{mg/dL}$$

In vitro determination of plasma glucose was performed for all subjects. It was determined by the use of Infinity glucose oxidase liquid stable reagent assay (Thermo Electron Corporation). Fasting plasma insulin was measured via the use of the Linco Human Insulin RIA Kit (Linco research) in accordance with the manufacturer's instructions.

## 2.9 Genetic investigation

DNA was collected in order to investigate the polymorphisms of the FABP2, CAD and WNK1 candidate genes and their relationship with the prevalence of Metabolic Syndrome in female Pakistanis. It is hoped that an investigation into the genetic link that underpins Metabolic Syndrome will assist in identifying at risk individuals and thus lead to targeted treatments that reduce the effects of Metabolic Syndrome (and hence preventing or delaying the onset of various chronic diseases) in the target group.

### **2.9.1 Buccal swab sampling**

Collection of DNA samples is often achieved through buccal swab sampling, as it is non-invasive and painless; accordingly, it is becoming increasingly more popular (Gracia-Closas et al. 2001, Milne et al. 2006, Lum and Le Marchand 1998). Therefore buccal swab sampling was used in order to collect the DNA of participants for genetic analysis.

### **2.9.2 DNA extraction**

Subjects were requested to rinse their mouths thoroughly twice with water. Tissue was collected by subjects rolling the buccal brush firmly on the inside of the cheek, approximately 20 times on each side, making certain to move the brush over the entire cheek. Buccal brush was air dried for 10-15 minutes at room temperature. The dry brushes were stored in the original packaging at 22-37°C until the next day for analyses. The buccal brush was placed into a tube containing DNA Extraction Solution and rotated a minimum of 5 times. The brush was then pressed against the side of the tube and rotated while removing it from the tube to ensure most of the liquid remained in the tube. The cap was then screwed on the tube tightly and Vortex mixed for 10 seconds, then incubated at 60°C for 30 minutes. The tube was then vortex mixed for 15 seconds. The tube was then transferred to 98°C and incubated for 8 minutes. The tube was then Vortex mixed for another 15 seconds. The tube was returned to 98°C and incubated for an additional 8 minutes. The tube was then Vortex mixed for 15 seconds. The tube was then chilled on ice briefly to reduce the temperature. Next, it was necessary to pellet cellular debris by centrifugation at 4°C for 5 minutes. The supernatant containing the DNA was then carefully transferred to a clean tube without including any of the beads. The DNA was then stored at -80°C for the long term.

### **2.9.3 Storage and disposal**

After extraction, the DNA was stored in a freezer at -80°C, until analysis. Once analysis was performed, DNA samples were autoclaved and disposed of as bio-hazardous material. The collection, storage and disposal of any genetic material or information was in accordance with NHMRC guidelines (NHMRC Part 16, Human Genetic Research).

### **2.10 Genetic investigation for single nucleotide polymorphism genotyping (SNP)**

For the purposes of this study, two standard techniques were performed. The FABP2 genetic variant was analysed via the thermal cycler polymerase chain reaction procedure. Other genetic variant CAD, and WNK1 were analysed via real-time polymerase chain reaction procedure. DNA was collected in order to investigate the polymorphisms of 3 specific genetic SNPs (FABP2, CAD, and WNK1,) and the prevalence of Metabolic Syndrome in female Pakistanis. Collection of DNA samples was achieved through buccal swab sampling as it is non-invasive and painless (Milne et al. 2006).

## **2.10.1 Protocol for the detection of (SNP) polymorphisms of the FABP2 gene by thermal cycler-polymerase chain reaction (PCR).**

### **2.10.1.1 Principle of the PCR**

The following description of methodology relating to PCR work used in this study was adapted from the manufacturer's instructions. (Getting Started guide, Biosystems 2004.)

PCR is a technique widely based on molecular biology to increase the number of copies of certain DNA fragments; these fragments are easily detectable by the use of gel electrophoresis technique. PCR is carried out in a single test tube simply by mixing DNA with a set of reagents and placing the tube in a thermal cycler, a piece of equipment which enables the mixture to be incubated at a series of temperatures which are varied in a programmed manner. Every PCR has three basic steps: denaturation, annealing of the primers and extension.

#### **2.10.1.1.1 Denaturation**

The mixture is heated to 94°C at which the hydrogen bonds that hold together the two strands in the double DNA molecule are destroyed, causing the DNA to denature.

#### **2.10.1.1.2 Annealing**

Annealing of the primers. The mixture is allowed to cool down to 50-60° C. The 2 strands of each molecule may join back together at this temperature, but most do not as the mixture contains a large excess of short single stranded DNA sequence (that are built complementary to a target sequence that flanks the beginnings and end of the DNA template to be amplified called oligonucleotides/primers. These primers represent the starting points for building new DNA strands. These primers anneal to the DNA molecule at specific positions.

### **2.10.1.1.3 Extension**

The temperature is raised to 72° C . This is the optimum working temperature for the taq DNA polymerase that is present in the PCR mixture. Taq DNA polymerase attaches to one end of each primer and synthesis new strands of DNA which is complementary to template DNA molecule, during this step of the PCR. Now we have 4 strands of DNA instead of the two original strands there were to start of with.

The temperature is increased back to 94°C. The double stranded DNA molecule, each of which consists of one new and one old DNA stand, denatures into 2 single strands. This begins a second cycle of denaturation, annealing, extension at the end of this cycle there are 8 DNA strands. By repeating the cycle 25-30 times, the double stranded molecule which we began with is converted to over 50 million new double stranded molecules.

## **2.11 Polymerase chain reaction (PCR) on FABP2 gene**

The Gotaq PCR Core System II (Promega USA) was used to carry out PCR on the DNA of all the subjects, following manufacturer's instructions. The Gotaq PCR Core System II provides all of the reagents necessary for amplification of specific region of DNA (target gene) using the PCR. It uses *Taq* DNA polymerase to enhance the amplification of the selected regions of DNA. The following primers of the targeted genes were purchased from Promega:

Forward primer 5-ACAGGTGTTAATATAGTGAAAAG-3

Reverse primer 5-TACCCTGAGTTCA GTTCCGTC-3.

Each primer was rehydrated by adding nuclease-free water to make a final concentration of 100µM (primer stock solution), and then each primer was further diluted to 10µM to use in PCR reaction mix.

### 2.11.1 Sample preparation

The PCR reaction mix was added to 0.5 ml micro-Eppendorf tubes. Each 50 µl of PCR reaction mix comprised the components in the proportions specified in Table 2.1 for each sample.

**Table 2.1** Composition of Each 50 µl of PCR Reaction Mix.

<b>Component</b>	<b>Amount (µl)</b>
MgCl <sub>2</sub> (25mM)	3
Green go taq flexi buffer 10X	10
PCR nucleotide mixture (10mM)	1
Forward control primer 15uM	3.3
Reverse control primer 15uM	3.3
Sample DNA 500ng/ul	1
Go taq DNA polymerase	0.25
Nuclease-free water(to a final volume)	29.4
<b>Total</b>	<b>50</b>

PCR Reaction Mix

### 2.11.2 PCR amplification conditions

The micro-Eppendorf tubes were briefly spun in a bench top centrifuge and then placed into a thermal cycler (Bio Rad) that had been preheated to 95°C and programmed to the correct settings, as specified in Table 2.2

**Table 2.2** Thermal Cycler Settings for PCR Amplification.

Step	Temp. (°C)	Time	Number of cycles
Initial Denaturation	95	10 minutes	1
Denaturation	95	1 minute	35
Annealing	55	1 minute	
Extension	72	1 minute	
Final Extension	72	5 minutes	1
Soak	4	Indefinite	1

### 2.11.3 Digestion of the FABP2 gene with restriction enzyme

On completion of PCR samples underwent restriction enzyme digestion prior to agarose gel electrophoresis and analysis. Restriction enzymes cut the DNA at a specific location along its length. These enzymes cleave the DNA at the recognized site referred to as the restriction site. Each type of restriction enzyme recognizes a particular base sequence of the DNA and cuts precisely at the same point each time i.e the restriction enzyme EcoR1 recognizes the sequence GATTC and cuts between the G nucleotide and adjacent A nucleotide. This results in smaller fragments of DNA which, as mentioned above, are then visualized via gel electrophoresis.

#### **2.11.4 Sample preparation for digestion of the FABP2 gene with restriction enzyme**

The HinPII Restriction enzyme (New England Biolabs) was used for the restriction digestion of PCR amplified DNA. In this study. The restrictions sites for the DNA samples were 5' G C G C 3' and 3' C G C G 5' , 2 units of HinPII enzyme was mixed with 1 µl of 1x nuclear extract (NE) buffer this was then added in the PCR product (refer to Table 2.2). This reaction mix was then incubated at 37°C for 90 minutes, after the incubation HinPII Restriction enzyme cut the DNA (FABP2) genotype ala/ala ( homozygous) into two bands, and genotype ala/thr ( heterozygous) into three bands.

#### **2.11.5 Sample analysis**

Following the digestion of DNA (FABP2) gene, the expected DNA digested products were detected on 1% gel electrophoresis.

### **2.12 Gel electrophoresis**

#### **2.12.1 Principle of gel electrophoresis**

Gel electrophoresis is an easy way to separate DNA fragments by their sizes and visualize them.

Gel electrophoresis is a common technique used molecular biology, for the separation of DNA/RNA, or protein molecules using an electric current applied to a gel. It is usually performed for analytical purposes, but may be used as a preparative technique prior to use of PCR. Organic molecules such as DNA carry a negative charge due to the presence of the phosphate group. These negatively charged molecules migrate through the gel towards the positive electrode in the gel. The gel matrix acts as a sieve for DNA molecules. The smaller the DNA molecule, the faster it travels towards the positive electrode of the chamber.

### **2.12.2 Gel electrophoresis methodology**

To visualise and detect the DNA segments, the PCR products were placed onto a 1% agarose gel. The gel was prepared by mixing 0.5g of agarose powder 50ml of 1xTAE. The agarose gel solution was then heated in a microwave oven until the agarose particles had dissolved; the mixture was then cooled to 60°C. 4µl of ethidium bromide (10mg/ml) was added to the agarose gel solution (to visualize the DNA fragments) and then poured into a tray and allowed to set. The ends of the gel tray were sealed with laboratory tape; a gel comb was placed at one end of the tray. (This would create wells within the gel, into which the samples would be loaded.) Once the gel was set, the tape and the comb were removed, and the gel was placed into an electrophoresis chamber. The chamber was then filled with 1xTAE buffer until the top of the gel was covered; this allowed for an electrical current to travel through the gel.

### **2.12.3 Sample preparation**

Molecular weight markers are used for estimating the size of DNA fragments in the gel electrophoresis. The low molecular weight marker ranged from 25 to 766 base pairs. We have used the 200 base pairs band as our reference point. Each sample (digested PCR product of FABP2 gene) was Vortex mixed into separate Eppendorf tubes. 16 µl of PCR product was loaded into each well. 1 µl of low molecular weight DNA ladder (Biolabs) was diluted with distilled water and mixed with 1 µl of blue loading dye and loaded in the first well. Electrophoresis was run at 55 V for 90 minutes. The procedure was repeated several times, with minor adjustments to the amount of DNA template added to the reaction mix every time. The concentrations of diluted DNA used were 200ng/300ng/400ng/500ng. This PCR procedure was necessary to visualise the DNA segments on the agarose gel. Analysis was performed by the use of the Chemidoc RS-digital camera system and Quantity One software package (Biorad).

### **2.13 Genetic investigation for genes with no lysine kinase (WNK) for hypertension and coronary artery disease (CAD) by real time-polymerase chain reaction (RT-PCR)**

The genotyping of two SNPs (CAD and WNK1,) were determined via using TaqMan (allelic discriminations) genotyping assays (Applied Biosystems). SNP analysis was performed by the use of TaqMan ready-made assays with the following ID numbers: C\_1754666\_10648612 for alleles C/G and C\_7561749\_20648611 for alleles C/T. Allelic discriminations assays for PCR includes a specific fluorescent dye-labeled probe for each allele. The probes contain different fluorescent reporter dyes (FAM™ dye and VIC® dye) to differentiate the amplification of each allele. Each TaqMan probe contains a reporter dye at the 5' of each probe. VIC dye is linked to the 5' end of the allele 1 probe, while FAM dye is linked to the 5' end of the allele 2 probes, and a non fluorescent quencher is attached to the 3' end of each of these probes. The probe lies between the two primers and overlays the SNP of interest. When both dyes are attached to the probe, reporter dye emission is quenched. During each extension cycle the AmpTaq Gold DNA polymerase cleaves the reporter dye from the probe dye. After being separated from the quencher the reporter dye emits its characteristic fluorescence. The fluorescence signals generated by PCR amplification indicate the alleles that are present in the sample. Allelic discrimination was measured automatically on the ABI Prism 7500HT (Applied Biosystems) with Sequence Detection Systems 2.1 software (auto caller confidence level, 95%).

### **2.13.1 Real time-polymerase chain reaction (RT-PCR); principle used in SNP genotyping**

The Real Time-Polymerase Chain Reaction (RT-PCR) (Applied Bioscience, 7500 Real Time PCR) is a technique used in validating gene polymorphism expression. RT-PCR allows detection of PCR products on a real-time basis. Real Time Polymerase Chain Reaction (RT-PCR) is a method used to detect and quantify defined DNA sequences of interest. RT-PCR uses a fluorescent reporter dye as a marker for the amplification and detection steps of the PCR reaction (Applied Biosystems 2004).

TaqMan reagent based chemistry uses a fluorogenic probe to enable detection of specific PCR product as it accumulates during PCR cycles. A reporter dye and a quencher are attached to the 5' and 3' ends of the TaqMan probes. When both dyes are attached to the probe reporter dye emission is quenched. During each extension cycle the AmpTaq, Gold DNA polymerase cleaves the reporter dye from the probe dye. After being separated from the quencher the reporter dye emits its characteristic fluorescence.

#### **2.13.1.1 Preparing the reaction mix**

An allelic discrimination assays were used to investigate the SNP genotyping (Applied Biosystem).The reaction mix was made from 40X SNP genotyping Assay, TaqMan Universal PCR Master Mix, and Dnase-free water. The final reaction volume used per well was 25  $\mu$ L (96-well plate). Refer to Table 2.3. Thermal Cycle Conditions (setup) were as in Table 2.4 each reaction plate was kept on ice until placed in the thermal cycler. The thermal cycler was run for ninety minutes, for each reaction plate.

**Table 2.3** PCR -Reaction Mix Per Well.

<b>Component</b>	<b>96-well plate</b>
TaqMan Universal PCR Master Mix (2x) Universal PCR	12.50µL
20 x working stock of SNP genotyping assay.	1.25µL
Sample DNA (200ng/ul) diluted with Dnase-free water.	11.25µL
<b>Total Volume per Well</b>	<b>25µL</b>

**Table 2.4** PCR - Thermal Cycle Conditions.

	<b>Times and Temperatures</b>	
<b>Initial Steps</b>	<b>Denature</b>	<b>Anneal/Extend</b>
Hold	40 cycles	40 cycles
10 min @ 95°C	15 sec @ 92°C	1 min @ 60°C

### **2.13.1.2 Sample analysis**

The generated data was analysed by the use of absolute quantification (AQ) software, as default setting on PCR equipment. The AQ plate document software was used to store real time data (Applied Biosystems 2004).

The presence of two primer/probe pairs in each reaction allows genotyping of the two possible variants at the single-nucleic polymorphism (SNP) site in a target template sequence. The actual quantity of target sequence is not determined.

For each sample in an allelic discrimination assay a unique pair of fluorescent dye detectors is used, for example, two TaqMan MGB probes that target an SNP site. One fluorescent dye detector is a perfect match to the wild type (allele 1) and the other fluorescent dye detector is a perfect match of the mutation (allele 2). (Applied Biosystem 2007).

The AD assay measures change in fluorescence of the dyes associated with the probes and shown as different colours. The allelic discrimination assay classifies unknown samples as:

Homozygotes (samples having only allele 1 or allele 2),

Heterozygotes (samples having both allele 1 and allele 2).

### **2.13.1.3 Statistical analysis**

The SPSS statistical package (version 12.0, SPSS Inc.) was used for the purposes of statistical analysis. All data were expressed as mean  $\pm$  standard error of mean (SEM). Data collected at weeks 0, 12, 24 was analysed using T-Tests. Categorical data obtained from the food frequency questionnaire was also analysed with use of the SPSS statistical using chi square to determine statistical significance between categories of frequency of consumption. A p-value of  $< 0.05$  was considered statistically significant. The recruitment of volunteers was based on a calculation of statistical power (solved for

n.  $1.96 \times SE(p) = 5$ , Where  $SE(p) = \sqrt{p(1-p)/n}$ , with a 95% confidence level shows that a sample size of 79.45 is required. (Armitage 1987) Therefore a statistically viable sample must consist of at least 80 individuals. A larger sample will be recruited with consideration of drop outs.

### **Reflection on Methodology**

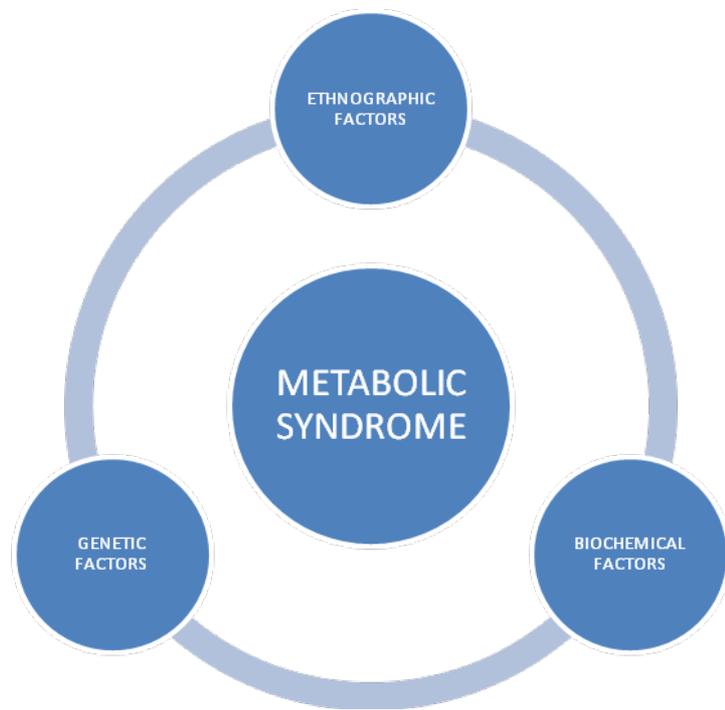
This study aimed to answer the following research questions: What are the metabolic characteristics of Pakistani women residing in Melbourne displaying the risk factors of Metabolic Syndrome? Do genetic markers that have been associated with Metabolic Syndrome exist in Pakistani females? Is a culturally appropriate diet and lifestyle intervention an effective mechanism for preventing the onset, or reducing the severity of Metabolic Syndrome in migrant Pakistani women?

It has been shown that losing weight or stopping weight gain via improved diet and increased physical activity may reduce the overall risk of the disorder (Stone and Sexon, 2005). The treatment of the disorder must be a multi-faceted process, that is, a process with a primary focus on therapeutic lifestyle change, in more advanced cases however, pharmacological intervention may also be required. (Deedwania 2002) As many of the symptoms of Metabolic Syndrome have a direct correlation with weight and body fat levels, the best non-pharmacological methods for controlling the syndrome relate strongly to lifestyle intervention, furthermore, the inclusion of pharmacological intervention is more effective once a diet and lifestyle intervention is already in force (Rosenson RS, Reasner 2002). It is clear that any individual or group may attempt to prevent or reduce the severity of Metabolic Syndrome by implementing a diet and lifestyle intervention; however, many factors need to be taken into account in order to design an effective intervention for a specific individual or group, in this case, for South Asians. The primary objective for recognising Metabolic Syndrome sufferers is to

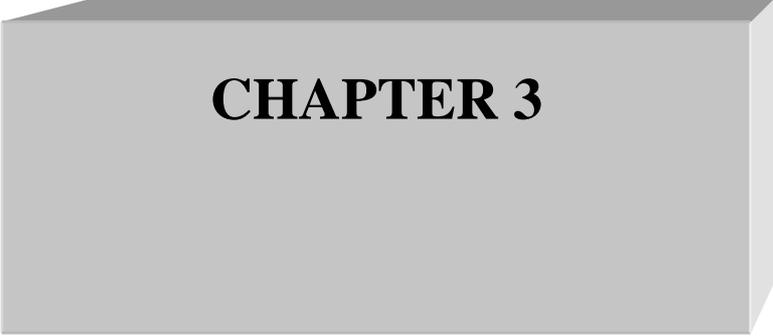
prevent cardiovascular disease and Type II diabetes. In this study an intervention was developed in order to treat female Pakistani Metabolic Syndrome sufferers. It has been suggested that a straight forward approach to reduce the risk of these diseases should be used, with a focus on dietary modification and physical activity (Adult Treatment Panel III, 2002 ). The implementation of dietary advice and lifestyle modification are influenced by various factors including ethnic, cultural and religious background. In this study dietary and behavioural modification was developed and used in the culturally sensitive target population successfully. Dietary and lifestyle modification was tailored to suit the target population's cultural and religious needs. It is possible that the selection sample for this study was biased, as we specifically targeted individuals displaying at least one component of Metabolic Syndrome, however, this possible bias was necessary as the overall purpose of this study was indeed to reduce the overall severity or prevent the onset of Metabolic Syndrome in participants by the implementation of a culturally appropriate diet and lifestyle intervention.

Biochemical, genetic and ethnographic factors were examined in this study. As elucidated in the conceptual map below.

**FIGURE 2.2**



**FIGURE.** Conceptual map of this study



## **CHAPTER 3**

### **STUDY 1 PILOT AND BASELINE STUDY OF RISK FACTORS.**

### **3. Pilot Study**

#### **3.0 Pilot and Baseline Study of Risk Factors**

##### **3.1 Introduction**

Metabolic Syndrome is currently a major health issue, particularly so in South Asian populations. Not much data exists examining the relationship between Metabolic Syndrome and female Pakistani populations. We sought to develop a study that investigated Metabolic Syndrome in this high risk population, we also aimed to develop a diet and lifestyle intervention that could help negate the severity, or prevent the occurrence of Metabolic Syndrome. A pilot study was required to assess the feasibility of a larger study.

##### **3.2 Aim**

The pilot study was used as a means of assessing the feasibility of a major study, it served as a small scale or trial version of the major study. A key aim of the pilot study was to assist in identifying major problems in advance, indicative of where the research might fail, or where research protocols would either not be followed, or prove to be inappropriate for this particular population.

##### **3.3 Methodology**

17 female Pakistani participants were selected (please refer to sections 2.2 and 2.2.2 of the Methods chapter for further detail) of whom 4 dropped out (and had no further bearing on study data). The 13 remaining participants engaged in our pilot diet and lifestyle intervention. The diet and lifestyle intervention program was implemented over a 12 week period. The program was based on 12 weekly modules, each module with a different focus and goal to achieve. The goals of the intervention program were multifaceted, including an overall decrease in energy intake and increase in physical

activity. Each module consisted of individual dietary counselling and researcher-participant interaction. The modules aimed to increase the participants' self efficiency and self regulatory skills (refer to section 6.3.1). Anthropometric, blood pressure and physical activity measurements were taken at week 0, and remeasured at week 12. The blood lipid profile was only analysed at week 0, however, it was not reanalysed at week 12 because it was Islamic fasting month (Ramadan) and the women refused to give blood while fasting. Measurements were taken according to standard techniques and equipment (International Study of Obesity, WHO 1998). (refer to the method chapter 2)

### **3.4 Inclusion Criteria**

Participants had a mean age of  $35.8 \pm 3.5$  years. Results were derived from 13 subjects in total. The attrition rate prior to the commencement of the pilot study was 23.5% (reasons for attrition included family commitments, relocating overseas and health issues such as pregnancies.)

Participants had been resident in Australia for 5 to 17 years. All subjects were married, with 2-6 children and residing with their husbands and/or in-laws. All subjects had completed primary and secondary education in Pakistan; half the participants had also some form of tertiary education. 46% of participants were fluent (able to communicate, read and write efficiently) in English, 23% were average (able to communicate, read and write at a basic level) and the final 30% had poor (unable to communicate, read or write) English skills.

#### **3.4.1 Sampling strategy**

Subjects that participated in the pilot study were selected from different geographical areas, such as Coburg, Dandenong, St Albans and Taylors Lakes. All subjects followed the Islamic faith. (refer to Method Chapter 2 section 2.2.2).

### 3.5 Protocols

All protocols followed in the pilot study were the same as protocols followed in the main study, (refer to Method Chapter 2 section 2.6).

### 3.6 Anthropometric analysis

Anthropometric measurements for all subjects were taken at weeks 0 and 12; parameters measured were height, weight and waist circumference. The results are summarised in Table 3.1. Results indicated that prior to the intervention the average BMI of the participants was classified as obese and participants had a high waist circumference. After the diet and lifestyle intervention, significant changes were observed. Average BMI was significantly reduced (Pre:  $32.6 \pm 1.2$  kg/m<sup>2</sup> Post:  $29 \pm 1.5$  kg/m<sup>2</sup>  $P < 0.05$ ). Average waist circumference was also reduced (Pre:  $102. \pm 2.5$ /cm Post:  $96. \pm 2.5$ /cm  $P < 0.05$ ).

**Table 3.1** Anthropometric Analysis Results Summary: ( n=13)

<b>Parameter Measured</b>	<b>Week 0 (Before Intervention)</b>	<b>Week 12 (After Intervention)</b>	<b>P-Value (Week 0-12)</b>
Body Mass Index (kg/m <sup>2</sup> , SEM)	$32.6 \pm 1.2$	$29 \pm 1.5$	$P < 0.05$
Weight (kg, SEM)	$74.2 \pm 1.4$	$71.8 \pm 1.3$	$P < 0.05$
Waist circumference (cm, SEM )	$102. \pm 2.5$	$96. \pm 2.5$	$P < 0.05$

Anthropometric results summary: BMI, body weight, and waist circumference measurements. Week 0 represents baseline data, before the beginning of the intervention. Week 12 represents data at the end of the intervention. Data is presented as mean  $\pm$  SEM. T-test statistical analysis was performed.

### **3.7 Blood pressure, physical activity and biochemical analysis**

In this study blood pressure was measured for all subjects according to the WHO criteria for the diagnosis of Metabolic Syndrome (NCEP-ATP III, 2001). The results are summarised in Table 3.2. Participants were considered hypertensive if their blood pressure was greater than 130/85 mmHg. Results indicate that prior to intervention participants were hypertensive. After the diet and lifestyle intervention, significant changes were observed in subjects blood pressure values and physical activity levels. Average blood pressure was significantly reduced (Pre: systolic  $145 \pm 2$  mm/Hg, diastolic  $112 \pm 2$  mm/Hg Post: systolic  $110 \pm 2$  mm/Hg, diastolic  $83 \text{ mm/Hg} \pm 2$   $P < 0.05$ ). Participants were also sedentary, taking (Pre:  $3140 \pm 200$  steps per day Post:  $9490 \pm 500$  steps per day  $P < 0.001$ ).

In this study blood biochemical profiles were measured only at week 0 only; including total serum cholesterol, HDL-C, LDL-C and serum triglycerides, plasma insulin and glucose measured for all subjects. The results showed that participants had high levels of cholesterol and triglycerides ( $6.56 \pm 0.2$  mmol/L and  $2.44 \pm 0.18$  mmol/L respectively). Consistent with dyslipidaemia, HDL levels were also low, at  $1.33 \pm 0.21$  mmol/L. Furthermore plasma glucose ( $6.9 \pm 0.5$  mmol/L) and fasting blood insulin ( $29.8 \pm 1.7$   $\mu\text{U/m}$ ) levels were high. We were unable to get the blood sample at the end of intervention week 12 because the Muslim fasting period started, and subjects felt uncomfortable with blood testing.

**Table 3.2** Blood Pressure and Physical Activity Analysis Results Summary: ( n=13)

<b>Parameter Measured</b>	<b>Week 0 (Baseline data)</b>	<b>Week 12 (Intervention Commencement)</b>	<b>P- Value (Weeks 0-12)</b>
Blood Pressure (Systolic/Diastolic, mm/Hg, SEM)	145. $\pm$ 2 110 $\pm$ 2	112 $\pm$ 2 83. $\pm$ 2	P<0.05
Physical Activity (steps per day, SEM)	3140 $\pm$ 200	9490 $\pm$ 500	P<0.001

Blood pressure systolic and diastolic (mm/Hg). Physical activity measurements (steps/day). Week 0 represents baseline data; before the beginning of the intervention week 12 represents the end of the intervention. Data is presented as mean  $\pm$  SEM. T-test statistical analysis was performed.

### **3.8 Alterations for the major study**

Key alterations in the study were required. It was established that transportation must be provided for subjects in any future study, so as to ease any difficulties that may arise in having all participants in the same place at the same time. Furthermore, in the major study weekly sessions would be held in a participant's house in small groups of 3 or 4; this would make it much easier to coordinate the project. Importantly, the blood collection location was changed from a pathology centre, to the Victoria University research laboratory for convenience and cost effectiveness. It was also established that a food frequency questionnaire was necessary for the purposes of this study, in order to establish any dietary changes that may have occurred as a result of the intervention. Finally, walking and the use of a pedometer were established as the best type of physical activity and its monitoring given their cultural and religious constraints.

### **3.9 Conclusion**

The pilot study assisted in amending the nature of the future study and intervention, thereby making any such future study more efficient and effective in terms of preventing or improving Metabolic Syndrome in the target population, and also in terms of time and cost.

Major risk factors for Metabolic Syndrome were reduced in this study. At week 12 (end of the intervention period) blood pressure, average BMI and waist circumference were significantly reduced. Physical activity levels had also significantly increased. The pilot study was very successful in its objectives; it helped us identify potential problems that could arise in any proposed future research. As the results of this study were positive and significant it was established that a longer, in-depth study would be beneficial and feasible. The pilot study helped shape the major study. It identified that walking (with the use of the pedometer) was the most convenient physical activity for participants: complications in arranging transport and establishing group meetings were also identified.

### **3.10 Major Study**

#### **Study Design**

The basic study design was a 12 week control period no intervention, followed by 12 week diet and exercise intervention. Baseline data for this study was collected at week 0. Anthropometric, blood pressure measurements and blood samples were obtained. Information on the physical activity levels and medical history of the participants was also obtained through a questionnaire in Urdu. The above parameters were also remeasured at week 12, before the commencement of the planned intervention. The diet and lifestyle intervention program was implemented for 12 weeks, after which all the above parameters were remeasured (week 24)

#### **3.10.1 Major Study**

Various psychosocial, environmental and unknown genetic factors have been associated with the development of the components of Metabolic Syndrome such as social isolation, depression and stress. Furthermore, psychosocial factors have been linked to environmental factors such as physical inactivity, poor diet and smoking. The interaction between psychosocial factors and environmental factors can often result in the excess accumulation of fat, which leads to the development of Metabolic Syndrome components (De Vogli et al. 2007). It was originally proposed by Bjorntrop and Rosmond that visceral adiposity could indeed be associated with stress given the implications of stress on the hypothalamic pituitary adrenal (HPA) axis (Goldbacher and Matthews 2007).

Metabolic Syndrome has not been investigated thoroughly in Pakistani populations, particularly, in Pakistani women. Cardiovascular disease however has been researched adequately in Pakistani populations. Furthermore, Metabolic Syndrome has been investigated to some extent in South Asian populations. The risk factors for Metabolic

Syndrome and cardiovascular disease that were identified in research conducted on South Asians, in particular Pakistani populations, have been used as determinative factors for Metabolic Syndrome in this study. No studies particularly focus on Pakistani populations; studies that have investigated Pakistanis have concurrently investigated Indians and/or Bangladeshis, that is, South Asians in general. Our study aimed to either draw parallels or distance Pakistanis and the Metabolic Syndrome/cardiovascular disease risk factors that they might suffer, from Indian, Bangladeshi and Sri Lankan populations, thereby clarifying some of the ambiguity that exists in differentiating between 'South Asians.'

All biochemical risk factors were investigated and classified according to WHO recommendations for the Metabolic Syndrome risk factors. Environmental factors that were investigated included dietary pattern, physical activity levels as well as the effect of social influence and attitude towards diet and physical activity. Participants were investigated for any change in diet and lifestyle after migration.

3.10.2 Methodology All typographical and grammatical errors have been addressed.

Baseline data for this study was collected from 60 participants (please refer to sections 2.2 and 2.2.2 of the Methods chapter for further detail). All parameters were measured at week 0 and remeasured at week 12, prior to the commencement of the planned intervention. . We actively sought migrant women with at least one component of Metabolic Syndrome (information was ascertained on a self-reported basis). Thus we aimed to validate and confirm the presence of Metabolic Syndrome in our subject population in order to design and implement a culturally appropriate diet and lifestyle intervention. Anthropometric, blood pressure measurements and blood samples were obtained from all participants. Information on the physical activity levels, dietary

pattern and medical history of the participants was also obtained through a questionnaire in Urdu.

### **3.11 Demographics and lifestyle habits**

This study investigated 60 participants. 150 women were invited to participate in this study. 90 potential participants responded to the invitation (our response rate was thus 60%). Of these 90, 15 did not meet eligibility criteria. Thus, the final 75 remaining potential participants were selected for this study, however 15 of the selected potential participants dropped out prior to the commencement of the intervention, leaving a total of 60 participants. The response was considered to be fairly positive, ethnic minorities and Pakistanis in particular are known to be reluctant in their participation of research studies (Rankin and Bhopal 2001).

Participants were selected from different areas of Melbourne; however, most of the women were from areas with a high concentration of Pakistanis (the Western, North Western and Outer Eastern suburbs of Melbourne, the exact numbers from each suburb follow) such as Taylors Lakes (10), St Albans (10), Deer Park (5), Werribee (9), Hoppers Crossing (7), Coburg (7), Fawkner (3), Dandenong (2) and Doncaster (7) (ABS, Australia 2001-b).

The group selected for participation in this study had a mean age range of  $37.6 \pm 4.3$  years. The number of years residing in Australia ranged from 5 to 22 years, most subjects having migrated to Australia in the mid 80s and early 90s. All of our participants were married and lived with their husbands; some participants also lived with their in-laws. Participants on average had 2-6 children refer to table 3.3 and 3.4.

Subjects were classified according to their education level, participants who had completed primary school only were classified as having 'poor' English skills; those who had completed secondary college were classified as having 'average' English

skills, and participants who had completed a degree at university were classified as having 'fluent' English skills.

All subjects had completed primary school education in Pakistan, all participants that had completed secondary school education, had also done so in Pakistan. Half the participants had some form of tertiary education. 50% of participants were fluent (able to communicate, read and write efficiently) in English, 25% were average (able to communicate, read and write at a basic level); the final 25% had poor (unable to communicate, read or write) English skills.

Voluntary conversations were conducted with many participants. It was observed that participants were generally aware of the benefits associated with regular exercise. Conversely, some participants had little knowledge as to what constitutes a healthy diet; while they were aware of certain health problems (such as hypertension and high cholesterol levels) they were not entirely sure as to the causes and repercussions of such problems. Participants were aware of the health problems associated with smoking, with none of our participants declaring themselves as a smoker. In general, participants had an average understanding of health and well-being (according to current Australian standards).

**Table 3.3** Demographic Results Summary (n=60)

<b>Parameter</b>	<b>Subject Population Statistics</b>
Age (years)	37.6 ± 4.3 years. Range: [19,59]
Length of residency in Australia	10.09 ± 0.43 year. Range: [5,22]
Marital status: Married.	100%
Living with husband and/or in-laws.	100%
1 child or more.	100%
Completed tertiary education.	50%
Completed primary/secondary education.	100%
Fluent English Skills.	50%
Average English Skills.	25%
Poor English Skills.	25%
Currently working.	75%

Demographic of Pakistani Women

**Table 3.4** Self Reported Health Conditions (n=60)

<b>Parameter</b>	<b>Subject Population Statistics</b>
BP checked within the last 3 months.	25%
Cholesterol checked within the last 3 months.	25%
Suffering from hypertension.	60%
Diagnosed with angina.	10%
Diagnosed (or told) they had diabetes.	43%
Diagnosed with high cholesterol.	40%
Diagnosed with high triglyceride levels.	83%
Diagnosed with obesity.	70%
On BP medication.	15%
On cholesterol medication.	5%
On diabetes medication.	5%
On oral contraceptive.	31%

Self reported health conditions of Pakistani women.

