Depression, Anxiety and Metabolic Syndrome
in Farm Men and Women

By

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Submitted in fulfilment of the requirements for the award of

Doctor of Psychology (Health)

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I am the author of the thesis entitled *Depression, Anxiety and Metabolic Syndrome in Farm Men and Women*

submitted for the degree of *Doctor of Psychology (Health)*

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Abstract

Over the past two decades, there has been increasing attention in medical research and literature into ‘metabolic syndrome’ due to the important role it plays in the aetiology and clinical course of non-insulin dependent diabetes and coronary heart disease. However, due to large individual differences seen in treatment outcomes of metabolic syndrome factors, which currently include lifestyle and pharmacological interventions, it has been proposed that other underlying risk factors are involved in the aetiology and pathogenesis of metabolic syndrome. The Hypothalamus-Pituitary-Adrenal cortex (HPA) axis dysregulation hypothesis posits that psychological factors place additional stressors on the endocrine system, which in turn may elevate the results of the biological indicators of metabolic syndrome in individuals. Therefore, both physical and psychological factors may be important in the development and maintenance of metabolic syndrome, and this has implications for targeted interventions and treatment. This study aimed to further expand on the current body of research of interrelationships between physical and psychological symptomatology and to determine predictive relationships between Depression, Anxiety and/or Stress and metabolic syndrome in farm men and farm women, a cohort that is known to have poorer health outcomes and to experience unique psychological stressors. The results revealed that the prevalence of metabolic syndrome is greater in farm men and women than the general population, and that Depression is related to the emergence of metabolic syndrome, whilst both Depression and Anxiety are involved in its maintenance. It is concluded that psychological evaluation and interventions should be included in the detection and treatment of metabolic syndrome.
Overview

Metabolic syndrome can best be described as a clustering of metabolic, anthropometric and haemodynamic abnormalities that play an important role in the aetiology and clinical course, and increased risk of, morbidity and mortality from Type II diabetes and cardiovascular disease (Eckel, Grundy & Zimmet, 2005; Reaven, 1993). The word ‘syndrome’ is used as the term defines a group or recognizable pattern of symptoms or abnormalities that indicate a particular trait or disease (International Diabetes Foundation (IDF), 2006). Whilst metabolic syndrome is currently used as a clinical tool to identify and inform treatment for individuals who are at significantly increased risk of cardiovascular disease or Type II diabetes, the aetiology and pathogenesis of metabolic syndrome is still unclear, partially due to large individual differences in the magnitude of intervention and treatment effects.

A variety of factors have been suggested to play a role in the aetiology and pathogenesis of metabolic syndrome including genetic influences, insulin resistance, environmental factors and Hypothalamic-Pituitary-Adrenal cortex (HPA) Axis dysregulation. Increasingly, research findings suggest that there is a complex interrelationship between these factors and that the interaction between physical and mental ill health places individuals at increased risk of developing and maintaining metabolic syndrome. As current interventions target the physical metabolic syndrome factors, if a relationship between physical and mental ill health can be identified it is possible to expand on current interventions to reduce the burden of disease and achieve better health outcomes. This thesis investigates these issues. It evaluates the role of Depression, Anxiety and Stress in the pathogenesis of metabolic syndrome as well as the reverse relationship, using a
sample of Australian farm men and farm women, a population known to have poorer physical health outcomes and to be susceptible to unique and sometimes catastrophic individual, financial and environmental stressors.

Chapter 1 begins with a description of the conceptualisation, definitions and criteria of metabolic syndrome. A review of the prevalence, course, burden of disease and opportunity for recovery from metabolic syndrome is presented. Aetiological models are discussed, and the HPA axis dysregulation hypothesis of metabolic syndrome is reviewed.

Chapter 2 provides a review and evaluation of research into the link between metabolic networks and mood disorders. The co-occurrence of metabolic syndrome and Depression, and evidence of the predictive values of each of these factors in determining poorer outcomes is reviewed and the limitations within the current body of research are discussed.

Chapter 3 reviews the status of rural physical and mental health in Australia, provides an overview of Australian farm men and women, and the determinants of poorer health outcomes amongst this population explored.

Chapter 4 details the aims of the proposed study, including details about the program from which the data was drawn from. Chapter 5 details the methodology used to achieve the research aims. It also describes the setting and participant demographics for this project.
Chapter 6 outlines the results of the cross-sectional study conducted on data collected at the commencement of the program, including comparisons to results from previous research. Results pertaining to metabolic syndrome and its individual factors, Depression, Anxiety and Stress, and the relationships between these are also presented.

Chapter 7 outlines the results of the longitudinal study conducted approximately 12 months later. Results pertaining to predictive relationships between Depression, Anxiety and Stress and metabolic syndrome, as well as the role of Depression, Anxiety and Stress in the maintenance of metabolic syndrome are also presented.

Chapter 8 discusses the results of the project and examines key findings. Significant relationships between Depression, Anxiety and metabolic syndrome are explained and the role of the HPA axis dysregulation hypothesis and environmental factors are reviewed. Limitations of the study and recommendations for future research are explored, followed by the implications of the findings and concluding remarks.
1.1 Conceptualisation of Metabolic Syndrome

Over the past two decades, 'metabolic syndrome' has gained increasing attention in medical research and literature due to the important role it plays in the aetiology and clinical course of non-insulin-dependent diabetes and coronary heart disease (Eckel et al., 2005; Reaven, 1993). Also known as ‘Syndrome X’, or ‘The Insulin Resistance Syndrome’, it can best be described as a clustering of metabolic, anthropometric and haemodynamic abnormalities that, when occurring together, significantly increase the risk of morbidity and mortality from Type II diabetes and cardiovascular disease (CVD). The word ‘syndrome’ is used as the term defines a group or recognizable pattern of symptoms or abnormalities that indicate a particular trait or disease (IDF, 2006). However, whilst metabolic syndrome is currently used as a clinical tool to identify and inform treatment for individuals who are at significantly increased risk of cardiovascular disease or Type II diabetes, it is not predictive of these events.

Metabolic syndrome is not a new condition. As early as 1923 a Swedish physician, Kylin, described a condition associated with the presence of hyperglycaemia, hypertension and gout (Ford & Giles, 2003; Zimmet, Alberti & Shaw, 2005). During the 1940s, it was noted that Type II diabetes and CVD were often observed in those with upper body adiposity (male-type or android obesity) (Eckel et al., 2005; Zimmet et al., 2005). However, it was not until 1988 that real interest was generated when Gerald M. Reaven, a Medical Doctor with the Department of Medicine at Stanford University, noted that several metabolic and hemodynamic abnormalities including hyperglycaemia, hypertriglyceridemia,
hypertension, abdominal obesity and low high-density lipoprotein cholesterol concentration, commonly cluster together in individuals with diabetes or coronary heart disease, (Gami et al., 2007; Reaven, 1993). In an effort to emphasise these relationships and to assist in understanding their importance in the aetiology and clinical course of coronary heart disease, high blood pressure and non-insulin-dependent diabetes, Reaven called this clustering of risk factors *Syndrome X* (Grundy, Brewer, Cleeman, Smith & Lenfant, 2004; Reaven, 1993).

Reaven (1993) also postulated that the primary defect associated with the risk factors of this syndrome was insulin-resistance. Insulin is a hormone produced by the pancreas which assists the body’s use of glucose for energy. A simplified explanation of insulin resistance is that it is a condition in which the body’s liver, muscle and fat cells do not respond properly to the uptake of insulin, usually due to an overabundance of circulating fatty acids derived from adipose tissue; thus the body requires more insulin to help glucose enter cells. The pancreas must therefore work harder to produce more insulin and eventually fails to keep up with the body’s requirements. This results in excess glucose building up in the bloodstream which significantly increases the chance of developing Type II diabetes and cardiovascular disease (Eckel et al., 2005; Groop, 2000; National Institute of Diabetes and Digestive and Kidney Diseases, 2011).

In the context of the increasing global burden of ‘diseases of modern civilization’ (e.g. obesity, Type II diabetes, hypertension, high cholesterol and cardiovascular disease), it was suggested by Reaven (1993) and subsequently
others, that the pathogenesis of Syndrome X was embedded in interactions between genetic factors, diet, and a sedentary lifestyle.

Due to the amplified risk of morbidity and mortality associated with the clustering of metabolic syndrome factors, the increased clinical interest and research in metabolic syndrome, and the need to allocate research and health care resources, a standard definition of metabolic syndrome risk factors was required. In 1997, the American Diabetes Association (1998) proposed that the ‘insulin resistance syndrome’ comprised a number of risk factors including central obesity, glucose intolerance and dyslipidaemia; however no specific thresholds or definitions were proposed (Gami et al., 2007). In 1999, the World Health Organization (WHO) proposed a unifying definition of these risk factors and chose to call it ‘metabolic syndrome’ rather than ‘insulin-resistance syndrome’, partly due to the fact that the underlying cause of the factors had not been established (Groop, 2000). This definition included the WHO’s perception of the components of metabolic syndrome and associated cut-off criteria, including high blood pressure, elevated plasma triglyceride levels, low HDL cholesterol, high BMI and/or high waist-hip ratio, and overnight urinary albumin excretion. However, it was discovered that the proposed cut-off for waist-hip ratio alone would classify 80-90% of the population as obese and therefore disagreement continued amongst the different international healthcare stakeholders (including the WHO, the National Cholesterol Education Program – Adult Treatment Panel III (ATP-III), the European Group for the Study of Insulin Resistance (EGIR), and International Diabetes Federation) in reaching a consensus on the unique
components of metabolic syndrome and threshold values that should be assigned (Eckel et al., 2005; Gami et al., 2007; Groop, 2000).

In 2001, an expert panel was convened to provide a new working definition for metabolic syndrome which, in 2006, was the most widely used in the United States to identify and research metabolic syndrome (Sierra-Johnson et al., 2006). The proposal by the third report of the National Cholesterol Education Program-Adult Treatment Panel III (ATP-III) suggested that three or more of the following 5 criteria must be met to identify metabolic syndrome in individuals: high waist circumference, hypertriglyceridemia (elevated levels of triglycerides), low High-Density Lipoprotein (HDL) cholesterol, high fasting glucose, and high blood pressure. Table 1 (below) provides a list of the criteria and threshold values of the ATP-III criteria.

Table 1.

*ATP III Metabolic Syndrome Criteria*

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Values (Male)</th>
<th>Values (Female)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Waist Circumference</td>
<td>&gt;102cm</td>
<td>&gt;88cm</td>
</tr>
<tr>
<td>Hypertriglyceridemia</td>
<td>≥150mg/dL</td>
<td>≥150mg/dL</td>
</tr>
<tr>
<td></td>
<td>(1.69mmol/L)</td>
<td>(1.69mmol/L)</td>
</tr>
<tr>
<td>Low High-Density Lipoprotein (HDL) Cholesterol</td>
<td>&lt;40mg/dL (1.04mmol/L)</td>
<td>&lt;50mg/dL (1.29mmol/L)</td>
</tr>
<tr>
<td>High Fasting Glucose</td>
<td>≥110mg/dL (6.1mmol/L)</td>
<td>≥110mg/dL (6.1mmol/L)</td>
</tr>
<tr>
<td>High Blood Pressure</td>
<td>≥130/85 mmHg</td>
<td>≥130/85 mmHg</td>
</tr>
</tbody>
</table>

*Three or more of the above criteria must be met for a diagnosis of metabolic syndrome*
In 2003, the ATP III cut-off point for fasting glucose level was modified to ≥ 100mg/dL (5.6 mmol/L) to refocus the primary cause of metabolic risk factors as insulin resistance (Grundy et al., 2005).

Whilst there is a general consensus amongst various stakeholders that the factors shown in Table 1 are appropriate for the diagnosis of metabolic syndrome, there is still some contention as to cut-off values for each of the risk factors due to the effects on prevalence rates and also the risk of under-identification of individuals or ethnic sub-groups who are at risk of cardiovascular disease and diabetes. For example, within the United States of America (USA) a comparison of the WHO criteria and the ATP-III criteria resulted in substantially different prevalence estimates for African-American men; 24.9% & 16.5% respectively (Ford, Giles & Dietz, 2002). Further, research into the diagnostic accuracy of ATP-III criteria for the metabolic syndrome among asymptomatic Caucasian adults ($N = 256$) has shown that this criteria set provides good specificity but low sensitivity for the identification of insulin resistance amongst this cohort, and that waist circumference alone appears to provide greater overall diagnostic accuracy than the combined components of metabolic syndrome (Sierra-Johnson et al., 2006).

In 2006, the International Diabetes Foundation (IDF) (2006) released what it termed the ‘worldwide definition of metabolic syndrome’ to address both clinical and research needs. This definition does not rely on measurements only available in research settings, and it includes a comprehensive list of additional criteria for use in epidemiological studies and other research. Table 2 provides a list of the
IDF criteria. Central to this definition is central obesity (defined as waist circumference), plus any two of the four factors of raised triglycerides, reduced HDL cholesterol, raised blood pressure and raised fasting plasma glucose.

Table 2.

**IDF Metabolic Syndrome Criteria**

<table>
<thead>
<tr>
<th>Central obesity (defined as waist circumference* with ethnicity specific values) plus any two of the following four factors:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Raised serum triglycerides ≥150mg/dL (1.7mmol/L) or specific treatment for this lipid abnormality</td>
</tr>
<tr>
<td>Reduced (HDL) serum cholesterol</td>
</tr>
<tr>
<td>&lt;40mg/dL (1.03mmol/L) in males</td>
</tr>
<tr>
<td>&lt;50mg/dL (1.29mmol/L) in females or specific treatment for this lipid abnormality</td>
</tr>
<tr>
<td>Raised Blood Pressure Systolic BP ≥130 or diastolic BP ≥85 mmHg or treatment of previously diagnosed hypertension</td>
</tr>
<tr>
<td>Raised Fasting Plasma Glucose (FPG) ≥100mg/dL (5.6 mmol/L), or previously diagnosed type 2 diabetes</td>
</tr>
<tr>
<td>If above 5.6 mmol/L or 100 mg/dl, Oral Glucose Tolerance Test is strongly recommended but is not necessary to define presence of the syndrome.</td>
</tr>
</tbody>
</table>

*If BMI is >30kg/m², central obesity can be assumed and waist circumference does not need to be measured.

Table 3 (below) provides the IDF ethnic group specific values for waist circumference. This is an important inclusion as previous research has shown significant differences in the prevalence rates of metabolic syndrome between ethnic groups when using the ATP III criteria for research purposes (Ford et al., 2002) and accommodates the anthropometric differences between ethnic groups.
(Magliano, Shaw & Zimmet, 2006). The IDF also recommends that these ethnic group specific cut-off criteria should be used for members of the same ethnic group no matter where they are residing. Therefore, the criteria recommended for South Asian populations should also be used in expatriate South Asian communities for research and clinical purposes. As a result of these improvements, the IDF definition is increasingly being used in research.

Table 3.

**IDF Ethnic Specific Values for Waist Circumference**

<table>
<thead>
<tr>
<th>Country/Ethnic group</th>
<th>Waist circumference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Europids</strong>*</td>
<td>Male</td>
</tr>
<tr>
<td>In the USA, the ATP III values (102cm male; 88cm female)</td>
<td>Male</td>
</tr>
<tr>
<td>are likely to continue to be used for clinical purposes</td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>≥ 90cm</td>
</tr>
<tr>
<td></td>
<td>≥ 80cm</td>
</tr>
<tr>
<td><strong>South Asians</strong></td>
<td>Male</td>
</tr>
<tr>
<td>Based on a Chinese, Malay and Asian-Indian population</td>
<td>Male</td>
</tr>
<tr>
<td></td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>≥ 90cm</td>
</tr>
<tr>
<td></td>
<td>≥ 80cm</td>
</tr>
<tr>
<td><strong>Chinese</strong></td>
<td>Male</td>
</tr>
<tr>
<td></td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>≥ 90cm</td>
</tr>
<tr>
<td></td>
<td>≥ 80cm</td>
</tr>
<tr>
<td><strong>Japanese</strong></td>
<td>Male</td>
</tr>
<tr>
<td></td>
<td>Female</td>
</tr>
<tr>
<td></td>
<td>≥ 90cm</td>
</tr>
<tr>
<td></td>
<td>≥ 80cm</td>
</tr>
<tr>
<td><strong>Ethnic South and Central Americans</strong></td>
<td>Use South Asian recommendations until more specific data are available</td>
</tr>
<tr>
<td><strong>Sub-Saharan Africans</strong></td>
<td>Use European data until more specific data are available</td>
</tr>
<tr>
<td><strong>Eastern Mediterranean and Middle East (Arab) populations</strong></td>
<td>Use European data until more specific data are available</td>
</tr>
</tbody>
</table>

*In future epidemiological studies of populations of Europid origin, prevalence should be given using both European and North American cut-off points to allow better comparisons.

**Originally different values were proposed for Japanese people but new data support the use of the values shown above.

In 2009, representatives from the International Diabetes Federation Task Force on Epidemiology and Prevention held discussions in an attempt to resolve differences in the definitions of Metabolic Syndrome (Alberti et al., 2009). It was
agreed that the categorical cut-off points for triglycerides, HDL, blood pressure and fasting blood glucose would remain the same as for the IDF definition (see Table 2 above), and that meeting any three of the five criteria would establish a clinical diagnosis of metabolic syndrome. However, it was also recommended that, rather than the adoption of an International criteria, waist circumference would require further cross-sectional and longitudinal research to determine sex and ethnic differences, which would allow particular health systems to adopt local population cut-off points for economic and pragmatic reasons.

Although there are other definitions of metabolic syndrome, such as the WHO definition and the European Group for the Study of Insulin Resistance (EGIR) (a modified version of the WHO definition to be used with subjects without a diagnosis of diabetes) (Cameron, Magliano, Zimmet, Welborn & Shaw, 2007), a review of the literature demonstrates that the most widely used definitions in research within the USA and Australia are those of the ATP III and IDF.

Controversy around the definitions and specific values associated with metabolic syndrome appear to reflect the various conceptual aetioloical frameworks (Magliano et al., 2006). Whilst initially proposed as a means of identifying insulin resistance, research has shown that not all of those meeting the criteria for Metabolic syndrome have insulin resistance which has, in turn, called into question the clinical usefulness of the syndrome (Cameron et al., 2007; Grundy et al., 2004; Khan, 2008). However, as Khan (2008) notes, even amongst critics, there is no doubt that the combination of the metabolic syndrome risk factors for Type II diabetes and CVD are “found more often in combination than
chance alone would dictate” (p. 1892). Therefore, metabolic syndrome is an advantageous indicator in the research, and the identification and treatment of those individuals and populations at increased risk of CVD or Type II diabetes.

1.2 Prevalence and Course of Metabolic Syndrome

Due to the relatively recent addition, and differing definitions of metabolic syndrome, prevalence data are somewhat limited. However, the available data are alarming as they provide evidence that not only is metabolic syndrome highly prevalent, it is also rapidly increasing in prevalence.

1.2.1 Prevalence

To initially estimate the prevalence rates in the USA, Ford et al. (2002) used data from the Third National Health and Nutrition Examination Survey (NHANES III), a cross-sectional, multistage, stratified sampling design survey conducted during 1988 and 1994 using a representative sample of the non-institutionalized civilian population aged 20 years and older (N=8814). Using the ATP III criteria (see Table 1) the overall unadjusted and age-adjusted prevalence rates of metabolic syndrome were 21.8% and 23.7% respectively. From age 20 through to 29 years, the prevalence rate of 6.7%, continued to increase over the life-span to 43.5% for participants aged 60 through 69 years. Little difference was found in the prevalence rates for men (24.0%) and women (23.4%).

Further research conducted by Ford, Giles and Mokdad (2004) that included data (N = 1677) from the NHANES 1999-2000 survey, found that the age-adjusted prevalence rates had increased by 23.5% (p = 0.021) amongst women
and 2.2% among men ($p = 0.831$), with rises in waist circumference, high blood pressure and hypertriglyceridemia accounting for much of this increase.

In an attempt to understand world-wide prevalence of metabolic syndrome, Eckel et al., (2005) presented the findings from various countries using the ATP III criteria (Figure 1). Although these various studies differed in design, precise metabolic syndrome definitions used, sex and age structure of the populations, year they were undertaken and sample selection, Eckel et al. (2005) believe that certain inferences can be drawn about prevalence of metabolic syndrome in urban populations.

![Figure 1. World-wide Prevalence of Metabolic Syndrome from ATP III Definition](image)

A comparison of four populations within the Asia-Pacific Region with distinctly different ethnic backgrounds (‘Asian’ for Japan and Korea; ‘Europid’ for Australia; and ‘Pacific Islander’ for Samoa) aged >35 years, also found
distinctly different prevalence rates of metabolic syndrome between countries (Lee et al., 2008). Table 4 (below) provides a summary of the age-adjusted prevalence rates using both the ATP III and IDF criteria. Lee et al. (2008) ensued that the inclusion of the ethnic specific waist circumference of the IDF definition appears to capture more of the at-risk population than the ATP III criteria would.

Table 4.

Summary of Age-Adjusted Prevalence (%) of Metabolic Syndrome in Adults Aged ≥ 35years

<table>
<thead>
<tr>
<th></th>
<th>Australia</th>
<th>Japan</th>
<th>Korea</th>
<th>Samoa</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Male</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATP III</td>
<td>35.7</td>
<td>8.1</td>
<td>12.5</td>
<td>39.2</td>
</tr>
<tr>
<td>IDF</td>
<td>42.2</td>
<td>7.5</td>
<td>12.7</td>
<td>45.3</td>
</tr>
<tr>
<td><strong>Female</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATP III</td>
<td>28.3</td>
<td>9.9</td>
<td>22.8</td>
<td>57.4</td>
</tr>
<tr>
<td>IDF</td>
<td>33.3</td>
<td>11.3</td>
<td>28.9</td>
<td>59.5</td>
</tr>
</tbody>
</table>

Within Australia, research of the Australian adult population aged 25 years and over has estimated metabolic prevalence rates to be between 22.1% (95% CI, 18.8-25.4) and 30.7% (95% CI, 27.1-34.3) using the ATP III and IDF criteria respectively (Cameron et al., 2007). A nationally representative, cross-sectional study of the Australian population (N = 11,247) by Cameron et al. (2007), using data collected in 1999-2000 for the Australian Diabetes, Obesity, and Lifestyle Study (AusDiab), also found that prevalence rates increased with age, with
estimates peaking amongst those participants aged 65-74 at approximately 43% and 52% using the ATP III and IDF criteria respectively. Gender differences were also found, with prevalence amongst males significantly greater than amongst females (APT III – 24.4% versus 19.9%, \( p < 0.001 \); IDF 34.4% versus 27.2%, \( p < 0.001 \)).

Another urban Australian cross-sectional study, using a representative random sample of predominantly European adults from Adelaide, South Australia (\( N = 5,850 \)) found that the rate of metabolic syndrome was 22.8% (15.7% women; 26.4% men) using the IDF definition, and 15.0% using the ATP III definition (14.4% women; 19.4% men) (Adams et al., 2005). Congruent with findings from previous studies, the prevalence rate increased with age, with 45.7% (IDF definition) and 29.5% (ATP III definition) of men between the ages of 55 and 69 meeting the criteria for metabolic syndrome. For women, those 70 years of age and over had the highest prevalence rates for both the IDF and ATP III definitions; 45.5% and 24.8% respectively.

Within rural Australia, the prevalence rates for metabolic syndrome are even greater than those of their urban counterparts and substantially higher than those reported in the 1999-2000 AusDiab survey. A study by Janus et al. (2007) conducted in rural areas of south-eastern Australia using a stratified random sample of the population aged 25-74 years (\( N = 806 \)), found the prevalence of metabolic syndrome, as defined by ATP III, was 27.1% (95% CI, 22.7-31.6) in men and 28.3% (95% CI, 24.0-32.6) in women. When using the IDF definition, prevalence rates for men and women were 33.7% (95% CI, 29.0-38.5) and 30.1%
(95% CI, 25.7-34.5) respectively. Fundamental to the observed differences in prevalence rates was the incidence of central obesity using the IDF waist circumference criteria of $\geq 94$ cm for men and $\geq 80$ cm for women (as opposed to the ATP III criteria of $\geq 102$ cm for men and $\geq 94$ cm for women), which resulted in 61.9% of men and 72.4% of women being classified as overweight or obese. It was also found that when using waist circumference as the criterion, prevalence of obesity ranged from 46.4% in regional centres to 49.8% in smaller towns. Of note was the comparison conducted by Janus et al. (2007) of their data with that of the 1989 National Heart Foundation of Australia (NHFA) Risk Factor Prevalence Study, which found that whilst mean height had not increased in either sex, mean weight had increased by an average of 10 kg for both sexes, and waist circumference had increased by an average of 12 cm in men and 16 cm in women. Janus et al. (2007) also reported that these increases were even greater than those of the 1999-2000 AusDiab study, which indicated an increase of 7 cm in men and 8 cm when compared with the NHFA 1989 study. These findings are consistent with trends of increased central obesity and development of myocardial infarction and diabetes (Janus et al., 2007).

1.2.2 Course

As noted above, those defined as having metabolic syndrome have a substantially elevated risk of developing Type II diabetes and CVD. This is not surprising as the different factors included in the definitions of metabolic syndrome are all risk factors for these diseases. Within a European population, relative hazard ratios for CVD outcomes in people with metabolic syndrome compared to those without it ranged from 2 to 5 (Eckel et al., 2005). Prospective
studies of middle-aged persons have also shown that of all new-onset CVD cases, metabolic syndrome alone was able to predict around 25% of cases (Grundy et al., 2004; Lakka et al., 2002).

In an attempt to quantify the impact of metabolic syndrome on early-onset coronary artery disease (CAD), Iribarren et al. (2006) conducted a case-controlled study of early-onset CAD subjects ($N = 393$) and individually matched control subjects ($N = 393$). It was found that after adjusting for external risk factors such as smoking, body mass index (BMI), educational attainment and alcohol consumption in men $>46$ years and women $>56$ years, the odds of early-onset CAD in the presence of metabolic syndrome by ATP III criteria was a 5-fold increase in the absence of diabetes and an 8-fold increase when combined with a diagnosis of diabetes.

A meta-analysis of 37 longitudinal studies that included 43 cohorts and 172,573 individuals has also been conducted to assess the association between metabolic syndrome and cardiovascular events and mortality (Gami et al., 2007). This study found the overall pooled relative risk (RR) for cardiovascular events and death in people with metabolic syndrome was 1.78 (95% CI, 1.58-2.00). More interesting were the results of the pooled analysis of studies that simultaneously adjusted multivariate models for both metabolic syndrome and its components. This analysis demonstrated that patients with metabolic syndrome have around a 50 per cent increased risk of cardiovascular disease beyond its individual components when compared with patients without metabolic syndrome (RR = 1.54, 95% CI 1.32-1.79) (Gami et al., 2007). This finding has important
implications for both the clinical usefulness of metabolic syndrome and the identification, treatment and prevention planning of those at risk of CVD.

Several studies have also indicated the predictive value of metabolic syndrome in the development of Type II diabetes, which accounts for 90 per cent of all diabetes (Eckel et al., 2005; IDF, 2006). However, given that glucose intolerance was initially purported in the aetiology of metabolic syndrome, this association is not surprising. A British study that investigated outcome data from two prospective studies on non-diabetic individuals found that metabolic syndrome was associated with an increased risk of diabetes in elderly persons aged 70-82 years ($N = 5,804$) ($HR = 4.41$, 95% CI, 3.33-5.84) and also in middle aged men aged between 40-59 years ($N = 7735$) ($HR = 7.47$, 95% CI, 4.90-11.46) (Sattar et al., 2008). However, Sattar et al. (2008) concluded that metabolic syndrome was not required to identify those at risk of developing Type II diabetes within the younger cohort as fasting glucose alone was able to predict the onset of Type II diabetes. Nevertheless, amongst the older cohort in their study, there was a significant association between meeting the criteria for metabolic syndrome and the incidence of diabetes ($HR = 4.41$, 95% CI, 3.33-5.84), whilst fasting glucose alone was only able to account for less than 50% of participants developing overt diabetes. No other individual risk factor alone was able to account for diabetes.

Beyond Type II diabetes and CVD, individuals with metabolic syndrome are purported to be susceptible to conditions such as polycystic ovary syndrome, sleep disturbances, some forms of cancer, fatty liver, cholesterol gallstones, and stroke (Grundy et al., 2004; Rosmond & Bjorntorp, 2000), although no causality has
been established. Again, the purporting of these associations is not surprising given that individual metabolic syndrome risk factors have been implicated in their aetiology previously. What has not been researched to date is the increased risk of these conditions when combined with meeting the criteria for metabolic syndrome. Nevertheless, the development of Type II diabetes and CVD, along with their respective complications, creates a global burden that impacts profoundly on healthcare systems.