### 3.12 Anthropometric analysis

Anthropometric measurements for all 60 subjects were taken. The results are summarised in Table 3.5. Measurements taken included weight, height and waist. BMI and waist circumference were used as the primary indicators of obesity (central/abdominal) in the target population. BMI and waist circumference were classified according to WHO Asian specific guidelines. According to these guidelines a waist circumference of 88 cm and a BMI of 23 is considered overweight, and a BMI of 25 or over is considered obese (WHO/IASO/IOTF 2000). Results indicate that prior to intervention at baseline the average BMI of participants was classified as obese ( $30.4 \pm 0.4$  kg/m<sup>2</sup>). Participants also had a high waist circumference on average ( $107.5$ /cm  $\pm$   $0.88$ / cm), 83 % (50) of participants were classified as obese. No significant differences were observed from weeks 0 to week 12.

**Table 3.5** Anthropometric Analysis Results Summary (n=60).

<b>Parameter Measured</b>	<b>Week 0 (Control period/ Baseline data)</b>	<b>Week 12 (End of Control period)</b>	<b>P- Value (Week 0-12)</b>
Body Mass Index (kg/m <sup>2</sup> , SEM)	$30.4 \pm 0.4$	$30.7 \pm 0.4$	P = 0.959
Weight (kg, SEM)	$75.26 \pm 1.4$	$74.78 \pm 1.4$	P = 0.805
Waist circumference (cm, SEM)	$107.5 \pm 0.7$	$105.2 \pm 1.8$	P = 0.922

Anthropometric results summary: BMI, body weight, and waist circumference measurements. Week 0 represents baseline data; week 12 represents the end of the control period prior to the intervention. Data is presented as mean  $\pm$  SEM. T-test statistical analysis was performed.

### **3.13 Biochemical analysis**

In this study blood biochemical profiles (including total serum cholesterol, HDL-C, LDL-C and serum triglycerides, insulin and glucose) were measured for all 60 subjects. The results are summarised in Table 3.6. Measurements were taken according to the WHO criteria for the diagnosis of Metabolic Syndrome (NCEP-ATP III 2001). All sixty subjects were included in the biochemical analysis, even subjects on medication for diabetes and elevated cholesterol levels. Our results showed that 36 participants (60%) had elevated levels of cholesterol ( $6.65 \pm 0.2$  mmol/L), and 50 participants (89%) had elevated levels of triglycerides ( $2.63 \pm 0.09$  mmol/L). Results indicate that, 25 participants (41%) also had elevated levels of plasma glucose ( $6.4 \pm 0.10$  mmol/L), and 57 participants (97%) had elevated levels of insulin ( $56.3 \pm 1.3$   $\mu$ u/ml). It can be concluded that the Pakistani subject population investigated in this study had high cholesterol, glucose, and insulin levels. No significant differences were observed from weeks 0 to week 12.

**Table 3.6 Biochemical Analysis Results Summary (n=60).**

Parameter Measured	Reference Value	Week 0 (Control period/Baseline data)	Week 12 (Intervention Commencement)	P- Value (Weeks 0-12)
Total Cholesterol (mmol/L, SEM)	4.5-5.5 mmol/L	6.65 ± 0.2	6.78 ± 0.2	P = 0.591
HDL Cholesterol (mmol/L, SEM)	0.90-2.2 mmol/L	0.87 ± 0.06	0.96 ± 0.07	P = 0.349
LDL Cholesterol (mmol/L, SEM)	1.2-1.51 mmol/L	1.55 ± 0.07	1.47 ± 0.07	P = 0.421
Triglycerides (mmol/L, SEM)	0.40-1.35 mmol/L	2.63 ± 0.09	2.49 ± 0.09	P = 0.259
Plasma Glucose (mmol/L, SEM)	4.5-5.5 mmol/L	6.4 ± 0.10	6.4 ± 0.2	P = 0.613
Plasma Insulin (µU/ml, SEM)	18-20 µU/ml	56.3 ± 1.2	52.88 ± 2.2	P = 0.349

Biochemical analysis results summary: Total cholesterol, HDL/LDL-C, triglyceride, plasma glucose/insulin measurements (mmol/L) (µU/ml). Week 0 represents baseline data; week 12 represents the end of the control period prior to the intervention. Data is presented as mean ± SEM. T-test statistical analysis was performed.

### 3.14 Blood pressure and physical activity analysis

In this study blood pressure was measured for all 60 subjects according to the WHO criteria for the diagnosis of Metabolic Syndrome (NCEP-ATP III 2001). The results are summarised in Table 3.7. Participants were considered hypertensive if their blood pressure was greater than 130/85 mmHg. A small number of subjects were on blood pressure medication; however, the inclusion of these subjects did not affect our outcomes. No significant differences were observed from weeks 0 to week 12. Baseline data revealed that 35 participants (59%) were hypertensive, with mean systolic blood pressure of (136.45±0.97, p=0.284123mm/Hg) and diastolic blood pressure of (89.08±0.75mm/Hg, p=0.123mm/Hg).

Participants' daily physical activity levels were also measured via the use of pedometers. Our results revealed that participants were sedentary; 58 (95%) of our participants were sedentary (a person is defined as sedentary if they take less than or equal to 5000 steps daily) taking  $4084 \pm 116$  steps/day as their daily step count was less than or equal to 5000 steps per day. It can be concluded that the Pakistani subject population investigated in this study were hypertensive and sedentary.

**Table 3.7 Blood Pressure and Physical Activity Analysis Results Summary (n=60)**

<b>Parameter Measured</b>	<b>Week 0 (Commencement of control period/baseline data)</b>	<b>Week 12 (End of the control period, commencement of the intervention)</b>	<b>P- Value (Weeks 0-12)</b>
Blood Pressure (systolic/diastolic, mm/Hg, SEM)	$136.5 \pm 0.9$ $89.1 \pm 0.8$	$134.4 \pm 1$ $86.8 \pm 0.5$	P = 0.284 (systolic) P = 0.123 (diastolic)
Physical Activity (steps per day, SEM)	$4084.10 \pm 115.$	$4427. \pm 117.$	P = 0.93

Blood Pressure and Physical Activity Analysis Results Summary. Blood pressure systolic and diastolic (mm/Hg). Physical activity measurements (steps/day). Week 0 represents baseline data; week 12 represents the end of the control period prior to the intervention. Data is presented as mean  $\pm$  SEM. T-test statistical analysis was performed.

### 3.15 Food pattern analysis results

In this study all 60 subjects were required to list the frequency at which they consumed each of the 54 food items listed. Data obtained via the food frequency questionnaire (FFQ) was categorical. Data was collected at both the commencement (week 12) and completion (week 24) of the intervention. Analysis was performed by the use of SPSS statistical software. Results are summarised in Table 3. 8.

Our results indicate that at baseline, 48% participants were consuming the processed foods, such as, preserved meats and finger foods and unhealthy snacks (snacks high in sugar, fat and energy). 56% participants were consuming fast foods, 47% participants were consuming soft drinks. Participants were consuming bread (staple food), 91% of participants were consuming breads prepared in butter or ghee such as parathas and 2% of participants were consuming vegetables and fruits. We could not establish any relationship between the these factors and the risk factors that participants displayed, for example, there is no link between the participants that were consuming processed foods and participants that suffered from hypertension. Please refer to Chapter 6, Table 6.4 for further insight into the relationship between risk factors.

**Table 3.8.** Food Pattern Analysis Prior to the Commencement of the Intervention.

<b>Food Item</b>	<b>Proportion (%) of subjects consuming the food item prior to the intervention.</b>
<b>Fruits and Vegetables</b>	
Seasonal fruits	2%
Canned fruits	3%
Salad	2%

Vegetables (excluding green vegetables.)	50%
Green vegetables	2%
Potatoes	52%
<b>Bread, Rice, Pasta</b>	
Bread (including Naan, Chapati and Parathas)	91%
Rice	54%
Pasta	49%
<b>Dairy Products</b>	
Full cream milk	52%
Low fat milk	2%
Tasty cheese	49%
Fetta cheese	49%
Coon cheese	47%
Mozzarella cheese	59%
Bread spreads	45%
Yoghurt	28%
Ice-cream	31%
<b>Meat, Poultry and Fish</b>	
Lamb	23%
Beef	52%
Goat	52%
Sausages	30%
Salami	36%
Fish	2%

Chicken	59%
Duck	3%
<b>Fast Food</b>	
Fast food (Pizza Hut, McDonalds, KFC etc.)	56%
<b>Fat and Oils</b>	
Vegetable oil	21%
Olive oil	16%
Canola oil	58%
Ghee	51%
<b>Lentils and Beans</b>	
Channa (dried lentils)	13%
Chickpeas	13%
Moong (green lentil)	11%
Other beans	2%
<b>Snacks, Processed and Finger Foods.</b>	
Chocolate (including cake.)	17%
Nuts (Coated with sugars and salts.)	28%
Potato, chips, ready food (packaged)	48%
Biscuits	46%
Pakorras (chickpea flour fried dumpling.)	31%
Samosas (pastry with meat or vegetable filling.)	34%
Processed / Canned Foods	55%
<b>Beverages</b>	
Soft drinks	47%

Diet drinks	2%
Tea	96%
Coffee	32%
Fruit juices	45%
Cordial	56%

Food categorical data obtained and presented as proportion (%) of subjects consuming the food item prior to the commencement of the intervention. (n=60).

### 3.16 Discussion

The baseline data of this study confirmed that our sample female Pakistani population residing in Melbourne suffers from multiple risk factors of Metabolic Syndrome. Our results of the risk factors study suggest that the average BMI of participants was classified as obese (Table 3.5); participants also had high waist circumference. Participants also had hypertension, dyslipidaemia and increased levels of blood cholesterol, fasting plasma insulin and glucose. Thus, all the requisite components of Metabolic Syndrome were observed in the female Pakistani population investigated. According to the literature these subjects are four times more likely to develop cardiovascular disease and its related complications when compared to non-obese women (Tillin et al 2005).

The biochemical results of this study confirmed that female Pakistani subjects had a dyslipidemic lipid profile (Table 3.6). The relative contributions of individual lipoproteins to overall cardiovascular risk have been investigated thoroughly over the last several decades. It has been established that high triglyceride levels and low levels of HDL-C are important independent risk factors for cardiovascular disease (Bhopal et al. 1999, Enas and Senthilkumar 2001, Enas 2002). Our subjects also showed elevated

fasting plasma glucose and insulin levels. These elevated parameters may be indicative of a state of insulin resistance in the subject population, which in turns leads to type 2 diabetes, as high plasma glucose levels have previously been associated with type 2 diabetes and central/abdominal fat (Haffner 2006). A combination of a dyslipidemic lipid profile and high plasma glucose levels can predispose participants to an increased risk of cardiovascular disease and type 2 diabetes (Haffner et al. 2006, Enas 1998, Stone and Saxon 2005).

Baseline data from this study also confirmed that our female Pakistani subjects were hypertensive (refer to Table 3.7). Hypertension has been closely associated with sufferers of Metabolic Syndrome; the nature of the link between Metabolic Syndrome (and its components) with hypertension is dependant on various factors (such as obesity, diet, physical activity). Many studies have shown that the majority of overweight patients display hypertension, furthermore, it has been suggested that one of the crucial contributing factors related to obesity and its complications is indeed hypertension (Davy and Hall 2004). Significant scientific controversy exists regarding the exact nature of the relationship between hypertension and related metabolic disorders; despite this researchers generally agree that obesity/physical inactivity is the driving force behind most of the risk factors for cardiovascular disease and Metabolic Syndrome (Ferrannini et al 1997, Sharma 2005, Tillin et al. 2005 Wilson et al. 2005).

Our results also indicate that our participants were sedentary. Physical inactivity is a major contributing factor in certain high risk ethnic populations (such as Indians, Pakistanis and Bangladeshis) in the development of cardiovascular disease (Jafar et al. 2004). It is well documented that a regular amount of moderate physical activity on most days of the week will confer health benefits for individuals of all ages (Irwin et al. 2000, Thompson 2003, Kolt et al. 2007). Regular physical activity, such as brisk walking for 30-40 minutes has been associated with a 30-50% reduction in the risk of

developing cardiovascular disease, obesity and diabetes (Wannamethee et al. 2000, 2003, Fischbacher et al. 2004).

We observed that participants' diets were atherogenic. Participants were consuming full cream dairy products, potato chips, and clarified butter ghee (ghee contains a substantial amount of cholesterol oxide which has previously been linked to atherosclerosis) (Jacobson 1987).

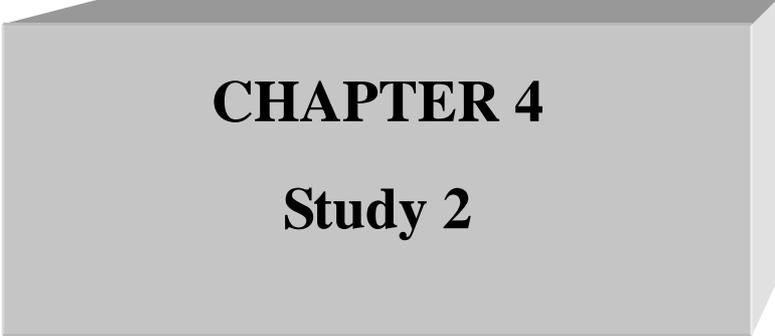
A diet high in saturated fat and low in fibre has been associated with obesity and thus elevated BMI (Patel et al. 2006). An elevated BMI and an obese waist circumference are indicative of Metabolic Syndrome. Furthermore, an atherogenic diet has been associated with an increase serum cholesterol and triglyceride levels, as well as insulin resistance. Thus, the typical diet observed in our subject population may have been contributory towards Metabolic Syndrome.

### **3.17 Conclusion**

The outcomes of this study have demonstrated that Pakistani females in our sample suffer from multiple risk factors of Metabolic Syndrome (after having been selected for the presence of at least one factor of the Syndrome) and are at risk of developing cardiovascular disease and type 2 diabetes. Our results have shown that our participants average BMI can be classified as obese, generally had an obese waist circumference and suffered from hypertension; all these components serve as major risk factors for Metabolic Syndrome. It was also revealed that participants were consuming energy dense foods and unhealthy fats, such as fried pakoras, potato chips etc (refer to Table 3.8). Participants originally had an atherogenic diet (high in saturated fats and low in fibre). Furthermore, participants were physically inactive and engaged in a very sedentary lifestyle. The biochemical results of this study demonstrated the presence of dyslipidaemia, elevated levels of blood cholesterol, fasting plasma insulin and glucose.

Increased levels of fasting insulin and the presence of dyslipidaemia could be the indicatives of future cardiovascular disease. All the requisite components of Metabolic Syndrome were observed in the target population.

The underlying causes of Metabolic Syndrome may be attributable to both biochemical and environmental factors. However, it is not entirely clear whether biochemical or environmental factors are of greater importance in terms of the incidence and severity of Metabolic Syndrome and/or cardiovascular disease. Consistent with other studies (Stewart-Knox 2005), we may also conclude that an unhealthy diet and physical inactivity are the most considerable contributory components of Metabolic Syndrome, thus, specifically targeting diet and physical inactivity in a high risk population can yield significant results in terms of treatment and prevention if detected at an early stage. It follows that the most logical approach in preventing/treating the underlying causes of Metabolic Syndrome is an appropriate diet and lifestyle intervention.



**CHAPTER 4**

**Study 2**

Genetics

## **4. GENETICS**

### **4.0 Single nucleotide polymorphism (SNP) genetic investigation on Pakistani females**

#### **4.1 Introduction**

In recent years Metabolic Syndrome has become a key focus for cardiovascular research. The high prevalence of Metabolic Syndrome is largely attributable to diet and lifestyle. The genetic basis for the Syndrome is inconclusive; however variants of key candidate genes have been implicated in the aetiology of the Syndrome (Guettier et al. 2004, Sipilainen et al. 1997).

Variants of key candidate genes have been linked to various components of Metabolic Syndrome. Variants of genes known as single nucleotide polymorphisms (SNP) are strongly associated with heritable phenotypes. It has been suggested that the presence of these variants may be responsible for the high prevalence of dyslipidaemia, hypertension, type 2 diabetes and cardiovascular disease in South Asians (Topol et al. 2006).

The key SNPs that we sought to investigate were present in the fatty acid binding protein gene (FABP2); this gene has been associated with dyslipidaemia (Vimalaswaran et al. 2006), the WNK (with no K [lysine] protein kinase) gene (associated with hypertension) (Tobin et al. 2005), and a SNP associated with the genetic variant of coronary artery disease (CAD), the gene containing the SNP is currently unknown. As South Asians, and Pakistanis in particular, have an increased propensity to suffer from hypertension and dyslipidaemia (and thus more likely to suffer from Metabolic Syndrome), it is probable that the above-mentioned genes may contribute to the increased prevalence of Metabolic Syndrome in South Asians. It was therefore important to investigate the nature of the relationship that exists between the candidate

gene SNPs and the occurrence of Metabolic Syndrome in the female Pakistani population.

As part of this study, we investigated the SNPs of three candidate genes for Metabolic Syndrome. The genes we selected have been most strongly associated with components of Metabolic Syndrome (Georgopoulos et al. 2000). The selected genetic SNPs have been identified in various high risk populations, namely, Pima Indians and South Indians. These SNPs have previously not been investigated in a Pakistani population.

There have been marked differences in the prevalence of Metabolic Syndrome amongst various ethnic groups. The genetic propensity to develop dyslipidaemia, type 2 diabetes and obesity has been shown to be higher in South Asians when compared to other populations (Abate and Chandalia 2003). Comparative studies have consistently shown a higher likelihood of type 2 diabetes and dyslipidaemia in South Asian populations, suggesting a genetic predisposition (Yajnik et al. 2004, Abate and Chandalia 2003).

In this study, SNP genetic investigation was performed. Due to time constraints and resource limitations we limited our research to the three genes that have been most strongly associated with Metabolic Syndrome. To the best of our knowledge, the association between our selected candidate genes and Metabolic Syndrome has not been investigated previously in a Pakistani population. As such, an investigation into the genetic variations of the FABP2, WNK4 and the CAD (SNP) and the association between Metabolic Syndrome and Pakistani women was considered necessary. Our study aimed to investigate the presence of genetic variants of the FABP2, WNK4 and CAD (SNP) in our subject population

A discussion of the SNP of the genes we chose to investigate follows.

## 4.2 The FABP2 genetic SNP

The FABP2 gene has been proposed as a candidate gene for Metabolic Syndrome and type 2 diabetes as products of this gene are involved in lipid absorption and metabolism and therefore may be associated with various components of Metabolic Syndrome (Vimalleswaran et al. 2006, Albala et al. 2004).

The FABP2 gene encodes intestinal fatty acid binding protein which is expressed only in the intestinal cells (in the cytosol of columnar absorptive epithelial cells of human small intestine). Intestinal FABP2 is the member of the intracellular lipid binding protein. FABP2 is believed to bind and transport long fatty acids in the cytoplasm of the intestinal cells. The protein contains a single ligand-binding site that non-covalently binds saturated and unsaturated long-chain fatty acids with high affinity; as such this protein plays an important role in the intercellular transportation of fatty acids (Albala et al. 2006). These long fatty acid chains are required by cells to maintain the cell membrane and as a metabolic substrate, as a precursor of cell signalling and as a mediator of gene expression. In order to perform all these tasks, fatty acids are required to enter and leave the cell in an efficient and effective manner (Topol et al. 2006).

The FABP2 gene consists of 3.4kb and is located on chromosome 4 (4q28-4q31). A polymorphism has been observed in the coding sequence of the FABP2 gene which alters the FABP2 protein. A single nucleotide base substitution in the coding sequence has previously been reported that encodes an amino substitution in the protein. A common polymorphism observed is the Guanine to Adenine (G→A) substitution at codon 54 which results in the substitution of the amino acid threonine for alanine (Ala54→Thr54) (Baier et al. 1995). This polymorphism is associated with increased triglyceride transport from the intestinal cells. Studies have suggested that Thr54 allele carriers have a two-fold greater affinity for long chain fatty acids than Ala54 carriers, as the FABP2 protein increases its affinity for long-chain fatty acids. Furthermore

intestinal cells expressing the threonine form of fatty acid binding protein transport long fatty acid chain and secrete triglycerides at significantly higher rates than those expressing the alanine form of this protein (Pratley et al. 2000). A variation in the transportation and metabolism of fatty acid regulation serves as the basis for dyslipidaemia and insulin resistance, both major components of Metabolic Syndrome (Pratley et al. 2000).

Overall, the literature suggests that polymorphisms in the FABP2 gene could contribute to the higher prevalence of Metabolic Syndrome in South Asians. Indian population carriers of the Thr54 allele had higher fasting plasma triglyceride concentrations when compared with Ala54 carriers (Boullu-Sanchis et al. 1999, Vimalaswaran et al. 2006). The FABP2 gene has been associated with Metabolic Syndrome in South Indians as well as other ethnic populations.

#### **4.3 The WNK genetic SNP (rs880054)**

Metabolic Syndrome has been closely associated with hypertension. Studies have suggested that the occurrence of hypertension may have a genetic link (Wilson et al. 2003). Studies investigating the genetic basis of hypertension are inconsistent in their findings, as hypertension is a complex trait and many genes are suggested to contribute to its occurrence (Erich et al. 2003). Studies have identified various candidate genes that are associated with hypertension to an extent; however, no such gene has been shown to contribute substantially to the development and occurrence of hypertension in the general population. Studies also reveal that South Asians are more likely to suffer from hypertension, when compared to the general population. South Asians are also more likely to suffer from Metabolic Syndrome when compared to the general population (Williams et al. 1993).

The WNK gene encodes a protein in the WNK family of kinases. The WNK kinases are a small group of serine-threonine kinases with unique catalytic domain that lack the lysine which is present in other kinases in their sub domain 11 residue (associated with ATP). However lysine (K) is replaced by cystine (C), therefore encoded with no K (WNK) (Turner et al. 2005). The WNK gene consists of four members: WNK1, WNK2, WNK3 and WNK4. WNK kinases are regulatory proteins and predominately expressed in distal convoluted and collecting ducts of kidney. WNK kinases are associated with renal electrolyte homeostasis and belong to a protein kinase super family of serine-threonine with no lysine K. A single nucleotide base substitution in the coding sequence has previously been reported. A common polymorphism observed is Cytosine / Thiamine (C/T). This mutation in WNK1 protein has been shown to play a role in the prevalence of hypertension in the general population; studies have identified mutations in the genes encoding WNK1 and WNK4 proteins which are associated with hypertension. Mutations in WNK kinase family have been associated with an autosomal-dominant form of inherited hypertension, known as Gordon's syndrome (Tobin et al. 2005)

The WNK1 and WNK4 genes have been identified as candidate genes for the occurrence of essential hypertension as many studies (primarily in the UK, USA and France) on various population sibling pairs, have strongly suggested that a region on chromosomes (12,17q), harbouring key genes, plays an important part in blood pressure regulation (Julier et al. 1997, Baima et al. 1999). The WNK1 gene maps to chromosome 12 and is encoded by 28 exons which span over 150 kb genomic DNA. To date, over 100 variances have been found on WNK1 gene. There are 9 common variants of the WNK1 gene that are associated with hypertension in humans (Tobin et al. 2005). WNK1 and WNK4 have been identified in association with hypertension. We sought to investigate the presence of a SNP in the WNK1 gene in our target population. As the

Pakistani population has been classified as a high risk population for cardiovascular disease (Abate and Chandalia 2003) and its risk factors, we investigated the SNP which has been shown to be closely associated with cardiovascular disease and its risk factors, particularly hypertension. Many participants in our study suffered from hypertension

#### **4.4 The CAD genetic SNP (rs1333049)**

Metabolic Syndrome has been closely associated with cardiovascular disease including coronary artery disease. Coronary artery disease and its main complication atherosclerosis, have shown a genetic basis (Samani et al. 2007, Helgadottir et al. 2005). Genetic variants are thought to influence the risk of CAD, both directly and through effects on known CAD risk factors including hypertension, and elevated levels of lipids (Dyslipidaemia) and atherosclerosis. Ultimately, these risk factors, if left untreated, lead to the development of cardiovascular disease (Helgadottir et al. 2005).

Intensive research has been conducted on, the molecular basis underlying the inherited component of myocardial infarction but this component has remained largely unexplained. Numerous candidate genes have been implicated, but few displayed reproducible results. However recent genome-wide association studies revealed several novel loci that are strongly associated with cardiovascular disease, coronary artery disease and atherosclerosis. A locus on chromosome 9p21.3 for genetic variants has been identified, and has been shown to be strongly associated with coronary artery disease. Specifically, the 9p21.3 locus itself does single gene which has been implicated in disease identified (Welcome Trust Consortium 2007). However recent studies (Samani et al. 2007) have shown that this locus encodes a large ribosomal RNA which is expressed in atherosclerotic tissues, suggesting that this locus can be associated with candidate genes known to affect cellular senescence, apoptosis, and stem-cell function which could substantially modify the basic models of atherogenesis (Schunkert et al.

2008). Atherosclerosis is a complex and degenerative disease; it is a central characteristic feature of coronary artery disease. In the absence of functional data such hypotheses remain inconclusive.

Coronary artery disease SNP is present on chromosome 9p21.3 a single nucleotide base substitution in the coding sequence has previously been reported, a common polymorphism observed is Cytosine / Guanine (C/ G). This polymorphism has been associated with the start up the enzymatic steps in the leukotrine pathway (oxidation of arachidonic acid to leukotrine) which plays a major role in the production of leukotrine, and ultimately in atherosclerosis. Leukotrines are inflammatory cytokines secreted by various types of inflammatory cells. In this process, inflammatory cells are attracted to the injured site in the blood vessel and are implicated in initiation and progression of atherosclerosis (Assimes et al. 2008).

As Metabolic Syndrome is often closely associated with the development of cardiovascular disease, an investigation into the genetic variants of a gene that may be linked to the development of cardiovascular disease (and thus may eventually cause heart attacks) (Mathieu et al. 2006) was given considerable importance. South Asians in particular are known to be a high risk population for the development of cardiovascular disease. We tried to ascertain if our subjects displayed the genetic variant of the coronary artery disease and whether the presence of the variant rendered our subjects more susceptible to Metabolic Syndrome and cardiovascular disease.

## **4.5 Methodology**

DNA was collected from 57 female Pakistani participants residing in Melbourne, (although 60 women participated in the study, 3 were not available for DNA collection). SNP genotyping was performed via 2 different methods: for the determination of FABP2: genetic variant thermal-cycler PCR was used followed by gel electrophoresis. For the determination of WNK1 and CAD genetic variant, real time PCR was used (refer to section 2.9).

## **4.6 Results**

### **4.6.1 FABP2 SNP Genetic Analysis**

In this study all data is derived from 57 participants as 3 participants were moved to Sydney. Our study has shown an association between the Metabolic Syndrome related Ala54→Thr54 and Thr54→Thr54 genetic variant (Topol et al. 2006) of the FABP2 and our subject population (refer to Table 4.1). 25 (44%) of our subjects were homozygous carriers for the presence of Thr54→Thr54 genetic variant. (For the detection of homozygous/heterozygous traits refer to section 2.11.3 ) However, the remaining 32 (56%) of subjects showed the presence of the wild type Ala54→Ala 54. We observed that participants that displayed the homozygous Thr54→Thr54 genotype displayed at least 5 components of Metabolic Syndrome by comparison with subjects that displayed the Ala54→Ala54 trait suffered from a maximum of 3 components of the Syndrome.

**Table 4.1** Component Prevalence and Genetic Analysis Summary (FABP2, Thr-Thr, Ala-Thr, Ala-Ala, SNP)

<b>Genotyping</b>	<b>Thr-Thr</b>	<b>Ala-Thr</b>	<b>Ala-Ala</b>
Number (n)	25/57	0	32/57
Percentage (%)	44	0	56
No. Of Metabolic Syndrome Components.	5	0.	3

Distribution of FABP2 SNP (n=57)

#### **4.6.2 WNK1 SNP Genetic Analysis**

For the purposes of this study, hypertension was defined as “the presence of a systolic blood pressure of 135mm/Hg or greater and a diastolic blood pressure of 85mm/Hg or above, or the use of hypertensive medication” (NCEP-ATP-III 2001). Note that no participants were on medication. All of our 57 subjects, participants displayed variants of the WNK1 gene. 19 subjects were homozygous (C-C) carriers and 38 were heterozygous (C-T) carriers. Our results revealed that 35 of our 57 subjects suffered from high blood pressure. Of the subjects that suffered from hypertension, it was established that 19 were homozygous carriers for the presence of polymorphism of the WNK1 SNP. The remaining 16 subjects were heterozygous carriers for the presence of the polymorphism of the WNK1 SNP. Importantly all homozygous carriers (i.e. 19/19) suffered from hypertension, whereas only 16 (i.e. 16/38) heterozygous carriers suffered from hypertension. This suggests that homozygous carriers are more likely to suffer from hypertension when compared to heterozygous carriers.

However, no difference was observed in blood pressure values between homozygous and heterozygous carriers (P=0.281(systolic), 0.121(diastolic) (refer to Table 4.2).

Similarly, no difference was observed in the number of Metabolic Syndrome components present when homozygous and heterozygous carriers were compared.

**Table 4.2** Hypertension and Genetic Analysis Summary (WNK1 SNP)

<b>Genotyping</b>	<b>Homozygous (C-C)</b>	<b>Heterozygous (C-T)</b>
Number (n)	19	38
Percentage of population (%)	19/57 (33%)	38/57 (67%)
Number and Percentage of carriers that suffered from hypertension.	19/19 (100%)	16/38 (42%)
Average Blood Pressure (Systolic, Diastolic.) mmHg	136.85 ± 0.98, 88.81 ± 0.72	135.95 ± 0.95, 88.79 ± 0.69
No. Of Metabolic Syndrome Components.	3 or 5	3 or 5

Distribution of WNK1 SNP (n=57)

#### **4.6.3 Coronary Artery Disease SNP Genetic Analysis**

Our study confirmed the presence of the coronary artery disease SNP in all participants. 15 of our subjects were homozygous (C-C) for this trait; the remaining 42 were heterozygous (C-G). Of our 57 participants, 50 of suffered from high triglyceride levels and 36 suffered from high cholesterol levels. We observed that all homozygous carriers had high cholesterol and triglyceride levels; furthermore, the prevalence of high cholesterol and triglyceride levels was significantly less in the heterogeneous group. Thus we can suggest that the coronary artery disease SNP may be associated with dyslipidaemia; we further suggest that homozygous carriers are much more likely to

show high cholesterol or triglyceride levels when compared to heterozygous carriers. Additionally, homozygous carriers are more likely to have higher cholesterol ( $P < 0.05$ ) and triglyceride ( $P < 0.05$ ) levels when compared to heterozygous carriers (refer to Table 4.3). No differences were observed in the number of Metabolic Syndrome components present in heterozygous and homozygous carriers.

**Table 4.3** Coronary Artery Disease Genetic Analysis Summary (CAD SNP)

<b>Genotyping</b>	<b>Homozygous (C-C)</b>	<b>Heterozygous (C-G)</b>
Number (n)	15/57	42/57
Percentage of population (%)	26%	74%
Number and Percentage of carriers that suffered from elevated cholesterol levels	15/15 (100%)	21/42 (50%)
Number and Percentage of carriers that suffered from elevated triglyceride levels	15/15 (100%)	35/42 (83%)
Average Cholesterol Level	7.5 mmol/dl	6.5 mmol/dl
Average Triglyceride Level	3.5 mmol/dl	2.5 mmol/dl
No. Of Metabolic Syndrome Components.	3 or 5	3 or 5

Distribution of CAD SNP. (n=57)

#### 4.6.4 Discussion

Many studies have reported that an association exists between the presences of genetic variants for the components of Metabolic Syndrome with the development of the Syndrome itself. Unfortunately there are no such studies that are based on a Pakistani population.

Out of 57 participants, 25 displayed all 5 components of Metabolic Syndrome. The remaining 32 participants displayed 3 components. Of the 25 participants that displayed 5 components, all 25 were homozygous for the FABP2 genetic variant, 16 were homozygous for the WNK1 genetic variant and 15 were homozygous for the CAD genetic variant. All our participants displayed SNPs of candidate genes; this was of little significance, greater differences were dependent on whether the variant was expressed as a homozygous or heterozygous trait. We can associate homozygous carriers of the FABP2 presence of all 5 components of Metabolic Syndrome, we can associate homozygous carriers of the WNK1 SNP with an increased likelihood to suffer from hypertension and we can associate homozygous carriers of the CAD SNP with increased cholesterol and triglyceride levels, an elaboration of these associations follows.

All of our subjects showed a SNP for the FABP2 gene. We observed that all 32 heterozygous carriers displayed 3 components of Metabolic Syndrome, while all 25 homozygous carriers displayed 5 components. We can therefore associate homozygous carriers with an increased propensity to show more components of Metabolic Syndrome (and thus the propensity to display the Metabolic Syndrome in greater complexity).

Our results link the WNK1 SNP to hypertension in our target population. Of our 57 subjects, 19 carried the homozygous trait and 38 the heterozygous trait for the WNK

polymorphisms. All homozygous carriers displayed hypertension, whereas only 16 of heterozygous carriers showed hypertension. These figures suggest that while there is no clear link between the presence of WNK1 polymorphisms and the occurrence of hypertension in this population, there is an increased likelihood for the homozygous carriers to display hypertension when compared to a heterozygous carrier. It can be suggested that the occurrence of hypertension in this population may not necessarily be associated with a single gene; it is likely that other genes as well as environmental factors influence the occurrence and development of hypertension. We were also unable to link SNP of the WNK1 gene with an increased propensity to suffer from more components of Metabolic Syndrome.

The influence of the CAD genetic variant is hard to gauge, as atherosclerosis itself is difficult to measure and evaluate. In our population no participants reported any form of cardiac event. As our study did not investigate the presence of coronary artery disease, we cannot make any definite conclusions. We can suggest that the CAD genetic variant can influence the degree of severity of hypercholesterolemia: of the 36 participants who had high cholesterol, 15 were homozygous carriers of the CAD genetic variant and 21 were heterozygous carriers. We observed that homozygous carriers had a higher cholesterol level (7.5 mmols/dl,) when compared to heterozygous carriers (6.5 mmols/dl,  $P < 0.05$ ). Of the 50 participants who had high triglyceride levels, 15 displayed the homozygous trait and 33 the heterozygous trait; homozygous carriers had a higher triglyceride level (3.5mmol/dl) when compared to heterozygous carriers (2.5mmol/dl,  $P < 0.05$ ). Thus homozygous carriers had high cholesterol and triglycerides levels; furthermore, they are likely to show higher cholesterol and triglyceride levels when compared to heterozygous carriers who also showed higher cholesterol and triglyceride levels. It is thus possible that a polymorphism of the CAD gene can influence factors

determinative of atherosclerosis, particularly cholesterol and triglyceride levels, as these factors contribute to dyslipidaemia.

Although our participants may be genetically susceptible to the development of Metabolic Syndrome, our diet and lifestyle intervention results suggest that the components of Metabolic Syndrome can be reduced. It is again submitted that the presence of Metabolic Syndrome is largely determined by the interplay between genetic and environmental factors.

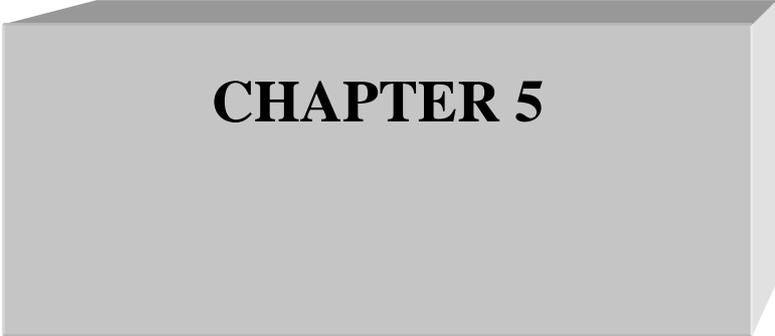
#### **4.7 Conclusion**

Our SNP analysis results suggest that the female Pakistani population investigated has an increased genetic propensity to suffer from components of Metabolic Syndrome. SNP of the three genes we investigated were present in this population.

Our results do not conclusively establish a genetic basis for Metabolic Syndrome; however, we can conclude that variants of the key candidate gene have an impact on the development of the components of Metabolic Syndrome. It can be concluded that the FABP2 gene is associated with the severity and complexity of Metabolic Syndrome. We cannot strongly associate the WNK1 gene with the presence of hypertension; however, we have established that homozygous carriers of this SNP are much more likely to show hypertension when compared to heterozygous carrier.

We cannot clearly link the CAD genetic variant with coronary heart disease in our subject population; however, it is likely that this gene has some influence on cholesterol and triglyceride levels and the occurrence of dyslipidaemia as homozygous carriers of this SNP are more likely to suffer from higher cholesterol and triglyceride levels, in greater severity, when compared to heterozygous carriers. It is well documented that sufferers of Metabolic Syndrome who display all the components of the Syndrome are

more likely to suffer from cardiovascular disease and type 2 diabetes when compared to sufferers who possess two components or less (Misra et al. 2007). Further investigation into the SNP we investigated, particularly the investigation of the difference between heterozygous and homozygous carriers of the SNP, would be beneficial in establishing the future direction of this area of research. As current research suggests, it is likely that the development of the components of Metabolic Syndrome is the result of the interplay between various genes and key environmental and psychosocial factors, as discussed in the next chapter.



**CHAPTER 5**

**STUDY 3 ETHNOGRAPHIC**

## **5 ETHNOGRAPHIC STUDY**

### **5.0 Predisposing, reinforcing and engaging determinants of changes in diet and physical activity in migrant Pakistani women.**

#### **5.1 Introduction**

Psychosocial factors such as chronic stress, loneliness, social isolation, health status, disease susceptibility, beliefs, attitudes, social trends and social situations have been associated with Metabolic Syndrome (De Vogli et al. 2007). Psychosocial factors have been suggested to play a role in metabolic deregulation (Vitaliano et al. 2002), for example, psychosocial distress has been associated with greater fasting blood glucose, elevated blood pressure and central obesity (De Vogli et al. 2007, Bush et al. 2001, Jonnalagadda and Diwan 2005). Furthermore, psychosocial factors have been suggested to influence behavioural factors, such as physical inactivity and emotional eating (De Vogli et al. 2005, Syed et al. 2006).

A straight forward approach to reduce the risk of chronic diseases in migrant population is to modify lifestyle, which includes changing dietary patterns and introducing/increasing physical activity (Stone and Saxon 2005). Diet and lifestyle modification approaches are also required in order to reduce the severity of chronic diseases. In order to design such an approach, it is crucial to understand the cultural and social aspects in the target migrant population, it is also important to pay attention to the gradual changes in the behaviour of the population, due to acculturation. Many researchers have suggested that the identification of cultural influences and ethnic dietary practises is essential, particularly in the development of an effective intervention strategy. Recently researchers have paid great attention to the psychosocial determinants of diet and lifestyle changes in migrant populations. The psychosocial determinants influence dietary change and food preference (Jonnalagadda and Diwan 2005, Syed et al. 2006).

### **5.1.1 Aim**

The main objective in this part of the study was to obtain qualitative information on the views and perceptions of the participant women on diet and physical activity and how various factors have influenced change in their dietary habits since migration. A qualitative approach was used to gather the views, thoughts and beliefs of the migrant women in regards to dietary and lifestyle change.

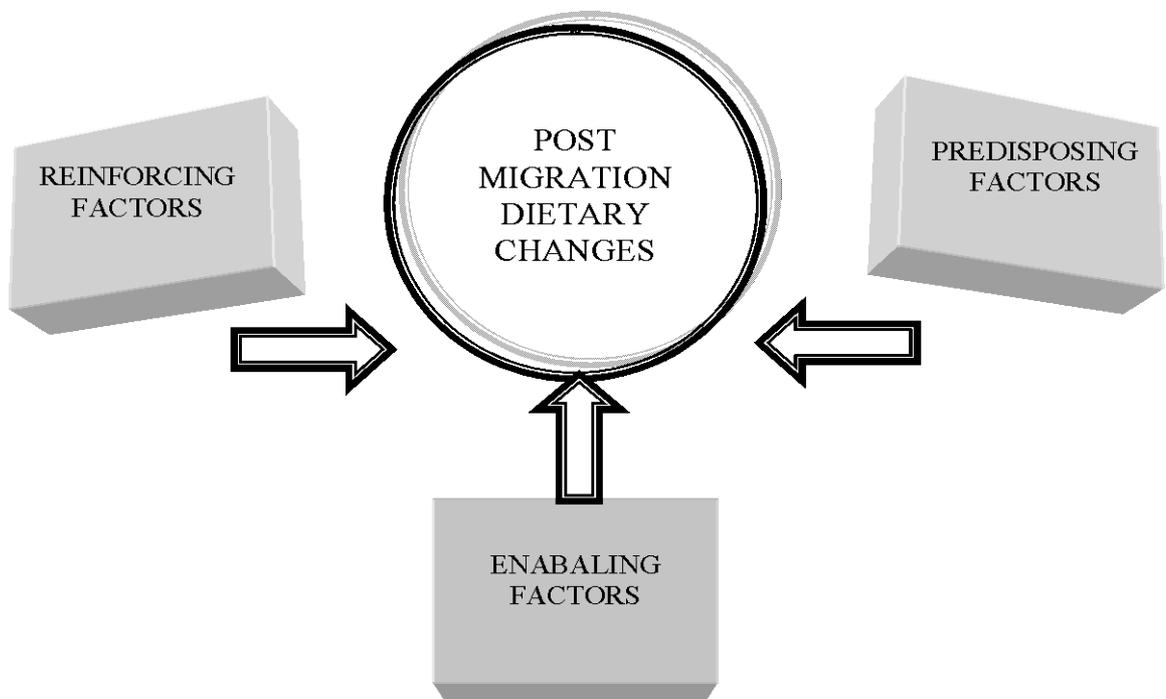
### **5.1.2 Methodology**

Cultural anthropology (a field of anthropology that examines culture as a meaningful scientific concept ) and ethnography (methodological strategy used to provide descriptions of human societies) served as the basis for our investigation of qualitative factors. In order to organise the psychosocial factors that became apparent in our research through voluntary conversation with participants, a record was kept by the researcher of discussions and questions about diet and physical activity from the intervention mini-focus groups. These comments were transcribed and analysed thematically.

These analyses explored the nature and scope of participants' experiences. Major themes were identified. The theoretical framework for the study was the PRECEDE – PROCEED model described in next section refer to Figure 5.1. This model has successfully been used on migrant Israeli, Chinese (Satia-Abouta et al. 2002) and Pakistani migrant populations (Mellin-Olsen and Wandel 2005). The PP model is founded on epidemiology, behavioural and social sciences. The model suggests that health risks emerge from multiple risk factors, thus, as health risks are determined by various factors, these factors affecting behaviour can be broadly grouped as predisposing, reinforcing and enabling constructs. These constructs are used in educational diagnosis and evaluation. Predisposing factors are defined as “any

characteristics of a person or population that motivates behaviour prior to the occurrence of the behaviour” (Green and Kreuter 1999). Reinforcing factors are defined as “any reward or punishment following or anticipated as a consequence behaviour, serving to strengthen the motivation of the behaviour after it occurs” (Green and Kreuter 1999). Enabling factors are defined as “any characteristics of the environment that facilitates action and any skill or resource required to attain a specific behaviour (Green and Kreuter 1999, Gielen and McDonald 2002).

**Dietary changes according to the precede-proceed (pp) mode:**



**Figure 5.1** Psychosocial Determinants for Shift in Dietary Pattern and Preference in Migrant Women (Gielen and McDonald 2002)

### 5.2.1 Discussion

The following discussion relates to statements made to the researcher by participants as part of the intervention phase.

#### 1. Food attitude and traditional values: food is important (Pre-disposing factors)

Through the course of our research differing views on diet and lifestyle emerged. However, a common attitude towards food was seen in the focus group, namely, that food is important and is part of the Pakistani culture; it is an integral part of socialisation. It was also observed that hospitality plays a major role in the Pakistani social/food culture. Notably, the role of hospitality in immigrants is important, for example, Italian immigrants are also known to be very hospitable (Bush et al. 2001). Hospitality is thought to be a big part of many cultures (particularly the Pakistani culture) as it tends to reflect on the social status and well-being of a family (Williams et al. 1993). A woman who prepares good food and a variety of dishes is thought to be a good wife, who works and labours hard and thus upholds the family reputation. Furthermore, a family that serves a variety of dishes to its guests is considered to be prosperous. Conversely, if only a few dishes are offered, or if the dishes served are of poor quality, the family is seen as being disrespectful towards its guests and will also be considered miserly by community.

“We often have social gatherings with our family friends, at least two or three times a week. Everyone enjoys the food and it is a good way to socialise” Participant 16

Many of the participants suggested that traditional foods were a big part of diet and lifestyle for them. Although, as mentioned earlier, strong notions of hospitality are present in many migrant populations, it is especially strong in migrant Pakistani populations (Mellin-Oslen and Wandel 2005). Many women mentioned that they felt

lonely as they lacked the family solidarity that exists in Pakistan. Instead, Pakistani families will socialise and eat large traditional meals almost every weekend, on top of the many traditional foods consumed during the week, and this could lead to the excess consumption of food, sweets and soft drink. A typical Pakistani gathering will involve a large number of guests (a minimum of 20 guests is not uncommon). Once all the guests have arrived, food is served; there are no portion sizes guests/family help themselves to food directly from large bowls or the cooking pot. After seconds and often thirds, the family and guests help themselves to a large variety of sweet dishes, traditional Pakistani tea (thick and creamy) is served at least 2-3 times during such a gathering.

“When we invite guests, I try and make very nice, prestigious dishes such as chicken Biryani (spicy rice with chicken), Tandoori chicken, Nihari (beef curry delicacy) and Nargasi Kofta (egg-filled meat ball curry). This way, I respect the guests and our family will not be seen as money savers.” Participant 24

“I know that eating a lot at almost every social gathering isn’t a good thing, we eat far too much food than we ought to, however, if I don’t eat I may offend the hosts, actually, I will offend the hosts and I mean no disrespect, I would hate it if I were to invite a family over for dinner and they only ate a little bit!” Participant 45

## **2. Traditional beliefs about food and health**

Many of the participants believed that specific foods have certain properties. The most common properties that were mentioned were that of “hot and cold.” It is believed that hot foods are very important for health and well-being; these foods include, meat, eggs, butter, nuts, certain herbs, ghee, homemade sweets etc. These foods are thought to be very healthy for growing children, pregnant women and elderly people. Subjects suggested that hot foods should be consumed more in the winter months in order to maintain ongoing good health. Cold foods include drinks, dairy products, juices and

fruits. These foods are thought to have cooling properties and are consumed more in summer, although, unlike hot foods, they are not believed to have any specific health benefits (Mellin-Olsen and Wandel 2005).

“Eggs and nuts should not be consumed in summer as they are a hot food, it is better to eat plenty of yoghurt and milk as these are cool foods.” Participant 33

### **3. Nutritional understanding in the subject population**

Data on the Pakistani population and its interpretation of ‘western’ nutrition and nutrition messages about food and health is lacking. However, a few studies have been conducted on South Asians and their attitudes towards food; this data is primarily based on Indian populations in the UK. Of the studies that have been conducted, controversy exists in terms of the populations’ beliefs and understandings, as well as their awareness of nutritionally related diseases. Beishan and Nazroo 1997 suggest that there is a general awareness of nutritionally related disorders in South Asians populations, whereas (Lip et al. 1995) have indicated that migrant Indian women residing in Birmingham, UK, have very little knowledge about cholesterol and diet- related disease. More research is required in order to properly determine the South Asian perspective on nutritionally related diseases.

Our study suggests Pakistani women residing in Melbourne generally lack nutritional knowledge. Although many of the women were aware of the fundamentals of good nutrition, many did not place importance on healthy eating. The women did not have in depth knowledge of nutrition and held many incorrect beliefs. In Pakistan, little emphasis is placed on nutrition knowledge and the majority of the population would lack information on various nutritional concepts. Therefore, although the women were aware that the excess consumption of foods, such as pizza or fish and chips could be

detrimental to long term health, they were unaware that the excess consumption of foods such as poultry, meat and dairy can also have a negative impact on health.

“Back home (In Pakistan) nobody cares about healthy eating; you simply eat food that tastes good or food that is affordable.” Participant 3

“It is clear that you shouldn’t eat too much junk food, but you can eat plenty of natural foods, such as poultry or meat, these are good for you.” Participant 8

“One should limit their consumption of junk food and consume wholesome home cooked foods in large amounts instead” Participant 22

Following the observations of food attitude and behaviour in the participants, it became clear that attitudes towards food, the consumption of traditional foods, and the social aspects surrounding the consumption of such foods, is clearly having an adverse nutritional impact on the Melbourne Pakistani population Melbourne, particularly regular social gatherings. Although such gatherings are socially important, they are perhaps detrimental in terms of nutrition and health; such gatherings result in an excess consumption of energy. These attitudes are believed to have contributed towards the poor health seen in this migrant population prior to the intervention. Therefore, the issue is merely one of education; this population was very receptive to nutritional information and the correction of bad habits.

“Excessive food is consumed at our social gatherings, everyone eats plenty and it is strange if you eat little, or tell others that you are dieting.” Participant 1

#### **4. Traditional and religious beliefs**

According to our observations, most of the Melbourne Pakistani community have very strong traditional beliefs in terms of diet. All the women were very religious and many quoted the Quran and Hadeeth (sayings and actions of the Prophet Muhammad). Some of the women said:

“The Quran mentions that olives, honey and dates are good and you should consume these foods when possible.” Participant 50

“You should limit your meat consumption and exercise early in the morning as well as in the afternoon.” Participant 37

When Participant 37 was questioned further, it was revealed that she had indeed limited her meat consumption, but was unable to exercise due to various constraints (refer to section 6.3 for further details.) As such, this intervention aimed to provide participants with ample opportunity to engage in physical activity.

## **5. Attitudes to body size and shape**

A general misconception that was observed in the population was that of a large body size. Many of the participants suggested that a larger body size (i.e., a person carrying more body fat) is an indication of good health, particularly in the case of children. In fact, it was suggested that a thin child should be fed extra in order to “fatten them up so that they can reach a healthy weight” Participant 19. Furthermore it was suggested that a person that appears to be “well-fed” is usually of good status with a strong socio-economic background. However, some of the participants suggested that although being overweight is not a good thing, neither is being too thin (or underweight.) These women also suggested that although it is necessary to maintain a healthy weight at a young age, once a certain age is reached (most suggested 40) nothing can be done and diseases will generally start to show and therefore no precautionary measures are required in their prevention. It was suggested that health starts to decline rapidly and diseases such as arthritis, cardiovascular disease and diabetes may become apparent (Greenhalgh et al. 1998, Chowdhury et al. 2003).

“Children should eat a variety of different foods in order to achieve a healthy physique, it is okay for kids to look fat, this means they are well fed, skinny kids are unhealthy and get sick easily.” Participant 12

“Bigger kids are healthy, thin kids are underfed and more susceptible to diseases and illness.” Participant 55

#### 5.4 Reinforcing factors

##### **5.4.1 Cooking: a reward based system**

Many of the women stated that purchasing good food and preparing meals was enjoyable for them; particularly so if they were to receive praise from their friends and family. Thus many of the participants stated that cooking food in a social context and being seen as a good cook was important to them. Some participants suggested that they enjoyed being “in control” of food and diet, a big part of their life.

“I enjoy it when I make a nice dish and either my guests or family make nice comments about it.” Participant 19

“I am famous for my Jelebi (Pakistani sweet). I absolutely love it when people ask me to make it on special occasions or when I am invited over for dinner. Sometimes, I’ll be at a social gathering and many people will ask me whether I’ve made my Jelebi or not.” Participant 5

“Sometimes I get tired of cooking, but I feel like I am expected to cook for guests and the family, I can’t make the family look bad.” Participant 31

Participants also informed us that they made many dishes at the request (or persistent hassling) of their children. The demands of children were a key reason behind change in diet, many of the women learnt to cook new western type foods to keep their children happy. This also suggests that the women were adaptive to change; this was a positive attitude for the purposes of our diet and lifestyle modification.

#### **5.4.2 The effect of loneliness and stress**

Subjects stated that loneliness and stress has resulted in many of the women cooking and eating in their spare time. Participants informed us that they often felt lonely at home, when their husbands are at work and the children at school. They also felt depressed and stressed about life in Australia and their families back in Pakistan.

“In Pakistan, there is always someone home with you; everything is done as a family. If everyone in your family is busy, at least, the neighbours will come over, sit down for some tea and a chat. Here I don’t even know my neighbours well and I haven’t got any family” Participant 49

“I get very bored and lonely during the day, the only solution is to do housework, prepare food and eat it. I often eat when I am bored, lonely or depressed and this has resulted in weight gain and poorer health.” Participant 60

“I have many worries, about myself, my family, my parents and siblings back home and I feel as if I can’t talk to anyone about it, I feel isolated. So I guess this has resulted in me eating a lot more food than I used to, or need to.” Participant 16

Subjects also stated that they felt stressed under community expectations to develop good Pakistani kids in the Australian society.

“I get stressed thinking about how my kids will turn out, everyone expects them to maintain their Pakistani culture, but in this day and age it is hard. Already, families talk about other family’s problems and disgraces behind their backs. I know it seems like a lot, but this stress also contributes to bad eating habits in my opinion” Participant 58

## **5.5 Enabling factors**

### **5.5.1 Budgeting costs**

In the traditional Pakistani culture women are responsible for purchasing and preparing meals, as well as other domestic household duties. In comparison, men play the role of the bread winner and are required to provide for the family. Young girls begin helping their mother with household duties at as young as 10 years old.

As women are responsible for purchasing food items and meal preparation, they are allocated a limited food budget in which they must meet the family's requirements. The women are often in pursuit of "bulk buys" as they get more products per dollar. Therefore, the women spend a lot of time searching for a new or "cheap outlet for their bulk buys, furthermore, they enjoy shopping for food as it is a good pastime, or leisure activity in which they can have fun with their friends. Many women mentioned that they enjoyed shopping as a pastime, as they felt stressed under the budget allocated for food shopping. As a consequence of the aforementioned budget and bulk buying, the nutritional value of foods is almost always neglected in favour of cost factors.

"I'll admit I don't always buy the healthiest option, I usually go for the cheapest option, I would prefer to get more product for less money." Participant 6

"I like looking for a good deal, shopping is a fun way to pass the time when they kids are at school, this sometimes results in me buying too much food though." Participant

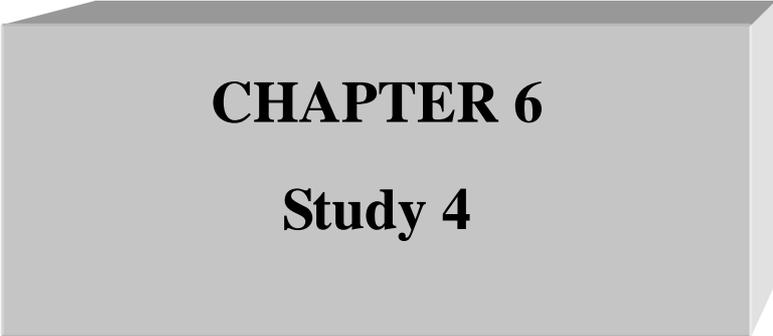
## 5.6 Conclusion

Psychosocial factors may play an important role in the development of Metabolic Syndrome; therefore, in designing an intervention not only should physical activity and diet be taken into account, but also psychosocial influences.

We identified the factors predisposing to lifestyle behaviour change in this population as; traditional values around food, traditional beliefs around food and health, interpretation/understanding of nutrition messages, religious beliefs, attitudes to body size. The reinforcing factors in this group were; reward gained from cooking and the effect of loneliness and stress. The enabling factor was budgetary cost.

In this study information pertaining to various cultural beliefs and attitudes towards food were ascertained via voluntary discussion with participants. As such, it is difficult to determine the exact association between psychosocial factors, Metabolic Syndrome and migration. According to literature, stress can be associated with migration, which could possibly contribute to Metabolic Syndrome and cardiovascular disease (Stewart-Knox 2005).

Our participants displayed a number of unsubstantiated beliefs, highlighting the importance of a proper health and nutrition education. It is also important to note that any education must be made in the context of cultural beliefs i.e. developed in a culturally competent way. The beliefs identified in this qualitative study must be considered in developing future interventions in this population. Another important outcome was that the participants indicated that they were experiencing stress and social isolation. The impact of this on Metabolic Syndrome needs to be further explored. The salience of these stressful experience to the women however supports the use with this population of peer-lead group education as a means of engaging the target group and enhancing social networks. These aspects were important in the implementation of our diet and lifestyle intervention, as discussed in the next chapter.



**CHAPTER 6**

**Study 4**

**Culturally appropriate diet and lifestyle intervention**

## **6 DEVELOPMENT OF INTERVENTION**

### **6.0 Culturally appropriate intervention**

#### **6.1 Introduction**

##### **6.1.1 South Asians (Pakistanis) at high risk for Metabolic Syndrome.**

A high risk population for the development of Metabolic Syndrome are South Asian migrants (people from Pakistan, India and Bangladesh). The prevalence of Metabolic Syndrome has been shown to be much higher in South Asian migrants when compared to African-Caribbean and white European migrants (McKeigue et al. 1989). It has been established that Metabolic Syndrome can increase the risk of developing cardiovascular disease four-fold in Punjabi Indians (Lakka et al. 2002). According to reports (primarily from the UK), the prevalence of Metabolic Syndrome ranges from 29-50% in migrant South Asians (Stone and Saxon 2005). It has also been shown that South Asians are twice as likely to develop cardiovascular disease when they have Metabolic Syndrome when compared to others. (Kolt et al. 2007, Balasubramanyam et al. 2008). Although studies on migrant South Asian women are limited, the data that does exist suggests that the rate of mortality due to cardiovascular disease is higher in migrant South Asian women when compared to men (William et al. 1993). As a sub-sample of the South Asian population, Pakistanis tend to have a higher rate of hyperinsulinemia and insulin resistance when compared to people of other ethnic backgrounds. Furthermore, the prevalence of cardiovascular disease in migrant Pakistanis living in the UK, USA and South Africa is 50% higher when compared to the general population of the host country (Misra and Vikram 2004).

### **6.1.2 Risk factors for Metabolic Syndrome in South Asians (Pakistanis)**

#### **a) Diet**

There are large South Asian communities (Pakistani, Indian and Bangladeshi) in many Western countries, particularly the UK, USA, Australia and Canada. Despite this, little attention has been paid to migrant South Asians' dietary patterns, particularly Pakistanis. The South Asian diet is typically an atherogenic diet, which is high in fat and salt, and low in fibre, with minimal vegetable consumption and excessive carbohydrate consumption (Simmons and Williams 1997, Thomas 2002, Anderson et al. 2005). This atherogenic diet has been associated in South Asians with an increased propensity to suffer from various disorders, such as the components of Metabolic Syndrome, particularly obesity and hypertension (Nicolaou et al. 2006). There is strong evidence to suggest that unhealthy dietary habits/behaviours including the excess consumption of dietary salt have been linked to the occurrence of hypertension in individuals (Stamler 1997, Law 2000, Whelton et al. 2002, Davy and Hall 2004).

#### **b) Physical activity**

It has been well documented that South Asians tend to live a sedentary lifestyle which may be seen as a major contributing risk factor for Metabolic Syndrome and cardiovascular diseases (Tillin et al. 2005).

Physical inactivity is a major contributing factor in certain ethnic high risk populations (such as Indians, Pakistanis and Bangladeshis) in the development of cardiovascular disease (Jafar et al. 2004). The level of physical activity has been reported to be very low in migrant South Asian populations, in comparison with the general population. Not many studies have specifically investigated physical activity levels in a Pakistani population. However, Hays et al. measured physical activity via the use of a pedometer

in a cross-sectional population-based study in the UK. It was reported that 87% of Pakistanis did not meet the guidelines for moderate physical activity levels (minimum of 5 times every week for duration of 30 minutes) (Hayes et al. 2002).

### **c) Migration**

It has been documented that a shift occurs in the types of foods individuals consume on migration. An increased consumption of food, particularly processed energy-dense foods, could possibly expose migrant populations to a higher risk of obesity, Metabolic Syndrome and cardiovascular disease (Ahmad and Frossard 2005, Patel et al. 2006, WHO 2006, Kolt et al. 2007). Strong evidence suggests that, upon migration, migrant populations undergo a transition in dietary pattern; often this transition period results in the formation of poor dietary habits, which in turn leads to obesity.

Studies on populations such as Tokelauana natives and South Asians in New Zealand and UK have suggested that this acculturation in migration results in the degradation of healthy cardiovascular profiles (William 1993, Lee et al. 2001). It has also been shown that the health of migrants generally deteriorates in proportion to the length of stay in the host country (Lee et al. 2001, Kolt et al. 2007). The author highlights the fact that while many Australians are trying to implement a healthier diet, many immigrants are doing the opposite, in that they are adopting “Western” dietary patterns. Such changes result in weight gain and obesity related complications (Lee et al. 2001).

### **6.1.3 The importance of diet and exercise in treating Metabolic Syndrome**

Recently, great importance has been placed on the prevention of cardiovascular disease and Metabolic Syndrome. The World Health Organisation has responded to the rise in these diseases by setting guidelines for the prevention of these diseases by the improvement of diet and physical activity at a population level (WHO 2003). Many

authorities and health professionals have recommended diet and lifestyle therapy as the best non-pharmacological therapy for the treatment of cardiovascular disease and related disorders (NCEP-ATP III 2001, Thompson 2003, Grundy 2005, Eckel et al. 2005, Cameron et al. 2007, Levesque and Lamarche 2008). Recently the American Heart Association/National Heart, Lung, and Blood Institute (Grundy et al. 2006) issued a scientific statement regarding the options for the management of Metabolic Syndrome (WHO 2003, Grundy et al. 2006). This document states that the underlying causes of Metabolic Syndrome are central obesity and insulin resistance; also, physical inactivity and an atherogenic diet were considered as contributing factors. This statement recommended that diet and lifestyle modification should be used as a first line of defence; furthermore, emphasis was placed on the early identification of Metabolic Syndrome sufferers in order to prevent the possible onset of cardiovascular disease. Health authorities generally agree that Metabolic Syndrome should be treated via a multi-factorial approach, with diet and lifestyle modification as the foundation of such an approach.

#### **6.1.4 Culturally appropriate and effective treatments for Metabolic Syndrome**

Over the last two decades many nutrition interventions have been developed aimed at reducing the burden of non-communicable diseases. However, suitable interventions have not been developed for culturally and linguistically diverse populations (Redman 1996).

Although adequate dietary guidelines exist for the general population, many migrant populations do not follow suggested guidelines primarily due to the presence of language barriers and a general lack of importance placed on nutrition (Barnett et al. 2006). Other barriers include economical hardships, addressing culturally specific dietary needs, traditional constraints and religious practices etc. (Yancey et al. 2003).

When considering diet and lifestyle interventions to be implemented on migrant populations, it is essential that interventions undertaken should be culturally competent (Kumanyika 2002, Yancey ET al.2004), and overcome certain restricting barriers. To be effective, health interventions for ethnic populations should employ multiple strategies, be consistent with the principles of cultural competence and should facilitate change in individuals and communities.

To date, no intervention has specifically targeted Metabolic Syndrome in a migrant Pakistani population. Chowdhury and colleagues (Chowdury et al. 2003) have highlighted the importance of the prevention of diabetes, cardiovascular disease, and implicitly Metabolic Syndrome, in South Asians. However these authors also commented on the limited work into improved education of this community in a healthy lifestyle, improved diet and increased physical activity (Chowdury et al. 2003, Kolt et al. 2007).

There have been limited intervention studies on South Asian populations in general investigating some of the contributing components of Metabolic Syndrome. These studies were primarily directed towards type 2 diabetes and many were quite successful. A randomised control trial conducted in the UK on a South Asian population (O'Hare et al. 2004) noted a 58% reduction in the incidence of type 2 diabetes after the implementation of a year-long effective dietary education and pharmacological intervention. Bilingual health workers and diabetes specialist nurses from within the South Asian community were used in the education and enhanced care of subjects. Another UK study on type 2 diabetic South Asians observed a reduction in blood pressure and cholesterol values; this study focused on education intervention. This research suggests that interventions with a focus on diet and lifestyle education can be successful (O'Hare et al. 2004, Diabetes Prevention Program Group 2002). It can be suggested that patients with diabetes, cardiovascular disease or Metabolic Syndrome

will reap health benefits from diet and physical activity interventions provided that these interventions are tailored according to the needs of the target population (Hawthorne et al. 2001, Gordon et al. 2004).( Daskalopoulous et al. 2004). The therapeutic strategy (diet and lifestyle) likely to confer the greatest benefit to a South Asian individual is one of moderate weight loss through regular exercise and dietary restriction. Reduction of abdominal obesity through lifestyle measures can improve all components of the Metabolic Syndrome and is likely to delay the development of both diabetes and atherosclerosis (Gupta et al. 2006).

In the current study, the principles of cultural competence were applied to overcome cultural barriers and sustain behavioural change for Pakistani women to make the lifestyle changes necessary to lose weight and reduce the risk of Metabolic Syndrome.

## **6.2 Framework of the intervention**

We aimed to formulate and implement a culturally competent diet and lifestyle intervention. Our intervention was based on simple approach that was culturally appropriate, convenient and financially feasible. All these factors were embodied in our “Step to Good Health “programme, a straight forward approach to reduce the risk of these diseases by modification of lifestyle, which included changing dietary patterns and increasing physical activity. This was a 12 week educational intervention program focusing on diet and physical activity. The programme combined basic nutrition and health education, as well as behavioural and supportive motivational strategies (such as interactive personal contact with subjects) to help the target group to achieve and maintain dietary change.

It is recognised that “cultural competence” is required when tackling the development of such interventions in culturally and lingually diverse communities. “Cultural

competence” is defined as “a set of congruent behaviours, attitudes and policies that come together in a system, agency, or among professionals and enable that system, agency or those professions to work effectively in cross cultural situations (NHMRC 2005, NHMRC 2006). Previous literature and interactions with our target population indicated that various cultural and gender barriers existed for the participants of our study. Apart from limited English language proficiency, poor access to transport, limited financial resources, family responsibilities and social isolation, our subjects placed little emphasis on physical activity and generally had a poor understanding of diet and nutrition. Furthermore, our subjects did not freely participate in unisex recreational activities and were restricted due to religious/cultural beliefs (Farooqi et al. 2001).

### **6.3 Intervention Details**

#### **6.3.1 Development of the Intervention**

For diet and lifestyle intervention, written materials were delivered as modules. These were adapted from Australian resources, the “Easing the Transition” food and nutrition program for refugees and the “Change of Heart” study, a behavioural programme for the treatment and prevention of cardiovascular disease (Burns et al. 2000).

Our intervention was based on a peer education model in which a facilitator led small groups of migrant women through a specially designed educational and behavioural programme (Burns et al. 2000). Our programme was titled “Step to Good Health”. The facilitator for our programme was a trained nutritionist with expertise in obesity management who has previously conducted interventions of this nature in ethnic populations, namely Dr Cate Burns.

The diet and lifestyle intervention program was implemented over a 12 week period. The program was based on 12 weekly modules, each module with a different focus and

goal to achieve. The goals of the intervention program were multifaceted, including an overall decrease in energy intake and increase in physical activity. Each module consisted of individual dietary counselling and researcher-participant interaction. The modules aimed to increase the participants' understanding of diet and nutrition. On average participants were counselled for 4 hours per week; 3 hours constituted one to one interaction, with the remaining hour of contact occurring via the telephone. The weekly counselling sessions included a half hour introduction to an allocated "objective of the week." The physiological and pathological aspects relating to the objective were then discussed. Later on in the session, the previous week's objective would ordinarily be reviewed, following a discussion on why it was or was not achieved.

The overriding objective of this study was not short-term weight loss, but rather, the achievement of a long term lifestyle change. While only short term effects were measured, follow-up programs are planned to assess the long term effectiveness of the lifestyle changes achieved beyond this research project. In the programme a combination of basic nutrition and health education, as well as behavioural and supportive motivational strategies (such as personal interaction with subjects) were used to help the participants to achieve and maintain dietary and physical activity changes. This strategy was used in order to overcome the barriers previously noted in literature for health promotion in culturally and linguistically diverse populations (NHMRC 2005). In the current study, the principles of cultural competence were applied to overcome the cultural barriers and sustain behavioural change for Pakistani women to make the lifestyle changes necessary to lose weight and improve their health and well-being.

The development of the programme, including the written modules, was based on the principles of cultural competence to facilitate health promotion in culturally and linguistically diverse communities. According to NHMRC 2005, these principles

involve establishing a strong relationship with the target community (in this case the female Pakistani participants) and understanding the culture of the community in depth is important to identify and understand risk factors that a population may be susceptible towards.

In this intervention, cultural competence was achieved by using a bilingual educator/facilitator. Differences in cultural understandings were addressed with the use of a bilingual educator (the researcher) from the same cultural group as the participants. This facilitator had an in-depth knowledge of the Pakistani culture and predominant religion. As our participants generally had poor access to transport, time restrictions and family commitments, the sessions were conveniently held at the participants' homes.

The topics covered in the modules are outlined below please refer to ( Appendix-B-1-B12 , pages,250-319) for further details on each module.

**1) Variety is the spice of life:** This module aimed to emphasise the importance of variation in the types of foods consumed, so as to ensure an overall balanced diet, with an appropriate amount of carbohydrates, fats, protein, vitamins and minerals. Participants were educated about different food groups and how often to consume different types of foods. (Refer to Appendix B-1, page, 250 )

**2) Which fat to choose:** This module aimed to discuss “good” (a food source with low saturated and high unsaturated fat content) and “bad” (a food source with high saturated fat and low unsaturated fat) content fats and how to make healthier food choices. Participants were informed that products such as ghee, coconut oil, palm oil, full-cream milk, cheese, butter, cream, deep fried foods (including samosas and pakoras), and chips tend to have a higher bad fat content. Similarly, participants were advised that olive oil, flaxseeds and fish are sources high in good fat content. A checklist was also

provided to subjects informing them on how to reduce “bad” fats in one’s diet. ( Refer to Appendix B-2, page 257 ).

**3) Achieving a healthy weight:** This module aimed to educate participants about how to achieve a healthy weight, as stipulated in the current WHO guidelines. Participants were informed that the overweight tend to have higher blood pressure and cholesterol levels than normal, further being overweight can be associated with chronic diseases such as cardiovascular and type 2 diabetes. Subjects were advised lowering and maintaining ones bodyweight can reduce blood pressure, cholesterol and plasma glucose levels. The module explained how to lose weight effectively with diet and physical activity modification by lower energy consumption and increased physical activity levels. The module promoted permanent dietary change as opposed to short periods of dieting. Refer to ( Appendix B-3, page 274 ).

**4) Let’s go shopping:** This module aimed to educate subjects about making healthy food choices while shopping. Participants were encouraged to make healthy food choices while shopping (such as purchasing margarine instead of butter, or purchasing low fat dairy products). The module emphasised the importance of reading the ingredient listings and nutrition panels in order to make informed choices. Participants were also taught to be watchful of seemingly healthy products, with high sugar content (such as fruit juice and white bread) (Refer to Appendix B-4, page 274 ).

**5) Cooking up a storm:** The aim of this module was to promote healthier methods of cooking (such as baking instead of frying etc) and positive ingredient modifications (such as using olive oil instead of ghee.) The participants were taught the Three R’s, Reducing, Removing or Replacing ingredients that may be detrimental to health. Refer to Appendix B-5, page 284).

**6) Eating away from home:** This module aimed to assist participants in making health-conscious choices while eating out. Healthy foods that can be consumed while eating out were suggested. Participants were advised that they could request restaurants not to add creamy sauces, for example, to their meals. Subjects were informed that selecting lean meats with vegetables and salads while eating out indeed constitutes healthy eating. ( Refer to Appendix B-6, page 288). .

**7) Fill up on fibre:** This module aimed to educate participants about the importance of fibre in the diet and its role in the body in reducing the high cholesterol. High fibre sources including fruits, vegetables, legumes (i.e. dried peas, dried beans and lentils), oats, oat bran, barley bran and rice bran were identified and discussed. (Refer to Appendix B-7, page 291)

**8) Flavour without salt:** This module discussed the implications of consuming too much salt in the diet; it encouraged subjects to add less salt and make low-salt purchases where possible. Participants were advised that certain foods have a high salt intake, particularly soy sauce, canned soups, stock cubes and snacks (including chips, salted nuts etc.) Participants were taught to purchase foods with no salt, or foods labelled no added salt, low salt and salt reduced. Subjects were encouraged to use freshly ground pepper, fresh or dried herbs, vinegar, lemon juice, fresh mustard or fresh garlic in lieu of salt. ( Refer to Appendix B-8, page 301). .

**9) Water and our body:** This module discussed the importance of drinking plenty of water and the role of water in the body. Participants were informed how much water to drink daily, they were also advised to drink water more often instead of soft drinks, juice etc. Refer to ( Appendix B-9, page 307). .

**10) New food and healthy eating habits:** This module discussed the new foods that subjects encountered in Australia (such as bread varieties, breakfast cereals, and various fast food); it also discussed foods that participants no longer consumed since migration to Australia, as well as whether subjects had formed healthy/unhealthy eating habits. ( - Refer to Appendix B-10, page 312).