1.3 Burden of Disease and Negative Outcomes

There are currently no figures available for the burden of disease related to metabolic syndrome, however the IDF (2006) provides the most comprehensive information regarding the burden of disease associated with the negative outcomes of metabolic syndrome: Type II diabetes (and associated complications such as amputation, blindness and kidney failure) and cardiovascular complications. It is predicted that by the year 2025, the incidence of diabetes will double, suggesting a comparable rise in cardiovascular-related morbidity and mortality.

At present, it is conservatively estimated that diabetes in those aged between 20 and 79 years of age has an annual global health care cost of 286 billion international dollars (ID) and that if the prevalence continues to rise as predicted, by 2025 this figure will increase to 396 billion ID, the equivalent of 13 per cent of the world’s healthcare budget (IDF, 2006). However, in countries where there is high prevalence of diabetes, this figure could be as much as 40 per cent of their healthcare budget. The IDF (2006) also specifies that these figures do not include
the co-morbid burden of CVD associated with metabolic syndrome where clinical
diabetes is not yet present. Thus, the burden of disease from metabolic syndrome
and associated risk factors is likely to be much greater than both the current
figures and future estimates. However, there is an opportunity to reduce the risk
factors associated with metabolic syndrome and possibly an opportunity for
recovery.

1.4 Opportunity for Recovery

The strength of the metabolic syndrome diagnosis is that it identifies the
clustering of metabolic abnormalities that are associated with an increased risk
of developing CVD or Type II diabetes. However, it has been suggested that once
detected, this risk can be significantly reduced through relatively simple and
inexpensive therapeutic lifestyle changes.

Core to the IDF’s definition, and included in the ATP III’s definition of the
metabolic syndrome, is central obesity. Obesity alone is associated with higher
CVD risk and contributes to high serum cholesterol, hyperglycaemia,
hypertension and low HDL cholesterol and can lead to a decrease in insulin-
mediated glucose uptake (IDF, 2006; Reaven, 1993). Whilst metabolic syndrome
can be found in non-overweight or non-obese individuals, suggesting that it is not
solely determined by environmental factors but also genetic and endocrine factors,
appropriate levels of habitual physical activity (at least 30 minutes of brisk
walking per day) and weight management appear to be the cornerstone in any
attempt to reduce risk factors associated with metabolic syndrome (Eckel, Khan,
Robertson & Rizza, 2006; Ford et al., 2002; Grundy et al., 2005; Lakka &
Laaksonen, 2007; Reaven, 1993). Indeed, research has demonstrated that an increase in physical activity when combined with dietary modifications improves insulin sensitivity, reduces insulin resistance, decreases blood pressure, increases high-density lipoprotein (HDL) cholesterol, decreases triglyceride levels, and may prevent or delay the onset of Type II diabetes, CVD and premature mortality. These reductions can often occur independent of weight loss (Ford et al., 2002; Grundy et al., 2004; Lakka & Laaksonen, 2007; Shaw & Chisholm, 2003). An uncontrolled study on sedentary individuals with metabolic syndrome ($N = 105$) showed that after 20 weeks of aerobic exercise training around 30% ($N = 32$) no longer met the criteria for metabolic syndrome, with no sex or ethnic differences in the efficacy of this treatment (Katzmarzyk et al., 2003).

As epidemiological data suggest that many components of metabolic syndrome are modifiable through diet and exercise, primary interventions should aim to include healthy therapeutic lifestyle changes including moderate calorie restriction (to promote weight loss) and moderate increase in physical activity (Feldeisen & IDF, 2006; Lakka & Laaksonen, 2007; Shaw & Chisholm, 2003; Tucker, 2007). However, research has also shown large individual differences in the magnitude of the effect of regular exercise on metabolic syndrome (Lakka & Laaksonen, 2007). There is also currently no consensus regarding the most appropriate dietary recommendations for metabolic syndrome, although it has been suggested that gene-diet interaction research may clarify this in the future and result in tailored dietary recommendations (Feldeisen & Tucker, 2007). These conclusions appear to confirm that the underlying aetiological mechanisms and pathogenesis of metabolic syndrome are still ill-defined and that metabolic syndrome may have a
multifactorial aetiology, including genetic, environmental and endocrine factors which will, in turn, affect the pathogenesis for individuals. To be able to refine interventions and treatments it is important to understand the underlying risk factors and how metabolic syndrome may develop.

1.5 Aetiological Models of Metabolic Syndrome

There are currently a number of proposed aetiological models for metabolic syndrome, with research suggesting that the development of the syndrome may be individualistic and dependent on a number of genetic and environmental influences. For example, a twin study using a sample of 125 monozygotic (MZ) twins and 178 dizygotic (DZ) twins aged between 55-74 years in Denmark found that, amongst the risk factors associated with metabolic syndrome, BMI, glucose intolerance and HDL cholesterol concordance rates were significantly higher amongst monozygotic (MZ) twins than dizygotic (DZ) twins (Poulsen, Vaag, Kyvik & Beck-Nielsen, 2001). However, gender accounted for much of the differences observed, with glucose intolerance among male twins primarily explaining the different concordance rates and no evidence of any genetic influence on glucose intolerance amongst female twins. Conversely, the difference in concordance rates for HDL-cholesterol was only detected amongst female twins. Poulsen et al. (2001) also found no differences in concordance rates for abdominal obesity, triglycerides, hyperinsulinaemia or hypertension, suggesting these particular risk factors of metabolic syndrome are substantially environmentally influenced. Therefore, it is important to examine some of the major theoretical aetiology models to further understand some of the underlying
influences on metabolic syndrome and how these may affect the pathogenesis of the syndrome.

1.5.1 Insulin Resistance

The original, and possibly most accepted, conceptualisation of metabolic syndrome and its pathophysiology is that of ‘insulin resistance’, as first suggested by Reaven in 1988 (Reaven, 1993). As stated earlier, insulin resistance occurs when the pancreas fails to keep up with the body’s increasing requirement of insulin (usually due to an overabundance of circulating fatty acids derived mainly from adipose tissue that interfere with the ability of the cells in the liver, muscle and fat to absorb insulin), resulting in a build-up of glucose in the bloodstream. In turn, these abnormalities increase lipid and glucose levels and elevate blood pressure, which significantly increases the risk of developing Type II diabetes and CVD. Whilst this is an overly simplified explanation of the mechanism of insulin resistance, it does clarify why, regardless of differing opinions on appropriate cut-off levels, the five risk factors of metabolic syndrome (particularly central obesity) were chosen by the major stakeholders given the role that adipose tissue fatty acids play in the development of insulin resistance.

Nevertheless, insulin resistance does not occur in isolation and various theoretical models of the pathogenesis of insulin resistance over the years have included such factors as age, obesity, foetal malnutrition, genetic inheritance and/or abnormalities, and visceral adiposity (Cameron et al., 2007; Ford et al., 2002; Groop, 2000). Also, whilst it is common for individuals with metabolic syndrome to have insulin resistance, a significant proportion do not (Ford et al.,
2002), suggesting that insulin resistance is not the principal underlying factor of metabolic syndrome, even though it may play an important role in the clustering of risk factors and may contribute towards outcomes (Grundy et al., 2004; Khan, 2008).

1.5.2 Environmental Factors

As previously stated, the clustering of abnormalities associated with metabolic syndrome is associated with increased risk of glucose-intolerance, dyslipidaemia, high blood pressure and CVD, which have been referred to as ‘diseases of modern civilization’ (Reaven, 1993). It is possible that these diseases have become more prevalent in modern society due, in particular, to two environmental determinants: an increase in sedentary behaviour (such as watching television) and/or decrease in physical activity; and poor dietary habits, including higher caloric intake and saturated fat content and lower quality of nutritional intake, known as ‘empty calories’ (Eckel et al., 2005; Feldeisen & Tucker, 2007; Ford et al., 2002; Ford, Giles & Mokdad, 2004; IDF, 2006; Lakka & Laaksonen, 2007; Poulsen et al., 2001).

These two environmental factors are well established as contributing to an overweight or obese status, which alone has been implicated in the elevation of risk factors in many fatal and nonfatal conditions including low HDL cholesterol, hypertension, hyperglycaemia, coronary heart disease, CVD, heart failure and stroke, even in the absence of glucose intolerance (Eckel et al., 2006; Grundy et al., 2004). There is also evidence of a correlation between abdominal obesity and other metabolic risk factors, including insulin sensitivity, although there is the
possibility of these factors being a coincidental expression of an unknown third factor (Groop, 2000; Grundy et al., 2004). Nevertheless, obesity has been growing steadily in prevalence over the past three decades and has become a significant world-wide problem with no socio-economic, ethnic or racial group spared (Cameron et al., 2007; Eckel et al., 2006). Obesity is also a simple-to-measure and modifiable risk factor that can be targeted by clinical and public health practitioners to decrease the associated risks (Janus et al., 2007; Magliano et al., 2006).

However, under the ATP III definition which does not require central obesity to be included in diagnosis, metabolic syndrome can also develop independently of obesity and sedentary activity (Reaven, 1993), and the evidence relating to genetic heritability of factors, and gender and age differences, cannot be ignored in the aetiology and pathogenesis of metabolic syndrome. Therefore, it appears that there is a complex interrelationship between genetic and environmental factors in the development of metabolic syndrome that is perhaps triggered by a third variable.

1.5.3 Hypothalamic-Pituitary-Adrenal Cortex Axis Activity

More recently, Hypothalamic-Pituitary-Adrenal cortex (HPA) Axis activity has been implicated in the aetiology and pathogenesis of metabolic syndrome. Prolonged stress activates the hypothalamus which then induces the anterior pituitary gland to secrete adrenocorticotropic hormone (ACTH), which in turn stimulates the adrenal cortex to secrete cortisol, enhancing metabolic activity and elevating blood levels of glucose and other nutrients (see Figure 2 below) (Kalat,
2007). Compared to the autonomic nervous system, which prepares the body for the brief emergency ‘fight or flight’ responses, the HPA axis reacts more slowly and plays a central role in the homeostatic processes of the endocrine system (Nevid, Rathus & Rubenstien, 1998; Rosemond & Bjorntorp, 2000).

In non-stressful situations, cortisol levels should vary throughout the day in a circadian, pulsatile pattern of high and varying levels in the morning and decreasing between 1600 hours and midnight to less than 75% of the morning values (Rosemond & Bjorntorp, 2000; Tsigos & Chrousos, 2002). Whilst temporary increases in cortisol levels enhance immune system activity, in situations of high levels of prolonged stress the HPA axis becomes the dominant response, continually releasing elevated levels of cortisol at the expense of the immune system (Kalat, 2007; Nevid et al., 1998). Continued secretion of elevated cortisol levels may suppress the production of antibodies to fight illness and disease and has been linked to increased vulnerability to the common cold and
influenza, and increased risk of chronic diseases including cancer and heart disease (Kalat, 2007; Nevid et al., 1998).

Cortisol levels, as regulated by the HPA axis, also play an important role in lipid and glucose metabolism, with evidence of prolonged elevated levels leading to Type II diabetes, a redistribution of body fat as characterised by central adiposity, and hypertension. A study by Rosemond and Bjorntorp (2000) on men chosen from the National Population Register and born within the first six months of 1944 ($N = 284$), demonstrated that a pathological HPA axis function (determined by low cortisol variability) in men aged 51 years, was significantly inter-correlated with established anthropometric, metabolic and haemodynamic risk factors for CVD, Type II diabetes and stroke, including the metabolic syndrome risk factor variables, with the exception of low HDL cholesterol.

As metabolic syndrome risk factors and psychological stress share the same endocrine system, HPA axis pathology may be a moderating factor in the development of CVD and Type II diabetes (Rosmond & Bjorntorp, 2000; Weber-Hamann et al., 2002). There is evidence to suggest that HPA axis pathology associated with stress is a plausible underlying pathway to increased risk of metabolic syndrome as psychological characteristics such as anger, hostility, Depression and Anxiety also influence the risk of developing CVD and diabetes (Goldbacher & Matthews, 2007; Weber-Hamann et al., 2002). Evidence of the co-occurrence of high levels of circulating cortisol and depressive symptoms, also known as hypercortisolemic Depression, has also supported this underlying pathway as a specific risk factor for metabolic syndrome. Research by Vogelzangs
et al. (2007) found a significant interaction between urinary cortisol, Depression and metabolic syndrome in an older population ($N = 867, M = 74.1$ years), with the odds of metabolic syndrome in persons with both urinary cortisol excretion in the highest tertile and depressed mood to be $1.84$ ($95\% \ CI = 1.02-3.34, p = 0.003$), when compared to persons without either condition. It was also found that an increased prevalence of metabolic syndrome was significantly associated with higher levels of depressive symptoms, which was partially mediated by urinary cortisol levels. Similarly, Weber-Hamann et al. (2002) found that when comparing post-menopausal female in-patients with a diagnosis of Major Depression ($N = 22$) to an aged-matched healthy control group ($N = 23$), cortisol concentrations were significantly higher in patients than in the control group ($26.3 \pm 9.9 \text{ vs. } 10.5 \pm 4.6 \text{ mmol/L, } F(1,37) = 32.9, p < 0.001$).

However, the above findings do not address the relationship between psychological distress and subsequent detrimental effects on positive health behaviours, including an increase in harmful behaviours and poor compliance with treatment regimes which, like metabolic syndrome, are also associated with insulin resistance and increased central adiposity (Goldbacher & Matthews, 2007).