**11) Let's understand the food pyramid:** This module aimed to educate subjects about the Australian food pyramid. The food pyramid groups were discussed, as well as the frequency at which various foods should be consumed on a daily basis, such as fruits and vegetables etc. ( Refer to Appendix B-11, page 318)

**12) Revision:** All modules were briefly revised. ( Refer to Appendix B-12, page, 319).

### **6.3.2 Cultural considerations.**

The results of this study (refer to section 6.3.3) indicate that the diet and lifestyle intervention was successful; this can largely be attributed to the degree of cultural sensitivity achieved. In terms of diet this essentially meant that Pakistani-style food was still being cooked, albeit, using healthier ingredients and techniques. For example, ghee (clarified butter) or butter, was replaced with olive or vegetable oil, and subjects were encouraged to grill chicken rather than frying it. In relation to physical activity, as previously discussed, subjects were encouraged to participate in an environment that they found comfortable (not a mixed gender gathering or confined environment) and more importantly, they were encouraged to participate in such activities often.

In our diet and lifestyle intervention, physical activity was clearly an important component and it was essential that this activity be organised in a way which was suitable to the participants. As this Pakistani female population is highly influenced by cultural and religious factors they were restricted from participating in public mixed

health facilities where men were present. This made it difficult to choose a suitable physical activity as no public and not many private health facilities were able to cater for this population's needs. Thus, the first viable option seemed to be privately-owned ladies- only gymnasiums. However, on further investigation, it was revealed that, even at a discounted group rate, attending the gym would not be financially feasible for most of the women.

Our subjects indicated that they would participate or incorporate physical activity into their daily life, but it was essential that such activity take place in a women only environment and at a lower cost to them. Thus, the local Brimbank City Council was contacted and discussions were held with the Mayor of that time. The City Council was requested to establish ladies-only sessions at the local gymnasium, however due to factors such as the availability of the female gym staff and excessive costs, the women-only gym sessions were thus not a viable option for the participants or the local council. It was suggested that women-only swimming sessions would be a more feasible option. In order to organise women-only swim sessions, we were required to lodge an application with the city council and we were required to receive an exemption from the Human Rights and Equal Opportunities Commission for legal purposes, as males could argue that by establishing women-only swim sessions, the leisure centres were actively discriminating against men. After 24 months of negotiation and effort the Brimbank City Council was granted permission to conduct women-only swimming session at local leisure centres in Sunshine and St Albans on a fortnightly basis. Once the swimming sessions were established, the outcome was extremely positive with the majority of our subjects participating, as well as many female members from the general public.

As this arrangement took longer than expected, we organised an alternative type of physical activity. Participants were encouraged to walk daily and were provided with pedometers to record their step counts (refer to section 2.5). The women were motivated

to walk and increase their levels of physical activity as the pedometers were an exciting and interesting gadget for them. Soon after the commencement of physical activity these women strived to reach 10,000 steps. The women reported that they felt proud and excited when they managed to increase their daily step count from day to day as they were continually getting closer to the goal of 10,000 steps per day. The women were requested to walk a minimum of 10,000 steps daily as this amount to roughly 30-40mins of brisk walking; the goal of 10,000 steps aimed to get the participants walking for at least half an hour a day.

Our participants agreed activity or exercise had some importance in leading a healthy life, yet they indicated that it was difficult to manage and incorporate a regular exercise routine into their daily lives. Walking with the use of pedometers was an easy alternative for the participants as it was convenient; participants could monitor their average steps on a daily basis which encouraged them to increase their steps, and thus many participants looked forward to checking their step count at the end of every day. After a few weeks, the participants had informed us that they had formed walking groups which would walk around their surrounding areas daily. We had observed a significant increase physical activity levels and it can be concluded that walking in their own time was an easy option for subjects as they were able to accommodate walking into their daily routines.

Another important factor was that all information was presented to participants in small groups of no more than four women. This was quite time consuming, but it was necessary to educate the individuals about diet and lifestyle modification at a level where they would actually absorb and retain the information. Importantly, in the Pakistani culture it is appreciated if discussions are held at a more personal level, rather than one person addressing a large group.

The diet and lifestyle intervention implemented in this study successfully increased the levels of activity in participants and achieved a reduction in the overall severity of all the major components of Metabolic Syndrome (Refer to section 6.3.3.4). We consider that a prerequisite to the success of this intervention lies in the application of the principles of cultural competence. The programme overcame many of the barriers previously noted in the literature for health promotion in culturally and linguistically diverse populations.

### **6.3.3 Post intervention results**

#### **6.3.3.1 Anthropometric analysis**

Anthropometric measurements for all 60 subjects were taken. Measurements taken included weight, height and waist circumference. The BMI of individual participants was calculated and the average of all 60 participants was calculated and used for statistical analysis. BMI and waist circumference were classified according to WHO Asian specific guideline, where a female waist circumference of 88cm and BMI of 23 are classified as overweight, and a BMI of 25 or over is considered obese (WHO/IASO/IOTF 2000). The results are summarised in Table 6. 1.

Results indicate that prior to intervention, the average BMI of participants was classified as obese and that after the completion of the intervention the average BMI was significantly reduced (Pre:  $30.4 \pm 0.44 \text{ kg/m}^2$   $P > 0.05$  Post:  $28.73 \pm 0.42 \text{ kg/m}^2$   $P < 0.05$ ). We observed an average of 5-6% reduction in body weight in all subjects; thus while overall BMI was significantly reduced; participants were still outside the desirable range for BMI according to WHO standards.

Prior to the intervention, participants of our study demonstrated a high waist circumference. The average waist circumference was significantly reduced on completion of the intervention. Overall we observed a reduction of 5-6% in waist

circumference (Pre:  $107.5 \pm 0.88$  cm Post:  $101.67 \pm 1.50$  cm  $P < 0.05$ ). At the commencement of our intervention 51 of our 60 participants (85%) classified as obese, according to the WHO standards. 3 participants had a normal waist circumference on completion of the intervention. No significant differences were observed from weeks 0 to week 12. All significant differences were observed at the completion of the diet and lifestyle intervention at week 24.

**Table 6. 1** Anthropometric Results Summary (n=60)

<b>Parameter Measured</b>	<b>Week 0 (Control period/ Baseline data)</b>	<b>Week 12 (End of Control period)</b>	<b>P- Value (Week 0-12)</b>	<b>Week 24 (Intervention Completion)</b>	<b>P- Value (Week 12-24)</b>
Body Mass Index (kg/m <sup>2</sup> , SEM)	$30.4 \pm 0.44$	$30.7 \pm 0.44$	0.959	$28.73 \pm 0.42$	<0.05
Weight (kg, SEM)	$75.26 \pm 1.40$	$74.78 \pm 1.39$	0.805	$70.94 \pm 1.42$	<0.05
Waist circumference (cm, SEM)	$107.5 \pm 0.88$	$105.2 \pm 1.78$	0.922	$101.67 \pm 1.50$	<0.05

BMI, body weight, and waist circumference measurements. Week 0 represents baseline data, week 12 represents the beginning of the intervention and week 24 represents the end of the intervention. Data is presented as Mean  $\pm$  SEM.  $P < 0.05$  indicates a significant change due to the intervention. T-test statistical analysis was performed.

### **6.3.3.2 Biochemical analysis**

In this study blood biochemical profiles were measured for all 60 subjects. Results are summarised in Table 6. 2. Our results showed that 36 of 60 (60%) participants had hypercholesterolaemia and 50 of 60 (89%) participants had elevated levels of

triglycerides. Results also indicated that 30 of 60 (41%) participants had hyperglycaemia and 57 of 60 (97%) of participants had hyperinsulinemia.

On the completion of the 12 week intervention period, significant changes were observed in the subject biochemical profiles. Subjects' average cholesterol levels were reduced by approximately 10% (Pre:  $6.65 \pm 0.16$  mmol/L, Post  $5.96 \pm 0.32$  mmol/L  $P < 0.05$ .) Similarly triglyceride levels were reduced by approximately 24% (Pre:  $2.63 \pm 0.09$  mmol/L, Post  $2.01 \pm 0.09$  mmol/L  $P > 0.05$ ). Average plasma glucose levels were reduced by approximately 8% (Pre:  $6.4 \pm 0.10$  mmol/L Post:  $5.9 \pm 0.33$  mmol/L  $P < 0.05$ ), and insulin levels by approximately 46% (Pre:  $45 \pm 6.3$   $\mu$ u/ml, Post:  $24.14 \pm 1.8$   $\mu$ u/ml,  $P < 0.001$ ). No significant differences were observed from weeks 0 to week 12. Participants in this study had shown a significant reduction in most of the biochemical risk markers for Metabolic Syndrome. All significant differences were observed at the completion of the diet and lifestyle intervention at week 24.

We did not observe any patterns or correlation in changes of the biochemical profile, for example, participants who had significantly reduced their cholesterol levels, did not necessarily reduce their glucose or insulin levels; similarly, participants who had significantly reduced their triglyceride levels did not necessarily reduce their cholesterol or glucose levels. We cannot establish a sub-group in which we observed significant changes across all biochemical factors analysed.

**Table 6. 2.** Biochemical Analysis Results Summary (n=60)

<b>Parameter Measured</b>	<b>Week 0 (Control period/Baseline data)</b>	<b>Week 12 (End of Control period)</b>	<b>P- Value (Weeks 0-12)</b>	<b>Week 24 (Intervention Conclusion)</b>	<b>P- Value (Weeks 0-24)</b>
<b>Total Cholesterol</b> (mmol/L, SEM)	6.65 ± 0.16	6.78 ± 0.15	0.591	5.96 ± 0.32	<0.05
<b>HDL Cholesterol</b> (mmol/L, SEM)	0.87 ± 0.06	0.96 ± 0.07	0.349	1.19 ± 0.20	0.265
<b>LDL Cholesterol</b> (mmol/L, SEM)	1.55 ± 0.07	1.47 ± 0.07	0.421	1.24 ± 0.06	0.013
<b>Triglycerides</b> (mmol/L, SEM)	2.63 ± 0.09	2.49 ± 0.09	0.259	2.01 ± 0.09	<0.05
<b>Plasma Glucose</b> (mmol/L, SEM)	6.4 ± 0.10	6.47 ± 0.12	0.613	5.75 ± 0.10	<0.001
<b>Plasma Insulin</b> (µU/ml, SEM)	56.25±1.86	52.88±2.26	0.349	28.90±0.95	<0.001

Total cholesterol, HDL/LDL-C, triglyceride, plasma glucose (mmol/L) / insulin measurements (µU/ml). Week 0 represents baseline data, week 12 represents the beginning of the intervention and week 24 represents the end of the intervention. Data is presented as Mean ± SEM. P< 0.05 indicates a significant change due to the intervention. T-test statistical analysis was performed.

### **6.3.3.3 Blood pressure and physical activity analysis**

In this study blood pressure was measured for all 60 subjects. Results are summarised in Table 6.3. Participants were considered hypertensive if their blood pressure was greater than 130/85 mmHg. On the completion of the 12 week intervention period, significant reductions were also observed in the subject blood pressure values. Our results revealed that prior to the intervention 35 out of 60 (59%) participants were hypertensive. We observed approximately an 8% reduction in systolic blood pressure (Pre: 136 ± 1.0 mmHg, Post: 126 ± 1.0mm/Hg P<0.001), and a 9% reduction in diastolic blood pressure

(Pre:  $89.1 \pm 0.8$ mm/Hg  $P=0.1$ , Post:  $81.2 \pm 0.6$ /Hg  $P<0.001$ ) in all 35 hypertensive participants: furthermore, 15 were now classified as normotensive.

On the completion of the 12 week intervention period, significant changes were also observed in the subjects' physical activity levels. Our results revealed that prior to the intervention 58 of our 60 (98%) participants were sedentary. We observed a 231% increase in physical activity (Pre:  $4084.1 \pm 115.9$ steps/day, Post:  $13523 \pm 1957$   $P<0.001$ ). Thus at the completion of the intervention, participants were no longer classified as sedentary. No significant differences were observed from weeks 0 to week 12. All significant differences were observed at the completion of the diet and lifestyle intervention at week 24.

**Table 6. 3** Blood Pressure and Physical Activity Analysis Results Summary

<b>Parameter Measured</b>	<b>Week 0 (Control period/Baseline data)</b>	<b>Week 12 (End of Control period)</b>	<b>P- Value (Weeks 0-12)</b>	<b>Week 24 (Intervention Conclusion)</b>	<b>P- Value (Weeks 12-24)</b>
<b>Blood Pressure (Systolic /Diastolic, mmHg, SEM)</b>	$136 \pm 0.1$ $89.1 \pm 0.8$	$134.3 \pm 1.1$ $86.8 \pm 0.1$	0.284/0.123	$126 \pm 1.0$ $81.2 \pm 0.6$	<0.001 <0.001
<b>Physical Activity (steps per day, SEM)</b>	$4084.1 \pm 115.9$	$4427.00 \pm 117.5$	0.93	$13523.7 \pm 1957.6$	<0.001

Blood pressure systolic and diastolic (mm/Hg). Physical activity measurements (steps/day). Week 0 represents baseline data, week 12 represents the beginning of the intervention and week 24 represents the end of the intervention. Data is presented as Mean  $\pm$  SEM.

$P < 0.05$  indicates a significant change due to the intervention. T-test statistical analysis was performed.

#### **6.3.3.4 Metabolic Syndrome component analysis results summary**

At the commencement of the intervention all 60 subjects suffered from Metabolic Syndrome according to the NCEP-ATP III definition. Results are summarised in Table 6.4. Of our 60 participants, 25 showed all 5 components of Metabolic Syndrome (elevated blood pressure, triglycerides and plasma glucose levels, obese waist circumference and decreased HDL-C levels.). The remaining 35 subjects showed any 3 of the 5 recognised components of Metabolic Syndrome.

At the completion intervention, 52 participants showed 1 component less than prior to the intervention. All the 25 participants that showed 5 components, now showed 4, but were still classified as Metabolic Syndrome sufferers. 27 participants originally showed 3 components but on completion showed only 2 and thus were no longer considered as Metabolic Syndrome sufferers. No change was observed in the remaining 8 participants who initially showed 3 components. Of the 52 participants that no longer displayed a particular component, 3 (5%) no longer had a high waist circumference, 15 (29%) no longer displayed hypertension, 14 (27%) no longer displayed elevated triglyceride levels, 5 (10%) increased HDL-C levels and 15 (29%) no longer displayed elevated plasma glucose levels. It is submitted that hypertension, triglyceride levels and blood glucose levels were easier to modify in comparison to waist circumference and HDL-C levels which were less responsive to our intervention. To establish any correlation among subjects and their measured pre and post parameters. A statical analysis was performed. A one way ANOVA with repeated measure was performed to compare the means of parameters. F- test was performed to detect any statically significance. However we did not find any co relation between the subject and measured parameter. The number of participants that suffered from Metabolic Syndrome was reduced from 60 prior to the intervention, to 32 (54%) on intervention completion.

**Table 6.4** Pre and Post Intervention Changes in Metabolic Syndrome Components

(n=60)\_

<b>Participant Number:</b>	<b>5 Components Displayed.</b>	<b>3 Components Displayed</b>	<b>Component no longer displayed.</b>
1	WC, EBP, ETR, DHL, EPG		EBP
2		WC, DHL, EPG	EPG
3	WC, EBP, ETR, DHL, EPG		ETR
4		WC, ETR, DHL	WC
5	WC, EBP, ETR, DHL, EPG		EBP
6		ETR, DHL, EPG	HDL
7		WC, ETR, DHL	ETR
8	WC, EBP, ETR, DHL, EPG		EPG
9		WC, EBP, ETR	EBP
10	WC, EBP, ETR, DHL, EPG		EBP
11		WC, EBP, ETR	WC
12	WC, EBP, ETR, DHL, EPG		EPG
13		WC, ETR, DHL	EBP
14		WC, ETR, DHL	ETR
15	WC, EBP, ETR, DHL, EPG		EPG
16		WC, EBP, ETR	ETR
17		WC, ETR, DHL	ETR
18	WC, EBP, ETR, DHL, EPG		EPG
19		WC, EBP, ETR	WC
20		WC, ETR, DHL	ETR
21	WC, EBP, ETR, DHL, EPG		EPG
22	WC, EBP, ETR, DHL, EPG		EPG
23	WC, EBP, ETR, DHL, EPG		ETR
24		WC, EBP, ETR	EBP
25	WC, EBP, ETR, DHL, EPG		ETR
26	WC, EBP, ETR, DHL, EPG		EBP
27		WC, EBP, ETR	ETR
28		EBP, ETR.,DHL	
29	DHL, EPG WC, EBP,		EBP
30	ETR, WC DHL EPG		EBP

<b>Participant Number:</b>	<b>5 Components Displayed.</b>	<b>3 Components Displayed</b>	<b>Component no longer displayed.</b>
	EBP		
<b>31</b>		EBP, ETR.,DHL	
<b>32</b>	WC, EBP, ETR, DHL, EPG		ETR
<b>33</b>		WC, EBP, ETR	
<b>34</b>	WC, EBP, ETR, DHL, EPG		EBP
<b>35</b>		WC, EBP, ETR	ETR
<b>36</b>	WC, EBP, ETR, DHL, EPG		ETR
<b>37</b>		WC, EBP, ETR	
<b>38</b>	WC, EBP, ETR, DHL, EPG		EBP
<b>39</b>		WC, ETR, DHL	
<b>40</b>	WC, EBP, ETR, DHL, EPG		EPG
<b>41</b>	WC, EBP, ETR, DHL, EPG		ETR
<b>42</b>		WC, ETR, DHL	
<b>43</b>		WC, ETR, DHL	
<b>44</b>	WC, EBP, ETR, DHL, EPG		EPG
<b>45</b>		EBP, ETR.,DHL	EBP
<b>46</b>		WC, EBP, ETR	ETR
<b>47</b>		WC, EBP, ETR	
<b>48</b>	WC, EBP, ETR, DHL, EPG		EPG
<b>49</b>		WC, EBP, ETR	HDL
<b>50</b>		WC, EPG ETR	HDL
<b>51</b>		EPG ETR.,DHL	EBP
<b>52</b>	WC, EBP, ETR, DHL, EPG		EPG
<b>53</b>		EPG DHL, WC	EBP
<b>54</b>		, EPG DHL, WC	HDL
<b>55</b>	WC, EBP, ETR, DHL, EPG		EBP
<b>56</b>		EPG DHL, WC	EPG
<b>57</b>		EPG ETR.,DHL	EPG
<b>58</b>		WC, ETR, DHL	EPG
<b>59</b>		WC, EPG, ETR	EPG
<b>60</b>		WC, EPG, ETR	HDL

A comparison between the number of components displayed by subjects both prior, and on completion of the intervention. The abbreviations used are defined below:

WC: Increased Waist Circumference. EBP: Elevated Blood Pressure. ETR: Elevated Triglyceride Levels DHL: Decreased HDL-C Levels.EPG: Elevated Plasma Glucose.

**Table 6.5** Component Sub-group Specific Post Intervention Changes (n=60)

<b>Component:</b>	Number of participants no longer displaying component
Waist Circumference	3
Elevated Blood Pressure	15
Elevated Triglyceride Levels	14
Decreased HDL-C Levels	5
Elevated Plasma Glucose	15

Post intervention sub-group of total subjects.

**Table 6.6 Correlation between anthropometric and biochemical parameters (n=60)**

Source	Type III Sum of Squares	df	Mean Square	Partial Eta Squared	Noncent Parameter	Observed Power <sup>a</sup>
BMI						
Intercept	4.750E9	1	4.750E9	.843	317.845	1.000
Error	8.817E8	59	1.494E7			
Waist Line						
Intercept	2655747.200	1	2655747.200	.728	157.863	1.000
Error	992564.800	59	16823.132			
Physical Activity						
Intercept	3130250.939	1	3130250.939	.998	27140.660	1.000
Error	6804.728	59	115.334			
Blood Pressure						
Intercept	1322179.606	1	1322179.606	.999	42379.214	1.000
Error	1840.728	59	31.199			
Total Cholesterol						
Intercept	7515.177	1	7515.177	.974	2252.253	1.000
Error	196.867	59	3.337			
HDL						
Intercept	183.068	1	183.068	.684	127.909	1.000
Error	84.443	59	1.431			
LDL						
Intercept	363.520	1	363.520	.907	575.616	1.000
Error	37.260	59	.632			
Triglyceride						
Intercept	1017.133	1	1017.133	.955	1250.118	1.000
Error	48.004	59	.814			
Blood Glucose						
Intercept	6967.893	1	6967.893	.988	4894.867	1.000
Error	83.987	59	1.424			
Insulin						
Intercept	254051.577	1	254051.577	.940	613.760	1.000
Error	16143.139	39	413.927			

**ANOVA statistical test was performed. The F ratio was used to test significance.**

As above, the ANOVA statistical test was used to detect the differences in the means of various parameters at 3 points in time (weeks 0, 12 and 24). The statistical F ratio was not significant, thus, no statistical significance or correlation was observed between the any of the parameters tested.

#### **6.3.3.5 Pre and post intervention changes in dietary consumption pattern.**

Data obtained via the food frequency questionnaire (FFQ) was collected from 60 subjects at the commencement and completion of the intervention. The food frequency questionnaire listed 54 food items. Subjects were required to list the frequency at which they consumed each food item listed. The frequency of consumptions of the foods in the FFQ was collapsed to include consumption at all frequencies greater than once or twice a week i.e. Table 6.5 proportion of subjects consuming the food item prior or after the intervention refers to the number of participants who consumed that food at least once at week. The results were collapsed because numbers were small and they would otherwise have been difficult to analyse. Results are summarised in Table 6.5, as a percentage (the percentage of subjects consuming a particular food item either prior to, or on the completion of the intervention).

At the completion of the intervention we noticed a significant shift (significant shift was defined as  $p < 0.05$ ) in food consumption pattern; more participants were consuming fresh vegetables and fruit; subjects had also increased their consumption of fish and healthy fats, as well as reducing their intake of unhealthy foods, particularly take away food. Our results indicate that prior to the intervention, 48% of participants were consuming unhealthy foods, such as potato chips, on the completion of intervention this figure was reduced to 32%. Prior to the intervention, 56% of participants were consuming fast foods; on completion of the intervention this was reduced to 25%. Our

results also indicate that 47% of participants were consuming soft drinks; after the intervention this number reduced to 30%. 91% of subjects were consuming Pakistani style bread (such as pratha) prepared in ghee or butter as their staple food prior to the intervention; after the intervention this was reduced to 83%. Only 2% of participants were consuming green vegetables prior to the intervention (green vegetables such as broccoli, silver beet, green beans and asparagus are not freely available in Pakistan) and thus are not part of the typical Pakistani diet; after the intervention subjects reported a 47% increase in the consumption of green vegetables.

**Table 6.7.** Pre and Post Intervention Changes in Dietary Consumption Pattern.

<b>Food Item</b>	<b>Proportion (%) of subjects consuming the food item prior to the intervention.</b>	<b>Proportion (%) of subjects consuming the food item after the intervention.</b>
<b>Fruits and Vegetables</b>		
Seasonal fruits	2%	8%
Canned fruits	3%	2%
Salad	2%	32%
Vegetables (excluding green vegetables.)	50%	67%
Green vegetables	2%	47%
Potatoes	52%	6%
<b>Bread, Rice, Pasta</b>		
Bread (including Naan, Chapati and Parathas)	91%	83%
Rice	54%	48%
Pasta	49%	24%
<b>Dairy Products</b>		
Full cream milk	52%	31%
Low fat milk	2%	20%

<b>Food Item</b>	<b>Proportion (%) of subjects consuming the food item prior to the intervention.</b>	<b>Proportion (%) of subjects consuming the food item after the intervention.</b>
Tasty cheese	49%	2%
Fetta cheese	49%	9%
Coon cheese	47%	8%
Mozzarella cheese	59%	16%
Bread spreads	45%	39%
Yoghurt	28%	11%
Ice-cream	31%	10%
<b>Meat, Poultry and Fish</b>		
Lamb	23%	20%
Beef	52%	18%
Goat	52%	31%
Sausages	30%	27%
Salami	36%	25%
Fish	2%	46%
Chicken	59%	34%
Duck	3%	2%
<b>Fast Food</b>		
Fast food (pizza, McDonalds, KFC etc.)	56%	25%
<b>Fat and Oils</b>		
Vegetable oil	21%	12%
Olive oil	16%	36%
Canola oil	58%	46%
Ghee	51%	43%

<b>Food Item</b>	<b>Proportion (%) of subjects consuming the food item prior to the intervention.</b>	<b>Proportion (%) of subjects consuming the food item after the intervention.</b>
<b>Lentils and Beans</b>		
Channa (dried lentils)	13%	39%
Chickpeas	13%	53%
Moong (green lentil)	11%	33%
Other beans	2%	77%
<b>Snacks, Processed and Finger Foods.</b>		
Chocolate (Including Cake.)	17%	20%
Nuts (Coated with sugars and salts.)	28%	21%
Potato chips, ready food (packeted)	48%	32%
Biscuits	46%	41%
Pakorras (Chickpea flour fried dumpling.)	31%	49%
Samosas (Pastry with meat or vegetable filling.)	34%	52%
Pappadum	55%	44%
<b>Beverages</b>		
Soft drinks	47%	30%
Diet drinks	2%	41%
Tea	96%	97%
Coffee	32%	14%
Fruit juices	45%	40%
Cordial	56%	40%

Food categorical data obtained and presented as proportion (%) of subjects consuming the food item prior to and after the intervention.

## 6.4 Discussion

The major outcome of this study was the development and trialling of a successful intervention for the treatment of the Metabolic Syndrome in a high risk ethnic population. To our knowledge this is the first intervention directed at treating the risk factors of the Metabolic Syndrome using a culturally appropriate lifestyle intervention in Pakistani immigrant women. The outcomes of this study have demonstrated that our female Pakistani population sample in Melbourne suffers from multiple risk factors of Metabolic Syndrome and are at a particularly high risk of type 2 diabetes and cardiovascular disease. However, the diet and lifestyle intervention implemented in this study successfully improved diet and increased the level of activity in participants, thereby reducing the overall severity of all the major components of Metabolic Syndrome. It can be concluded that the root causes of Metabolic Syndrome (central obesity and physical inactivity) are reversible and the individual components of the Syndrome can be treated via a culturally appropriate intervention.

Our subjects initially showed central/abdominal obesity, hypertension, dyslipidaemia, elevated plasma glucose and insulin levels. Our subjects were not physically active and generally maintained an atherogenic diet (refer to section 3.6). Our findings are consistent with other reports on South Asian populations (McKeigue et al. 1996, Zaninotto et al. 2007, Barnett et al. 2006).

On completion of the 12 week culturally appropriate lifestyle intervention, we observed significant and beneficial changes in average waist circumference, blood biochemical profiles, hypertension, physical activity levels, and dietary pattern (refer to sections 6.3.3).

Analysis of our results revealed that participants originally had a diet high in saturated fat; however on completion of the intervention program less subjects were consuming saturated fat and more subjects were consuming healthy fats (particularly from fish),

contributing to a notable improvement to participant blood pressure, blood lipid profiles, and insulin levels. However, no significant changes were observed in HDL-C or LDL-C levels. It has been reported that low energy diets have the potential to improve BMI, the lipid profile and insulin sensitivity and that such changes can generally be observed within 5-8 weeks. Therefore low calorie diets reduce the effects, or severity of the components of Metabolic Syndrome. Our study has validated the above mentioned findings (Goldstein 1992, Ginsberg et al. 1998, WHO 2000, Case et al. 2002, Gordon et al. 2004, Daskalopoulou et al. 2004).

Our analysis further revealed that prior to the intervention participants were consuming minimal amounts of fibre; on completion of the intervention, more participants were consuming fibre. Participants were consuming more lentils, legumes, beans, various fruits and oats. It has been reported that increased fibre consumption results in lower serum cholesterol and triglyceride levels. Fibrous foods also promote weight loss as they are generally low in fats and sugars.

The changes we observed in this population as a result of the dietary modification were positive. It is submitted that by increasing the consumption of fruit and vegetables, fibre and healthy fats, and by further limiting the consumption of saturated fats and salt positives changes can be observed in bodyweight, the lipid profile, hypertension and insulin sensitivity. (Refer to section 6.3.3)

The results of this study suggest that the reduction in BMI was clinically significant although average BMI was still considered high. Participants lost approximately 5-6% of total body weight and on average 3.5cm from the waist circumference. The reduction in body weight and waist circumference can be associated with the improvement in Metabolic Syndrome risk factors. According to research, a moderate weight loss of 5-10% can be associated with the improvement in risk factors for Metabolic Syndrome

(Case et al. 2002, Park et al. 2004) and thus a reduction in the severity of Metabolic Syndrome (Refer to section 6.3.3.4).

Our study also suggests that an improvement of diet and increase of physical activity can reduce the major components of Metabolic Syndrome and is thus comparable to other studies that have achieved a 5% or greater weight loss (Daskalopoulou et al. 2004). At the conclusion of our diet and lifestyle intervention, the percentage of subject classified with Metabolic Syndrome was reduced from 100% to 53%; this reduction could be largely attributable to the healthy diet, weight loss and increased physical activity observed in our subject population.

#### **6.4.1 The unique nature of our intervention.**

The results of this study showed the efficiency of diet and lifestyle intervention as a first line of defence against various components of Metabolic Syndrome. (Refer to section 6.3.3.4) It is emphasised that this study was designed specifically for migrant Pakistani women. Pakistani women are primarily responsible for food preparation, thus, the education of women was seen as the best method in providing overall benefits to the entire family. It is submitted that the success of this intervention lies in strategies implemented to educate subjects; it was not simply a weight loss programme, rather it created guidelines for a group of migrant women who suffer from language barriers and also had difficulty to access the public leisure activities. As part of the intervention a substantial amount of time was spent with our subjects on their dietary modifications; subjects were shown how to make dietary modifications (refer to section 6.3.2) to a significant level, thus encouraging self education and self help, a more long term solution when compared to a mere weight loss program.

To the best of our knowledge, this is the only study to have implemented a culturally appropriate diet and intervention lifestyle programme in a female Pakistani population.

Studies investigating cardiovascular disease and type 2 diabetes in South Asians make little discrimination between Indians and Pakistanis and Bangladeshi men and women.

A small number of studies have investigated diet and lifestyle interventions directed to type 2 diabetes in South Asians (Chowdury et al. 2003, O'Hare et al. 2004). Of the studies that aimed to improve blood biochemistry or participant knowledge, only a few have proved successful. Chowdury et al. have suggested that a successful intervention is dependant on nutritional education in the context of health and well being in South Asian communities. It is further commented that knowledge of the risk associated with increased obesity/fatness is poor in South Asians, suggesting that culturally appropriate interventions are required. Chowdury et al. also comment on the success of "Project Dil." This project investigated the prevention of cardiovascular disease in the South Asian community residing in the UK. Primary care programs were developed in order to identify and manage cardiovascular risk factors in this high risk population. A focus group was used to identify the needs of South Asian communities. Training and education programs were implemented for patients as well as healthcare professionals together with the use of peer educators assisted in 'spreading' the project message.( Farooqi and Bhavsar 2001)

Our diet and lifestyle intervention is unique; unlike other studies it focused solely on Pakistani women, promoted nutritional education in the subject population, encouraged increased physical activity levels and did not employ the use of pharmaceuticals. The study was entirely a therapeutic diet and lifestyle intervention.

## 6.5 Conclusion

The target population has been identified as a high risk population for cardiovascular disease and Metabolic Syndrome, where Metabolic Syndrome is often considered as a preliminary stage for cardiovascular disease and type 2 diabetes. Although cardiovascular disease and Metabolic Syndrome have multiple causes, the predominant possible causes have been identified as obesity, poor diet, and a sedentary lifestyle.

The significant changes that we observed after the implementation of the short 12 week intervention suggests that most of the risk factors for Metabolic Syndrome can be positively modified. The subjects' improved diet (revealed through our analysis of the food frequency questionnaire), in conjunction with increased physical activity levels in our subject population, have been shown to reduce the incidence of the components of Metabolic Syndrome.

In this intervention, cultural competence was achieved by using a bilingual educator/facilitator. Differences in cultural understandings were addressed with the use of a bilingual educator (the researcher) from the same cultural group as the participants. This facilitator had an in-depth knowledge of the Pakistani culture and predominant religion. As our participants generally had poor access to transport, time restrictions and family commitments, the sessions were conveniently held at the participants' homes.

This intervention entails a community development approach to promoting well being and prevention of chronic diseases. Their families and communities will also have reduced risk of Metabolic Syndrome as women and their families gained a greater understanding of the concepts of chronic disease, the need to undertake prevention activities. Results have indicated that participants have reduced their total 'risk index' by losing weight, reducing their waist lines, eating more healthy food (while still retaining cultural and religious requirements), exercising more often and decreasing blood glucose and lipid levels, and blood pressure.

Our study has shown the importance of culturally competent interventions; the success of this intervention lies in the fact that we had used a simple approach for modification of the diet on a daily basis and the addition of a simple form of physical activity. We can conclude that a culturally appropriate diet and lifestyle intervention can indeed act as an effective mechanism for preventing the onset, or reducing the severity of Metabolic Syndrome in Pakistani female's sufferers.

### **6.5.1 Limitations of the study**

The intervention developed in this study could be applied to any other ethnic groups with certain limitations, including the stipulation that the intervention must be modified to suit the population being examined. Another limitation is the general gender bias observed in this study. Men were not examined for cultural and social reasons. The researcher responsible for data collection in this project was female; this presents difficulties in terms of communication and interaction with men. However, it is emphasised that this study was designed specifically for women. Pakistani women are primarily responsible for food preparation; thus, the education of women was seen as the best method in providing overall benefits to the entire family.

Another limitation of this study was the type of physical activity that participants could engage in. As discussed previously, participants were not able to access public leisure facilities and were therefore limited to walking. It would be possible to achieve even more significant results if the physical activity component of the intervention program was more vigorous in nature.

## **6.6 Future research**

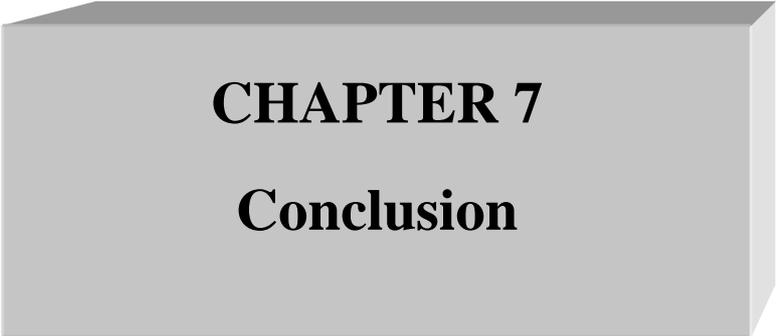
Pakistanis are a particularly vulnerable group who are said to be hard to access. Many do not speak English, and indeed may have little education from their home country, are very isolated from mainstream society, and have variable knowledge of how to use health services in western metropolitan Melbourne. Often their first presentation to a health service will be at a very late stage and at the emergency department.

The results of the study demonstrated that once the Pakistani women were approached in a sensitive and inclusive manner, they were eager and willing participants in the wellbeing program. Each had their own personal story (eg, a family member; their own health; a community member) which contributed to their motivation to improve and change. They wanted to learn; and the 'out-reach' gave them a supportive environment in which they could also contribute to their own and others' lifestyle modification experiences.

There is a need to obtain better information about Pakistani migrants' changes in food consumption, factors/reasons influencing dietary choices, as well as understanding of the environmental factors that lead to changes in dietary patterns. Many studies have recommended lifestyle modification as a means of losing weight and maintaining overall good health but more research is required to develop best practice with these populations. There is a need for further research in the area of relationship between dietary pattern and the accumulation of abdominal/central fat.

Culturally appropriate diet and lifestyle intervention programs ideally should be implemented at an early stage (preferably on migration) so as to encourage healthy eating and physical activity. Education in the migrants' native language is necessary in order to create awareness about Metabolic Syndrome and preventative measures, so as

to reduce the impact and effects of obesity on various populations. Furthermore, leisure activities in which individuals from culturally sensitive backgrounds can participate, should be promoted as a means of endorsing a physically active lifestyle; only then will the financial burden on health systems ease and overall health of the aforementioned populations improve. It is strongly suggested that future studies of similar nature should be conducted on a larger scale in affiliation with medical centres. Furthermore, such studies should be conducted over a longer time period, thus allowing for an assessment of the long term sustainability of any such diet and lifestyle intervention.