1.6 Summary

In summary, metabolic syndrome is a clustering of metabolic, anthropometric and haemodynamic abnormalities. With increasing clinical and research interest over the past two decades, the definition of metabolic syndrome continues to evolve and currently the ATP III and IDF definitions are the most widely used.
Limited, global prevalence studies have found that the risk of developing metabolic syndrome increases with age, whilst gender differences appear to be related to ethnicity. The course of metabolic syndrome places an individual at increased risk of development of Type II diabetes and CVD which, along with their respective complications, creates a global burden that impacts profoundly on healthcare systems. It has been suggested that this risk can be significantly reduced through relatively simple and inexpensive therapeutic lifestyle changes such as diet and exercise; however large individual differences in the results of interventions have been found, suggesting a multifactorial aetiology of metabolic syndrome. The differences in intervention results may possibly be explained by the HPA axis pathology conceptualisation, which suggests that psychological factors may be placing additional stress on an endocrine system that is shared with the biological indicators of metabolic syndrome. It is important to understand this interrelationship between psychological and biological risk factors in the aetiology and pathogenesis of metabolic syndrome as the current treatment paradigm is focused on interventions aimed at reducing only physical symptomatology. Further understanding of the psychological factors associated with metabolic syndrome is imperative for determining those populations at higher risk of developing the syndrome and for guiding the development of more effective interventions and treatments. Psychological factors associated with metabolic syndrome will be examined and evaluated in the following chapters.
Chapter 2: Psychological Factors Associated with Metabolic Syndrome

2.1 Rationale for Increased Research Interest

Over the past decade there has been an increasing volume of research into the associations between cortisol, mood disorders (including Depression, Anxiety and Stress) and the component factors of metabolic syndrome. From the literature reviewed, it appears that this shift has occurred for a number of reasons, including the inability to ascertain a direct cause and effect relationship between physiological factors and the pathogenesis of metabolic syndrome. The formulation of the HPA axis pathology hypothesis, which suggests that a shared endocrine system that is influenced by both physical and psychological factors is a plausible pathway in the aetiology of metabolic syndrome. This has led to a paradigm shift amongst psychologists and other health professionals towards developing an understanding of relationships between psychological factors (e.g. behaviours, attitudes, and beliefs) and health and illness.

Along with physical illnesses, Depression has been identified by the WHO as one of the top 10 worldwide health problems. It is responsible for the greatest proportion of burden attributable to non-fatal health outcomes, accounting for almost 12% of total years lived with disability worldwide (Muhtz, Zyriax, Klahn, Windler & Otte, 2009). In fact, it is estimated that by 2020 unipolar Depression will account for the second largest burden of disease worldwide, after ischemic heart disease (Viinamaki et al., 2009). Depression has also been associated with an increased risk of developing a number of physical illnesses, including cardiovascular disease (Whooley, 2006). A number of authors have suggested a link between metabolic and psychological factors and suggest the risk estimates
may be additive if a person has both metabolic syndrome and Depression. Recent explanatory theories, such as that of HPA axis activity (as discussed in Chapter 1), have also posited that alterations in metabolic networks mediate allostasis, that is, the process of achieving homeostasis through behavioural or physiological change. In the short term, they are essential for adaptation, maintenance of homeostasis, and survival (allostasis). Yet, over longer time intervals, they exact a cost (allostatic load) that can accelerate disease processes, such as metabolic derangements. Therefore, awareness of depressive symptoms could be important in the detection and clinical management of metabolic syndrome. This chapter will evaluate and discuss the research available on the relationship between psychological and metabolic risk factors, and identify those individuals or populations who are at increased risk of metabolic syndrome.

2.2 Metabolic Networks and Mood Disorders

A systematic review by McIntyre et al. (2007) of 40 years of research conducted between 1966 and 2006 found evidence that disturbances in metabolic networks are implicated in depressive disorders in a number of ways, including pathophysiology (changes in the normal mechanical, physical and biochemical functions, caused by disease or resulting from an abnormal syndrome), brain volumetric changes, symptomatic expression (e.g., neurocognitive decline), and medical comorbidity. The authors also found a number of shared abnormal metabolic process overlaps between Major Depressive Disorder and Type II diabetes, suggesting shared pathophysiological mechanisms. In fact, the authors went as far as proposing that alternations in metabolic networks are such a defining component in pathophysiology, that a neuropsychiatric syndrome of
‘metabolic syndrome Type II’ be conceptualised as a basis for testing metabolic influence and therapies for mood disorders. Whilst initially this may appear a brave proposal, a study of 60 acute depressive adult inpatients found that those patients with metabolic syndrome ($n = 15$) had significantly higher triglyceride blood levels in acute Depression than those without metabolic syndrome ($n = 45$), $t(56) = 4.83, p < 0.01$ (Richter, Juckel & Assion, 2010). Richter et al. (2010) also found a significant positive correlation between meeting the criteria for hypertriglyceridemia and severity of Depression, as measured by the Clinical Global Impression Scale (CGI) score, as a characteristic of the group with metabolic syndrome ($r = 0.65, p < 0.01$). Richter et al. (2010) posited that Depression and elevated triglycerides may be linked by an assumed activation of the HPA axis, citing research by Glueck et al. (1993) as support for their argument. Glueck et al. (1993) suggested a reversible causal relationship between high triglycerides and symptoms of Depression as the authors found after a 54-week single blind treatment with triglyceride-lowering diet and medication, a concurrent major shift toward amelioration or absence of depressive symptoms, as measured by the Beck Depression Inventory, was observed.

Whilst it would be pragmatic to suggest that Depression and/or metabolic syndrome could be treated by focusing on metabolic factors alone, it does suggest an interrelationship between the two, and provides some further support of the HPA axis hypothesis.
2.3 Co-occurrence of Metabolic Syndrome and Depression

Research on the co-occurrence of metabolic syndrome and Depression is typically undertaken using standard clinical means and measures for metabolic syndrome factors, and self-report questionnaires for psychological symptoms of Depression and Anxiety, such as the Beck Depression Inventory (BDI-21) or the Hospital Anxiety and Depression Scale (HADS), and the Kessler 10 measure of Psychological Distress (K10). Whilst self-report measures have their limitations, particularly the possibility of exaggeration or minimisation of symptoms depending on the motivation of the respondent, they are more economically viable for larger scale research than clinical interviews and their use is considered acceptable practice (Gregory, 2007).

Most studies have also used the ATP III definition of metabolic syndrome which has allowed for comparison between the studies. However, they may not have captured a significant proportion of those who may meet the criteria for metabolic syndrome under the more sensitive IDF definition, which could be considered a possible limitation of these studies.

Nevertheless, research supporting the co-occurrence of metabolic syndrome and Depression is growing. One study in Finland (Miettola, Niskanen, Viinamaki & Kumpusalo, 2008), using eight adult birth cohorts (N = 416) found the prevalence rate of metabolic syndrome was 37% (39% in men and 35% in women). The mean BDI-21 total score was 7.07 among the subjects with Metabolic Syndrome and 5.49 among the subjects without Metabolic Syndrome (mean difference −1.585 and 95% CI −2.917; −0.253). Miettola et al. (2008) also
examined items within the BDI-21 and found that participants with metabolic syndrome were significantly worse off than those without on the items relating to irritability ($p = 0.006$), work inhibition ($p = 0.003$), fatigability ($p = 0.011$), weight loss ($p = 0.013$), and loss of libido ($p = 0.014$). Gender comparisons found that whilst men with metabolic syndrome scored significantly higher on all of the above items than those men without, for women the item ‘loss of libido’ was the only significant difference. Logistic regression analysis, using a BDI-21 cut-off point of 14/15, and adjusted for age, vocational education, marital status and working status, found significant associations between self-reported Depression and elevated blood glucose amongst men (OR = 1.697, 95% CI, 1.185-2.430, $p = 0.004$) and large waist circumference amongst women (OR = 1.066, 95% CI, 1.009-1.127, $p = 0.024$).

Another study conducted in France by Skilton, Moulin, Terra and Bonnet (2007) on 1598 patients (aged 30-80 years) referred to an outpatient centre on the basis of possessing at least one CVD risk factor, found metabolic syndrome present in 61.5% of the study population, Anxiety in 62.6% and Depression in 24.5%, when using the Hospital Anxiety and Depression Scale (HADS) as the self-report measure. When comparing those with metabolic syndrome with those without, there was a significantly higher rate of Depression in men (22.5% vs. 15.5%, $p = 0.007$) and women (38.6% vs. 23.3%, $p < 0.001$), which persisted after adjusting for age, prior cardiovascular disease, employment status and marital status, and was only slightly altered by the addition of smoking status, dietary score and physical activity. No gender differences were found when examining these associations. The association of metabolic syndrome and Depression was
consistently observed across BMI categories and was of a similar magnitude in both the youngest and oldest quartiles. Skilton et al. (2007) also found that the primary metabolic syndrome component associated with Depression was abdominal obesity, although the association was found to be markedly stronger among women than among men. Interestingly, no association was found between metabolic syndrome and Anxiety. However the prevalence of metabolic syndrome in those with both Anxiety and Depression was similar to those with Depression alone, suggesting a relatively greater association with Depression and metabolic syndrome than Anxiety and metabolic syndrome.

Similarly, an association with Depression, but not Anxiety or psychological distress in men and women from rural Australia aged 25-84 years ($N = 1,345$) was reported by Dunbar et al. (2008). When comparing those participants with metabolic syndrome using the ATP III criteria ($n = 409, 30.4\%$) with those without, those with metabolic syndrome had higher scores for Depression (mean scores 3.41 vs. 2.95, $p=0.013$) and were more likely to have moderate to severe Depression (10% vs. 6.0%, $p = 0.069$). Two components of metabolic syndrome, waist circumference and HDL cholesterol, were independently associated with Depression. Of those participants with metabolic syndrome ($n = 409$), 338 did not have diabetes, however the same associations remained after controlling for this variable, which is important as diabetes has previously been shown to be independently associated with Depression (Lustman & Clouse, 2005). Dunbar et al. (2008) also conducted the analyses using the IDF criteria for metabolic syndrome and found the association between Depression and metabolic syndrome was consistent across definitions.
The above findings from the different studies not only support an association between metabolic syndrome and Depression, but also suggest possible gender differences in the pathophysiology or symptomatology of metabolic syndrome.

2.4 Depression as a Predictor of Metabolic Syndrome

Much of the research to date assumed that Depression predicts the onset of metabolic syndrome, based presumably on the need to improve aetiological models for metabolic syndrome. Research conducted in Finland on a sub-group ($n = 223$) from a random general population sample ($N = 3004$), interviewed participants once a year for three years between 1998 and 2001 and then again in 2005 (seven years from baseline) on self-reported measures of mental symptoms and the Structured Clinical Interview for DSM-IV (Viinamaki et al., 2009). Metabolic syndrome criteria were measured by health professionals at a medical laboratory. It was found that the overall prevalence of metabolic syndrome at the 2005 follow-up was 32% (49% in men and 21% in women, $p < 0.001$). Viinamaki et al. (2009) also categorised participants into groups that self-reported either high mental symptoms (HMS) ($n = 106$) or low mental symptoms (LMS) ($n = 117$) based on the Hamilton Rating Scale for Depression (HDRS) and BDI, and conducting a Structured Clinical Interview for DSM-IV to confirm diagnoses of personality disorders and Major Depressive Disorder (MDD). Cumulative depression burden was then calculated by summing the BDI scores from the three previous assessments (1998, 1999 & 2001), and used as an estimate for symptom severity. Participants were also divided into categories (HMS or LMS) based on the median of the total burden score. It was found that those participants in the HMS group had significantly higher prevalence of metabolic syndrome than those
in the LMS group (46% vs. 27%, \( p < 0.001 \)), and that those with metabolic syndrome presented overall with more signs of depressive symptoms and suicidality in the HDRS and BDI than those without. When analysed by gender, these associations were only statistically significant in men. When the pooled sample was observed, it was found that psychiatric diagnoses were not associated with metabolic syndrome, however a statistically significant association was found between the diagnosis of MDD and metabolic syndrome in men (30.4%, \( p = 0.02 \)). Conversely, the frequency of metabolic syndrome amongst those with chronic Depression (total Depression burden score above the median) was markedly higher than those under the median (41% vs. 24%, \( p = 0.01 \)). When analysed by gender, this difference was observed in men (61% vs. 36%, \( p = 0.01 \)) but not in women (25% vs. 17%, \( p = 0.22 \)). Further analyses found significant relationships between the different descriptors of mental illness in relation to fulfilled metabolic syndrome criteria for men were: glucose level and major depressive disorder (26.2%, \( p < 0.05 \)) and BDI (8.8%, \( p < 0.05 \)); Triglyceride level and HMS (71.4%, \( p < 0.05 \)); suicidality (33.3, \( p < 0.01 \)) and BDI (10.3%, \( p < 0.05 \)); and HDL cholesterol and suicidality (20.8%, \( p < 0.05 \)). Amongst women, the only significant relationship was between HMS and glucose level (54.0%, \( p < 0.05 \)). Based on hypocortisolemic depression of the HPA axis activity theory, Viinamaki et al. (2009) suggest that these gender differences, whilst peculiar, could possibly be explained by some men reacting to long-term mental distress by developing a metabolic syndrome phenotype. Further research would be required to test this hypothesis, particularly as the authors do not report controlling for medication. However, the findings that cumulative scores above the means on
High Mental Symptoms and chronic Depression were associated with metabolic syndrome highlights the clinical importance of a relationship.

Similar results relating to the severity of Depression affecting metabolic syndrome emergence was found in a study conducted by Raikkonen, Matthews and Kuller (2007) on middle aged women ($N = 432$) who were followed for an average of 15 years from baseline. The potentially confounding factor of HRT on metabolic effects was statistically controlled for. Using the IDF criteria, it was found that 9.7% ($n = 42$) had metabolic syndrome at baseline, and this rose to 38% ($n = 164$) by follow-up. In logistic regression models, ‘depressive symptoms’ and ‘severity of stressful life events’ were associated with the cumulative prevalence of metabolic syndrome (including those with metabolic syndrome at baseline), (OR 1.31, 95% CI 1.08-1.61; OR 1.71, 95% CI 1.15-2.54). The analyses also demonstrated that each one standard deviation (SD) increase in depressive symptoms was associated with 1.21 to 1.43 fold increases in risk for developing metabolic syndrome over the follow-up period.

Another longitudinal study ($N = 488$) to evaluate the risk for developing metabolic syndrome when having depressive symptoms (as self-reported in the BDI) was conducted over seven years in Finland by Vanhala, Jokelainen, Keinanen-Kiukaanniemi, Kumpusalo and Koponen (2009). At baseline, 16 men (8.2% of the baseline study population) and 48 women (16.3%) had depressive symptoms. At the 7 year follow-up, logistic regression analysis showed a 2.5-fold risk (95% CI 1.2-5.2) for middle-aged women with depressive symptoms at baseline to develop metabolic syndrome after adjusting for age, physical activity,
education, smoking, alcohol use, marital status and the use of antidepressant medication and hormone replacement therapy. Whilst there was no significant finding for men, males reported less severe depressive symptoms when compared with females, suggesting symptom severity or gender differences may play a role in the reporting of depressive symptoms. However, symptom severity could also perhaps explain the findings of the research by Viinamaki et al. (2009) reported above, as the males in their study reported more severe symptoms when compared to females.

2.5 Metabolic Syndrome as a Predictor of Depression

There has also been research examining whether metabolic syndrome is associated with the onset of depressive symptoms. Akbaraly et al. (2009) conducted a longitudinal study of middle-aged British civil servants (N = 5,232, mean age 49.5 ± 6.1 years) from the Whitehall II prospective cohort study. This study was conducted over a six-year period and used the Depression subscale from the 30-item General Health Questionnaire to measure depressive symptoms. At baseline, prevalence of metabolic syndrome was 10.4% (n = 547) and those participants with metabolic syndrome were more likely to be men, non-white, have a low education level and to undertake low physical activity levels. Three years later, 428 participants (8.2%) had developed depressive symptoms and it was found to be more common amongst participants with metabolic syndrome than those without (13.5% vs. 10.2%, respectively). A higher rate of new-onset depressive symptoms was observed in those with high triglycerides (29.4% vs. 24.9%), low HDL cholesterol (22.7% vs. 17.8%) and central obesity (14.3% vs. 10.4%). Multivariate models using data from both the three and six year follow-
ups, and controlling for age, sex and ethnicity, found that the odds ratio of new-onset depressive symptoms within those participants with metabolic syndrome relative to those without was 1.47 (95% CI = 1.09-1.99, \( p = 0.01 \)), which remained statistically significant after further adjustment for health behaviour, coronary heart disease and other socio-demographic factors (OR 1.38, 95% CI = 1.02-1.87, \( p = 0.04 \)). Interestingly, none of the components of metabolic syndrome had a significant independent effect on the onset of depressive symptoms, however the percent attenuations between depressive symptoms and metabolic syndrome by individual components were 23.4% for low HDL, 37.5% for obesity, and 38.0% for triglycerides; together explaining 93% of the association. Notably, Akbaraly et al. (2009) also found no evidence to support bidirectional associations between depressive symptoms and metabolic syndrome, and suggested that depressive symptoms may be a consequence rather than a cause of metabolic syndrome, and that a combination of abnormal lipids and central obesity constitutes a risk factor for the onset of depressive symptoms. As nearly half of the participants with metabolic syndrome were overweight (49.8%), the authors offer a possible explanation of the obesity-depression relationship as being the experience of stigma and devaluation by overweight and obese individuals. These experiences may cause individuals to suffer from lower self-esteem and higher levels of Depression and, whilst there are a number of studies to support this explanation, Akbaraly et al. (2009) note that the onset of depressive symptoms appears to be the product of the cumulative effect of central obesity, high triglycerides and low HDL components. The finding that high triglyceride levels are associated with depressive symptoms is similar to that of Richter et al. (2010) and provides further support for the HPA axis pathology
hypothesis. Interestingly, Akbaraly et al. (2009) found that participants with elevated fasting blood glucose levels were less likely to develop depressive symptoms at the six year follow-up, which suggests that insulin resistance may not be a core mechanism in the metabolic syndrome-depressive symptoms hypotheses.

Conversely, in a study that examined gender effects and the role of cortisol in the association between depressive symptoms (as measured by the Patient Health Questionnaire) and metabolic risk, Muhtz et al. (2009) found no association between depressive symptoms and metabolic syndrome as an entity \( (p > 0.3) \) in either men \( (n = 107) \) or women \( (n = 108) \). However, when the single components of metabolic syndrome were analysed using MANOVA, there was a significant effect for gender \( (F = 9.2, p < 0.01) \), and it was found that women with depressive symptoms had a larger waist circumference \( (F = 4.7, p = 0.03) \), higher fasting blood glucose \( (F = 7.9, p < 0.01) \), diastolic blood pressure \( (F = 5.7, p = 0.02) \), evening salivary cortisol, and lower HDL cholesterol \( (F = 5.0, p = 0.03) \) when compared to those without depressive symptoms. A significant effect of depressive symptoms on cortisol levels was also found in women \( (F = 2.7, p = 0.03) \) and the adjusted regression analyses found that evening cortisol levels attenuated the association between depressive symptoms and fasting glucose, waist circumference, diastolic blood pressure and HDL cholesterol, and partially mediated the association between metabolic syndrome and depressive symptoms. No significant associations emerged for men. Whilst these findings partially support the HPA axis hypotheses of metabolic syndrome, it is unclear why this would be the case for only women.
2.6 No Evidence of a Relationship

There are also studies that have reported no association between Depression and metabolic syndrome. One study, conducted by Hildrum, Mykletun, Midthjell, Ismail and Dahl (2009) was cross-sectional in nature and generous in power ($N = 9,571$). The researchers concluded there were no consistent associations between either Depression or Anxiety and metabolic syndrome. However, after adjusting for age and gender, a weak positive association was found for Depression (as measured by the HADS) as a continuous measure and metabolic syndrome as defined by IDF criteria (OR 1.07 per standard deviation increase in symptom load, 95%CI = 1.02-1.12, $p = 0.007$). This finding would suggest a relationship between the number of factors of metabolic syndrome and the severity of depressive symptoms. Hildrum et al. (2009) also found a weak relationship between both Depression and comorbid Anxiety and Depression with central obesity and high triglycerides; two factors which are becoming regularly apparent in the association between Depression and metabolic syndrome. These findings suggest an integrated approach to the pathogenesis of metabolic syndrome is required to further understand the relationship between metabolic syndrome factors and depressive symptoms.

2.7 Chronic Stress, Distress and Metabolic Syndrome

According to the HPA axis pathology hypothesis, chronic stress could also be an underlying factor in the aetiology and pathogenesis of metabolic syndrome, as stress is known to adversely affect the neuroendocrine system through sympathetic dysregulation that may result in metabolic and anthropometric changes over time (Kalat, 2007; Matthews & Kuller, 2002; Nevid et al., 1998;
Raikkonen, Tsigos & Chrousos, 2002; Rosemond & Bjorntorp, 2000). There is also a relatively large body of evidence to suggest a relationship between stress and CVD or CHD, diseases that those individuals with metabolic syndrome are at increased risk of developing (Vitaliano, Scanlan, Zhang, Savage, Hirsch & Siegler, 2002). Unfortunately, few studies have examined this relationship. As noted by Vitaliano et al. (2002), this may be because there is an absence of theoretical models to guide hypotheses formulation and research. However, with the inclusion and subsequent momentum of the HPA axis hypothesis, chronic stress and distress should be included as possible factors in the research around the aetiology and pathogenesis of metabolic syndrome.

In a longitudinal study of 425 middle-aged women aged 42-50 years at study entry, Raikkonen et al. (2002) measured metabolic syndrome factors and also psychological risk attributes using the BDI, the Framingham Tension Scale, the Spielberger Trait Anxiety Questionnaire, the Cohen Perceived Stress scale, and the Spielberger Trait Anger Questionnaire at baseline. Participants were re-tested after 7.4 years, at which time, relative to those without metabolic syndrome, women who met the criteria for metabolic syndrome at baseline also had significantly higher scores seven years later on the BDI ($p = 0.002$), Trait Anxiety ($p = 0.01$), Tension ($p = 0.02$), and Trait Anger ($p = 0.01$), although perceived stress was found to be not significant ($p = 0.16$). Results also showed that women who were classified at baseline as having metabolic syndrome when compared to those who did not, experienced a greater increase in Anger ($M = 1.37$ vs. $-0.67$, $p < 0.001$) and Anxiety ($M = 1.68$ vs. $-0.55$, $p < 0.001$) at follow up. Furthermore, the risk for developing metabolic syndrome during the 7.4 years was increased for
women who experienced an increase in Trait Anger from baseline to follow-up (OR = 2.02, 95%CI = 1.50 – 2.72). Statistical controls for physical activity and alcohol did not significantly alter associations, ruling them out as mediational influences.

In a further follow-up study on the same cohort at 15 years post baseline, Raikkonen et al. (2007) found that intense and frequent feelings of anger and tension predicted the risk of metabolic syndrome using both the IDF and ATP-III criteria. Using the WHO criteria for metabolic syndrome, global perceived stress was predictive of the risk for the development of metabolic syndrome. This finding highlights the important role of psychological risk factors in the development of metabolic syndrome and also the requirement of a global definition of metabolic syndrome, as results can differ according to the criteria used.

In response to the lack of theory linking chronic stress, psychophysiology and CHD, Vitaliano et al. (2002), used structural equation modelling to test a path model of chronic stress in the development of metabolic syndrome and CHD. The model was evaluated using a sample of caregivers whose partners had Alzheimer’s disease, as the course of this disease is a prototypic chronic stressor. Caregivers were categorised as older men, women using hormone replacement therapy (HRT), and women not using HRT. The authors controlled for HRT as its use in postmenopausal women is believed to reduce CHD risk factors and is associated with greater HDL cholesterol and lower LDL cholesterol, insulin resistance and central adiposity. Participants were measured for chronic stress,
personal resources, vulnerability (less controllable and relatively enduring influences), social resources, psychological distress (negative affect, burden, hassles and the absence of positive experiences in response to stress) and metabolic syndrome factors at time of entry (Time One) and 15-18 months later (Time Two). Vitaliano et al. (2002) also measured poor health habits during this time, as there is greater risk for some people to increase fat and caloric intake and engage in alcohol consumption, smoking, and sedentary behaviours during times of stress. The cross-sectional pathway between distress and metabolic syndrome was significant both at Time One and Time Two for men, at Time Two only in women not using HRT, and non-significant in women using HRT at either time point. Positive relationships between distress and metabolic syndrome were three to 12 times larger in men than in women overall in a follow-up of medical records that was conducted 27-30 months after study entry. It was also found that metabolic syndrome was positively associated with CHD prevalence in men at Time Two. In women not using HRT, cross-sectional analyses found that poor health habits were related to metabolic syndrome at Time One only, distress was related to metabolic syndrome at Time Two only, and psychological distress was related to poor health habits at both Times One and Two, suggesting both psychological attributes and health behaviours were important. The use of the two time points also showed that these relationships grew stronger over time and that some variables, such as health habits, may have lagged effects on the development of metabolic syndrome. Vitaliano et al. (2002) also measured anger and hostility in this study and found that whilst they contributed to distress, there was a more direct relationship over time between distress and metabolism as measured by the metabolic syndrome factors. Therefore, chronic stress appears to activate the
distress reaction, which may precipitate metabolic reactions and lead to cardiovascular events, particularly in males experiencing chronic stress and a negative change in health behaviours. However, the results also suggest that future metabolic syndrome cannot be directly predicted by prior psychological distress, although it may predict metabolic changes due to poor health habits in the 15 to 18 months before metabolic syndrome is detected.

2.8 Limitations of Research Reviewed

Whilst there are a number of limitations amongst the reviewed research, including, in some cases, small sample sizes and predominantly Caucasian samples based in Europe, one of the major limitations of the research reviewed is the use of the HADS as a measure of Depression in participants. The HADS has been designed for, and validated on, medical patients and excludes many of the somatic symptoms of Depression that overlap with physical problems such as fatigue, sleep disturbance, psychomotor changes and loss of appetite and weight. Other measures, such as the PHQ and the BDI, are based on DSM-IV criteria for major Depression. As participants identified as having metabolic syndrome in these studies were generally not seeking or undertaking medical treatment for this clustering of risk factors, nor necessarily meeting the criteria cut-off for a major depressive episode, a large proportion of participants may have been experiencing sub-acute levels of Depression that were not recognised. Future research should consider the use of a measure that has been normed on a general population and is continuous in its measure to ascertain cut-off levels that include sub-acute experiences by participants.
Most of the research has also used the ATP III definition of metabolic syndrome which, while allowing for comparisons between the studies, may not have captured a significant proportion of those who may meet the criteria for metabolic syndrome under the more sensitive IDF definition. This is also a possible limitation of these studies.

2.9 Summary

In summary, over the past decade there has been a paradigm shift in research that has resulted in an increasing focus on associations between cortisol, mental health problems (particularly Depression) and the component factors of metabolic syndrome. Based on the HPA axis activity hypothesis, neuro-endocrine and metabolic networks mediate both homeostasis and allostasis; as such, disturbances within these networks have been implicated in the pathogenesis, and also recovery from, depressive disorders. In particular, elevated triglyceride levels, a risk factor of metabolic syndrome, have been shown to have a reversible relationship with depressive symptoms, although this can also be confounded by such factors as alcohol use and body weight.

There is a growing body of evidence to suggest the co-occurrence of metabolic syndrome and Depression; although it appears that gender differences occur not only in prevalence of both Depression and metabolic syndrome, but also the pathophysiology or symptomatology of metabolic syndrome. From the research reviewed, it appears that hypertriglyceridemia and waist circumference are more closely associated with Depression in men with metabolic syndrome. Amongst women with metabolic syndrome, the more closely associated indicators appear to
be low HDL cholesterol levels and waist circumference. A reciprocal relationship between metabolic syndrome and psychological attributes may also therefore be dependent on gender related physiological manifestations or self-perceived severity of symptoms.

There is also evidence that the greater the number metabolic syndrome factors met, the higher the likelihood of co-morbid depressive symptoms, particularly in men. Interestingly, one study in which these associations were found was undertaken in a semi-rural community in eastern Finland. It has also been observed that the mean score for metabolic syndrome and Depression was around two to three times higher in research conducted in rural regions of Australia when compared with other studies reviewed. These findings suggest that those living within rural areas are at increased risk of co-morbidity of metabolic syndrome and Depression.

Whilst it would be pragmatic to suggest a causal relationship between metabolic syndrome and mood disorders, the evidence at present suggests that, whilst there are associations between these factors, there is currently no consensus on direction or causation. However, awareness of symptoms of psychological distress could be important in the clinical management of metabolic syndrome, and vice versa.

When data from prevalence studies are incorporated with the findings from research into the co-occurrence of metabolic syndrome and mood disorders, it appears that certain characteristics may increase a person’s risk in developing
metabolic syndrome. These characteristics include middle-age, lower education level, low levels of physical activity, and higher levels of psychological distress. There are also gender differences amongst these characteristics as men are more likely report psychological distress as depressive symptoms or feelings of suicidality, whereas females report experiencing higher levels of Depression, Anxiety, tension and anger. In relation to the co-occurrence of psychological distress and metabolic syndrome factors, males are more likely to have a large waist circumference and have a greater number of other metabolic syndrome factors, particularly high triglyceride levels, whereas for women, a larger waist circumference is the most prominent co-occurring risk factor.