**CHAPTER 7**

**Conclusion**

## **7 CONCLUSION**

### **7.0 Conclusion**

The purpose of the identification of Metabolic Syndrome is to recognise high risk individuals and populations in order to assist in the management of cardiovascular disease. Effectively limiting or preventing the onset of Metabolic Syndrome may delay or prevent the onset of cardiovascular disease. The best strategy for the management of the Syndrome entails modifying underlying risk factors, namely, central obesity, physical inactivity and an atherogenic diet: genetic factors also contribute to the prevalence and development of Metabolic Syndrome, but clearly genetic factors cannot be modified. Such modification is possible via effective diet and lifestyle changes. Evidence suggests that weight reduction, and subsequent weight maintenance, can be achieved via reduced energy intake, increased levels of physical activity and behaviour modification. Rapidly losing weight over a short period of time is not an effective approach for Metabolic Syndrome sufferers. After an initial 5-10% reduction in body weight, priority should be placed on long term weight reduction via the use of an effective intervention tailor-made to the needs of any individuals/communities.

Various approaches for the treatment of obesity in ethnic communities have been less than successful (Case et al. 2002). Studies have commented on the importance of culturally appropriate interventions for “underserved populations” (Yancey et al. 2006, Farooqi and Bhavsar 2001). Data on Metabolic Syndrome and ethnic communities, Pakistanis in particular, is significantly lacking. Some studies have suggested that one reason for the lack of data on ethnic minorities is due to the major difficulties involved in recruiting and retaining ethnic subjects. It has been suggested that South Asians are

less inclined to participate in research studies as they generally display a lack of awareness about the significance and importance of research (Yancey et al. 2004, Kumanyika 2002). Various studies have clearly stated that emphasis should be placed on involving ethnic populations in research, and researchers should tailor a “culturally specific message” for each ethnic population (Yancey et al. 2006). The outcomes of this study suggest that a culturally appropriate programme with a clearly defined purpose and function, if properly explained to potential candidates, is an adequate means of overcoming recruitment barriers commonly observed in ethnic populations.

South Asians are a heterogeneous group yet many studies fail to differentiate amongst various South Asians groups. However, it has been shown that the prevalence of Metabolic Syndrome and cardiovascular disease is highest in Pakistanis and other South Asian migrants residing in the UK, Australia, Canada and New Zealand when compared to the respective host population (Al-Mousa 2005, Dhawan et al. 1997). It has also been documented that Pakistani women are more likely to show elevated cardiovascular risk factors when compared to Pakistani men. Thus Pakistani women have been labelled an “at risk” group; as such, the implementation of appropriate intervention strategies is required for Pakistani female populations globally (Williams et al.1993, Zaininotto 2007).

All the requisite components of Metabolic Syndrome were observed in the female Pakistani population investigated, including biochemical risk factors (i.e. cholesterol, triglyceride, fasting plasma insulin and glucose levels) as well as anthropometric factors (such as elevated BMI and waist circumference) (refer to section 6.3.3.4). It is difficult to ascertain precisely when the components of Metabolic Syndrome began to develop in the subject population; participants mentioned that their weight had increased most

significantly after migration to Australia. Many studies have reported that upon migration/relocation to another country, migrants show key changes in their diet and lifestyle patterns as part of the process of acculturation. Generally, migration and lifestyle change (such as an increase in energy dense foods and a reduction in physical activity) leads to a higher prevalence of Metabolic Syndrome in migrant populations (Patel et al. 2006). It is suggested that migration and acculturation may have hastened (although not guaranteed) the occurrence of Metabolic Syndrome in our subject population.

Our results showed a significant reduction in most of the risk factors for Metabolic Syndrome after the implementation of our culturally appropriate diet and lifestyle intervention. Although we are not sure as to the precise mechanism(s) contributing towards the reduction in most of the biochemical risk factors, it is suggested that these risk factors may have been reduced due to an overall improved healthy diet and increased levels of physical activity. It is submitted that the increase in physical activity levels that was observed in our subject population may have significantly contributed to the improvement of most Metabolic Syndrome risk factors. Additionally, the overall shift in dietary habits we observed in our target population is believed to have further improved participant health. For example, the majority of subjects had increased their consumption of fresh fruit and vegetables, switched to the use of healthier fats and limited their consumption of fast foods. Other contributory factors include subject motivation and determination to participate in this study.

Our data therefore completely supports the view that dietary interventions are the best non-pharmacological preventive strategy for the prevention of Metabolic Syndrome and associated chronic diseases. Our results suggest that Pakistani females residing

elsewhere may also benefit from such a program. However it cannot be ascertained whether the changes observed in our target population are permanent long term changes, as our intervention period was for 12 weeks only and as such we could not adequately assess the permanency of any change in biochemical and physical measurements. Ideally, reassessment of the subject population should occur one year after the completion of any such intervention program.

The genetic basis for Metabolic Syndrome is ambiguous(refer to section 4.1). Our genetic analysis has revealed that our subjects may be genetically predisposed to components of Metabolic Syndrome Genetic variants of key candidate genes for Metabolic Syndrome were detected in our population. An investigation into the variants of candidate genes for Metabolic Syndrome revealed that our subject population may be genetically susceptible to the development of the disorder. It was established that subjects who displayed homozygous traits for variants of all candidate genes were likely to suffer from more components of Metabolic Syndrome (5) when compared to non-homozygous participants (3). Similarly , it was also established that subjects that displayed homozygous traits for variants of the WNK1 and CAD genes were likely to suffer from hypertension and dislipidemia, basic components of cardiovascular disease( refer to section 4.7). We can thus suggest that some of our participants may be predisposed to genetic factors which can be determinative of Metabolic Syndrome, given that they are overweight and engage in a sedentary lifestyle. As such, obesity in this population may have predisposed participants further to the various components of Metabolic Syndrome. Overall, however, the major factors are unhealthy diet and physical inactivity; our study suggests that a diet and lifestyle intervention can greatly negate the components of Metabolic Syndrome in a possibly genetically susceptible population. Overall, the genetic association of Metabolic Syndrome in our subject

population was apparent but ambiguous. The nature of this genetic association remains inconclusive.

Analysis of our qualitative data.( refer to section 5.2) suggests that psychosocial factors such as anger, anxiety, depression and loneliness (social isolation) can result in physical inactivity and poor diet It has been reported that impact of these psychosocial factors on obesity and disease risk have highlighted the fact that under stress, excessive cortisol release could create an imbalance in the physiology of fat deposition such that there is a tendency for a greater proportion of fat to be deposited (De Vogli et al. 2007). Through voluntary discussion with participants it was ascertained subjects were less inclined to exercise and more likely to consume excess food as a result of various psychosocial factors. In particular, key contributory psychosocial factors which include migration, stress and loneliness have may influenced the development of Metabolic Syndrome in our subject population.

We can suggest that unhealthy diet, (refer to section 3.16) physical inactivity, genetic predisposition and psychosocial factors all contribute to the development of Metabolic Syndrome and its associated disorders. We have demonstrated that the implementation of a culturally appropriate diet and lifestyle intervention can actively reduce and limit the effect and severity of Metabolic Syndrome. Our findings further suggest that our subject population was somewhat genetically predisposed to a few components of the Metabolic Syndrome. We submit that a culturally appropriate diet and lifestyle intervention can positively contribute to the management of risk factors for Metabolic Syndrome and its associated disorders in a high risk migrant population.

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## **APPENDICES**



## APPENDIX- A

### Attachment A-1

# MEMO

TO Ms Rizwana Kousar DATE 25/11/03

---

FROM Dr Alan Hayes  
Chair, Human Research Ethics Committee  
Faculty of Health, Engineering and Science

---

SUBJECT **SET 04/03 Metabolic Syndrome: Effects of diet and physical activity in female immigrants**

---

At its meeting in November 2003 the Human Research Ethics Committee administered by the Faculty of Health, Engineering and Science considered your application for the project

***Metabolic Syndrome: Effects of diet and physical activity in female immigrants***

The committee resolved to **approve** your application until 31 December 2004.

Please ensure that the survey instrument has the University contact details and letterhead.

You are reminded that an annual report (the form for which can be downloaded from the Office for Industry and Research webpage) must be submitted within a year of approval or at the completion of the study, whichever comes first.

If you have any questions or queries about the requirements of the Committee's deliberations please do not hesitate to contact me via email or on 9919 4658.

Sincerely,

Dr Alan Hayes  
Chair, Human Research Ethics Committee  
Faculty of Health, Engineering and Science



# MEMO

TO Assoc Prof Jack Antonas  
Biomedical and Clinical Sciences  
St Albans Campus

DATE 11/12/2007

FROM Dr Alan Hayes  
Acting Chair  
Victoria University Human Research Ethics Committee

SUBJECT Ethics Application – HRETH 07/222

Dear Assoc Prof Antonas,

Thank you for submitting this application for ethical approval of the project:

**HRETH 07/222** Establishing a genetic link between Metabolic Syndrome and female Pakistani immigrants residing in Melbourne (HREC 07/179)

The proposed research project has been accepted by the Acting Chair, Victoria University Human Research Ethics Committee. Approval has been granted from 11 December 2007 to 11 December 2008.

Please note that the Human Research Ethics Committee must be informed of the following: any changes to the approved research protocol, project timelines, any serious or unexpected adverse effects on participants, and unforeseen events that may effect continued ethical acceptability of the project. In these unlikely events, researchers must immediately cease all data collection until the Committee has approved the changes.

Continued approval of this research project by the Victoria University Human Research Ethics Committee (VUHREC) is conditional upon the provision of a report within 12 months of the above approval date (by 11 December 2008) or upon the completion of the project (if earlier). A report proforma may be downloaded from the VUHREC web site at <http://research.vu.edu.au/hrec.php>

If you have any queries, please do not hesitate to contact me on 9919 4658.

On behalf of the Committee, I wish you all the best for the conduct of the project

  
Dr Alan Hayes  
Acting Chair  
Victoria University Human Research Ethics Committee

**Attachment A-3**

**METABOLIC SYNDROME:  
EFFECTS OF DIET AND PHYSICAL ACTIVITY IN FEMALE IMMIGRANTS**

*Female Participant Brief Questionnaire (screening tool)*

**DIRECTIONS:**

- Indicate your answer by ticking the appropriate box  or by writing your answer in the space provided
- If you are uncertain about the answer to any of the questions leave them blank and ask me to help you when you have reached the end of the questionnaire.

Date   /   /

**Participant Id**

/ \*

**1.0 Personal Details**

---

**1.1 Age:** \_\_\_\_\_

**1.2 Marital Status**

- Never Married.....  1.
- Married .....  2.
- Divorced .....  3.
- Widowed .....  4.

**1.3 How Many Children Do You Have?**

- None.....
- Children 0-14 years    \_\_\_ \_\_\_ number
- Children 15-17 years   \_\_\_ \_\_\_ number
- Children 18+ years     \_\_\_ \_\_\_ number

**1.4 Living Arrangements**

- Living with husband .....  1.
- Living with other person(s) (such as children, parents).....  2.
- Living alone .....  3.

**1.5 Country of Birth**

Where were you born\_\_\_\_\_

(Write Country )

**1.6 Level of Education**

- Never Attended School .....  1.
- Primary School .....  2.
- Some High School .....  3.
- Completed High School (Year 12 or equivalent)...  4.
- University, C.A.E, or other tertiary education.....  5.

**1.7 Current Employment**

Do you have a full, part or casual job of any kind?

- NO      YES**
- 1     2

**2.0 Medical History**

---

**2.1 When Did You Last Have Your Blood Pressure Measured?**

- In The Last Three Months .....  1.
- In The Last Six Months .....  2.
- In The Last Year.....  3.
- In The Last Three Years.....  4.
- More Than Three Years Ago.....  5.
- Never Measured .....  6.
- Don't Know.....  7.

**2.2 When Did You Last Have Your Blood Cholesterol Measured?**

- In The Last Three Months .....  1.
- In The Last Six Months .....  2.

- In The Last Year.....  3.
- In The Last Three Years.....  4.
- More Than Three Years Ago.....  5.
- Never Measured .....  6.
- Don't Know.....  7.

**2.3 Have You Ever Been Told That You Have Any Of The Following By Your Doctor?**

- |   | NO                          | YES                         |
|---|-----------------------------|-----------------------------|
| High Blood Pressure .....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Angina .....  | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Heart Attack (a "Coronary", coronary occlusion,<br>coronary thrombosis, myocardial infarction)..... | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Diabetes.....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| High Cholesterol.....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| High Triglycerides.....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Obesity.....  | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |

**NO      YES**

**2.4 Are You On Tablets For Blood Pressure?**

1       2

**NO      YES**

**2.5 Are You Having Treatment to Lower your cholesterol?**

1       2

**NO      YES**

**2.6 Are You On Tablets Or Other Treatment for Angina?**

1       2

**2.7 Has a Doctor or Nurse Ever Told You That You had Diabetes?**

**NO      YES**

If yes, please state the year you were first told 19 \_\_ (YEAR)

1       2

**2.8 Are you currently taking blood thinning medication like "WARFARIN"?**

**NO      YES**

1       2



# APPENDIX- A

Attachment -4

<b>METABOLIC SYNDROME: EFFECTS OF DIET AND PHYSICAL ACTIVITY IN FEMALE IMMIGRANTS</b>
---

*Female Participant Detailed Questionnaire*

<b>DIRECTIONS:</b>
<ul style="list-style-type: none"><li>• Indicate your answer by ticking the appropriate box <input checked="" type="checkbox"/> or by writing your answer in the space provided</li><li>• If you are uncertain about the answer to any of the questions leave them blank and ask me to help you when you have reached the end of the questionnaire.</li></ul>

	/		/	
--	---	--	---	--

**Participant Id**

	/*			
--	----	--	--	--

**1.0 Personal Details**

---

**1.1 Age:** \_\_\_\_\_

**1.2 Marital Status**

Never Married.....  1.

Married .....  2.

Divorced .....  3.

Widowed .....  4.

**1.3 How Many Children Do You Have?**

None.....

Children 0-14 years     \_\_\_ \_\_\_ number

Children 15-17 years   \_\_\_ \_\_\_ number

Children 18+ years     \_\_\_ \_\_\_ number

**1.4 Living Arrangements**

- Living with husband ..... 1.
- Living with other person(s) (such as children, parents)..... 2.
- Living alone ..... 3.

**1.5 Country of Birth**

Where were you born \_\_\_\_\_  
(Write Country )

**1.6 Level of Education**

- Never Attended School .....  1.
- Primary School ..... 2.
- Some High School .....  3.
- Completed High School (Year 12 or equivalent)... 4.
- University, C.A.E, or other tertiary education.....  5.

**1.7 Current Employment**

Do you have a full, part or casual job of any kind?

**NO      YES**

- 1     2

**2.0 Medical History**

---

**2.1 When Did You Last Have Your Blood Pressure Measured?**

- In The Last Three Months .....  1.
- In The Last Six Months ..... 2.
- In The Last Year.....  3.
- In The Last Three Years..... 4.
- More Than Three Years Ago..... 5.
- Never Measured .....  6.
- Don't Know.....  7.

**2.2 When Did You Last Have Your Blood Cholesterol Measured?**

- In The Last Three Months .....  1.
- In The Last Six Months ..... 2.
-

- In The Last Year..... 3.
- In The Last Three Years.....  4.
- More Than Three Years Ago.....  5.
- Never Measured .....  6.
- Don't Know.....  7.

**2.3 Have You Ever Been Told That You Have Any Of The Following By Your Doctor?**

- |   | NO                          | YES                         |
|---|-----------------------------|-----------------------------|
| High Blood Pressure .....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Angina .....  | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Heart Attack (a "Coronary", coronary occlusion,<br>coronary thrombosis, myocardial infarction)..... | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Diabetes.....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| High Cholesterol.....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| High Triglycerides.....   | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |
| Obesity.....  | <input type="checkbox"/> 1. | <input type="checkbox"/> 2. |

NO YES

**2.4 Are You On Tablets For Blood Pressure?**

1  2

NO YES

**2.5 Are You Having Treatment to Lower your Blood Cholesterol?**

1  2

NO YES

**2.6 Are You On Tablets Or Other Treatment for Angina?**

1  2

**2.7 Has a Doctor or Nurse Ever Told You That You had Diabetes?**

NO YES

If yes, please state the year you were first told 19 \_\_ (YEAR)

1  2

NO YES

**2.8 Are you currently taking blood thinning medication like "WARFARIN"?**

1  2

**2.9 Have you ever been given advice or treatment for diabetes or sugar trouble?**

NO YES

If yes, please state the year this advice or treatment was first given 19\_\_

1  2

year

Was this:

Diet Advice..... 1.

Insulin injections..... 2.

Diet advice and tablets..... 3.

Diet advice and injections..... 4.

**2.10 Have you ever taken the oral contraceptive pill?**

Yes..... 1.

No..... 2.

**2.11 Are you currently pregnant?**

Yes..... 1.

No..... 2.

**3.0 Recreation, Sport or Health-Fitness**

---

**3.1 In the Past 2 Weeks, Did you Engage in Vigorous Exercise – Exercise Which Makes you breathe harder or puff and pant.**

No..... 1.

Yes..... 2.

If yes , how many sessions of vigorous exercise did you have over the 2 week period? \_\_\_\_\_

Please estimate the TOTAL TIME spent exercising vigorously during the past 2 weeks: \_\_\_\_ / \_\_\_\_  
hours minutes

**3.2 In the PAST 2 WEEKS, did you engage in less vigorous exercise for recreation, sport or health-fitness purposes which did not make you breathe harder or puff and pant?**

No..... 1.

Yes..... 2.

If yes, how many sessions of less vigorous exercise did you have over the 2 week period? \_\_\_\_\_

**3.3 In the past 2 weeks, did you walk for recreation or exercise?**

No..... 1.

Yes..... 2.

If yes, how many times? \_\_\_\_\_

**3.4 Vigorous Tasks At Work And Around The House ( Paid or Unpaid Work)**

In the PAST 2 WEEKS, did you engage in vigorous activity, apart from exercise, which made you breathe harder or puff and pant? (e.g. carrying loads, heavy gardening, chopping wood, labouring- at home, during employment or anywhere else.)

No..... 1.

Yes..... 2.

If yes, how many sessions of these types of vigorous activity did you have over the 2 week period? \_\_\_\_\_

Please estimate the TOTAL TIME spent in these types of vigorous activity during the PAST 2 weeks

\_\_\_\_/\_\_\_\_  
Hours Minutes

**3.5 If you have the opportunity to swim, would you take it?**

Yes..... 1.

No..... 2.

**3.6 If you have the opportunity to participate in a “Group Walk”, would you take it?**

Yes..... 1.

No..... 2.

**4.0 Diet and Nutrition**

---

**4.1 Do you cook your own meals?**

**NO YES**

1  2

All the time

Yes.....

No.....

If No, how often \_\_\_\_\_  
(Number of Days a week)

**4.2 Who is responsible for meal preparation?**

Yourself..... 1.

A Family Member..... 2.

Other (Please Specify) .....\_\_\_\_\_

**4.3 How do you decide what meal to prepare?**

Cook Books..... 1.

Traditional Recipes..... 2.

Family / Friends..... 3.

Special / Religious Occasions..... 4.

**4.4 Do you have the same meal preparation methods as you did in Pakistan?**

Yes.....

No.....

**4.5 If your meal preparation method has changed, please specify how?**

-----  
-----  
-----

**4.6 What kinds of fat/oil do you use in meal preparation?**

Vegetable Oil.....

Canola Oil.....

Olive Oil.....

Ghee (Animal Fat) .....

Other .....

**4.7 Did you eat more meat or vegetables (lentils inclusive) in Australia?**

Meat.....

Vegetables.....

**4.8 Do you think since your arrival in Australia, your dietary practices have changed?**

Yes.....

No.....

**4.9 How do you think your meals differ from that of your neighbour?**

Please specify. -----  
-----  
-----

**4.10 What do you think has made your dietary practices change?**

Cultural Differences .....

Health.....

Employment.....

Time.....

Affordability/ Accessibility.....

Other, Please Specify. -----  
-----  
-----

**4.11 Do you consider your diet to be healthy?**

Yes.....

No.....

**4.12 Are you aware of the five food groups?**

Yes.....

No.....

**4.13 How Much oil/ fat do you purchase a week ?**

No Fat .....

2 Litres .....

4 Litres.....

Other (Please Specify in Litres)\_\_\_\_\_

**4.14 Do you add salt to your food after it is cooked?**

Rarely or never.....

Sometimes.....

Almost always or always.....

**4.15 How often do you eat the fat on meat?**

Usually.....

Sometimes.....

Rarely or never.....

**4.16 Do you consume any of the following sweets?**

	Yes	No
Kheer (Rice Pudding).....	<input type="checkbox"/>	<input type="checkbox"/>

Gajerallia (Carrot Halwa).....	<input type="checkbox"/>	<input type="checkbox"/>
--------------------------------	--------------------------	--------------------------

Any Other Sweet, please specify \_\_\_\_\_

**If yes, please specify number of times a week** \_\_\_\_\_  
(Number Of Times A Week)

**4.17 Do you consume any of the following home made breads?**

	Yes	No
Chappati.....	<input type="checkbox"/>	<input type="checkbox"/>

Pratha.....	<input type="checkbox"/>	<input type="checkbox"/>
-------------	--------------------------	--------------------------

Nan.....	<input type="checkbox"/>	<input type="checkbox"/>
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**If yes, please specify number of times a week** \_\_\_\_\_  
(Number Of Times A Week)

**Thank You for Your Time**

# APPENDIX -A

Attachment A-5

**METABOLIC SYNDROME:  
EFFECTS OF DIET AND PHYSICAL ACTIVITY IN FEMALE IMMIGRANTS**

## Food Frequency Questionnaire

In the table below, indicate the frequency at which you consume a particular food item (with a ✓) also, indicate whether there has been an increase or decrease in consumption of the particular food item since you have been in Australia, according to a scale of -3 to +3 (-3,-2,-1,0,+1,+2,+3) with -3 being a large decrease, +3 a large increase, and 0 being no change.

Food item	More than once per day	Once per day	3-6 times per week	Once or twice per week	Once per month or less	Never	Change in Consumption since in Australia
<i>Example :</i> Salad			✓				+2 ( An Increase)
Seasonal fruit (various)							
Canned fruit (various)							
Salads (various)							
Vegetables (various)							
Green vegetables (various)							
Potatoes							
Bread (various)							
Rice							
Pasta							
<b>Milk</b>							
Full cream							
Reduced fat							

<b>Cheese</b>							
Tasty							
Fetta							
Coon							
Mozzarella							
Butter / Margarine							
Spreads							
Yoghurt							
Ice-cream							
<b>Meat</b>							
Lamb							
Beef							
Goat							
Sausages							
Salami							
<b>Seafood</b>							
Fish							
Other							
<b>Poultry</b>							
Chicken							
Duck							
Fast Food (e.g. Hungry Jacks, Pizza, KFC)							
<b>Fats/Oil</b>							
Vegetable							
Olive							
Canola							
Ghee (animal fat)							
Other							

<b>Legumes</b>							
Channa							
Chickpeas							
Moong							
Other							
<b>Snacks</b>							
Chocolate/ Lollies							
Nuts							
Chips							
Biscuits							
Pakora							
Samosa							
Pappadum							
Other							
<b>Drinks</b>							
Soft drinks							
Diet soft drinks							
Tea							
Coffee							
Fruit juice							
Cordial							

# APPENDIX -A

Attachment A-6

## Names of Researchers:

### **METABOLIC SYNDROME: EFFECTS OF DIET AND PHYSICAL ACTIVITY IN FEMALE IMMIGRANTS**

Dr. Paul Lewandowski

Dr. Cate Burns

Mrs. Rizwana Kousar

### **Participant Explanatory Statement**

Victoria University conducts a number of research studies. I am a Ph.D student with Victoria University, (School of Biomedical Sciences). I am conducting a research study, which will explore the effect of diet and exercise on various health problems which include obesity, diabetes, blood pressure, changes in cholesterol levels. As our culture has a significant influence on the food choice, it is important to evaluate the change in diet and physical activity on our health. You are invited to participate in the research project. This study has the potential to benefit female immigrant's residing in Melbourne. Female population will gain the knowledge in terms of better understanding and perception of some cultural foods. The aim of the study is to collect baseline data on metabolic syndrome and Pakistani females.

I'll be contacting you via phone, to inquire you are willing to participate in the study. An initial introductory meeting with participants will be arranged, and an information kit (comprising of consent form, and participant explanatory statement). Researcher will introduce the study aims, requirements of participants, and information on data collection procedure. The participants will be given a chance to ask any questions, potential participants will read all the information in their own time, and then complete the consent form. The researcher will collect all the paperwork, after a few days. You can take part in the study by signing an informed consent form. At this stage, all the participants will have to fill another detailed questionnaire. This questionnaire will obtain information on your personal details and health condition (same as in first questionnaire). This questionnaire will also obtain information on dietary habits (what meals you prepare, what fat you use, and consumption of traditional foods) as well as physical activity levels (do you take any exercise, or not at all). To record the dietary habits, you need to complete the food frequency sheet. Please sign the consent form and give me permission to measure your height, and weight. I also need your permission to measure your blood pressure, and take a blood sample from your arm, to test the levels of sugar and cholesterol in your blood. In blood collection, there is a slight risk of the arm bruising as well as a risk of fainting and dizziness.

#### ***Maintaining Participants Confidentiality***

To ensure participants privacy, every female will be allocated with a unique numeric code, which will be used to identify participant's data and blood sample. No data identifying individuals will be published or made available to any unauthorised person. All data will be kept in a secure place. All the participants will be advised that participation is voluntary, and they can withdraw from the study at any time, without any consequence. If you require further information on any procedure, please contact Dr. Paul Lewandowski at Victoria University on 93652321. Thank you for your time and I hope that you will consider being a part of the project.

Principal Investigator's signature ..... Phone No: (03) 9365 2321

Date

Victoria University .

Any queries about your participation in this project may be directed to the researcher (Name: Dr. Paul Lewandowski ph. (03- 9365 2321), If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University.PO Box 14428 MC, Melbourne, 8001 (telephone no: 03-9688 4710).

## **APPENDIX - A**

## Consent Form for Participants Involved in Research

### INFORMATION TO PARTICIPANTS:

We would like to invite you to be a part of a study investigating  
METABOLIC SYNDROME:  
EFFECTS OF DIET AND PHYSICAL ACTIVITY IN FEMALE IMMIGRANTS

### **CERTIFICATION BY PARTICIPANT**

I, \_\_\_\_\_

Of, \_\_\_\_\_

certify that I am at least 18 years old\* and that I am voluntarily giving my consent to participate in the experiment entitled:

METABOLIC SYNDROME:  
EFFECTS OF DIET AND PHYSICAL ACTIVITY IN FEMALE IMMIGRANTS

being conducted at Victoria University of Technology by:

**Dr. Paul Lewandowski, Dr. Cate Burns, and Mrs. Rizwana Kousar**

I certify that the objectives of the experiment, together with any risks to me associated with the procedures listed hereunder to be carried out in the experiment, have been fully explained to me by:

**Mrs. Rizwana Kousar**

and that I freely consent to participation involving the use on me of these procedures.

### **Procedures:**

- **Completion of questionnaire**
- **Completion of written food frequency questionnaire**
- **Physical measurements**
- **Blood pressure measurement**
- **Blood sample**

I certify that I have had the opportunity to have any questions answered and that I understand that I can withdraw from this experiment at any time and that this withdrawal will not jeopardise me in any way.

I have been informed that the information I provide will be kept confidential.

Signed: ..... }

Witness other than the experimenter: ..... } **Date:** .....

Any queries about your participation in this project may be directed to the researcher (Name: Dr. Paul Lewandowski ph. (03- 9365 2321).If you have any queries or complaints about the way you have been treated, you may contact the Secretary, University Human Research Ethics Committee, Victoria University . P O Box 14428 MC, Melbourne, 8001 (telephone no: 03-9688 4710).

### **APPENDIX -A**

## Revocation of Consent Form for Subjects Involved in Research

Used for participants who wish to withdraw from the project

I, \_\_\_\_\_

of (address), \_\_\_\_\_

\_\_\_\_\_

hereby wish to WITHDRAW my consent to participate in the research proposal described in the Plain Language Statement for the research project called:

METABOLIC SYNDROME: EFFECTS OF DIET AND PHYSICAL ACTIVITY IN FEMALE IMMIGRANTS

and understand that such withdrawal WILL NOT jeopardise any treatment or my relationship with Victoria University.

Any data already collected may/may not be included in the research project.

**Signature:** \_\_\_\_\_ **Date:** \_\_\_\_\_

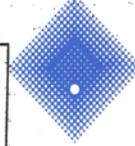
منسلک A-3

**Victoria University**

PO Box 14428  
Melbourne City MC  
VIC 8001 Australia

Telephone: METABOLIC SYNDROME  
(03) 9365 2667  
Facsimile:  
(03) 9365 2466

**VICTORIA UNIVERSITY**



OF TECHNOLOGY

**School of Biomedical Sciences**

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McKeechie Street  
St Albans

میٹابولک سڈروم  
خوراک اور جسمانی مشقت کے تارکین وطن خواتین میں اثرات

مختصر سوالنامہ برائے شرکت کنندہ خواتین

ہدایت نامہ

اپنے جواب کو مناسب باکس پر لگا کر ظاہر کریں یا مہیا کروا جگہ پر لکھیں  
اپنے جوابات سے غیر یقینی کی صورت میں خالی جگہ چھوڑ دیں اور سوال نامہ کے اختتام پر مجھ سے  
پوچھ کر مدد لیں

تاریخ ..... / ..... / .....

شرکت کنندہ کی شناخت .....

1.0 ذاتی تفصیلات

1.1 عمر .....

1.2 ازدواجی حیثیت

1 غیر شادی شدہ .....

2 شادی شدہ .....

3 طلاق یافتہ .....

4 بیوہ .....

1.3 آپ کے بچوں کی کل کتنی تعداد ہے

کوئی بھی نہیں ..... بچے صفر تا ۱۳ سال ..... تعداد، ۱۴ تا ۱۵ ..... تعداد، ۱۸ سے اوپر ..... تعداد



23 کیا آپ کو کبھی آپکے ڈاکٹر یا معالج سے معلوم ہوا ہے کہ آپ مندرجہ ذیل امراض میں سے کسی ایک میں

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McKechnie Street  
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بہلا ہیں (ہاں نہیں میں جواب دیں)

- 1 High Blood Pressure (فشار خون)
- 2 Angina (آضعف قلب)
- 3 Heart Attack (a "Coronary", Coronary Occlusion, Coronary thrombosis, myocardial infarction (دورائے قلب)
- 4 Diabetes (ذیابیطس)
- 5 High cholesterol (گھسٹروں کی زیادتی)
- 6 High Triglycerides
- 7 Obesity (موٹاپا/فریبی)

- 2.4 کیا آپ فشار خون / بلڈ پریشر کی کوئی دوا استعمال کرتی ہیں
- 2.5 کیا آپ گھسٹروں Cholesterol کی کمی کے علاج کراتی رہی ہیں
- 2.6 کیا آپ ضعف قلب Angina کے علاج کی دوا استعمال کرتی ہیں
- 19 2.7 کیا آپ کو معالج یا نرس سے معلوم ہوا ہے کہ آپ کو ذیابیطس تھی اگر آپ کو بتایا گیا ہے تو سال کا اندراج کریں
- 2.8 کیا آپ آجکل خون پتلا کرنے کی دوا جیسے "Warfarin" استعمال کر رہی ہیں

2.9 کیا آپ کو کبھی بھی ذیابیطس یا شوگر کی تکلیف کے بارے میں نصیحت یا علاج ہوا ہے

اگر آپ کا جواب ہاں ہے تو سال کا اندراج بھی کریں

کیا وہ:

- 1 نصیحت برائے خوراک تھی
- 2 انسولین انجیکشن
- 3 نصیحت برائے خوراک و دوا
- 4 نصیحت برائے خوراک و انجیکشن

2.10 کیا آپ نے کبھی مانع حمل کوئی دوا استعمال کی ہے

ہاں / نہیں ..... 2.11 کیا آجکل آپ حاملہ ہیں آپ کا جواب ہاں / نہیں

Victoria University

PO Box 14428  
Melbourne City MC  
VIC 8001 Australia

METABOLIC SYNDROME

Telephone:  
(03) 9365 2667  
Facsimile:  
(03) 9365 2465VICTORIA  
UNIVERSITYOF  
TECHNOLOGY

School of Biomedical Sciences

St Albans Campus  
McKechnie Street  
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میٹابولک سندروم

خود راک اور جسمانی مشقت کے تارکین وطن خواتین میں اثرات

تفصیلی سوالنامہ برائے شرکت کنندہ خواتین

ہدایت نامہ

اپنے جواب کو مناسب باکس پر لگا کر ظاہر کریں یا مہیا کر دیا جگہ پر لکھیں  
اپنے جوابات سے غیر یقینی کی صورت میں خالی جگہ چھوڑیں اور سوال نامہ کے اختتام پر تجھ سے  
پوچھ کر مدد لیں

تاریخ ..... / ..... / .....

شرکت کنندہ کی شناخت ..... / ..... / .....

1.0 ذاتی تفصیلات

1.1 عمر .....

1.2 ازدواجی حیثیت

1 غیر شادی شدہ .....

2 شادی شدہ .....

3 طلاق یافتہ .....

4 بیوہ .....

1.3 آپ کے بچوں کی کل کتنی تعداد ہے

کوئی بھی نہیں : بچے صفر تا ۱۳ سال ..... تعداد : ۱ تا ۵ ..... تعداد : ۱۸ سے اوپر ..... تعداد :

۱.۴ رہائشی طریقہ زندگی

- ..... ۱ شوہر کے ساتھ رہائش
- ..... ۲ دیگر لوگوں کے ساتھ رہائش
- ..... ۳ تنہا رہائش

۱.۵ جائے پیدائش

..... (ملک کا نام لکھیے) آپ کہاں پیدا ہوئی تھیں

۱.۶ تعلیمی حیثیت

- ..... ۱ کبھی اسکول میں نہیں پڑھا
- ..... ۲ ابتدائی اسکول پڑھا
- ..... ۳ کچھ ثانوی اسکول پڑھا
- ..... ۴ پائی اسکول درجہ ۱۲ مکمل کیا یا مساوی
- ..... ۵ یونیورسٹی، یا اعلیٰ مساوی تعلیم مکمل کی
- ..... ۱.۷ موجودہ روزگار

کیا آپ کوئی جزی، عارضی یا مستقل نوعیت کا کام کرتی ہیں  
ہاں..... یا نہیں.....

2.0 آپ کی متعلقہ طبی ہسپتال یا Medical History

2.1 آپ نے آخری بار کب اپنے خون کا دباؤ یا بلڈ پریشر معلوم کروایا

- ..... ۱ آخری ۳ مہینے کے اندر
- ..... ۲ آخری ۶ مہینے کے اندر
- ..... ۳ پچھلے سال
- ..... ۴ پچھلے تین سال کے اندر
- ..... ۵ پچھلے ۳ سال سے پہلے
- ..... ۶ کبھی نہیں
- ..... ۷ معلوم نہیں

2.2 آپ نے کب آخری بار اپنے خون میں تامل یا گلوٹروٹول کی جانچ کی تھی

آخری ۳ ماہ کے اندر.....، ۶ ماہ کے اندر.....، پچھلے سال.....، پچھلے ۳ سال کے اندر.....، کبھی نہیں کی.....

معلوم نہیں..... (ہاں یا نہیں میں جواب دیں)

3. کیا آپ کو کبھی آپکے ڈاکٹر یا معالج سے معلوم ہوا ہے کہ آپ کو ذیابیطس یا امراض میں سے کسی ایک میں مبتلا ہیں (ہاں نہیں میں جواب دیں)

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..... High Blood Pressure (فشار خون) 1

..... Angina (آضعف قلب) 2

Heart Attack (a "Coronary", Coronary Occlusion, Coronary thrombosis, myocardial infarction)

..... (دورائے قلب) 3

..... Diabetes (ذیابیطس) 4

..... High cholesterol کلسٹرول کی زیادتی 5

..... High Triglycerides 6

..... Obesity موٹاپا فریبی 7

2.4 کیا آپ فشار خون / بلڈ پریشر کی کوئی دوا استعمال کرتی ہیں

2.5 کیا آپ کلسٹرول Cholesterol کی کمی کے علاج کراتی رہی ہیں

2.6 کیا آپ صعف قلب Angina کے علاج کی دوا استعمال کرتی ہیں

2.7 کیا آپ کو معالج یا نرس سے معلوم ہوا ہے کہ آپ کو ذیابیطس تھی اگر آپ کو بتایا گیا ہے تو سال کا اندراج کریں

2.8 کیا آپ آجکل خون پتلا کرنے کی دوا جیسے "Warfarin" استعمال کر رہی ہیں

2.9 کیا آپ کو کبھی بھی ذیابیطس یا شوگر کی تکلیف کے بارے میں نصیحت یا علاج ہوا ہے

اگر آپ کا جواب ہاں ہے تو سال کا اندراج بھی کریں

کیا وہ:

1 نصیحت برائے خوراک تھی

2 انسولین انجیکشن

3 نصیحت برائے خوراک و دوا

4 نصیحت برائے خوراک و انجیکشن

2.10 کیا آپ نے کبھی مانع حمل کوئی دوا استعمال کی ہے

ہاں ..... / نہیں ..... 2.11 کیا آجکل آپ حاملہ ہیں آپ کا جواب ہاں ..... / نہیں

## ۳۔ تفریح کھیل صحت اور جسمانی چستی

۳.۱ پچھلے دو ہفتوں میں کیا آپ نے کوئی سخت ورزش کی ہے؟ ایسی ورزش جس میں آپ ہانپنے لگی ہوں یا تیز تیز سانس لیں لی ہوں ہاں / نہیں.....