The finding of higher risk ratios for metabolic syndrome and Depression amongst those living in rural areas is of particular interest given that rural populations are often under-researched (as evidenced by only two studies of this population) and under-serviced, not least, within Australia, due to their lower population density when compared to their urban counterparts and their geographical distance from research institutions and health care providers. Those living in rural areas also generally experience poorer health as reflected in levels of disease, mortality and health risk factors, than those living in major cities. Living and working on the land also exposes people to psychological stressors that are unique to this cohort. Therefore, it is important to understand if those living in rural areas may be more at risk for the co-occurrence of metabolic syndrome and mood disorders, and how this relationship may affect the pathogenesis of metabolic syndrome. This shall be explored in the next chapter.
Chapter 3: Rural Health in Australia

3.1 Health Issues in Rural Australia

The rural idyll has been depicted in novels, films, popular press and academic publications, and portrays the entrenched view of many that life in rural areas offers tranquillity, peace, open space, a sense of belonging, community integration (expressed in close-knit ties), reduced criminality and social disorganisation, fewer problems, low pollution and simplicity of life (Brumby, Chandrasekara, McCoombe, Kremer & Lewandowski, 2011; Sarantakos, 2000). In the words of one family, farm life is described as “Especially for the kids, because it's a great life for them. Yeah it's a good life. It's a hard life, but it's a good life.” (Department of Primary Industries, 2007).

However, the limited research available has found no significant differences between the subjective quality of life of farmers and metropolitan residents (Best, Cummins & Lo, 2000; Sarantakos, 2000). Furthermore, contrary to the agrarian myth described above of a healthy and peaceful way of life, an abundance research shows that people living in rural areas generally experience poorer health than their counterparts who reside in major cities as is reflected in levels of disease, mortality and health risk factors (AIHW, 2008a). For example, alcohol consumption, cigarette smoking, drink driving accidents, Depression and suicides are all significantly higher in rural communities (Wilson, 2007). Indeed, it is well established that farming is an occupation that is inherently stressful. High levels of Depression, Anxiety and stress have been acknowledged as significant psychological hazards associated with engagement in agricultural work (Fraser et al., 2005). Research also suggests that poor mental and physical health may in
turn impair the running of a farm business effectively (Gorgievski-Duijvesteijn, Giesen, & Bakker, 2000), leading to further stress.

Those residing in rural and remote communities are also 1.3 times more likely to report having diabetes, and 6% of all excess deaths each year are directly related to diabetes, not including for those deaths from diabetic complications (AIHW, 2008b). However many rural and remote residents may be unaware they have the condition as they are less likely to be tested than their metropolitan counterparts (AIHW, 2008b). Due to current methodological issues, the rates of cardiovascular disease in rural/remote and metropolitan residents are not directly comparable (AIHW, 2008b).

The poorer health status of rural residents when compared to their metropolitan counterparts may be occurring for a number of reasons. For example, rural communities often face many barriers to accessing health care services, not only because of the lack of availability of services within their community, but also because of transportation problems and/or associated costs of accessing health services located in another town or region (Berry et al., 2008; Hefflinger & Christens, 2006). Consistent with the lower availability of general and specialist health professionals in rural and remote communities, it has also been found that people living in rural and remote areas are more likely to be admitted to hospital with potentially preventable conditions, such as skin and other cancers which can be detected early through screening (AIHW, 2008a), with a mortality rate 42% higher for those living outside major cities (ABS, 2011).
Whilst there is a growing body of research surrounding health risk factors and barriers to accessing services in rural and remote areas, much of the research fails to differentiate between those who simply reside in rural and remote communities and those who work the land. There is currently very little research available on an important and unique cohort within this population: farm men and farm women.

3.2 Overview of Australian Farm Men and Women

Farmers are an important asset to Australia and its population, with 125,594 farms solely dedicated to agricultural production (NFF, 2009). Australian farms and closely related sectors generate approximately 12% of the Australian Gross Domestic Product (GDP), equating to approximately AUD$103 billion per year in production. Australian farms also produce around 93% of Australia’s domestic food supply, directly employ 317,730 people on the farms, and support jobs for approximately 1.6 million other Australians, or 17.2% of the national workforce (NFF, 2009). Farm men and women also possess a large transferable skill set. In comparison to the 17-18 skills that a factory worker may have, farmers have around 120 skills that are broadly based and can be transferred to different jobs or careers (Department of Primary Industries, 2007).

Despite these impressive figures and the positive aspects of farm life, the agricultural industry and particularly farmers, face unique challenges that could possibly have a number of repercussions, including physical, psychological, social and economic impacts. Agriculture has one of the highest mortality rates of any industry and exposes farm men and farm women to a range of physical and mental
health risks (McCurdy & Carroll, 2000). Farmers are now working longer and harder, are ageing and are increasingly relying on family members to provide extra labour on the farm (Brumby, Martin & Willder, 2006). Present-day circumstances such as increased economic distress, decline in family farming, a growing need for off-farm work, and a fall in both the number of farms and the income generated by farming activities, have been shown to be associated with a generally lower quality of life and wellbeing (Melberg, 2003). In this time of difficult economic and climatic conditions currently being experienced by the Australian agricultural sector, it is becoming increasingly apparent that research into the mental health of farm men and women and associated physical health outcomes is necessary.

3.3 Rural and Farmer Mental Health

As farm men and women are currently an under researched sub-population, it is necessary to defer to the research available for rural and remote Australian population mental health issues. One Australian cross-sectional population survey ($n=1563$) on the prevalence of psychological distress amongst people aged between 25-75 years living in the south-east of South Australia and the south-west and north-west of Victoria, found that 31.3% of men and women living in these area reported psychological distress (Kilkkinen et al., 2007). Kilkkinen et al., (2007) also found that 10% of the population surveyed reported elevated Depression and Anxiety, with the highest rates occurring in both men and women in the 45-54 year age bracket. Another report on rural, regional and remote health also found that males aged 45 to 65 years living in regional or remote areas were 1.2 times more likely to report high to very high levels of psychological distress
and 1.4 times more likely to report Depression than their Major City counterparts (AIHW, 2008b). Conversely, the AIHW (2008b) found that there were no significant inter-regional differences in the prevalence of Depression or Anxiety, and that women aged 45-64 years living in rural and remote areas were significantly less likely to experience Anxiety when compared to their Major City counterparts. However, in a study of Australian farm men and women ($N = 1813$) the incidence of psychological distress using the K10 was 45.9%, which was higher than rural Victorian (31.3%), state (32.9%) or national (35.6%) averages (Brumby et al., 2011; Kilkkinen et al., 2007).

It has also been argued that, unlike farm men, farm women experience stress related to both the farm, and the impact of farming stressors on the social, financial and physical wellbeing of all family members (Fraser et al., 2005). Farm women in the USA have been found to have higher levels of stress, Depression and fatigue than farm men, with high workloads and role conflict as consistent explanatory themes (Gallagher & Delworth, 1993).

Apart from the lack of research with a definite focus on farm families, much of the currently available Australian research on rural population health is over 10 years old. Since then, Australian farmers have experienced a 10 year drought, which was then followed by floods. Much is known about the link between mental health problems and acute weather disasters (such as cyclones or floods), however less is known about the effects of chronic climate-related disasters such as drought (Berry et al., 2008). Thus, older research may not be reflective of the current mental health experienced by Australian farmers. As the hardships of farming
lifestyle increase, the stress of property damage, lost income and debt will inevitably result in mental health problems for some (Berry et al., 2008).

Despite the suggestion that farmers may be at increased risk of mental health problems, research often shows that farmers self-report a lower prevalence of psychiatric morbidity than the general population (AIHW, 2008b). However, mortality rates from suicide are far greater for this cohort, reportedly being responsible for 1.7% of all deaths in Australia (AIHW, 2007). In regional and remote areas this proportion is 66% higher than in major cities and it is particularly elevated in males between the ages of 25 and 44 years (ABS, 2011), and higher still amongst farmers in both the United Kingdom and Australia. Research using descriptive and linear regression analysis of aggregated Australian mortality data from the period 1988 to 1997 found that of the 921 farm suicides identified, 67.4% were farm managers (including owners), with 97.3% of this group being males and 48.5% over the age of 55 years (Page & Fragar, 2002). The most common methods of suicide were firearms (51%), motor vehicle exhaust gas (12.3%) and hanging (16.4%), which collectively accounted for approximately 81% of completed suicides amongst farm managers and labourers (Page & Fragar, 2002). These suicide rates for farmers and farm managers are higher than those reported above for the wider rural population, and are believed to be associated with stressors such as financial or family problems and the administrative, legislative and production pressures of Australian agricultural processes and the rural economy (Page & Fragar, 2002).
Research by Thomas et al. (2003) has found that farmers are also more likely to report that life was not worth living (OR 3.26, 95%, CI 1.51 to 7.02). Whilst the research by Thomas et al. (2003) was conducted in Britain amongst a representative cohort of 606 farmers, farm workers and family members, the comparison of farm family members and workers and rural and semi-rural householders highlights the possibility of different experiences, or cognitive processing of these experiences, between cohorts residing in the same geographical location. This may be attributed, in part, to some of the determinants of poorer health outcomes for farmers, which is discussed in further detail in this chapter.

Psychological autopsies conducted by Malmberg, Simkin and Hawton (1999) on 84 farmers who had died by suicide in England and Wales found that rather than a reaction to an immediate crisis, suicide in farmers was the end point of an accumulation of difficulties over time. It was also found that depressive disorder was the most frequently occurring mental illness associated with suicide, with physical illness and relationship, occupational and financial problems important and common factors in these deaths. Anecdotal evidence of these contributing factors commonly occurring in Australian farming populations is also noted by rural community health care workers, teachers and religious leaders (Department of Primary Industries, 2007). It is therefore important to further understand the factors that place greater stress on farming families, and how they affect their mental health and wellbeing.
3.4 Determinants of Poorer Health Outcomes

Whilst there is little research on the mental health of farm men and women, the available research has identified a number of possible factors that may be particularly associated with the mental health and wellbeing of farming families. These factors include drought and climate variability, increased workload, engagement in off-farm work, and economic pressures.

3.4.1 Drought and Climate Variability

As stated earlier, farming is an inherently stressful occupation, which is only made more difficult in times of extreme weather events (Berry et al., 2008). Many farming families within Australia have been impacted by the effects of drought over the past few years, if not decades. For example, New South Wales (NSW) has been ‘drought-free’ for only four of the 30 years prior to 2007, with many new areas around Australia declared ‘exceptional circumstances’ areas between 2001 and 2007 (ABS, 2008; Morrissey & Reser, 2007). Exceptional Circumstances (EC) due to drought is determined on the basis of six criteria, including meteorological conditions, agronomic and stock conditions, water supplies, environmental impacts, farm income levels, and the scale of the event. Figure 3 (below) shows the changes in EC areas in Australia between 2000 and 2007.

It has been suggested by Wilson (2007) that as rural communities rely heavily on the success of agribusiness, drought places a particular stress on them. In trying to quantify the cost of drought on life satisfaction, it is estimated that the detrimental impact of drought in Spring equates to an AUD$18,000 annual reduction in mean rural household income (Carroll, Frijters & Shields, 2009).
The financial estimate in this research was calculated for only one spring drought in rural Australia and does not take into account the often chronic nature of drought, which can last for years. Lost income, damage to property and debt due to drought add more stressors which can accumulate and increase the likelihood and prevalence of mental health problems (Berry et al., 2008). The unpredictability of drought also often creates feelings of powerlessness (Sartore, Kelly, Stain, Albrecht & Higginbotham, 2008) and is commonly associated with higher levels of Anxiety and emotional distress amongst farm men and women when compared to those living in a drought-free city (Coelho, Adair & Mocellin, 2004).

![Exceptional Circumstances Areas 2000-02 to 2006-07](image)

Figure 3. *Exceptional Circumstances Areas 2000-02 to 2006-07*

The presenting symptomatology associated with drought is most often related to failure and chronic loss, such as generalised Anxiety, helplessness-induced
Depression, and ongoing emotional distress. A recent study surveying randomly selected adults from the Australian Electoral Roll measuring support networks and community attachment, recent stressors, and current health and functioning, found that 71.8% of farmers and farm workers in rural NSW reported high levels of perceived stress due to drought (Stain et al., 2008). This perceived stress was particularly associated with recent adverse life events, functional impairment and increased alcohol use.

In a report to NSW Agriculture on the social impacts of drought, it was noted that the poverty of farm families is more than relative deprivation in comparison with other Australians: there is also a psychological poverty, defined as a poverty of the spirit, that relates to increased workloads, lack of access to services, and a withdrawal from the community, which is exacerbated by a feeling that others do not understand what is currently being experienced (Alston & Kent, 2004). This report highlights a significant amount of stress amongst farming families which has resulted in increased mental illness and other stress related conditions. However, Alston & Kent (2004) alert readers to the difficulty in ascertaining whether these social impacts are drought-induced or related to ongoing structural adjustment within farming communities.

Alarmingly, a report by Berry et al. (2008) to the Garnaut Climate Change Review on the rural mental health impacts of climate change, predicts that regions in Australia previously considered to have low vulnerability and risk to adverse climate change will experience markedly larger downturns in agricultural productivity by 2030 than previously predicted. For those regions at high risk of
adverse climate changes, the effects on agriculture could be so great that grazing may not be feasible, thus excluding agriculture from many communities. This prediction and subsequent adverse mental health impacts that can be expected to arise highlight the necessity for both health and wellbeing education, and provision of services in rural communities, particularly amongst farm men and women, whose very livelihoods may be at stake.

Interestingly, the drought experienced by the farmers over the past ten years officially broke in a number of areas during the longitudinal research component of this study. Unfortunately, other natural disasters occurred including severe weather events that resulted in flooding, unseasonal rainfall that affected crops and/or livestock, cyclones and the biggest locust plague seen in decades (Department of Agriculture, Fisheries and Forestry, 2011). Therefore, it could be concluded that the chronic nature of environmental stressors encountered by farmers was maintained during this time period.

3.4.2 Economic Pressures

Economic hardship is of particular concern amongst farming Australians who are often asset rich and cash-flow poor (Berry et al., 2008). Specifically, the effects of these economic pressures on farming populations have been consistently identified as one of the major causes of stress, and they are associated with symptoms such as psychological dysfunction (any disruption in normal psychological functioning), cognitive impairment, Anxiety, physical aggressiveness, Depression, withdrawal, marriage problems, loss of temper,
increased substance use and suicidal ideation and completion (Fraser et al., 2005; Heflinger & Christens, 2006).