اگر سخت ورزش کی ہے تو دو ہفتوں کے دوران کتنی بار کی ہے؟.....

۳.۲ دو ہفتوں کے دوران آپ نے مجموعی طور پر ورزش میں کتنا وقت صرف کیا..... گھنٹہ / منٹ

۳.۳ پچھلے دو ہفتوں میں کیا آپ نے ہلکی ورزش کی ہے جس سے مقصد تفریح کھیل یا فٹنس مقصود ہو اور جس سے آپ نہ تو ہانپنے لگے ہوں اور نہ تیز سانس چلی ہو..... ہاں / نہیں

اگر ہلکی ورزش کی ہے تو پچھلے دو ہفتوں میں کتنی بار کی ہے؟.....

۳.۴ پچھلے دو ہفتوں میں کیا آپ نے تفریح یا ورزش کے مقصد کیلئے چہل قدمی کی ہے؟ ہاں / نہیں

اگر ہاں جواب ہے تو کتنی بار.....

۳.۵ محنت طلب فرائض ملازمت میں یا گھر میں (اجرت یا بغیر اجرت کام)

پچھلے دو ہفتوں میں کیا آپ نے گھریلو کام یا ملازمت کے سلسلے میں سخت محنت کی ہے اور اس کی وجہ سے ہانپنے لگی ہیں یا سانس پھول گئی ہے (مثال کے طور پر وزن اٹھا کر چلنا، محنت طلب باغبانی، کھاڑی

سے لکڑی کاٹنا یا گھر میں اور کوئی سخت محنت کا کام نہیں..... ہاں /

اگر ہاں تو پچھلے دو ہفتوں میں کتنی مرتبہ اس قسم کی محنت کی ہے.....

اندازہ کے طور پر مجموعی طور پر کتنا وقت پچھلے دو ہفتوں میں اس قسم کے کاموں میں صرف کیا

..... گھنٹے / منٹ

۳.۵ اگر آپ کو تیراکی کا موقع ملے تو کیا آپ تیراکی کریں گی؟..... ہاں / نہیں

۳.۶ اگر آپ کو گروپ کی صورت میں چہل قدمی کا موقع ملے تو کیا آپ کریں گی؟..... ہاں / نہیں

۳.۷ خوراک اور غذائیت

۳.۸ کیا آپ اپنا کھانا خود بناتی ہیں؟..... ہاں / نہیں، اگر ہاں تو ہمیشہ..... ہاں / نہیں

اگر نہیں تو ہفتے میں کتنے دن.....

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۳۴.۳ کھانا تیار کرنے کا ذمہ کس کا ہے؟

۱. آپ خود .....

۲. خاندان کا کوئی فرد .....

یا کوئی دوسرا ..... ( نشان دہی کریں )

۳.۳ آپ کیسے فیصلہ کرتی ہیں کہ کیا کھانا تیار ہوگا؟

۱. کھانے کی کتاب سے .....

۲. روایتی کھانے .....

۳. خاندان / یا دوستوں کے مشورے سے .....

۴. خاص موقعوں / مذہبی موقعوں پر .....

۳.۴ کیا آپ نے وہی کھانا تیار کرنے کے طریقے رکھے ہیں جو آپ پاکستان میں کرتی تھیں؟

ہاں / نہیں .....

۳.۵ اگر آپ کے کھانا تیار کرنے کے طریقے تبدیل ہو گئے ہیں تو کیسے؟ وضاحت کریں

۳.۶ آپ کھانا تیار کرنے میں کس قسم کی چکنائیاں استعمال کرتی ہیں؟

نباتی تیل .....

( کینولا ) سرسوں کا تیل .....

زیتون کا تیل .....

( جانور سے حاصل شدہ چکنائی ) گھی .....

کوئی دوسری چکنائی .....

۳.۷ کیا آپ خیال کرتی ہیں کہ جو غذا آپ پاکستان میں کھاتی تھیں وہ نوعیت اور مقدار کے اعتبار سے تبدیل ہو گئی ہے؟ ہاں / نہیں ..... اگر تبدیل ہو گئی ہے تو کیوں وضاحت کریں

کیا آپ یہ خیال کرتی ہیں کہ جب سے آپ آسٹریلیا آئی ہیں آپ کی غذائی عادات تبدیل گئی ہیں مثال کے طور پر کھانے کی قسمیں جو آپ بناتی ہیں اور کھانا پکانے کے طریقے وضاحت کریں

آپ کے خیال میں آپ کے اور آپ کے پڑوسی کے کھانے میں کیا فرق ہے؟ وضاحت کریں

آپ کے خیال میں آپ کی غذائی عادات کیوں تبدیل ہو گئی ہیں؟

تہذیبی اختلافات .....

تندرستی .....

ملازمت .....

وقت .....

مالی حالات .....

دوسری کوئی وجہ وضاحت کریں

کیا آپ اپنی خوراک کو صحت افزا خیال کرتی ہیں؟ ہاں / نہیں

کیا آپ غذا کے پانچ گروپ سے آگاہ ہیں؟ ہاں / نہیں

کتنا تیل یا چکنائی آپ ہر ہفتے خریدتی ہیں؟

کچھ نہیں / دو لیٹر

۴.۸ کیا آپ یہ خیال کرتی ہیں کہ جب سے آپ آسٹریلیا آئی ہیں آپ کی غذائی عادات تبدیل گئی ہیں مثال کے طور پر کھانے کی قسمیں جو آپ بناتی ہیں اور کھانا پکانے کے طریقے وضاحت کریں

۴.۹ آپ کے خیال میں آپکے اور آپکے پڑوسی کے کھانے میں کیا فرق ہے؟ وضاحت کریں

۴.۱۰ آپکے خیال میں آپکی غذائی عادات کیوں تبدیل ہو گئی ہیں؟

تہذیبی اختلافات .....

تندرستی .....

ملازمت .....

وقت .....

مالی حالات .....

دوسری کوئی وجہ وضاحت کریں

۴.۱۱ کیا آپ اپنی خوراک کو صحت افزا خیال کرتی ہیں؟ ہاں / نہیں

۴.۱۲ کیا آپ غذا کے پانچ گروپ سے آگاہ ہیں؟ ہاں / نہیں

۴.۱۳ کتنا تیل یا چکنائی آپ ہر ہفتے خریدتی ہیں؟

کچھ نہیں ..... دو لیٹر .....

چار لیٹر ..... دیگر (لیٹر میں وضاحت کریں) .....

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کیا آپ کھانا تیار ہونے کے بعد اس میں مزید نمک ڈالتی ہیں؟

بہت کم یا کبھی نہیں.....

کبھی کبھی.....

ہر دفعہ یا تقریباً ہر دفعہ.....

آپ گوشت کے ساتھ چکنائی کتنی دفعہ کھاتے ہیں؟

عام طور پر.....

کبھی کبھی.....

بہت کم یا کبھی نہیں.....

کیا آپ مندرجہ ذیل میٹھے میں سے کوئی استعمال کرتے ہیں؟

کھیر (چاول کی) ہاں / نہیں

گجریلا (گاجر کا علاوہ) ہاں / نہیں

کوئی دوسرا میٹھا وضاحت کریں.....

اگر ہاں تو بتائیں کہ ہفتے میں کتنی مرتبہ.....

کیا آپ مندرجہ ذیل..... سے کوئی استعمال کرتی ہیں؟

چپاتی ہاں / نہیں

پراٹھا ہاں / نہیں

نان ہاں / نہیں

اگر استعمال کرتی.....

سوالنامہ برائے متواتر غذائی تبدیلی: (فلسفہ ٹو اے)

METABOLIC SYNDROME

میٹابولک سنڈروم

تارکین وطن خواتین میں غذا اور جسمانی محنت کے اثرات

سوالنامہ برائے متواتر غذائی تبدیلی

مندرجہ ذیل چارٹ میں (صحیح) کا نشان لگا کر ظاہر کریں کہ کونسا غذائی جزو کتنی بار استعمال کرتی ہیں اور یہ بھی ظاہر کریں کہ اگر آسٹریلیا آنے کے بعد کسی غذائی جزو کے استعمال میں کمی یا زیادتی ہوئی ہو، +3 سے -3 کے پیمانے پر (-3, -2, -1, 0, +1, +2, +3) ، -3، انتہائی کمی +3، انتہائی زیادتی اور 0 بغیر کسی تبدیلی کو ظاہر کرے

غذائی جزو	ایک سے زیادہ روزانہ	روزانہ ایک بار	3-6 دفعہ ہفتہ وار	ایک یا دو گنا ہفتہ وار	مہینہ وار یا کم	کبھی نہیں	استعمال میں تبدیلی آسٹریلیا آنے کے بعد
مثال (سلاد)			✓				زیادتی +2
موسمی پھل (متعدد)							
ڈبہ میں پینک پھل							
سلاد (متعدد)							
سبزیاں (متعدد)							
ہری سبزیاں							
چائے							
کافی							
پھلوں کا جوس							
میٹھا جوس							

غذائی جزو	ایک سے زیادہ روزانہ	روزانہ ایک بار	3-6 دفعہ ہفتہ وار	ایک یا دو گنا ہفتہ وار	مہینہ وار یا کم	کبھی نہیں	استعمال میں تبدیلی آسٹریلیا آنے کے بعد
دیگر							
Legumes							
چنا							
چھولے							
مونگ							
دیگر							
اسٹیکس							
چاکلیٹ / لولی							+2
اخروٹ							
چپس							
بسکٹ							
پکوڑا							
سموسہ							
پاپ							
دیگر							
شربت							
سافٹ ڈرنک							
ڈائٹ سافٹ ڈرنک							

غذائی جزو	ایک سے زیادہ روزانہ	روزانہ ایک بار	3-6 دفعہ ہفتہ وار	ایک یا دو گنا ہفتہ وار	مہینہ وار یا کم	کبھی نہیں	استعمال میں تبدیلی آسٹریلیا آنے کے بعد
متعدد							+1
آلو		✓					
(متعدد) روٹی		✓					
چاول		✓					
(Pasta) پاستا		✓					
دودھ							
فل کریم		✓					+2
چکنائی کے بغیر پنیر							-1
ٹیسٹ (Tasty)			✓				+2
فٹا (Fetta)			✓				
کون (Coon)			✓				
موزاریللا			✓				
کھن / مارجرین		✓					
spreads		✓					
دھی		✓					
آلسکریم		✓					
گوشت							

غذائی جزو	روزانہ ایک سے زائد	روزانہ ایک	3-6 ہفتہ وار	ایک یا دو گنا ہفتہ وار	مہینہ وار یا کم	کبھی نہیں	استعمال میں تبدیلی اسٹریٹیا کرنے کے بعد
بھیڑ							
گائے کا گوشت							
بکرے کا گوشت							
sausages							
سلامی salami							
سمندری غذا							
مچھلی							
دیگر							
poultry							
مرغی							
بطخ							
e.g. فاسٹ فوڈ							
pizza, KFC							
چکنائی / تیل							
سبزی							
زیتون							
کینولا canola							
گھی							
(جانور کی چکنائی)							

۴.۸ کیا آپ یہ خیال کرتی ہیں کہ جب سے آپ آسٹریلیا آئی ہیں آپ کی غذائی عادات تبدیل گئی ہیں مثال کے طور پر کھانے کی قسمیں جو آپ بناتی ہیں اور کھانا پکانے کے طریقے وضاحت کریں

۴.۹ آپ کے خیال میں آپکے اور آپکے پڑوسی کے کھانے میں کیا فرق ہے؟ وضاحت کریں

۴.۱۰ آپکے خیال میں آپکی غذائی عادات کیوں تبدیل ہو گئی ہیں؟

تہذیبی اختلافات .....

تندرستی .....

ملازمت .....

وقت .....

مالی حالات .....

دوسری کوئی وجہ وضاحت کریں

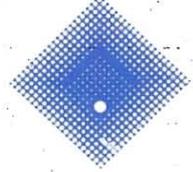
۴.۱۱ کیا آپ اپنی خوراک کو صحت افزا خیال کرتی ہیں؟ ہاں / نہیں

۴.۱۲ کیا آپ غذا کے پانچ گروپ سے آگاہ ہیں؟ ہاں / نہیں

۴.۱۳ کتنا تیل یا چکنائی آپ ہر ہفتے خریدتی ہیں؟

کچھ نہیں ..... دو لیٹر .....

چار لیٹر ..... دیگر (لیٹر میں وضاحت کریں) .....



ریسرچ کنندہ کے نام:

Dr. Paul Lewandowski ڈاکٹر پال لیوان ڈوسکی

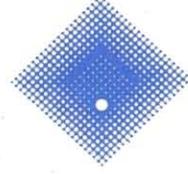
Dr. Cate Burns ڈاکٹر کیٹ برنز

Mrs. Rizwana Kousar مسز رضوانہ کوثر

تفصیل برائے شرکت کنندہ

وکتوریہ یونیورسٹی متعدد ریسرچ سے فسک ہے۔ میں وکتوریہ یونیورسٹی کی ایک پی ایچ ڈی طالب علم ہوں (اسکول برائے بائیومیڈیکل سائنسز)۔ میں ایک ریسرچ کر رہی ہوں جس سے غذائی استعمال اور ورزش کے اثرات جو صحت سے متعلق مسائل پر ہوتے ہیں انکا مطالعہ ہو سکے گا جیسے موٹاپا (فریبی)۔ ذیابلس، بلڈ پریشر (فشار خون) کلو سٹروں کے درجات کی تبدیلی (یہ تمام صحت سے متعلق مشکلات اجتماعی طور پر میٹابولک سنڈروم کہلاتی ہیں) جیسا کہ ہمارا ماحول یا کچر ہمارے غذائی انتخاب پر اثر انداز ہوتا ہے اس لیے اس بات کی اہمیت ہے کہ ہم غذائی تبدیلی اور جسمانی محنت کے صحت پر اثرات کو جانچنے کی کوشش کریں۔ آپ کو اس ریسرچ پراجیکٹ میں شرکت کی دعوت دی جاتی ہے۔ اس مطالعہ سے ملبورن کی تارکین وطن خواتین کو بے شمار فوائد حاصل ہو سکتے ہیں۔ خواتین کو کچرل غذائی آٹم کے متعلق ایک بہتر معلومات حاصل ہونگی۔ اس مطالعہ کا مقصد میٹابولک سنڈروم اور پاکستانی خواتین سے متعلق بنیادی معلومات فراہم کرنا ہے۔ میرا آپ سے فون پر رابطہ رہے گا۔ اگر آپ اس مطالعہ میں مزید کردار ادا کرنا چاہتی ہیں تو ایک تعارفی ملاقات کا انتظام کیا جائے گا اس ملاقات میں ضروری کاغذات (راضی نامہ اور تفصیل برائے شرکت کنندہ) مہیا کیے جائیں گے اس کے بعد میں اس مطالعہ کے مقاصد اور شرکت کنندہ سے متعلق ضروریات اور معلومات جمع کرنے کے عمل سے مکمل آگاہی مہیا کرونگی۔ شرکت کنندہ کو پورا موقع دیا جائے گا کہ وہ تمام سوالات جو اس کے ذہن میں ہوں کر سکے اور اپنے مناسب وقت میں وہ تمام مہیا کردہ معلومات پڑھ کر پھر راضی نامہ کو بھر سکیں۔ کچھ دنوں بعد ریسرچ کنندہ تمام پیپر ورک کو اکٹھا کرے گا۔ آپ ایک راضی نامہ فارم پر دستخط کر کے اس ریسرچ میں شرکت کر سکتے ہیں۔ اس مرحلے پر تمام شرکت کنندہ کو ایک دوسرا تفصیلی سوالنامہ مکمل کرنا ہوگا۔ یہ سوالنامہ آپکی ذاتی تفصیلات اور صحت سے متعلق حالت کے بارے میں ہوگا (جیسا کہ پہلے سوالنامہ میں تھا)۔ یہ سوالنامہ آپکے غذائی استعمال اور غذائی عادات سے بھی متعلق ہوگا (آپ کس طرح کی غذا تیار کرتی ہیں کتنی چکنائی کا استعمال کرتی ہیں اور کونسی روایتی غذا استعمال کرتی ہیں) اور اس کے علاوہ آپکی جسمانی محنت اور عادات سے بھی متعلق ہوگا (جیسے کیا آپ کوئی ورزش کرتی ہیں یا نہیں) اپنی غذائی عادات کو ریکارڈ کرنے کے لیے آپکو غذائی

Victoria University

PO Box 14428  
Melbourne City MC  
VIC 8001 AustraliaTelephone:  
(03) 9365 2667  
Facsimile:  
(03) 9365 2469VICTORIA  
UNIVERSITYOF  
TECHNOLOGY

School of Biomedical Sciences

St Albans Campus  
McKechnie Street  
St Albans

تحقیق میں شامل شرکت کنندگان کے لیے اجازت نامہ

اطلاع برائے شرکت کنندگان

ہم آپ کو اپنی تحقیق میں شرکت کی دعوت دیتے ہیں جو تحقیق برائے میٹابولک سنڈروم METABOLIC SYNDROME ہے  
تارکین وطن خواتین میں غذا اور جسمانی محنت کے اثرات  
شرکت کنندگان کی تصدیق

میں

کی (OF)

تصدیق کرتی ہوں کہ میری عمر کم از کم 18 سال ہے اور میں اپنی مرضی سے اس تجربہ میں شریک ہونے کی تصدیق  
کر رہی ہوں جس کا عنوان مندرجہ ذیل ہے

METABOLIC SYNDROME

تارکین وطن خواتین میں غذا اور جسمانی محنت کے اثرات  
یہ تحقیق وکٹوریہ یونیورسٹی آف ٹکنالوجی میں ہو رہی ہے جس کے نگران ہیں:  
ڈاکٹر پیال، ڈاکٹر کیٹ، اور مسز رضوانہ کوثر

( Dr. Paul Lewandowski, Dr. Cate Burns, &amp; Mrs. Rizwana Kousar )

میں تصدیق کرتی ہوں کہ تجربہ کے تمام مقاصد اور تجربہ کے عمل سے منسلک تمام خطرات کے بارے میں مجھے مسز رضوانہ کوثر  
نے پوری طرح آگاہ کیا ہوا ہے اور میں بغیر کسی دباؤ کے ان تمام عمل سے گزرنے کے لیے اپنی رضامندی ظاہر کرتی ہوں

طریقہ کار:

سوالنامہ کی تکمیل:

سوالنامہ برائے غذائی استعمال کی تکمیل

طبعی پیمائش:

بلڈ پریشر کی پیمائش:

خون کا نمونہ:

میں تصدیق کرتی ہوں کہ مجھے تمام مواقع حاصل رہے کہ میں اپنے تمام سوالات کے جوابات حاصل کر سکوں اور میں سمجھتی  
ہوں کہ میں جب بھی چاہوں اس تجربہ سے پیچھے ہٹ سکتی ہوں اور میرے اس عمل سے مجھے کسی بھی طور کسی نقصان کا احتمال نہیں

۴.۸ کیا آپ یہ خیال کرتی ہیں کہ جب سے آپ آسٹریلیا آئی ہیں آپ کی غذائی عادات بدل گئی ہیں مثال کے طور پر کھانے کی قسمیں جو آپ بناتی ہیں اور کھانا پکانے کے طریقے وضاحت کریں

۴.۹ آپ کے خیال میں آپکے اور آپکے پڑوسی کے کھانے میں کیا فرق ہے؟ وضاحت کریں

۴.۱۰ آپکے خیال میں آپکی غذائی عادات کیوں تبدیل ہو گئی ہیں؟

تہذیبی اختلافات .....

تندرستی .....

ملازمت .....

وقت .....

مالی حالات .....

دوسری کوئی وجہ وضاحت کریں

۴.۱۱ کیا آپ اپنی خوراک کو صحت افزا خیال کرتی ہیں؟ ہاں / نہیں

۴.۱۲ کیا آپ غذا کے پانچ گروپ سے آگاہ ہیں؟ ہاں / نہیں

۴.۱۳ کتنا تیل یا چکنائی آپ ہر ہفتے خریدتی ہیں؟

کچھ نہیں ..... دو لیٹر .....

چار لیٹر ..... دیگر (لیٹر میں وضاحت کریں) .....

۴.۸ کیا آپ یہ خیال کرتی ہیں کہ جب سے آپ آسٹریلیا آئی ہیں آپ کی غذائی عادات بدل گئی ہیں مثال کے طور پر کھانے کی قسمیں جو آپ بناتی ہیں اور کھانا پکانے کے طریقے وضاحت کریں

۴.۹ آپ کے خیال میں آپ کے اور آپ کے پڑوسی کے کھانے میں کیا فرق ہے؟ وضاحت کریں

۴.۱۰ آپ کے خیال میں آپ کی غذائی عادات کیوں تبدیل ہو گئی ہیں؟

تہذیبی اختلافات .....

تندرستی .....

ملازمت .....

وقت .....

مالی حالات .....

دوسری کوئی وجہ وضاحت کریں

۴.۱۱ کیا آپ اپنی خوراک کو صحت افزا خیال کرتی ہیں؟ ہاں / نہیں

۴.۱۲ کیا آپ غذا کے پانچ گروپ سے آگاہ ہیں؟ ہاں / نہیں

۴.۱۳ کتنا تیل یا چکنائی آپ ہر ہفتے خریدتی ہیں؟

کچھ نہیں ..... دو لیٹر .....

چار لیٹر ..... دیگر (لیٹر میں وضاحت کریں)

مجھے یہ بتایا گیا ہے کہ میری فراہم کردہ معلومات کو مکمل طور پر خفیہ رکھا جائے گا

دستخط .....

دیگر گواہان (شرکت کنندہ کے علاوہ)

تاریخ .....

کسی قسم کا بھی سوال جس کا تعلق اس پروجیکٹ سے ہے وہ ریسرچ کنندہ کے حوالے کر دیا جائے گا جو یہ ہیں

(نام: ڈاکٹر پال لیوان ڈوسکی، فون نمبر (03-93652321) یا مسز رضوانہ کوثر، فون نمبر (03-93652711)

اگر آپ کسی قسم کی شکایت یا کوئی سوال کرنا چاہیں تو آپ سیکریٹری، یونیورسٹی ہیومن ریسرچ برائے اخلاقی کمیٹی،  
وکتوریہ یونیورسٹی آف ٹکنالوجی سے رابطہ کر سکتے ہیں۔

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۴.۸ کیا آپ یہ خیال کرتی ہیں کہ جب سے آپ آسٹریلیا آئی ہیں آپ کی غذائی عادات تبدیل گئی ہیں مثال کے طور پر کھانے کی قسمیں جو آپ بناتی ہیں اور کھانا پکانے کے طریقے وضاحت کریں

۴.۹ آپ کے خیال میں آپکے اور آپکے پڑوسی کے کھانے میں کیا فرق ہے؟ وضاحت کریں

۴.۱۰ آپکے خیال میں آپکی غذائی عادات کیوں تبدیل ہو گئی ہیں؟

تہذیبی اختلافات .....

تندرستی .....

ملازمت .....

وقت .....

مالی حالات .....

دوسری کوئی وجہ وضاحت کریں

۴.۱۱ کیا آپ اپنی خوراک کو صحت افزا خیال کرتی ہیں؟ ہاں / نہیں

۴.۱۲ کیا آپ غذا کے پانچ گروپ سے آگاہ ہیں؟ ہاں / نہیں

۴.۱۳ کتنا تیل یا چکنائی آپ ہر ہفتے خریدتی ہیں؟

کچھ نہیں ..... دو لیٹر .....

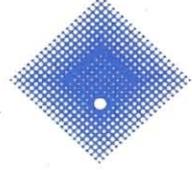
چار لیٹر ..... دیگر (لیٹر میں وضاحت کریں)

Victoria University

PO Box 14428  
Melbourne City MC  
VIC 8001 Australia

Telephone:  
(03) 9365 2667  
Facsimile:  
(03) 9365 2466

VICTORIA  
UNIVERSITY



OF  
TECHNOLOGY

School of Biomedical Sciences  
St Albans Campus  
McKechnie Street  
St Albans

وکتوریہ یونیورسٹی آف ٹیکنالوجی  
تحقیق میں شامل شرکت کنندگان کے لیے اجازت نامہ

اطلاع برائے شرکت کنندگان

ہم آپ کو اپنی تحقیق میں شرکت کی دعوت دیتے ہیں جو تحقیق برائے میٹابولک سنڈروم METABOLIC SYNDROME ہے  
تاریکین وطن خواتین میں غذا اور جسمانی محنت کے اثرات  
شرکت کنندگان کی تصدیق

میں

کی (OF)

تصدیق کرتی ہوں کہ میری عمر کم از کم 18 سال ہے اور میں اپنی مرضی سے اس تجربہ میں شریک ہونے کی تصدیق  
کر رہی ہوں جس کا عنوان مندرجہ ذیل ہے

METABOLIC SYNDROME

تاریکین وطن خواتین میں غذا اور جسمانی محنت کے اثرات  
یہ تحقیق وکتوریہ یونیورسٹی آف ٹیکنالوجی میں ہو رہی ہے جس کے نگران ہیں:  
ڈاکٹر پال، ڈاکٹر کیٹ، اور مسز رضوانہ کوثر

( Dr. Paul Lewandowski, Dr. Cate Burns, & Mrs. Rizwana Kousar )

میں تصدیق کرتی ہوں کہ تجربہ کے تمام مقاصد اور تجربہ کے عمل سے منسلک تمام خطرات کے بارے میں مجھے مسز رضوانہ کوثر  
نے پوری طرح آگاہ کیا ہوا ہے اور میں بغیر کسی دباؤ کے ان تمام عمل سے گزرنے کے لیے اپنی رضامندی ظاہر کرتی ہوں

طریقہ کار:

سوالنامہ کی تکمیل:

سوالنامہ برائے غذائی استعمال کی تکمیل

طبعی پیمائش:

بلڈ پریشر کی پیمائش:

خون کا نمونہ:

میں تصدیق کرتی ہوں کہ مجھے تمام مواقع حاصل رہے کہ میں اپنے تمام سوالات کے جوابات حاصل کر سکوں اور میں سمجھتی  
ہوں کہ میں جب بھی چاہوں اس تجربہ سے پیچھے ہٹ سکتی ہوں اور میرے اس عمل سے مجھے کسی بھی طور کسی نقصان کا احتمال نہیں

# APPENDIX-B

## ATTACHMENT B-1

### MODULE 1

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#### **Variety is the spice of life**

Variety in our diet is important to ensure we eat all the nutrients we require. No single food can give us all the nutrients we need. To have a balanced diet, different proportions of foods are to be eaten on a day to day basis.

#### **Eat A Lot**

Vegetables (especially green leafy vegetables), legumes (peas, beans, lentils and chickpeas), fruits, bread (paratha, roti), cereals, rice, pasta, noodles

These foods are important as they give us the vitamins and minerals we need for good health. These foods are rich in carbohydrates, fibre, vitamins A, B, C and a wide variety of minerals.

They help us to feel our best and protect us from a number of illnesses, including some forms of cancer, heart disease and diabetes.

They are low in fat and sugar and so help us control our weight.

They contain a lot of fibre which helps us to avoid constipation.

#### **Eat Some**

Milk, yoghurt, cheese, lean meat, fish, chicken, eggs, nuts

These foods give us protein and some important vitamins and minerals such as calcium for healthy teeth and bones and iron.

They also contain some fats and too much fat is not good for our health.

#### **Eat a Little**

Butter, margarine, oil, sugar

These foods add taste to food but need to be eaten in very small amounts.

Too much fat may give us trouble controlling our weight and will increase cholesterol.

Sugar gives us energy. But too much sugar can lead to tooth decay and may make it difficult to manage weight.

### ***Worksheet 1.1: Why should you change?***

We can all make some changes to reduce our risk of disease. The best way to reduce our risk of disease, or to prevent it getting worse, is to identify and reduce the risk factors that we can alter.

There are some risk factors for disease that we cannot change. These include:

- having a family history of disease
- increasing age.

#### **Circle the risk factors that apply to you.**

The good news is there are many risk factors that we can alter, including:

- high total blood cholesterol
- physical inactivity
- high blood pressure
- being overweight
- diabetes.

## Worksheet 1.2: How's your usual food intake?

Write down everything you ate yesterday:

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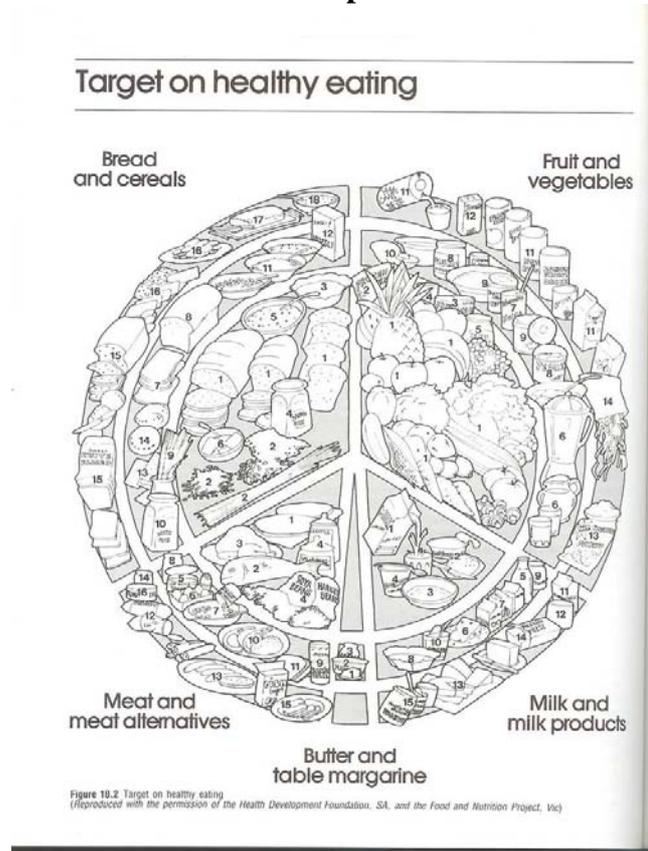
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Now compare your current intake with the list below. Tick the right hand column to highlight eating habits you feel you could improve.

Do you:	Could be improved
Eat a proper breakfast? Have 3 regular meals a day; breakfast, lunch, dinner ? Make grain-based foods such as breakfast cereals, bread, pasta, noodles and rice a major part of each meal?	
Have a serve of vegetables during each meal?	
Eat fresh fruit daily?	
Choose low or reduced fat milk and yoghurt or 'added calcium' soy beverages? Try to limit cheese and ice cream to twice a week.	
Have fish (any type, fresh or canned) at least twice a week?	
Select lean meat (meat trimmed of fat and chicken without skin)? Try to limit fatty meats including sausages	
Incorporate dried peas (e.g. split peas), dried beans (e.g. haricot beans, kidney beans), canned beans (e.g. baked beans, three bean mix) or lentils into two meals a week?	
Snack on plain, unsalted nuts?	
Use a variety of oils for cooking? Some suitable choices include canola, sunflower, soybean, olive and peanut oils. Use margarine spreads instead of butter or dairy blends?	
Try to limit takeaway foods to once a week? Takeaway foods include pastries, pies, pizza, hamburgers and creamy pasta dishes.	
Try to limit snack foods such as potato crisps and corn crisps to once a week?	

Try to limit cakes, pastries and chocolate or sweet biscuits to once a week.	
Use salad dressings and mayonnaise made from oils such as canola, sunflower, soybean and olive oils?	
Try to limit cholesterol-rich foods such as egg yolks and offal (e.g. liver, kidney and brains)?	

**Serve sizes of Food on a plate**



Taking steps to reduce some of these risk factors will help you have a healthy life. Healthy eating is especially important in the prevention of disease.

For example: Metabolic Syndrome

The constellation of abnormal levels of certain cholesterol, elevated blood pressure, impaired glucose tolerance, and central obesity is identified now as metabolic syndrome, also called syndrome X. Metabolic syndrome has been identified as an indication for vigorous lifestyle intervention. Effective interventions include diet, exercise, and judicious use of pharmacologic agents to address specific risk factors. Weight loss significantly improves all aspects of metabolic syndrome. Increasing physical activity and decreasing caloric intake by reducing portion sizes will improve metabolic syndrome abnormalities, even in the absence of weight loss. Specific dietary changes that are appropriate for addressing different aspects of the syndrome include reducing saturated fat intake to lower insulin resistance, reducing sodium intake to lower blood pressure, and reducing high-glycemic-index carbohydrate intake to lower triglyceride levels. A diet that includes more fruits, vegetables, whole grains, monounsaturated fats, and low-fat dairy products will benefit most patients with metabolic syndrome.

	EXCELLENT CHOICES	GOOD CHOICES	FAIR CHOICES
<b>Bread and cereals</b>	1. Wholemeal bread 2. Wholemeal pasta 3. Rolled oats and oatmeal 4. Brown rice 5. Unsweetened muesli 6. Ready to eat wholegrain low salt, low sugar breakfast cereal	7. White bread ★ 8. Brown bread ★ 9. Refined pasta ★ 10. White rice ★ 11. Refined, unsweetened breakfast cereal ★ 12. Wholegrain cereal high in added sugar or salt ● or ■ 13. Unsalted, refined, savoury biscuits, wholegrain salted crackers ★ or ■ 14. Crumpets ★	15. Refined sweet breads and buns ★ ● 16. Refined salty savoury crackers ★ ■ 17. Plain wholemeal cakes and biscuits ● ▲ 18. Refined, sweet breakfast cereal and refined, high salt breakfast cereal ★ ● ■
<b>Fruit and vegetables</b>	1. Raw or cooked fresh fruit and vegetables 2. Frozen fruit or vegetables 3. Canned, unsweetened fruit 4. Canned, unsalted vegetables 5. Dried fruit	6. Unsweetened fruit juice ★ 7. Unsalted vegetable juice ★ 8. Canned fruit with added sugar ● 9. Canned vegetables with added salt ■ 10. Avocado ▲	11. Fruit juice drinks ★ ● 12. Salted vegetable juice ★ ■ 13. Mashed potato ★ ▲ 14. Unsalted potato chips ★ ▲
<b>Meat and meat alternatives</b>	1. Lean, trimmed meat 2. Non-oily fish, trimmed 3. Lean poultry 4. Dried peas, beans, lentils	5. Canned fish in brine ■ 6. Eggs ▲ 7. Unsalted nuts ▲ 8. Unsalted peanut paste ▲ 9. Canned baked beans ■ 10. Lean, untrimmed meats and poultry ▲ 11. Salted lean trimmed meats e.g. corned beef ■	12. Processed meats eg. Fritz, Devon ▲ ■ 13. Sausage ▲ ■ 14. Salted peanut paste and salted nuts ▲ ■ 15. Fish fingers ▲ ■ 16. Canned salted fish in oil ▲ ■
<b>Milk and milk products</b>	1. Skim or low fat milk 2. Cottage cheese 3. Ricotta cheese 4. Low fat yoghurt	5. Whole plain milk ▲ 6. Swiss cheese ▲ 7. Flavoured, low fat milk ● 8. Low fat milk custard ● 9. Whole, plain yoghurt ▲ 10. Low fat, flavoured yoghurt ●	11. Flavoured whole milk ▲ ● 12. Evaporated milk ▲ ■ 13. Cheese: processed, cheddar, gouda, edam ▲ ■ 14. Cheese spreads ▲ ■ 15. Whole, flavoured yoghurt ● ▲
<b>Butter and table margarine</b>		1. Butter ▲ 2. Table margarine ▲ 3. Cream ▲	
<b>Symbols:</b> ■ High salt      ▲ High fat ● High added sugar      ★ Fibre reduced (plant foods only) The symbol is bigger if the food is very high in that factor.			

### Total amounts of food per day

Milk fat reduced - 600ml (300 ml =40g cheese = 1 tub yoghurt)

Breakfast cereal -1 cup (1 cup = 2 weetbix)

Bread -4 slices (1 roti or 1 pita bread)

Biscuits- 2 plain

Rice or potato (2/3 cup)

Vegetables- 5 different vegetables everyday (1 serve=1/2 cup)

Fruits-2 fresh fruit

Lean meat -2 pieces (1 serve = 100g=2/3 cup lentils)

Egg -1 (or extra slice cheese, small tin fish in brine)

Oil -2 tablespoons (include butter or margarine)

### Example of a Daily Menu

**Breakfast** Fruit Cereal + milk Toast + egg or cheese **Mid-morning** Fruit Tea or coffee

**Lunch** Sandwich with meat, fish, cheese and salad or 1 Roti and 1 cup cooked rice + vegetables + meat **Fruit** **Afternoon tea** 2 plain biscuits Tea or coffee **Evening meal** Meat 1 cup cooked Rice or potatoes / Bread ( 2 roti) Vegetables + salad Yoghurt

### Everyday foods

Milk and dairy, Meat, eggs, fish, chicken or lentils, Bread and cereal, Rice and potato, Vegetables, Fruits, Water, herbal tea, diet soft drinks

### Sometimes foods (once a week)

Cake, biscuits, pastries, Ice cream, Hot chips, Chocolate

### Rarely foods (once a month)

Soft drink, Fried foods in batter or crumbs, Mayonnaise

#### Did you know?

Over 200 studies have shown people who have a diet high in fruit and vegetables have a lower risk of stroke and heart attack. Even just one extra serve a day lowers your risk! (WHO Report: Diet, nutrition and the prevention of chronic disease 2003).

#### Now let's check how varied your usual eating pattern is.

Look back at what you ate yesterday and count the number of different foods you had.

If you scored more than 20: Excellent, you have a very varied eating pattern.

If you scored between 12 and 20: You probably had enough variety to meet your nutrition needs.

If you scored less than 12: You probably missed out on some nutrients yesterday. How could you increase your variety?

#### Did you know?

The Japanese dietary guidelines suggest eating at least 30 different foods each day!



**Pilot concept-**

**Worksheet 1.4: Decisional balance sheet**

If you are unsure whether you are ready to make lifestyle changes, it can be useful to compare the benefits and costs of making each change.

Behaviour you are thinking of changing \_\_\_\_\_

Continuing as before	<b>Benefits:</b> (what's good about not changing) ----- -----
	<b>Costs:</b> (what price you might pay for not changing) ----- -----
Making a change	<b>Benefits:</b> (what positives might result from changing) ----- -----
	<b>Costs:</b> (what negatives might result from changing) ----- -----

## ATTACHMENT B-2

### Which fat to choose

### Dietary fats and heart disease

#### **Worksheet 2.1: Let's review**

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_

\_\_\_\_\_

2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

\_\_\_\_\_

Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 2.2 for a new challenge.

Making and sustaining lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

\_\_\_\_\_

\_\_\_\_\_

Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track.

\_\_\_\_\_

\_\_\_\_\_



## **Worksheet 2.2: Dietary fats and heart disease**

### **What is cholesterol?**

Cholesterol is a fatty substance produced naturally by the body and found in the blood. It increases risk of heart problems when there is too much in the blood.

### **What's wrong with high blood cholesterol?**

High total blood cholesterol causes fatty deposits to build up in blood vessels, making it more difficult for blood to flow through. This can reduce blood flow to the heart and lead to a heart attack. Most of the total cholesterol in the blood is made up of 'bad' LDL cholesterol, which clogs blood vessels. Only a small part is made up of 'good' HDL cholesterol, which helps protect against heart disease.

### **What can I do if my blood cholesterol is high?**

High blood cholesterol can be treated by making changes to your lifestyle, and with medication. Making lifestyle changes, in particular making changes to the foods you eat, is very important for reducing your blood cholesterol level and improving your heart health—even if you are taking medication for high blood cholesterol!

*Saturated fat is the type of fat that raises blood cholesterol levels. To reduce your risk of heart disease, it's important to reduce your intake of foods high in saturated fats.*

### **Where is saturated fat found?**

It is found mainly in:

- full fat dairy products (especially milk and cheese)
- fatty meats
- butter and cream
- most deep fried foods like samosa, pakoras, chips
- most commercially baked products such as biscuits and pastries
- coconut oil and palm oil.

It is also found in fried eggs, oil that is heated and reused and in ghee.

### **What should I replace saturated fat with?**

There is good evidence that replacing saturated fat with polyunsaturated and monounsaturated fats will reduce the risk of coronary heart disease. Monounsaturated and polyunsaturated fats are 'healthy' fats.

**Foods high in monounsaturated fats are:**

Some margarine spreads  
Olive oil, canola oil, peanut oil  
Avocados  
Some nuts, e.g. almonds, peanuts

**Foods high in polyunsaturated fats are:**

Some margarine spreads  
Sunflower oil, safflower oil, soybean oil  
Seeds  
Some nuts, e.g. walnuts  
Fish

**What about dietary cholesterol?**

Cholesterol in food can also raise blood cholesterol, particularly in people who are at high risk of heart disease. Try to limit cholesterol-rich foods such as egg yolk and offal (e.g. liver, kidney, brains).

**Now you know which foods are high in saturated fat, let's see how good you are at spotting them!**

Circle the foods high in saturated fat on this menu. Then write an alternative food that is low in saturated fat next to each food you circled.

*Breakfast*

Croissants with jam  
Toast with butter  
Cornflakes with full cream milk

*Lunch*

Sausage roll  
Vegetable soup  
Cheese and salami sandwich

*Dinner*

Fried chicken  
Hot chips  
Chicken curry cooked in coconut milk  
Scone with cream and jam

**Making Healthy Choices**

It is not necessary to eat meat everyday or to eat it in large amounts. The amount that can be held in an adult palm per person per day is enough.

Choose meats with minimal visible fat or fat that can be trimmed before cooking.

Choose skinless chicken or remove skin before cooking, as the fat is in the skin.

Make meat dishes more satisfying by using beans, lentils, chickpeas or grains such as rice or pasta.

Try beef as it tends to be relatively low in fat.

It is best to buy less amounts of high quality, low fat meat than cheaper cuts of fatty meats.

Choose low fat cooking methods such as grilling, boiling, stir frying (without too much oil) or roasting (without added fat).

Fat can be reduced while making curries by boiling the meat the day before, allowing it to cool and skimming the fat off the top before completing cooking.

Nuts should be limited as they are relatively high in fat.

Use of animal fat like in butter increases cholesterol but use of plant fats like in margarine and vegetable oils help lower cholesterol

Fish is a good substitute for meat, as it is high in protein, low in saturated fat and contains a range of vitamins and minerals including Omega 3 fatty acids. These are healthy fats that act in several ways to reduce the risk of heart disease and stroke.

The Heart Foundation recommends that people should have fish (any type, fresh or canned) at least twice a week.

Avoid frying fish as this will make it very high in fat.

Fish with the highest amount of healthy fats include Atlantic salmon, mackerel, southern bluefin tuna, trevally and sardines. However, all types of fish are good choices. Enjoy it steamed, pan-fried, baked or barbecued.

**You can also use food labels to check a food's saturated fat content.** A simple rule when comparing similar food products is to choose the food with the lower saturated fat content.

Compare the following two products. Which is lowest in saturated fat? In total fat? Which is the best choice? (**Answers below.**)

Butter

Canola margarine spread

Nutrient	content per 100g
Energy	3060 kJ
Protein	Less than 1g
Fat—Total	82.2g
Saturated	54.2g
Polyunsaturated	21.5g
Monounsaturated	2.1g
Cholesterol	200mg
Sodium	640mg

Nutrient	content per 100g
Energy	2600 kJ
Protein	Less than 1g
Fat—Total	70 g
Saturated	17.6 g
Polyunsaturated	14.0 g
Monounsaturated	31.5 g
Cholesterol	Nil
Sodium	360mg

Answers: The canola margarine spread has the least saturated fat (17.6g) so it is the best choice.

Module 4 has more information on label reading.



## Worksheet 2.3: Check your saturated fat intake

Let's check whether you could reduce the amount of saturated fat in your usual food intake.

Circle Yes or No in response to each question.

Do you avoid eating meat everyday?	Yes / No
Do you trim the fat and skin from your meat and chicken?	Yes / No
Do you use a variety of oils for cooking, e.g. canola, sunflower, soybean, olive and peanut oils?	Yes / No
Do you use less than one table spoon of oil in your cooking in a day?	Yes / No
Do you limit your intake of fatty meats such as sausages and delicatessen meats?	Yes / No
Do you choose low or reduced fat milk and yoghurt?	Yes / No
Do you try to limit cheese and ice cream to twice a week?	Yes / No
Do you usually have margarine spread instead of butter?	Yes / No
Do you use salad dressings and mayonnaise made from oils such as canola, sunflower, soybean and olive oils?	Yes / No
Do you eat deep fried takeaway food (e.g. chips) less than once a week?	Yes / No
Do you eat commercially made cakes and biscuits only occasionally?	Yes / No
Do you limit potato crisps or chocolate to special occasional treats?	Yes / No

Adapted from *Enjoy healthy eating: A guide to keeping your blood cholesterol in check.* (2002). NHFA.

**If you answered 'Yes' to most questions then you are doing well. If you answered 'No' to most**

### Supermarket news #1

Have you noticed the 'cholesterol lowering' margarine spreads on the supermarket shelf? These margarine spreads contain plant sterols which inhibit the absorption of cholesterol. They can reduce your LDL ('bad') cholesterol level by 10%, but you need to eat around one and a half tablespoons each day. Even if you are taking medication to lower your blood cholesterol, these margarine spreads will still have a cholesterol lowering effect.

## **Supermarket news #2**

Heard of 'omega 3s'? These are a type of polyunsaturated fat that can reduce the risk of heart disease. These healthy fats are mainly found in fish, though they are also found in some plant oils, such as canola, walnut, soy and linseed oil. Aim to choose both plant-based and marine omega 3 food sources as part of a healthy eating pattern. Recently food manufacturers have begun adding omega 3 fats to other products. Next time you're shopping try to spot omega 3-enriched foods such as bread, eggs, deli meats, margarine spreads and milk. They may be worth considering if you do not eat fish.

questions, your saturated fat intake is probably higher than recommended.



## ATTACHMENT B-3

### MODULE 3

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#### **Achieving a healthy weight**

This module aims to help you improve your health, feel more self confident as well as lose weight and centimetres off your waist.

In this programme you will complete questionnaires that will help you to pinpoint the changes in your eating and exercise routines which you will need to make in order to improve your health. This programme will get you to get small achievable goals for eating, exercise and feeling good about yourself. These small steps will help you achieve your long term health goals. It is step by step.

Why diets don't work?

We all want to lose weight fast and nowadays there is no end to the new diets that are advertised in magazines. They are known as 'fad' diets and may provide short term results. But these diets are difficult to sustain and in the long run may do you more harm by depriving you of the essential nutrients your body needs. A fad diet is one that shares some or all of the following characteristics:

- Promises a quick fix
- Promotes magic foods or combination of foods
- Implies that food can change body chemistry
- Excludes or severely restricts food groups or nutrients such as carbohydrates
- Has rigid rules that focus on weight loss
- Makes claims based on a single study or testimonials
- By cutting out key foods, fad diets have been known to cause the following symptoms such as dehydration, weakness and fatigue, nausea and headaches, constipation and inadequate vitamin and mineral intake.

This is NOT a diet. In this programme you are making lifestyle changes which you will maintain long after the programme is finished. Think about any previous attempt you have had to lose weight. You can stick to it for a while but usually the food is not what you prepare for your family, the exercise programme involves a costly gym membership and the gym environment may not be appropriate for you. This may happen several times and with each time you get more depressed and put on more weight. You have to make small achievable changes to your lifestyle that you can keep up...for the rest of your life.

### **Worksheet 3.1: Let's review**

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_  
\_\_\_\_\_
2. \_\_\_\_\_  
\_\_\_\_\_
3. \_\_\_\_\_  
\_\_\_\_\_

Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 3.2 for a new challenge.

Making lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

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Think of different ways to overcome the barriers that stopped you achieving your goals. **These solutions will help you get your progress back on track.**

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## Worksheet 3.2: Healthy weight, healthy body

### Why your weight matters

Weight gain is a major health problem in Australia due to both our sedentary lifestyle and the ready availability of foods that are high in sugar and fat. Overweight people tend to have higher blood pressure and higher blood cholesterol. Being overweight is also associated with other serious diseases such as diabetes. These are all major risk factors for heart attack and stroke.

The good news is that being a healthy body weight can:

- lower high blood cholesterol
- lower blood pressure
- lower blood glucose levels
- reduce your risk of other health related problems.

### How can I tell if I'm overweight?

Measuring your waist circumference (with a tape measure around the level of your belly button) gives you an indication of how much fat you have stored around your middle. People who have a lot of fat stored there are at greater risk of developing diseases such as heart disease and diabetes.

#### My waist circumference is .....cm

Waist circumferences (cms)		Weight management program
Men	Women	
94-102	80-88	Avoid weight gain
> 102	> 88	Take action to lose weight

How does your measurement compare?

Another way to check your weight is to calculate your Body Mass Index (BMI). This examines whether your weight is healthy for your height. If you have a calculator handy, divide your weight (in kg) into your height (in metres<sup>2</sup>).

For example if someone weighs 80 kg and is 173 cm tall:

$$\begin{aligned}\text{BMI} &= 80 \text{ divided by } (1.73 \times 1.73) \\ &= 80 \text{ divided by } 2.9929 \\ &= 27 \text{ (so that person is overweight)}\end{aligned}$$

#### My BMI is .....

Weight categories by BMI are:

Healthy weight	BMI between 18.5 and 22
Overweight	BMI between 23 and 25
Obese	BMI over 25

Both waist circumference and BMI are simply guides. Check with a doctor or dietician if you are concerned about your weight.

### **Worksheet 3.3: Reaching your healthy weight**

If healthy eating and physical activity are part of your usual lifestyle, you have a good chance of reaching and maintaining a healthy weight! Remember, weight loss and weight management are not simple and need lifelong change, so aim for small, gradual, enjoyable changes.

#### ***It's all about balance.***

The keys to achieving and maintaining a healthy weight are to enjoy healthy eating and be physically active. To achieve a healthy body weight, balance the kilojoules coming into your body through food and drinks with the kilojoules being used up by your body through regular physical activity. To achieve weight loss, try to use up more kilojoules through physical activity and consume fewer kilojoules from food and drinks.

#### **If you are trying to lose weight:**

What you weigh is not a good indicator of weight loss, as weight naturally fluctuates from day to day. Also if you are doing more physical activity, your muscles which weigh more than fat, will develop. How your clothing fits, is much more important.

Aim for a steady, slow loss of around one kilogram a month. If you lose weight slowly you are more likely to keep it off.

Measure success not by what you weigh, but by the healthy eating and lifestyle changes made and kept (because these are the things that will keep your weight off in the long run)

#### **Tips on reducing kilocalories**

Foods high in fat are high in kilocalories, so if you are already working on reducing your saturated fat intake you are on the right track. Foods and drinks high in sugar and alcoholic drinks are also high in kilojoules.

**Use this checklist to see how you could reduce your kilocalories.**

<b>Do you:</b>	<b>Could be improved</b>
Choose mainly plant-based foods such as bread, cereals, rice, and pasta? Eat plenty of vegetables, fresh fruit, legumes (dried peas, dried beans and lentils)? These foods are generally low in kilojoules.	
Select lean meat (meat trimmed of fat, lean mince and chicken without skin)? Try to limit fatty meats like sausages and fatty mince meat	
Use cooking methods such as microwaving, grilling, baking (using healthier fats), stir frying (using healthier fats), steaming or boiling? Brush oil onto the frying pan with a brush and use a non stick frying pan?	
Try to limit take-away foods to once a week? Take-away foods include pastries, pies, pizza, hamburgers and creamy pasta dishes.	
Try to limit cakes, pastries, chocolate or cream biscuits to once a week?	
Try to limit snack foods such as potato and corn crisps to once a week?	
Snack on plain unsalted nuts? Note that nuts can contribute to excess	

kilojoules, so limit the amount and frequency of eating them.	
Choose low or reduced fat milk or yoghurt or 'added calcium' soy beverages? Try to limit cheese and ice cream to twice a week. Try yoghurt as a substitute for cream and sour cream? Use ricotta or cottage cheese or paneer as replacements for butter and margarine? Reduce the amount of sugar in your diet?	
Let soups, casseroles and stews cool so fat solidifies on top and then remove the fat before adding the vegetables and reheating?	
Drink water, plain mineral water, soda water or diet soft drinks rather than soft drinks, fruit juice and alcohol?	

## Physical activity

Now let's look at physical activity. Regular, moderate physical activity is vital for achieving a healthy weight. There are plenty of other benefits as well.

People who are regularly physically active tend to:

- have improved long term health
- be less likely to have a heart attack
- feel more energetic
- manage their weight better
- have a healthier blood cholesterol level
- have lower blood pressure
- have stronger bones and muscles
- recover better from a heart attack (if this has occurred)
- feel more confident, happy, relaxed and able to sleep

The Heart Foundation and other leading authorities recommend at least 30 minutes of physical activity at moderate intensity, such as brisk walking, on all or most days of the week. The amount of activity can be accumulated in bouts of ten minutes or more if this is more convenient.

## Check list for exercise

Think about your life in Australia. Think about your life in your homeland. Has your activity level changed since you arrived in Australia? How do you think it can be improved?

**Did you know?**

Research has confirmed that it is never too late to become active. People of all ages can improve their health and wellbeing by starting some moderate-intensity activity. The benefits start as soon as you start, regardless of your age.

**Tips on increasing physical activity**

The significant benefits of physical activity may have got you thinking about increasing your activity. This goal is more likely to be successful if you:

- choose activities you enjoy; in that way, you will be more likely to keep doing them
- vary the type of activity you do, so that you don't become bored with the one thing, therefore increasing your chance of maintaining your activity throughout life
- set yourself small, realistic goals for your activity (e.g. Tomorrow I'll start with a 10 minute walk)
- set aside certain times of the day or one period that suits you to be active—you're more likely to be committed if you schedule it into your routine
- be active with friends or family. This way you can motivate and encourage one another and you have a social reason to persist. Another way of gaining social support is to join a club or a group.

**Safety first!**

Take a moment to discuss being active safely with your health professional, and whether you should see a doctor before beginning a physical activity program. Wear proper and comfortable shoes for walking. Before intense physical activity, remember to do warm up exercises. If you feel any pain or discomfort, stop immediately. Your safety is paramount. Finally, use a pedometer to count your steps daily and to help you feel motivated.

**Worksheet 3.4: Setting goals**

Worksheet 3.2 explained why being a healthy weight is important for a healthy heart. Worksheet 3.3 gave you feedback on areas of your food intake and physical activity that you could improve to help you achieve a healthy weight. Do you feel ready to choose one or more areas to work on?

If you feel ready to improve some areas of your eating pattern and activity levels ...

**Write up to 3 goals here.** They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART). For example 'I will walk for 30 minutes after dinner 5 times each week for the next month.'

List them in order of importance.

1. \_\_\_\_\_  
\_\_\_\_\_

2. \_\_\_\_\_  
\_\_\_\_\_



## ATTACHMENT B-4

### MODULE 4

---

#### Let's go shopping

This module discusses how to make healthier choices while shopping. There is discussion on how to read the ingredients list and the nutrition panel and make informed choices. There are examples for you to try. This module also tells you how to look out for hidden fats and sugars.

#### **Worksheet 4.1: Let's review**

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_

\_\_\_\_\_

2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

\_\_\_\_\_

Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 2.2 for a new challenge.

Making and sustaining lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

\_\_\_\_\_

\_\_\_\_\_

Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track

## **Worksheet 4.2: Healthy value**

We have already discussed how important healthy eating is for good health and in the prevention of disease, and how making healthy food choices can reduce your risk of further disease.

The first step to healthy eating is making healthy food choices. You probably already check whether the products you choose at the supermarket are value for money. Now it's time to learn how to check their nutritional value.

### **Food labels**

There are two parts of a food label that will give you nutrition information: the ingredients list and the nutrition information panel.

### **Ingredients list**

Ingredients in the product are listed in order from the biggest to the smallest amount of ingredient used, based on weight.

Here is the ingredients list on a packet of strawberry yoghurt:

Ingredients: Skim milk, sugar, strawberries (4%), light cream, thickener (1422), halal gelatine, vegetable gums (410, 412, 440), flavour, colour (120), food acid (330, 332), culture, halal rennet.

**Which ingredient is present in the largest amount?**

---

**Which ingredient is present in the smallest amount? (check answers below)**

---

**Answers:** The ingredient present in the largest amount is skim milk and the ingredient present in the smallest amount is halal rennet.

High saturated fat ingredients include animal fat, hydrogenated fat, tallow, butter, palm oil, shortening, ghee, lard, dripping, coconut oil, coconut cream, copha and full cream milk solids. If one of these ingredients is listed in the first three ingredients or if the ingredient list contains several of these ingredients, then the food product is likely to be high in saturated fat. Try to find a lower saturated fat alternative. Examples of foods high in saturated fats include commercial biscuits and pastries, potato crisps and corn crisps and many takeaway foods.

High sodium (salt) ingredients include MSG (monosodium glutamate), sea salt, rock salt, garlic salt, celery salt, vegetable salt, sodium bicarbonate, sodium nitrate, stock cubes, baking powder and baking soda. If one of these ingredients is listed in the first three ingredients or if the ingredient list contains several of these ingredients, then the food product is likely to be high in sodium. Try to find a lower sodium alternative. Foods that are generally high in salt are meat paste, fish paste, tomato paste, commercial sauces, powdered sauce mixes, salad dressings, pickles, olives, soup powders, canned soup, packet seasonings, bacon and ham.

High fibre ingredients include wholegrain or wholemeal grains and cereals such as oats, rice, barley, rye and wheat, fruit, vegetables, legumes and lentils. If one of these ingredients is listed in

the first three ingredients or if the ingredient list contains several of these ingredients, then the food product is likely to be high in fibre. So they are good choices.

Look for foods low in saturated fat and salt, and high in fibre. The nutrition information panel can help you.

## What's the nutrition information panel?

The nutrition information panel shows the energy, protein, total fat, saturated fat, carbohydrate, sugars and sodium content, and any other nutrient for which a claim is made on the label. Here is the nutrition information panel on a packet of strawberry yoghurt:

Nutrient content per 100g	
Energy	87 Cal or 365kJ
Protein	5.7g
Fat - Total	0.5g
- Saturated	0.3g
Carbohydrate – Total	14.6g
- Sugars	13.5g
Dietary fibre	0.5g
Sodium	24mg

**What is the saturated fat content of this product? (check answer below)**

---

The manufacturer must state the saturated fat content of the product so choose the one with the lowest saturated fat content.

**Is this product low in salt? (check answer below)**

---

Salt is called sodium on labels. If a product contains less than 120mg of sodium per 100g then it is low in salt. There are not many products that are low in salt, but '*No added salt*' or '*Salt reduced*' products are better choices than the regular version. Module 8 explains why a low salt diet is important.

**How much fibre is in this product? (check answer below)**

---

It is useful to check the fibre content of the bread and cereal-based products you buy. If a product contains more than 3g of dietary fibre per 100g it is high in fibre. Look for '*High fibre*', '*Wholemeal*', '*Wholegrain*', '*Wholewheat*', '*bran*' '*wheatbran*' '*wheatmeal*' and '*rolled oats*' to find products that are higher in fibre. (Fibre content is not relevant for foods that are naturally low in fibre, such as dairy products, meats, fish, oils and dressings and drinks.)

Module 7 explains how a high fibre diet can reduce the risk of heart disease.

**Answers:** Saturated fat = 0.3g. It contains 0.5g total fat, which is <3.0 g per 100g so it is a low fat product. Sodium = 24mg so it is a low salt product. (<120mg per 100g). Fibre = 0.5g, so it is not a high fibre product, but as dairy products are naturally low in fibre you wouldn't make a decision based on fibre content in this case.

## What do the claims mean?

Manufacturers often put claims on their labels which give the impression that the product is low in fat. This may not be the case.

'Low fat' generally means the food has 3% or less fat (if solid) or 1.5% or less fat (if liquid). These foods are good choices.

'Reduced fat' may not mean low in fat. Reduced fat cheddar cheese, for example, contains 25% fat so is still considered a high fat food. Check the nutrition information panel of these foods for total fat content.

'Light' or 'lite' can mean anything. It may mean less salt, fat or sugar. Sometimes it means more than one of these. In most cases 'lite' or 'light' doesn't mean low in kilojoules or low in fat. For example, 'lite' olive oil is light on flavour, not light in fat or kilojoules.

'Cholesterol free' doesn't mean the food is necessarily particularly healthy, low in fat or kilojoules, or low in saturated fat. All it means is that the food is free of dietary cholesterol. Plant foods don't contain cholesterol but some, such as coconut and palm oil are high in saturated fat and aren't good choices.

## Looking for a shortcut? The important Tick



The Tick Program is the National Heart Foundation's guide to help you make healthier food choices quickly and easily. Foods with the Tick are healthier choices among foods of their type. Tick foods are lower in saturated fat, sodium (salt) and where appropriate kilojoules. Some are also higher in fibre.

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### Did you know?

An Australian study has found that food manufacturers now use 235 fewer tonnes of salt each year, to help some of their products qualify for a Heart Foundation Tick.



### Worksheet 4.3: Check the label

It's important to remember that the Tick foods are healthier choices among foods of their type. For example, you need to compare a loaf of bread with the tick to a loaf without the Tick:

#### Bread with Tick Bread without Tick

Nutrient content per 100g	
Energy	1121kJ
Protein	9.5g
Fat - Total	4.0g
- Saturated	0.6g
Carbohydrate – Total	46.6g
- Sugars	2.0g
Dietary fibre	2.4g
Sodium	520mg

Nutrient content per 100g	
Energy	1031kJ
Protein	8.6g
Fat - Total	2.0g
- Saturated	0.4g
Carbohydrate – Total	44.7g
- Sugars	2.9g
Dietary fibre	6.0g
Sodium	450mg

#### Bread without Tick

**Compare the saturated fat content of these breads.** They are very similar. Like most breads, both products contain very little saturated fat.

**Check the fibre content of each bread.** The bread with the Tick has more than twice as much fibre. Bread must contain at least 3g of fibre per 100g to qualify for the Tick.

**Compare the sodium (salt) level in each bread.** Bread is generally a high salt food, and both these breads are high in salt. However the bread with the Tick contains the least salt. Bread must contain less than 450mg per 100g to qualify for the Tick.

So for these breads, it is fibre and salt content that decides which is the healthier choice.

## Now let's compare milk

### Milk with the Tick

Nutrient content per 100g	
Energy	193kJ
Protein	3.3g
Fat - Total	1.5g
- Saturated	0.9g
Carbohydrate – Total	5.0g
- Sugars	5.0g
Sodium	50mg

### Milk without the Tick

**Compare the saturated fat content of these milks.** The milk without the Tick has more than twice the saturated fat content of the milk with the Tick.

**Check the fibre content of each milk.** Fibre is not listed on most dairy products, as they are naturally very low in fibre.

**Compare the sodium (salt) level in each milk.** Like most unprocessed food, milk is low in salt. Both these milks are low in salt.

So in this case, the saturated fat content decides which is the best choice.

**Now let's compare margarine with a butter/margarine 'easy spread' blend:**

### Margarine with the Tick

Nutrient content per 100g	
Energy	2620kJ
Protein	<1g
Fat - Total	70g
- Saturated	18g
Carbohydrate – Total	<1g
- Sugars	<1g
Sodium	340mg

## Butter/margarine dairy blend without the Tick labelled 'SALT REDUCED'

Nutrient content per 100g	
Energy	2630kJ
Protein	0.8g
Fat - Total	70.0g
- Saturated	29.8g
Carbohydrate – Total	1.5g
- Sugars	1.5g
Sodium	320mg

**Compare the saturated fat content of these spreads.** Both products contain quite a lot of total and saturated fat. However the margarine with the Tick has about half the saturated fat content than the other product.

**Check the fibre content of each margarine.** Fibre is not listed on most butters and margarines as they are very low in fibre.

**Compare the sodium (salt) level in each milk.** Even though the butter/margarine dairy blend is labelled 'salt reduced' it still contains a lot of salt. The margarine with the Tick is also high in salt. It contains slightly more salt than the dairy blend.

For these spreads, the margarine with Tick is the healthier choice. Even though it is slightly higher in salt, it contains much less saturated fat than the dairy blend.

Have you compared the saturated fat, fibre and salt content of the products you buy each week to make sure you are making healthy food choices

### ***When shopping at a supermarket***

Think about buying

Breads- Pita bread, raisin bread, whole meal bread, white hi-fibre, multi grain or fruit bread.

Dried biscuits- as a substitute for bread, particularly for children's snacks and lunches. Eg: premium 97% fat free biscuits

Cottage and Ricotta cheese- as a substitute for spreads such as butter or margarine

Margarine and Butter- Choose a poly unsaturated margarine

Fresh vegetables- packed with essential vitamins and minerals and plenty of fibre, they fall into the category 'Eat Most'. If buying tinned or packaged varieties, buy frozen vegetables with nothing added or varieties packed in water or juice. Select tinned varieties with 'no added salt' or the brand with the lowest salt content (using the nutrition panel).

Fruit- Most varieties. If buying packaged varieties, look for words 'in natural juice' or 'no added sugar' on the label.

Baked beans- are high in fibre and low in fat making a nutritious, inexpensive and convenient meal or snack. Select tinned varieties with 'no added salt' or the brand with the lowest salt content (using the nutrition panel).

Legumes- Convenient protein and fibre source. Select brands with 'no added salt' or the brand with the lowest salt content (using the nutrition panel).

Fish- as a substitute for meat. All varieties packed in brine or spring water. Limit varieties packed in oil if weight control is an issue.

Milk- reduced fat and skim milk varieties are healthy choices. Soy milk is an alternative to cow's milk, particularly those who are lactose intolerant. Full fat varieties are the best choice for children, especially those under 5.

Yoghurt- as a good calcium source and as a substitute for sour cream. Buy low fat varieties sweetened only with natural fruit.

Meats- choose low fat meats, trimmed meats, skinless chicken, beef strips, lean beef mince, trim lamb eye of loin. Cheaper cuts of meat may be fattier. Try and remove the visible fat before cooking or cook the meat the day before and let it stand. Remove the fat from the top before finishing the dish or serving.

**5 WATCH OUT for FOODS with Hidden SUGARS**  Indicates ADDED SUGAR in Level Teaspoons

CHOCOLATE BAR		
ICED DOUGHNUT (Ring)		
ICE CREAM		
CHOCOLATE COATED BISCUIT		
CAKE		
FRESH FRUIT		
PLAIN YOGHURT		
TINNED FRUIT		
DRIED BISCUITS		
SANDWICH WITH PEANUT BUTTER		

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## Worksheet 4.4: Setting goals

Worksheet 4.2 explained that the first step in healthy eating is buying healthy food. Worksheet 4.3 helped you to learn to make healthy choices by reading food labels.

Do you feel ready to improve some areas of your food choices? If so:

**Write up to 3 goals here.** They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART). For example 'I will buy low fat dairy products next time I am shopping.'

List them in order of importance.

1. \_\_\_\_\_

\_\_\_\_\_

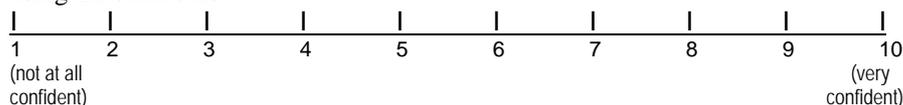
2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

\_\_\_\_\_

**How confident are you that you can achieve these goals?** Rate your confidence in achieving each goal using the scale below.



If you rated your confidence as 6 or less then you should probably readjust your goal. It is better to make small changes that will be successful and build on these, instead of setting goals that are destined to fail. If your confidence rated 9 or 10 then the goal may be too easy. Readjust your goal to make it more challenging.

**How will progress toward each goal be monitored?** (e.g. 'I will check my fridge at the end of the week to see if all dairy products are low fat')

\_\_\_\_\_

\_\_\_\_\_

**What could you do to increase your success in achieving your goal?** ( I will ask my husband to check and stop me buying full fat dairy products. I will use the Heart Foundation's Tick to help me when shopping.)

\_\_\_\_\_

\_\_\_\_\_

## Attachment B-5

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### MODULE 5

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#### **Cooking up a storm**

say you buy a 4 litre tin of oil from the supermarket for your family of four every month? That is 1 litre of oil per person per month. That amounts to 8100 kilocalories, 33300 kilojoules or 5 tubs of margarine or ghee.

#### **Worksheet 5.1: Let's review**

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_

\_\_\_\_\_

2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

\_\_\_\_\_

Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 5.2 for a new challenge.

Making lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track.

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

## Worksheet 5.2: Cooking class

Many recipes will only require a few simple changes to reduce the saturated fat content. The two steps to changing a recipe are:

1. Try healthier cooking methods
2. Change ingredients by Reducing, Removing or Replacing them (the tripple R).

**How do you usually cook your meat and fish? Use this checklist to see if there is a healthier way to do it.**

### Cooking methods

Cooking method	Healthier alternative	Could be improved
Deep frying	Roast in the oven on a lined tray or grill tray. Food can be lightly steamed or microwaved first, and brushed with oil such as canola, sunflower, soybean or olive oils for crispness. Crumbed fish, chicken and oven fries can be cooked in the oven.	
Shallow frying	Stir fry using reduced salt stock, or oil such as canola, sunflower, soybean or olive oil. Try using a non-stick frying pan.	
Roasting meat	Choose lean cuts of meat, or trim all visible fat and then place meat on a rack in a baking dish with 1–2cm of water. For extra flavour add herbs and wine to water.	
Casserole/stew	Trim fat off meat before cooking. Add legumes or lentils to add bulk and flavour. After cooking, chill food so any fat solidifies on the surface. Skim fat off the surface before reheating and serving.	

Adapted from: *Healthy eating for the heart: A guide to lowering your blood cholesterol*. (2002). NHFA.

**Now think about the ingredients you cook with. Use this checklist to get ideas for healthier alternatives.**

### **Ingredient modifications**

<b>Ingredient</b>	<b>Healthier alternatives</b>	<b>Could be improved</b>
Milk, yoghurt, cream	Use reduced fat varieties. Use ricotta cheese whipped with a little icing sugar, fruit and reduced fat milk as a substitute for cream.	
Sour cream	Use reduced fat natural yoghurt. Use reduced fat evaporated milk and lemon juice (e.g. in soups).	
Cheese	Use smaller amounts of reduced fat varieties. Use a smaller amount of a strongly flavoured cheese (e.g. grated parmesan cheese) instead of grated cheddar. Mix with breadcrumbs, bran or wheatgerm for a crispy topping on baked dishes.	
Butter or Ghee	Use margarine spreads or oils such as canola, sunflower, soybean, olive and peanut oils instead of butter, lard, cophera or cooking fats.	
Oil	Use oils such as canola, sunflower, soybean, olive and peanut oils. Use concentrated <i>reduced salt</i> stocks and juice to sauté.	
Mayonnaise/dressing	Make your own using ingredients such as reduced fat yoghurt, buttermilk, tomato paste, lemon juice, ricotta cheese, mustard and fruit pulp. Use salad dressings and mayonnaise made from oils such as canola, sunflower, soybean and olive oils.	
Meat/poultry	Trim fat off meat before cooking. Remove skin from poultry. Keep portions small and provide extra serves of vegetables and legumes (e.g. kidney beans, chick peas).	
Cakes/biscuits	The minimum amount of fat required for biscuits is about 2 tablespoons per cup of flour. This will retain crispness. Use margarine spreads or oils such as canola, sunflower or soybean oil instead of butter.	
Pastries	Use filo pastry instead of shortcrust. Bake before using as pastry base. Choose a pastry made with oil such as canola, sunflower or olive oil.	
Coconut cream/coconut milk	Use reduced fat yoghurt or reduced fat evaporated milk with a little desiccated coconut or coconut essence.	

## Worksheet 5.3: Modifying recipes

### Look at this recipe for Pakistani Sindh Biryani

<b>Ingredients:</b>	
Mutton/chicken 1 kg	Ginger 2 teaspoons
Basmati rice 1 kg	10 cloves
Potato 1/2 kg	10 pieces of black pepper
Tomato 1/2 kg	Cumin 1 teaspoon
Yogurt 250 gm	Cinnamon 1 stick
Red chili powder 1 teaspoon	2 bay leaves
Cardamoms (optional)	Oil or ghee
Salt to taste	Green chillies 6
Onions 2 medium	Coriander leaves 2
Garlic paste 2 tsp	tablespoons
	Mint leaves 2 tablespoons
	Yellow food color two

#### Method:

1. Slice the onion and fry it in oil until its light brown.
2. Add garlic, ginger, salt, chili powder, cloves, cardamoms, black peppers, bay leaves, cinnamon, and yogurt.
3. Fry this until the water is dry and then add the meat and fry it again.
4. Add some water and cook on low heat until the meat is cooked and the water has evaporated.
5. Peel the potatoes and cut them into large chunks. Boil them until they're half cooked.
6. Soak the rice in water for half an hour. Boil and drain the water off when half done.
7. Cut the tomatoes. Add green chillies (whole), mint and coriander leaves. Add this to the meat and throw in the potatoes as well.
8. Layer this with the rice in a pot.
9. Sprinkle a solution of food color and sugar.
10. Keep the lid closed and cook this on low heat until the rice is done. Serve.