Research into the stress experiences of farm men in the United States of America (USA) who were owner operators \( n=84 \) and had similar farming operations to Australian farmers, found that farmers had greater exposure to job-related and financial events than non-farmers \( n=295 \) (Swisher, Elder, Lorenz & Conger, 1998). These job-related and financial events were also the most significant predictors of Depression for farm men, as assessed by the Hopkins Symptom Checklist (SCL-90-R), six items from the positive affect sub-scale of the Mental Health Inventory, and the Psychiatric Epidemiology Research Interview (PERI) Life Events Scale.

As there is an abundance of research that has shown that social support, particularly family support, acts as a buffer to mental health problems, Johnson and Booth (1990) conducted a longitudinal study into the role of individual psychological distress in mediating the effect of economic decline on the erosion of family relations, (in particular, marital quality) on Nebraska farmers during the 1980’s farm crisis in the USA. It was found that whilst the close relationship between the family unit and the economic unit of the farm were likely to serve as a barrier to marital instability, serious economic problems threaten and weaken the economic unit which in turn weakens the barrier to marital breakup. Thus, economic distress had a significant effect on marital communication and thoughts of divorce, and created personal psychological distress, which in turn influenced
marital quality over a 5 year period. It was also found that half of the effect of thinking about divorce was due to increased levels of Depression.

3.4.3 Increased Workload and Engagement in Off-farm Work

Climate- and economic-related business stress is also associated with increased workloads which can, in turn, heighten risk factors such as social isolation, socio-economic strain and family separation, well-established factors to unfavourable to mental health outcomes (Berry et al., 2008; Stain et al., 2008). Statistics show that 59% of Australian farmers worked over 49 hours per week, with a median of 51 hours per week, in comparison to 41 hours for all self-employed people in all occupations within Australia (ABS, 2003). As farming has become less profitable over the past 20 years, many farm families have found it necessary for members to undertake more on- and off-farm work to supplement and stabilise the family income (ABS, 2003; Fraser et al., 2005). Recent figures show that 81% of farm women currently undertake off-farm work, with this income source accounting for between 44% and 48% of the farming family’s income (ABS, 2003; Fraser et al., 2005).

These changes in family roles and family separation can be difficult for farming families. For example, Gallagher & Delworth, (1993) suggest that there are three ‘shifts’ to a farm woman’s day: the first involves off-farm paid employment; the second shift includes child care and housework combined; and participation in farming operations such as managing and maintain the family farm comprises a third shift. Whilst women are more likely than men to move away from home for off-farm work, men have reported that staying behind to
work the farm increases their sense of not being able to escape and they feel the loss of companionship (Alston & Kent, 2004; Sartore et al., 2008). Although off-farm work was often initially undertaken to reduce financial stress, a comparison of farm spouses \( (n=1067) \) in seven states of the USA found that families with women engaging in this survival strategy fared worse on several dimensions of family and economic functioning when compared with families that remain living and working on the farm together, including higher debt loads, longer hours in all production, less satisfaction with marital relationships, and lower levels of life satisfaction amongst husbands (Godwin, Draughn, Little & Marlowe, 1991). Increased workload responsibilities and role conflicts for farm men and farm women may in turn contribute to higher levels of Anxiety and emotional distress in farm women and increased Depression in farm men.

3.4.3.1 Job Demand-Control (-Support) Model

The Job Demand-Control (-Support) (JDC(-S)) model (see Figure 4 below) is a theoretical framework aimed at describing the relationship between occupational stress and psychological distress. According to the strain hypothesis those workers in ‘high strain’ jobs, that is, workers with high (psychological) job demands in combination with low control (or decision latitude) are more likely to experience the most adverse psychological reactions, such as fatigue, Anxiety, Depression, and physical illness (Sanne, Mykletun, Dahl, Moen & Tell, 2005).
Figure 4. The Job Demand-Control(-Support) Model

The JDC(-S) model also recognises the importance of social support in buffering the negative effects of high strain, particularly when this support increases the control level above a certain threshold for the individual (known as the buffer hypothesis). The iso-strain hypothesis expands on these original hypotheses, by predicting that jobs characterised by high strain in combination with low support or social isolation have the most negative outcomes, such as Depression, Anxiety and stress (Sanne et al., 2005). Indeed, research has found that high demand, low support and low control, particularly when combined, are risk factors for both Depression and Anxiety.

In relation to farm men and farm women, the demands of the job are often high, as their work often becomes a 24 hour a day job. In addition to off-farm work that must be undertaken to ease economic pressures, there is the continual pressure to maintain their own farm. Consequently, farmers may perceive a low level of control over decisions as requirements surrounding crops or stock are often dependent on such factors such as climate and resource availability. Farm
men and women may also feel the effects of low support or social isolation as they may not be able to employ workers to assist them, or factors such as geographical isolation or a culture of stoicism makes them reluctant to seek assistance. Farm succession also places high demands on farm men and women: waiting for financial control and being expected to do the work (high demand) with little or no financial control (low control) is especially complicated in a relationship that spans both an emotional one (marriage and family) and an economic one (the farm business). These high demands and sense of low control, in combination with social isolation or low support, may lead to farm men and women becoming vulnerable to psychological distress.

3.5 Barriers to Help Seeking in Rural Areas

As previously mentioned, the increased incidence of health problems of rural residents may be problematic for a number of reasons including accessing health care services, not only because of the lack of availability of services within their community and large geographical distances between major regional centres, but also the perception of stigma associated with mental illness and a culture of stoicism.

3.5.1 Isolation

With regards to mental health, decreased population density in rural populations can create a sense of psychological as well as physical isolation, which also poses a significant barrier to early identification of, and intervention for mental health problems (Heflinger & Christens, 2006). Wilson (2007) found that within the rural northern New South Wales population, people with a mental
illness are only likely to enter services after a longer period of untreated psychosis than their urban counterparts, with the number of clients treated for first-episode psychosis seven-fold than expected. Many studies have also found that rather than seeking assistance when they recognise personal psychological distress, people in rural communities will tend to conceal their distress through means such as self-medicating with alcohol, and only access mental health services after a crisis occurs (Fraser et al., 2005; Philo, Parr & Burns, 2004; Wilson, 2007). Several other factors seem to be associated with decreased help-seeking behaviour. These include an absence of knowledge about mental illness, unavailability of providers and cost of care (Fox, Blank, Rovnyak & Barnett, 2001). These factors may further increase the sense of isolation in the event of psychological distress.

3.5.2 Stigma

The perceived stigma associated with mental illness can contribute to reluctance of members of rural communities to access mental health services. Stigma can be defined as the co-occurrence of its components; that is, labelling, separation, stereotyping, discrimination and status loss (Link & Phelan, 2001). However, power must be exercised for stigmatization to occur. Due to heightened visibility associated with small communities, it has been suggested that people fear being stigmatised if they use mental health services (Fraser et al., 2005). A largely qualitative study on communities in Scotland’s remote and rural Highlands found that those with mental health problems tended to feel stigmatised and hid their problems, often for many years, before seeking help, and then often tried to conceal formal service use (Philo et al., 2004). Another study on psychological distress in rural America (N = 1487) also found that the size of the town lived in
was negatively correlated with the depressive symptoms shown by men and the level of stigma towards mental health care: the smaller the town lived in, the higher the level of both depressive symptoms and stigma attached to mental health issues (Hoyt, Conger, Valde & Weihs, 1997). Within Australia, interviews with 22 key mental and general health professionals in rural South Australia found that mental health problems were associated with a high degree of stigma as people with mental health problems are allied with particular stereotypes and prejudices (Fuller, Edwards, Procter & Moss, 2000). In turn, this lead to an avoidance of mental health services, even when people recognised their distress.

3.5.3 Stoicism

There are also stereotypical cultural beliefs regarding the stoicism of Australian rural communities; that they endure times of adversity with patience and indifference, without asking for help, and just ‘get on with it’. Research by Fuller et al. (2000) found a culture of stoical self-reliance and mistrust of outsiders within rural South Australian communities, particularly of mental health workers, contributed to people only seeking ‘outside’ help in dire necessity. In his research on mental illness in rural New South Wales, Wilson (2007) also found that this culture of stoicism, particularly for males, increases the reluctance to seek assistance in times of need lest it be seen as a sign of weakness. Rural families do not wish to be viewed as anomalous by their neighbours and the combination of increased risk of mental health issues and decreased willingness to seek help creates an environment that is ripe for the development and exacerbation of mental health problems.
3.6 Summary

In summary, the physical and mental health of those living in rural and remote Australia is significantly worse than that of their major city counterparts in a number of areas, and is associated with higher levels of mortality from preventable diseases and higher suicide rates. Unfortunately, much of the research fails to differentiate between those who simply reside in rural and remote communities, and those who work the land. However, inferences can be made based on the rural data in conjunction with the research on farmers. The combination of Australia’s prolonged drought and climate change, increased economic pressures necessitating off-farm work, and the current barriers to help-seeking behaviours such as isolation, stigma and stoicism may increasingly jeopardise the health and wellbeing of many farm men and women.

If the HPA axis hypothesis previously discussed is correct, it would be expected that farm men and women, who may be enduring psychological distress related to the chronic nature of stressors associated with farm life, would also be experiencing detrimental physical health effects that could be measured via the metabolic syndrome factors. Thus, research is required to assess and review the current level of psychological functioning of Australian farm men and women as a distinctive cohort with unique challenges, and to determine if the prevalence of metabolic syndrome is significantly higher than that of the general population. However, from the research reviewed in Chapter 2, the relationship between metabolic syndrome and psychological distress is unclear. It has been suggested that psychological distress may be either a predictor or a consequence of
metabolic syndrome, or that there is no relationship between the two. This leads to the current study.
Chapter 4: The Current Study

4.1 Introduction

The prevalence of metabolic syndrome is continuing to rise globally and is considered to be a major risk factor in the development of Type II diabetes and CVD. The burden of disease for diabetes alone is currently estimated to be 286 billion International Dollars (ID) annually and is expected to risk to 396 billion ID by the year 2025. From a review of the research, it appears that the aetiology and pathogenesis of metabolic syndrome is an interaction between a number of factors including increased adipose fatty tissue, higher caloric diet, sedentary lifestyle, and a dysfunctional endocrine system possibly due to dysregulation of the HPA axis brought on by chronic stressors. Further, it is also unclear if gender differences in the prevalence and symptomatology of metabolic syndrome are associated with presentation and/or levels of psychological distress, or due to ethnicity, as suggested by previous research. Therefore, a greater understanding of the associations and interactions between Depression, Anxiety and Stress in the pathogenesis of metabolic syndrome is a research area worth pursuing.

Those living in rural and remote areas also have poorer physical health and health outcomes due to a number of environmental factors and barriers to health care and help-seeking behaviours. Due to the chronic nature of the stressors they endure, it could be expected that farm men and women would be experiencing higher levels of psychological distress than the general population. Consequently, if the HPA axis hypothesis is correct, it would also be expected that the prevalence of metabolic syndrome amongst this cohort would be higher than the general population and is perhaps related to the level of psychological distress.
they are experiencing. Therefore, studying the physical and mental health interactions can provide direction for future research, health promotion and disease prevention interventions for this particularly under researched, at risk population.

4.2 Aims of the Current Study

The current study was conducted in two phases, approximately one year apart. Study One was made up of a cross-sectional analysis from data collected at Time One. Study Two involved a longitudinal follow-up of participants in Study One.

4.2.1 Study One

The first study in the current research aimed to analyse baseline data on the prevalence of metabolic syndrome and psychological distress (Depression, Anxiety and Stress) amongst farm men and women. These data were then compared with existing prevalence data from the general population. Based on the evidence of poorer health outcomes in rural areas, it was hypothesised that the prevalence of metabolic syndrome amongst farm men and women would be higher than that of the general population. Furthermore, based on the research findings related to unique environmental stressors, it was hypothesised that levels of Depression, Anxiety and Stress would be higher amongst farm men and women when compared with the general population norms. Based on previous research findings and the HPA axis dysregulation hypothesis, it was hypothesised that elevated levels of Depression and Stress would be significantly associated with metabolic syndrome, but that Anxiety would not.
These proposed associations were further investigated at the level of the individual IDF criteria of metabolic syndrome (central obesity, raised triglycerides, reduced HDL cholesterol, raised blood pressure and raised fasting plasma glucose). Based on previous research findings it was hypothesised that waist circumference and elevated levels of triglycerides would be significantly associated with Depression, but that no factors would be significantly associated with Anxiety or Stress.

Based on previous research findings suggesting those with metabolic syndrome are more likely to have moderate to severe Depression, it was also hypothesised that there would be a significant association between Depression severity classification and the presence of metabolic syndrome.

Due to conflicting evidence surrounding gender differences, no hypotheses relating to gender differences in metabolic syndrome are proposed. However, the study will allow this issue to be investigated.

4.2.2 Study Two

The second study in the current research aimed to monitor both metabolic syndrome and psychological distress status over the course of approximately one year, and to assess the relationships between these variables over time. Due to conflicting evidence around the direction of these relationships, the current study aimed to investigate and clarify these relationships in three ways, with competing hypotheses being tested.
Firstly, metabolic syndrome as a predictor of psychological distress was investigated. Based on previous research findings, it was hypothesised that those participants with metabolic syndrome at Time One would display increased Depression scores at Time Two, when compared to those without metabolic syndrome. Due to the inconsistency of the research findings, no predictions are made in relation to gender differences.

Secondly, psychological distress as a predictor of metabolic syndrome was explored. Given the association between Depression and metabolic syndrome in the longitudinal studies of Raikkonen et al. (2007), Vanhala et al. (2009) and Viinamaki et al. (2009), in which it was suggested that Depression may be a predictor of metabolic syndrome, it was hypothesised that higher levels of Depression at Time One would be associated with a positive metabolic syndrome status at Time Two. Furthermore, based on previous research findings and the HPA Axis hypothesis on the effects of chronic Stress, it was hypothesised that higher levels of Stress at Time One would be associated with a positive metabolic syndrome status at Time Two. Due to the inconsistency of research findings, no predictions were made in relation to gender.