#### Now how can you make this recipe healthier?

Use oil instead of ghee. After frying the onions and meat, place them on an absorbent paper to remove excess oil. Meat can also be cooked prior and made to stand and the fat removed from the top later.

What do you think of these changes?

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Think about some of your favourite recipes. What changes could you make so they are healthier?

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Do you have some recipe books or magazines with healthy recipes in them?

The Heart Foundation has some great recipe books to help you enjoy healthy eating. These include:

*Cooking for Few*

*Deliciously Healthy*

*The Heart Foundation Cookbook*

Call *Heartline*, the Heart Foundation's national telephone information service, on 1300 36 27 87 to find out how to obtain these

## Worksheet 5.4: Setting goals

Worksheet 5.2 showed you how to cook and prepare your food in a healthy way. This is an important skill to help you make positive changes to your usual eating pattern. Do you feel ready to choose one or more areas of your food preparation to work on?

If you feel ready to make some changes:

**Write up to 3 goals here.** They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART), for example 'I will reduce the amount of oil that I use in my cooking'.

List them in order of importance.

1. \_\_\_\_\_

\_\_\_\_\_

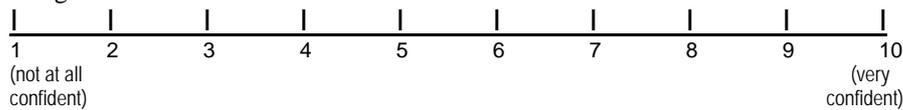
2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

\_\_\_\_\_

**How confident are you that you can achieve these goals?** Rate your confidence in achieving each goal using the scale below.



If you rated your confidence as 6 or less then you should probably readjust your goal. It is better to make small changes that will be successful and build on these, instead of setting goals that are destined to fail. If you rated your confidence at 9 or 10 then the goal may be too easy. Readjust your goal to make it more challenging.

**How will your progress toward each goal be monitored?** (e.g. 'I will note on my recipes each time I used lesser amounts of oil).

\_\_\_\_\_

\_\_\_\_\_

**What could you do to increase your success in achieving your goal?** (e.g. 'I will buy smaller cans of oil so I have a better idea as to how much oil I use in a month')

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## Attachment B-6

### MODULE 6

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#### Eating away from home

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The previous module discusses how you can choose healthier alternatives while cooking at home. There is a tendency to splurge on food when dining out as it is not an everyday event. But it is very important to make the same healthier choices when eating away from home. If you do splurge, try and exercise for ten more minutes or walk for half an hour more the next day. Eating out and eating with friends can be an enjoyable part of a healthy lifestyle. If you are trying to make healthy food choices, you may need to read the menu a little more carefully.

#### **Worksheet 6.1: Let's review**

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_

\_\_\_\_\_

2. \_\_\_\_\_

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3. \_\_\_\_\_

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Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 2 for a new challenge.

Making lifestyle changes is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

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Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track.

\_\_\_\_\_



## Worksheet 6.2: Eating away from home

Eating out and eating with friends can be an enjoyable part of a healthy lifestyle. If you are trying to make healthy food choices, you may need to read the menu a little more carefully.

Think about the last time you ate away from home.

**Write down what you had to eat and drink:**

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**Now use this checklist to decide whether you could have made a healthier choice:**

Did you:	Could be improved
Choose sauces that are not cream or butter-based or ask that these be not added?	
Try to choose lean meats with lots of salads or vegetables?	
Try to remove any saturated fat from foods served to you? For example, trying to avoid the oil in the curry or passing up on the fried chicken?	
Ask for plain, fresh bread instead of garlic and herb breads which can be high in saturated fat?	
Choose fresh or grilled seafood rather than crumbed and fried?	
Ask that no butter is added to vegetables?	
Ask for salad dressings and mayonnaise made from oils such as canola, sunflower, soybean and olive oils? Ask for these on the side so you can add them yourself.	
Choose a pasta dish with a vegetable or tomato-based sauce instead of a creamy one?	
Choose carefully from smorgasbords? Beware: Smorgasbords encourage you to eat more than you need.	
Go easy on rich desserts or go halves with someone? Choose fruit-based desserts such as fruit mousse, fresh, baked or canned fruit, or reduced fat custards, sorbets or gelati.	
Choose water as your preferred drink?	
Resist the temptation to have an extra serve of food?	
Order fruit juice? Fruit juices have the same kilojoules as regular beer. If you are trying to reach a healthy weight, drinking fruit juice may not help you.	

If you read a menu carefully you can usually spot some healthy options.

**Have a look at the menu below and circle the healthiest choice in each section of the menu:**

(Answers below)

Entrees, Vegetable samosa, Tandoori Chicken ,Fried chicken drumsticks

Main courses, Garlic naan, Lamb Vindaloo, Malai kofta, Fried rice

### **Side dishes**

Tomato, olive and feta cheese salad served with olive oil,Garden salad with lemon juice dressing,Desserts, Fresh fruit salad with cream or yoghurt ,Pistacchio kulfi,Drinks

Milkshake,Plain mineral water,Fruit juice

### **Answers**

Tandoori chicken would be the healthiest option as it is cooked in a traditional oven (Tandoor) and uses minimal oil. Samosas and drumsticks are deep fried snacks rich in saturated fats. Garlic naan is coated with a lot of butter during preparation. Ask for plain naan or roti instead. Lamb Vindaloo is the better choice than Malai Kofta even though Malai Kofta is a vegetarian dish. This is because Malai Kofta is made from deep fried cottage cheese and vegetables cooked in curry sauce and finished with cream. Steamed or boiled rice is a healthier option than fried rice. The garden salad is the healthiest side dish. The tomato, olive and feta cheese salad is okay, but contains more salt from the cheese and olives and also saturated fat in the cheese. The fruit salad is the best choice of dessert, and a reduced fat yoghurt would be the healthiest choice to have it with, as cream is very high in saturated fat. Plain mineral water is the best choice as it is low in kilojoule

## Worksheet 6.3: Takeaway foods

When choosing takeaway food, the important things to remember are:

- Look for takeaway foods that contain lean meats, lots of vegetables and cereals such as steamed rice, pasta and noodles
- try to limit takeaway foods such as pastries, pies, pizza, hamburgers and creamy pasta dishes to once a week.

### What are your favourite take-away meals?

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Look through the checklist below to see if you can make healthier choices.

### Takeaway alternatives

Takeaway foods	Healthier choices	Could be improved
BBQ chicken	Remove the skin and fat and choose the breast meat. Serve with salad or vegetables rather than hot chips and try to limit gravy and stuffing.	
Chinese/Indian/Thai	Choose steamed rice, mixed vegetable dishes, lean meats (beef, lamb, pork or chicken), fish and stir fries. Try to limit dishes based on fried noodles, battered or crumbed deep fried meats or seafood, coconut cream/milk and ghee.	
Hamburgers including Halal	Ask for lean grilled meat and lots of salad. Try to limit mayonnaise or choose one based on polyunsaturated or monounsaturated oil.	
Italian	Choose pasta with vegetable or tomato based sauces. Choose pizza with reduced saturated fat toppings such as lean meats, small amounts of reduced fat cheese and lots of vegetables.	
Lebanese	Choose donner or shish kebabs in pita bread with tabouli or Lebanese bread with salad.	
Salad bars	Try to choose salads with dressings or mayonnaise made from oils such as canola, sunflower, soybean and olive oils, or choose salads with dressing on the side.	
Sandwiches	Try to choose reduce saturated fat fillings such as lean meats, reduced fat cheese, salmon, tuna and plenty of salad vegetables. Try to limit high saturated fat processed meats such as salami. Choose margarine instead of butter or dairy blends. Try other spreads	

	such as avocado, jam, chutney, hommus or pickles.	
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**Did you know?**

In 2002 the average Australian ate out 83 times! The most popular fast foods were sandwiches, hot chips, hamburgers, cakes/pastries and pizza (BIS Shrapnel survey).

It isn't always easy to make healthy food choices when we are out and about. Here is an eating out scenario to think about.

You are visiting friends who have invited you for a dawat. The hostess tells you she has just spent the entire day in the kitchen making biriyani, roti, fried chicken , kababs, roghan josh, salad and kulfi just for you and that you have to do justice to the food. What will you do?

Stuck for ideas? Here are some approaches that might work:

- just have small servings of the food.
- politely decline the fried chicken but have a kabab instead.
- have less of the biriyani and more salad.
- avoid the temptation to over eat. Stop when you feel full how ever much any one forces you.

Try explaining to your friends that you really need to be careful about what you eat for the good of your health.

## Worksheet 6.4: Setting goals

Worksheet 6.2 gave you tips for making healthier food choices when you eat out. Worksheet 6.3 listed ways to make takeaway foods a healthier option. Do you feel ready to work on making healthier food choices when you eat out? If you are unsure, try doing worksheet 6.5.

If you feel ready to make some changes:

**Write up to 3 goals here.** They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART), for example 'I will eat only healthier foods when I eat out for the next month'.

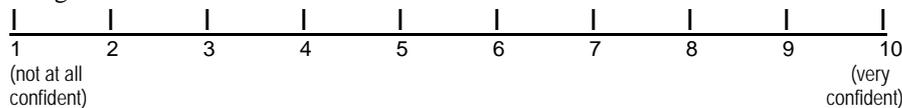
List them in order of importance.

1. \_\_\_\_\_

2. \_\_\_\_\_

3. \_\_\_\_\_

**How confident are you that you can achieve these goals?** Rate your confidence in achieving each goal using the scale below.



If you rated your confidence as 6 or less then you should probably readjust your goal. It is better to make small changes that will be successful and build on these, instead of setting goals that are destined to fail. If you rated your confidence at 9 or 10 then the goal may be too easy. Readjust your goal to make it more challenging.

**How will your progress toward each goal be monitored?** (e.g. 'I will make a note in my work diary each day I buy sandwiches with a healthier filling').

\_\_\_\_\_

\_\_\_\_\_

**What could you do to increase your success in achieving your goal?** (e.g. 'I will keep the checklist from worksheet 2 in the office to remind myself')

\_\_\_\_\_

## Attachment B-7

### MODULE 7

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#### Fill up on fibre

Dietary fibre is the part of food not digested by our stomach or intestines. Fibre is only found in plant foods.

There are two types, insoluble and soluble. Foods high in soluble fibre include fruits, vegetables, legumes (i.e. dried peas, dried beans and lentils), oats, oat bran, barley bran and rice bran. The soluble fibre in these foods can help lower blood cholesterol (but not as effectively as reducing the amount of saturated fat you eat).

Think about what foods you ate yesterday. Write them down as 'meals' and 'snacks'. Write another list of the same foods as one containing fibre and another containing no fibre. At the end of this module, think about the fibre containing foods in your diet yesterday. Do you think it is satisfactory?

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#### Worksheet 7.1: Let's review

Are you ready to check your progress toward the goals you have set to help yourself?

If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_

\_\_\_\_\_

2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

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Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 7.2 for a new challenge.

Making lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

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Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track.

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## Worksheet 7.2: Fabulous fibre

Dietary fibre is the part of food not digested by our stomach or intestines. Fibre is only found in plant foods.

There are two types, insoluble and soluble. Foods high in soluble fibre include fruits, vegetables, legumes (i.e. dried peas, dried beans and lentils), oats, oat bran, barley bran and rice bran. The soluble fibre in these foods can help lower blood cholesterol (but not as effectively as reducing the amount of saturated fat you eat).

Insoluble fibre helps to keep bowels regular but has little effect on cholesterol. It's found mainly in wholemeal bread, breakfast cereals (especially those based on wholegrains) and unprocessed bran.

There are 3 easy ways to increase the amount of fibre you eat each day. They are listed below.

### Could you use any of these ideas to improve your fibre intake?

Do you:	Could be improved:
Make vegetables, and grain based foods such as breakfast cereals, bread, pasta, noodles and rice the major part of each meal.	
Incorporate dried peas (e.g. split peas), dried beans (e.g. haricot beans, kidney beans), canned beans (e.g. baked beans, three bean mix) or lentils into two meals a week.	
Snack on plain, unsalted nuts and fresh fruit.	

### How much?

The Heart Foundation suggests a target of 30g fibre daily for Australian adults. Most of us eat about 20g fibre each day. Following the guidelines in the list above will help you meet that target. You can also check the labels of foods you buy. Look for fibre in the nutrition information panel and compare the amount of fibre per 100g in similar food products.

### Breakfast cereal

Nutrient content per 100g	
Energy	865kJ
Protein	5.7g
Fat—Total	5.5g
– Saturated	2.3g
Carbohydrate—Total	14.6g
– Sugars	13.5g
Dietary fibre	4.5g
Sodium	24mg

## **To increase the amount of fibre in your diet**

Leave the peel on fruit and vegetables where ever possible.

Choose breads with wholemeal or wholegrain listed first on the ingredients list.

Add grains (rice or pasta) and legumes (chickpeas, beans, lentils) to meals such as stews.

Eat more fruits and vegetables as snacks or include them in meals. (2 serves of fruit and 5 different vegetables everyday)

Choose a breakfast cereal that is high in fibre (Check the label).

Try wholemeal versions of products such as pasta, rice and dry biscuits. Check the labels on packets of instant noodles as some varieties use a lot of palm oil which is a saturated fat.

Use quick cooking methods for vegetables (such as stir frying and lightly steaming) as prolonged cooking may destroy some of the fibre in food.

Try baked beans as a quick meal or snack.

### **Did you know?**

Fibre helps to fill you up. A small glass of apple juice contains the juice of two apples, but is much less filling than two apples because it lacks the fibre in the apples. Try to eat the fruit instead of drinking the juice to increase your fibre intake

### **Worksheet 7.3: Fibre—including it everyday**

Do you include dried beans and peas, lentils or canned beans/peas in at least 2 meals a week? If you are trying to reach this goal, here are some ideas to help you:

- add lentils or dried peas or beans to soups
- add canned beans or peas to salads, e.g. 4 bean mix, chickpeas
- mix lentils or canned beans into meat dishes,
- use them as an alternative to meat e.g. use chickpeas instead of meat in a curry
- use them to make dips, e.g. chickpeas are a main ingredient in hummus
- they can make a quick, light meal, e.g. baked beans on toast.

What else could you cook with dried beans and peas, lentils or canned beans/peas?

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What suggestions can you make to increase the fibre in this menu? (Answers below)

breakfast, puffed rice cereal low fat milk 2 slices of white toast with margarine spread and jam

lunch 2 slices of white bread with margarine, chicken and cheese

Apple juice, Dinner Grilled steak, Boiled potato, Boiled broccoli, Boiled pumpkin Chocolate mousse

Answers

Breakfast: Choose a wholegrain breakfast cereal, e.g. porridge, use wholemeal or multigrain bread. Include some fruit either on your cereal or as an extra. For lunch, use wholegrain bread. Include salad in the sandwich. Have an apple instead of apple juice. For dinner, keep the skin on the potato, add some more vegetables and serve with couscous or wild rice. Have a fruit-based dessert.

## Worksheet 7.4: Setting goals

Worksheet 7.2 gave you tips for increasing your fibre intake. Worksheet 7.3 gave you ideas on how to do this. Do you feel ready to work on increasing the amount of fibre you eat each day? If you are unsure, try doing worksheet 7.5.

If you feel ready to improve your fibre intake

**Write up to 3 goals here.** They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART). For example: I will eat a fruit whenever I feel hungry between meals for the next month.

List them in order of importance.

1. \_\_\_\_\_

\_\_\_\_\_

2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

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**How confident are you that you can achieve these goals?** Rate your confidence in achieving each goal using the scale below.



If you rated your confidence as 6 or less then you should probably readjust your goal. It is better to make small changes that will be successful and build on these, instead of setting goals that are destined to fail. If you rated your confidence at 9 or 10 then the goal may be too easy. Readjust your goal to make it more challenging.

**How will your progress toward each goal be monitored?**

\_\_\_\_\_

\_\_\_\_\_

**What could you do to increase your success in achieving your goal?** (e.g. 'I will make sure I have fruits in the pantry)

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## Attachment B-8

### MODULE 8

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#### Flavour without salt

. This module reflects on ways in which salt usage can be reduced in the diet. Salt is an important addition to our foods. Without salt, our food does not taste as good. But too much salt has been proven to be linked with high blood pressure which can then lead to other diseases/ conditions eating too much salt can lead to high blood pressure. High blood pressure increases your risk of heart disease and stroke. If you already have high blood pressure, eating too much salt may make it worse. Enjoying a healthy eating pattern that is low in salt is one way to help control your blood pressure, or to help to avoid high blood pressure altogether. Enjoying regular physical activity and maintaining a healthy weight will also help to control blood pressure. Worksheet 8.1: Let's review

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_

2. \_\_\_\_\_

3. \_\_\_\_\_

Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 8.2 for a new challenge.

Making lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

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Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track.

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## **Worksheet 8.2: Salt and blood pressure**

### **What's wrong with eating too much salt?**

Eating too much salt can lead to high blood pressure. High blood pressure increases your risk of heart disease and stroke. If you already have high blood pressure, eating too much salt may make it worse. Enjoying a healthy eating pattern that is low in salt is one way to help control your blood pressure, or to help to avoid high blood pressure altogether. Enjoying regular physical activity and maintaining a healthy weight will also help to control blood pressure.

### **Which foods contain salt?**

Salt is found in almost every food we eat, but the amount present in different foods varies a great deal. Most of the salt we eat (about 75%) comes from processed foods. High salt processed foods include soy sauce, processed meats, canned soups, canned anchovies and stock cubes. About 15% of the salt we eat comes from the salt we add at the table or in cooking.

Most fresh foods (vegetables, fruits, nuts, unprocessed meat) are naturally low in salt and contribute little to the salt we eat.

Use the checklist below to decide how you could reduce your salt intake.

<b>Do you:</b>	<b>Could be improved:</b>
Try to limit processed foods such as processed meats, commercial sauces, soups, packet seasoning and stock cubes (other than those labelled 'no added salt', 'low salt' or 'salt reduced')?	
Try to limit potato chips and crisps, high salt takeaway foods and salted nuts?	
Enjoy plenty of fresh vegetables and fruit?	
Choose foods normally processed without salt and foods labelled 'no added salt', 'low salt', or 'salt reduced'?	
Choose low salt breads and cereals?	
Choose packaged foods with a sodium content of no more than 400 milligrams per 100grams	
Cook without adding salt?	

Salt is usually called 'sodium' on food labels. Look for sodium in the nutrition information panel and compare the amount of sodium per 100g in similar food products.



## Worksheet 8.3: Salt—how to cut back

As well as limiting the high salt foods listed on worksheet 8.2, you can also reduce your salt intake by not adding salt at the table

<b>Do you:</b>	<b>Could be improved:</b>
Flavour food using freshly ground pepper, fresh or dried herbs, vinegar or balsamic vinegar, lemon juice, fresh mustard (made from powdered mustard), and fresh garlic or garlic powder?	
Buy commercial no added salt sauces?	
Try to make your own sauces, pickles and chutneys without adding salt?	

Keep in mind that vegetable salt, rock salt, sea salt, garlic salt and celery salt are no different from ordinary salt and should be avoided.

Potassium chloride is another product often used as a salt alternative. You should check with your doctor before using it.

**What are some other ways of adding flavour to food instead of using salt, stock cubes and stock powder, gravy powder and soy sauce?**  
(Answers below)

What else can I do to help lower my blood pressure?

To help lower blood pressure there are other lifestyle changes you can make apart from limiting salt intake.

<b>Do you:</b>	<b>Could be improved:</b>
Need to achieve and maintain a healthy body weight? Reducing excess weight can lower blood pressure in most people.	
Keep physically active? The Heart Foundation recommends at least 30 minutes of moderate intensity physical activity, such as brisk walking, on all or most days of the week. The amount of activity can be accumulated in bouts of 10 minutes or more if this is more convenient.	

### **Did you know?**

High blood pressure affects almost a third of people who are over 50 years of age. Almost one half of Australians have high blood pressure before they reach the age of 70 .

## Worksheet 8.4: Setting goals

Worksheets 8.2 and 8.3 gave you tips for decreasing your salt intake. Worksheet 8.3 also listed other lifestyle changes you can make to lower your blood pressure. Do you feel ready to work on making some of these lifestyle changes? If you are unsure, try doing worksheet 8.5.

If you feel ready to make some changes:

**Write up to 3 goals here.** They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART). For example 'I will walk for 30 minutes after dinner 5 times each week for the next month.'

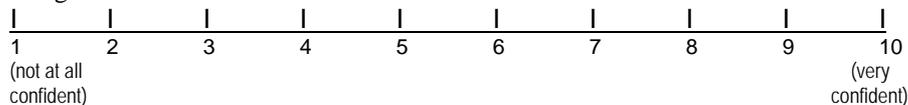
List them in order of importance.

1. \_\_\_\_\_

2. \_\_\_\_\_

3. \_\_\_\_\_

**How confident are you that you can achieve these goals?** Rate your confidence in achieving each goal using the scale below.



If you rated your confidence as 6 or less then you should probably readjust your goal. It is better to make small changes that will be successful and build on these, instead of setting goals that are destined to fail. If you rated your confidence at 9 or 10 then the goal may be too easy. Readjust your goal to make it more challenging.

**How will your progress toward each goal be monitored?**

\_\_\_\_\_

\_\_\_\_\_

**What could you do to increase your success in achieving your goal?** (e.g. 'I will put reduced salt canned foods on my shopping list')

\_\_\_\_\_

## Answers

Use vinegar, fruit juice, herbs, spices, garlic, onion, chives, spring onion, horseradish, wine, sherry, low salt stock cubes/powders.

## **Water**

In this module, we discuss water and its importance to the human body.

Our body gets water from many sources, beverages, certain foods etc. But there is a recommended daily requirement of water which is not often met. People usually drink water only when they are thirsty or during meals to help in the swallowing process.

Thirst is a conscious desire to drink and is actually a body defence mechanism. If you feel thirsty, chances are that the body is dehydrated and hence the desire to drink more water. It is wise to continue drinking beyond the point when the thirst feels to have been quenched.

The module also discusses ways in which you can increase your water intake.

## **Worksheet 9.1: Let's review**

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_

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2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

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Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 9.2 for a new challenge.

Making lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

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Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track.

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## **Worksheet 9.2: Water**

Water, after oxygen, is the second most important substance for human health. It is the basis of all biological processes in the human body. Our body is comprised of 70% water. Water is needed to regulate body temperature and plays a role in digestion, absorption and transport of nutrients to all our organs. Water also transports oxygen to our cells, removes waste products, and protects our joints and organs.

### **Why should you drink water?**

Our body loses a lot of water everyday via sweat and urine. The more physically active you are, the more water you lose. This lost water has to be replaced. If this is not done, the body gets dehydrated and may result in serious health problems.

Water can also help in weight loss. Drinking water before meals can fill you up and this will result in intake of lesser amounts of food.

Water is a good medium to flush out all toxins and keep you feeling refreshed and rejuvenated.

Drinking water can also help the skin retain its natural glow.

As it does not contain any sugars, it is a better choice to protect teeth from decay.

Drinking water can be an important source of a number of nutrients, especially calcium, magnesium, iodine and fluorine.

### **What is the recommended daily requirement of water?**

We get water from the foods we eat. For example: cucumbers, watermelon, oranges, milk are all nearly 90% water. Nearly all solid foods contain some water. But apart from this, it is recommended that you drink 1.5-2 litres a day or 8 glasses of water spread across the day.

### **Other sources of Water**

#### **Cordials and Fizzy drinks**

Most cordials and fizzy drinks contain sugar and can cause tooth decay.

As they contain sugar, they can also increase your kilojoules and result in weight gain.

When drinking fizzy drinks, opt for diet versions of the same.

#### **Fruit juices**

Fruit juices also like cordials and fizzy drinks contain a lot of sugar. If weight control is a problem, drinking fruit juices is not going to help.

Fruit juices contain no fibre and the sugars in some fruit juices may be hard to digest resulting in stomach problems.

When buying fruit juices, choose the ones which say 100% juice or no added sugar.

# 3 WATCH OUT for DRINKS with Hidden SUGARS



## LIMIT

CORDIAL (1 glass)



SOFT-DRINK (1 can)



FRUIT JUICE DRINK  
(1 mini carton with straw)

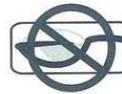


## BETTER

100% FRUIT JUICE



GLASS OF MILK



## BEST

WATER



© VFST

## Worksheet 9.3: Drinking More Water

There are many ways you can increase your water intake. Look at the checklist below for ideas:

Do you:	Could be improved:
Drink water before and after meals?	
Drink water in between meals?	
Drink herbal teas? (this will increase your water intake)	
Drink more tea/ coffee than water in a day?	
Ask for a jug of water when dining out instead of soft drinks?	
Take water breaks instead of coffee breaks?	
Eat more fruits that contain water?	

**Worksheet 9.4:**

### Setting goals

**Write up to 3 goals here.** They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART). For example 'I will drink a glass of water before and after each meal for the next month'.

List them in order of importance.

1. \_\_\_\_\_

\_\_\_\_\_

2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

\_\_\_\_\_

**How confident are you that you can achieve these goals?** Rate your confidence in achieving each goal using the scale below.

1	2	3	4	5	6	7	8	9	10		
(not at all confident)										(very confident)	

If you rated your confidence as 6 or less then you should probably readjust your goal. It is better to make small changes that will be successful and build on these, instead of setting goals that are destined to fail. If you rated your confidence at 9 or 10 then the goal may be too easy. Readjust your goal to make it more challenging.

**How will your progress toward each goal be monitored?**

\_\_\_\_\_

\_\_\_\_\_

**What could you do to increase your success in achieving your goal?** (e.g. 'I will carry a bottle of water with me where ever I go'.)

## **New food and healthy habits.**

### **Think about:**

How your eating habits have changed since you arrived in Australia? What foods from your homeland do you miss? What foods from your homeland do you find hard to buy in Australia? What are some new foods that you have started to eat since coming to Australia? What impact do you think these foods have had on your health? What are some Australian foods that you have tried and do not like? Why? What foods do your children like to eat here in Australia? Why are these popular with children?

## **Worksheet 10.1: Let's review**

Are you ready to check your progress toward the goals you have set to help yourself? If so, take this opportunity to discuss how you are going with your health professional.

Write your current goals here:

1. \_\_\_\_\_  
\_\_\_\_\_

2. \_\_\_\_\_  
\_\_\_\_\_

3. \_\_\_\_\_  
\_\_\_\_\_

Review any written records of progress towards your goals. If you are on track to achieve your goals, congratulations! Try the activities in worksheet 9.2 for a new challenge.

Making lifestyle change is difficult. If you are not happy with your progress, list the difficulties you encountered in meeting your goals (e.g. did you forget? did others not support your efforts?)

\_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

Think of different ways to overcome the barriers that stopped you achieving your goals. These solutions will help you get your progress back on track.

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\_\_\_\_\_

## **Worksheet 10.2:**

Many studies have reported that migrant women find it difficult to maintain some traditional cooking methods since they were time consuming and labour intensive. Also, in many countries the main meal of the day is taken at lunch time. Due to the structure of the working day in Australia, it is not often possible for families to maintain this practice. Many families then struggle to shape an alternative arrangement that ensures optimal food intake.

There is also an interest expressed by these women in learning about foods that are simple and quick to prepare and how to purchase and prepare foods that are unfamiliar.

### **Breakfast**

Breakfast is one of the most important meals of the day.

Studies indicate that people who eat breakfast perform better through out the day.

A healthy breakfast is also an important factor in weight control as it prevents mid morning hunger (when people's access to healthy food choices is more likely to be limited).

Breakfast foods are also an important source of vital nutrients, such as protein and fibre.

Price is not a good indicator of the quality of a breakfast cereal. Some of the cheaper products such as porridge oats and Weet-Bix are also among the most nutritious.

Check the nutrition panel when making your choice.

A good breakfast has most of its foods coming from the Eat Most category, in particular, grain base and something from the Eat Moderately category.

Breads, pancakes, muffins or crumpets

Pre prepared breakfast cereals eg: Mini wheats, Weeties, Ready wheat, Shredded wheat, Sultana Bran, Vita Brits, Rice flakes, Corn flakes, Oatbran.

Cooked cereals such as rice, noodles or porridge

Fresh or tinned fruit

Baked beans

Milk (on cereal or as a drink)

Yoghurt (with fruit or cereal)

Eggs (boiled or poached)

Adding fruits and yoghurt to cereals will enhance both their taste and nutritional value.

Limit juices unless having 100% fruit juices.

Use low fat milk for adults.

### **Introducing breads**

All bread is nutritious. By increasing the variety of bread in our diet, we broaden the range of grain sources from which we derive nutrients.

Bread contains vitamins and minerals and contains a lot of fibre. It is important to check for the salt and fibre content in the nutrition panel. Choose the brand with more fibre and less salt. Wholemeal bread and Multigrain bread are healthier options.

### **Lunches to take to school and work**

A balanced lunch includes bread or some other food from the cereals and grain group such as egg, meat or a dairy product; fruit and water.

Try different breads in lunches. For example: Pita or pocket breads, lavash, mountain bread, rice cakes, bagels, whole meal and whole grain bread rolls and white bread (fibre enriched). Whole meal and whole grain bread varieties are the best choices. It is not necessary to use butter and margarine on bread.

Some fillings can be: cheese, humus dip (home made or available from the supermarket, hard boiled egg mashed (this can be combined with cottage or ricotta cheese), cottage or ricotta cheese with salad vegetables, avocado. All the above fillings can be made with salad vegetables such as sliced tomato, cucumber, lettuce and grated carrot. Others include: peanut butter, almond paste, promite, marmite or vegemite.

Limit fruit juice drinks, cordials and soft drinks as they contain added sugar which contributes to weight gain and tooth decay. It is best to drink water. For children, the amount of vitamins and minerals provided by juice and dairy foods is provided in one small glass of juice and three cups of milk. After that it is best to encourage water. If using fruit juice, dilute it with water to promote health and save money. Aim for one part juice to three parts water.

Snack options: For children

Cheese slices/ sticks or cubes, celery cut into 4cm lengths with a little peanut butter or almond paste spread on it, dried fruits such as sultanas, apricots or dates (not too often), fruit and nut mixes, a small punnet of yoghurt (preferably natural), raisin or fruit bread, carrot sticks, breakfast cereals (such as Weet-bix or Vita Brits spread with peanut butter). Adding a high protein snack such as yoghurt may be particularly important if a protein source has not been used as sandwich filler (eg: vegemite).

Always include a fruit.

### **Introducing frozen and tinned foods**

Tinned and frozen foods can play an important part in our diets particularly when fresh produce is not available or there are no means to ensure safe storage.

They are useful for times when foods have run out.

They can be useful convenience foods, assisting to cope with time pressures often associated with a Western lifestyle.

Unlike other processed foods, tinned and frozen varieties contain minimal preservatives.

Fresh fruits and vegetables can be combined with frozen and tinned varieties.

They are nutritious.

It is very important to check nutrition panel and to select brands with 'no added salt' or the brand with the lowest salt content.

### **Snacks**

Avoid snacking on junk foods such as chips, cream biscuits and cheesy snacks.

Try and eat fruits or carrot sticks if hungry.

Have dried fruit such as sultanas, apricots or dates ( not too often).

Eat a small punnet of yoghurt (preferably natural or fruit based).

Snack on breakfast cereals.

**Some recipes for you to try. Check out the websites for more ideas.**

### **Vegetarian Black Bean Chili**

(healthycookingrecipes.com)

Black beans - 2 cans (19oz each drained and rinsed)

Onion - 1 (chopped)

Carrot - 1 (chopped)

Tomatoes - 3 (chopped)

Green bell pepper - 1 (chopped)

Jalapeno pepper - 1 (minded)

Chili powder - 1 tbsp

Ground cumin - 1 tsp

Crumbled dry rosemary - 1 tsp

Salt - 1 tsp

Garlic - 2 cloves (minced)

Vegetable oil - 1 tbsp

Heat oil to medium-high. Add in garlic, onion, jalapeno, carrot, and green pepper. Cook until the veggies are soft. Add in tomatoes, beans, chili powder, salt, rosemary, and cumin. Bring to a boil then reduce heat. Simmer for 20 minutes. Serve.

### **Tofu Stuffed Mushrooms**

(healthycookingrecipes.com)

Large mushrooms - 6 to 8 (hollowed out)

Tofu - 1 block (375 grams) (cut into 1/2cm square)

Green onions - 2 (chopped)

Crushed garlic - 2 cloves

Light soy sauce - 1 tablespoon

Frozen corn - 200 grams

Sesame oil - 2 teaspoons

Salt and black pepper

- Chop up the mushroom stalks and mix with the green onions and oyster sauce.
- Mix with the tofu and corn. Add in salt and pepper. Mix well.

- Stuff the mushrooms with this tofu mix, and brush the edge of the mushrooms with sesame oil.
- Bake in the oven for 12 to 15 minutes, and serve.

### **Easy Chicken Stir-Fry**

by Jackie Newgent in Food and Nutrition (prevention.com)

2 tsp reduced-sodium soy sauce

1 tsp honey

2 tsp sesame oil

1 lg bunch asparagus (about 1¾ lb), trimmed and cut on diagonal into 1" pieces

1 clove garlic, minced, or 1 tsp bottled minced garlic

2½ c sliced cooked chicken breast

1 tsp sesame seeds (optional)

Combine soy sauce and honey in small bowl. Set aside. Heat oil in large skillet or wok over medium-high heat. Add asparagus and garlic. Cook 4 minutes, stirring frequently. Toss in chicken and soy sauce-honey mixture. Heat thoroughly, remove from pan with tongs, and serve with steamed brown rice or whole wheat couscous. Sprinkle with sesame seeds, if desired.

### **Worksheet 10.3: Eating responsibly**

Look at the checklist below for ideas:

<b>Do you:</b>	<b>Could be improved:</b>
Make grain based foods such as bread, cereals a major part of your breakfast?	
Choose low or reduced fat milk and yoghurt or 'added calcium' soy beverages?	
Have a serve of fresh fruit for breakfast?	
Avoid mid morning snacking on junk foods?	
Check the nutrition panel before buying foods?	

## Worksheet 10.4: Setting goals

Write up to 3 goals here. They need to be **S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**ime targeted (SMART). For example 'I will eat a proper breakfast every day for the next month'.

List them in order of importance.

1. \_\_\_\_\_

\_\_\_\_\_

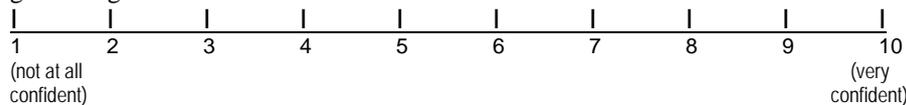
2. \_\_\_\_\_

\_\_\_\_\_

3. \_\_\_\_\_

\_\_\_\_\_

**How confident are you that you can achieve these goals?** Rate your confidence in achieving each goal using the scale below.



If you rated your confidence as 6 or less then you should probably readjust your goal. It is better to make small changes that will be successful and build on these, instead of setting goals that are destined to fail. If you rated your confidence at 9 or 10 then the goal may be too easy. Readjust your goal to make it more challenging.

**How will your progress toward each goal be monitored?**

\_\_\_\_\_

\_\_\_\_\_

**What could you do to increase your success in achieving your goal?** (e.g. 'I will ask my husband to encourage my efforts to eat a proper breakfast')

\_\_\_\_\_

\_\_\_\_\_

# Let, s learn the food pyramid.

Various activities involving all the subjects to learn the food pyramid

## 1 Healthy Eating Pyramid

**Eat**  
IN SMALL AMOUNTS

- OIL
- MARGARINE
- REDUCED-FAT SPREADS
- BUTTER
- SUGAR

**Eat**  
MODERATELY

- LEAN MEAT
- EGGS
- FISH
- CHICKEN (without skin)
- MILK
- YOGHURT
- CHEESE

**Eat**  
MOST

- VEGETABLES
- DRIED PEAS
- BEANS and LENTILS
- CEREALS
- BREAD
- FRUIT & NUTS

Pyramid concept used with permission of The Australian Nutrition Foundation Inc. 1999

## **Let's revise all modules.**

**The subjects revised all modules with the facilitator**