Finally, the continuity of participants’ metabolic syndrome over time and possible relationships between Depression, Anxiety and Stress were also explored both prospectively and retrospectively. It was hypothesised that the maintenance or development of a positive metabolic syndrome status over time would be associated with higher levels of Depression at Time One, as previous research has suggested that Depression may be predictor of metabolic syndrome (Raikkonen et
al., 2007; Vanhala et al., 2009; Viinamaki et al., 2009). Furthermore, based on the longitudinal research by Akbaraly et al. (2009), Dunbar et al. (2008) and Vanhala et al. (2009), it was hypothesised that those who maintained or developed metabolic syndrome would have higher levels of Depression at Time Two due to the cumulative effect of metabolic syndrome factors on Depression. Due to the unavailability of previous research findings, no predictions were made in relation to Anxiety or Stress.
Chapter 5: Method

5.1 Setting

Data were obtained from the Sustainable Farm Families (SFF) program, a health education and health promotion program specifically designed for, and delivered to, Australian farm men and farm women. Participants are recruited via agricultural industry groups, are aged over 18 years, and have worked on a farm for a minimum of five years (Brumby, Martin & Willder, 2006). It is unknown how many people were approached to participate as this was undertaken by Department of Agriculture and included newspaper advertisements, information at local agricultural shows and approaching people associated with local groups such as herd or pasture improvement groups. Therefore, response rates are unknown and unable to be documented due to the nature of these means. The program is delivered via a structured workshop each year for three consecutive years (two-day in the first year, one-day in the subsequent years) and is designed to increase farm family’s health knowledge and skills, whilst supporting them to change their lifestyle and reduce risk behaviours (Sustainable Farm Families Program, 2011). It is facilitated by Registered Nurses (Division One) who also collect biological samples and other data for both individual and research purposes (see Appendices A and B). Nurses delivering the program attended training facilitated by SFF program coordinators, to ensure both sample and data collection adhered to protocol, and that delivery of the program was standardised across sites.

Baseline data (Time One) were collected from 21 different workshop locations between July 2009 and August 2010, dependent on when the program was run within the community. Follow-up data (Time Two) were then collected at the
same locations between July 2010 and August 2011. Individuals were assigned a code by the nurse delivering the program that could not be used to identify the individual, but would allow data for each individual to be collated over time. Data was then collated centrally by the National Centre for Farmer Health Data Manager in Hamilton, Victoria and data entered twice to ensure accuracy. De-identified SFF data was provided for the current study from the National Centre of Farmer Health via an SPSS database.

5.2 Participants

Participants in the current study were 368 farm men and farm women from across the following regions in the state of Victoria, Australia: Barwon South Western (n=94, 25.5%), Gippsland (n=40, 10.9%), Grampians (n=114, 31%), Hume (n=18, 4.9%) and Loddon Mallee (n=102, 27.7%). Figure 5 (below) provides a pictorial representation of these rural regions (Department of Health Victoria, 2011).

Figure 5. Department of Health, Victoria - Rural Regions
5.2.1 Demographics

Demographic information was obtained from data collected from all participants at commencement of the SFF program and included age, sex, country of birth and Indigenous/non Indigenous status (see Appendix A). Participant ages ranged between 22 and 79 years, with a mean age of 51.5 years. Of the participants, 54.1% ($n = 199$) were male and 45.9% ($n = 169$) female.

Australia was nominated as the country of birth by 91% of participants ($n = 335$). Of the remaining nine per cent, 2.2% did not respond to the question and the remaining 6.8% were born in the following countries: England ($n = 3$), New Zealand ($n = 8$), Canada ($n = 1$), USA ($n = 1$), Germany ($n = 2$), Netherlands ($n = 1$), Scotland ($n = 1$), UK ($n = 1$), Other ($n = 6$). Two participants identified their status as Aboriginal, one participant identified as both Aboriginal and Torres Strait Islander.

Complete medical data and psychological data were available for 357 participants at Time One (“Sample Time One”) and 234 participants at Time Two (“Sample Time Two”). Table 5 (below) provides details of the demographic characteristics of the total sample. The data was collected in various community locations, for example meeting rooms in Shire offices, where the programs were delivered. Due to the nature of participant recruitment and lack of available data, it is uncertain if the SFF participants are representative of the farming communities.
5.3 Materials

5.3.1 Short Form of the Depression Anxiety and Stress Scale (DASS21)

The Short Form of the Depression, Anxiety and Stress Scale (DASS21) (Lovibond & Lovibond, 1995) was used to assess Depression, Anxiety and Stress (see Appendix G). This 21 item self-report measure contains three seven-item subscales that assess the core symptoms of these constructs. Respondents use a 4-point Likert scale to respond to items such as [over the last two weeks] “I couldn’t seem to experience any positive feeling at all” (Depression), “I was aware of dryness of my mouth” (Anxiety) and “I found it hard to wind down” (Stress). Responses range from 0 (did not apply to me) to 3 (applied to me very much or most of the time). For research and clinical purposes, the obtained scores from these subscales can be multiplied by two and compared to scores designed to characterise the degrees of severity relative to the population. Table 6 below outlines the guidelines for DASS severity ratings.
Table 5.

**Demographic Characteristics of Participants**

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<th>Total sample (n=368)</th>
<th>Sample Time One (n=357)</th>
<th>Sample Time Two (n=234)</th>
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<td>51.5 (11.8)</td>
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<td><strong>Gender (%)</strong></td>
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<td>164 (45.9)</td>
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<td><strong>Region of Victoria (%)</strong></td>
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<td>101 (28.3)</td>
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</tbody>
</table>
Crawford et al. (2011) provide normative data for an Australian general adult population sample and report reliabilities of 0.90, 0.79 and 0.89 for the Depression, Anxiety and Stress scales respectively. In the current study, the baseline Cronbach alpha coefficient for the DASS21 Depression, Anxiety and Stress subscales were 0.85, 0.70 and 0.83 respectively. A small number of studies have investigated other psychometric properties of this version of the DASS. In clinical studies Antony, Bieling, Cox, Enns and Swinson (1998) replicated the factor structure using exploratory factor analysis, but using confirmatory factor analysis, Clara, Cox and Enns (2001) were unable to find a three-factor model that met all the fit criteria. Daza, Novy, Stanley and Averill (2002) reported similar difficulties in a study of an Hispanic sample with Anxiety disorders with a Spanish version of the DASS-21, despite finding support for a three factor model and sound internal reliabilities and discriminant and convergent validity. However, when they re-analysed the non-clinical data reported in their 2003 study using only the 21 items of the DASS-21 rather than the 42 items of the DASS, Henry and Crawford’s (2005) confirmatory factor analysis revealed that the model...
with optimal fit had a quadripartite structure, which included a general factor of psychological distress in addition to the Depression, Anxiety, and stress factors. The internal reliability for the Depression subscale was 0.88, for the Anxiety subscale 0.82, and for the stress subscale 0.93. Although they did not report all of their findings related to the convergent and discriminant validity of the DASS-21, Henry and Crawford (2005) stated that their investigation indicated that, as for the full version of the DASS, the DASS-21 shows evidence good convergent and discriminant validity when compared with HADS and the Positive and Negative Affect Schedule (PANAS). They concluded that the purported DASS-21 subscales are valid, but that each of them also assesses a more general dimension of psychological distress or negative affect.

5.3.2 Health Conditions and Behaviours

Information about participants’ current health conditions and health behaviours was collected using the Victorian Department of Human Service Coordination Tools (Appendices E and F). These tools have their basis in health promotion literature and practice reviews, and incorporate key consumer information including medical, physical and social data that have been determined as useful in the determination of risk factors, further assessment and referrals.

5.3.3 Metabolic Syndrome Factors

A range of health data and samples are collected by registered nurses on the first morning of the SFF program in each consecutive year (see Appendix B). Participants are required to fast for a minimum of ten hours before attending the first day of each workshop. Under ethical guidelines, health information and
biometric measurements are collected in a private and confidential manner. Measurements were collected and assessed as per guidelines of the Australian Government National Health and Medical Research Council (NHMRC) (NHMRC, 2011), including those variables associated with metabolic syndrome, such as fasting cholesterol and blood glucose, weight for height, waist-to-hip ratio, blood pressure and pulse. Blood pressure is measured twice, with both readings and an average of the two readings recorded. The average reading for both Systolic and Diastolic was used in analysis. In data analysis, those participants who were previously diagnosed with either hypertension and/or Type II diabetes were identified as meeting the IDF metabolic syndrome criteria as a previous diagnosis by this definition equates to meeting the criteria.

5.4 Procedure

Ethics approval for the SFF program was obtained from the South West Health Care Ethics Committee (see Annex H).

Prior to the commencement of the first workshop, participants were mailed a package of documentation that included a Plain Language Statement (Appendix C) and a form consenting to the use of the data obtained from them to be used for research purposes (Appendix D). This package also included questionnaires recording demographic and health information (Appendices A, E, F). Participants were also asked to complete the DASS21 (Appendix G). All forms were returned prior to the workshop or handed to the program facilitator at the commencement of the workshop during the health assessment.
The program commenced with a two-day workshop, followed by a second one-day workshop approximately 12 months later and a further one-day workshop one year after that. Participants were required to fast for a minimum of ten hours before attending the first day of each workshop so that physical health assessments could be conducted on the first morning of each workshop. Assessment and collation of physical and psychosocial data and biometric measurements was undertaken in a private and confidential environment. The full SFF Physical Assessment Form can be viewed in Appendix B. Those participants who were identified as having results greater than acceptable levels as defined by NHMRC standards were referred for relevant further assessment or intervention.
Chapter 6: Results – Study One

6.1 Demographics

Of a total of 368 participants in the SFF program at Time One, eleven were excluded from analyses due to missing metabolic syndrome factor and/or DASS data. The age distribution of the remaining participants is summarised in Table 7 below.

Initial analyses assessed Time One gender differences in participants’ age and country of birth. There was no significant gender difference in mean age ($t(355) = 0.537, p = 0.59$).

Table 7.

*Age Distribution at Time One (N=357)*

<table>
<thead>
<tr>
<th>Age Group (Years)</th>
<th>$n$</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-24</td>
<td>3</td>
<td>0.8</td>
</tr>
<tr>
<td>25-34</td>
<td>35</td>
<td>9.8</td>
</tr>
<tr>
<td>35-44</td>
<td>63</td>
<td>17.6</td>
</tr>
<tr>
<td>45-54</td>
<td>100</td>
<td>28.0</td>
</tr>
<tr>
<td>55-64</td>
<td>110</td>
<td>30.8</td>
</tr>
<tr>
<td>65+</td>
<td>46</td>
<td>12.9</td>
</tr>
</tbody>
</table>
6.2 Health Conditions and Behaviours

Of those participants identified with existing health conditions at the commencement of the SFF program, nine had been previously diagnosed with Type II diabetes and 15 had been previously diagnosed with hypertension. As a previous diagnosis or specific treatment for raised triglycerides, reduced (HDL) cholesterol, raised blood pressure and raised fasting plasma glucose are to be considered when defining metabolic syndrome, these participants were controlled for in the analyses by manually coding them to reflect meeting these individual criteria of metabolic syndrome.

Participants reported their overall health in general to be ‘fair’ \((n = 34)\), ‘good’ \((n = 136)\), ‘very good’ \((n = 157)\) or ‘excellent’ \((n = 30)\). No participants reported their health as ‘poor’. When self-reporting how much their health interfered with their normal activities during the past four weeks, 63\% of participants \((n = 225)\) reported ‘not at all’, 26.1\% of participants \((n = 93)\) reported ‘slightly’, 6.2\% \((n = 22)\) reported ‘moderately’ and 4.5\% \((n = 16)\) reported ‘quite a bit’. There were no significant gender differences in the reporting of either overall health \((\chi^2(3, 357) = 1.94, p = 0.59)\), or interference with normal activities \((\chi^2(3, 357) = 1.33, p = 0.72)\).

Seventy-five per cent of participants \((n = 250)\) reported accumulating 30 minutes or more of moderate intensity physical activity on most days of the week. However, significant gender differences were found, with males \((n = 151, 78.2\%)\) more likely than females \((n = 99, 60.4\%)\) to report ‘yes’ to achieving this level of activity \((\chi^2(1, 357) = 13.5, p < 0.001)\).
6.3 Metabolic Syndrome Factors

In accordance with the IDF metabolic syndrome criteria, each participant was assessed to determine if they met the cut-off values for central obesity (defined as waist circumference), raised triglycerides, reduced HDL cholesterol, raised blood pressure and raised fasting plasma glucose (as previously described in detail in Chapter 1). The percentages of participants meeting the values for individual metabolic syndrome factors are presented in Table 8 below.

6.3.1 Metabolic Syndrome Factors and Gender

A Chi-square test for independence (with Yates Continuity Correction) indicated a significant association between gender and the categorical variable of meeting the IDF waist circumference cut-off or not, $\chi^2(1, 357) = 6.60, p = 0.007$, $\phi = 0.14$. A significantly higher proportion of females (78.7%) met the central obesity criteria for metabolic syndrome than males (65.8%). There was also an association between gender and the categorical variable of raised blood pressure, $\chi^2(1, 357) = 9.35, p = 0.002$, $\phi = -0.17$, with a significantly higher proportion of males (69.9%) meeting the IDF criteria than females (53.7%). There were no significant gender differences in the categorical variables of raised triglycerides, $(\chi^2(1, 357) = 2.46, p = 0.15, \phi = 0.08)$, reduced HDL cholesterol $(\chi^2(1, 357) = 2.33, p = 0.10, \phi = 0.09)$ or raised fasting plasma glucose $(\chi^2(1, 357) = 0.19, p = 0.67, \phi = -0.03)$. Table 8 below also includes Chi-square tests and significance level for gender differences in these criteria.
Table 8.

*Frequency of Participants who met Individual IDF Metabolic Syndrome Factor Criteria and Gender Differences (N=357)*

<table>
<thead>
<tr>
<th>Criteria</th>
<th>All Participants</th>
<th>Male (n=193)</th>
<th>Female (n=164)</th>
<th>Chi square</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>256</td>
<td>71.7</td>
<td>127</td>
<td>65.8</td>
<td>129</td>
</tr>
<tr>
<td>Raised triglycerides</td>
<td>72</td>
<td>20.2</td>
<td>33</td>
<td>17.1</td>
<td>39</td>
</tr>
<tr>
<td>Reduced HDL cholesterol</td>
<td>164</td>
<td>45.9</td>
<td>81</td>
<td>42.0</td>
<td>83</td>
</tr>
<tr>
<td>Raised blood pressure</td>
<td>223</td>
<td>62.5</td>
<td>135</td>
<td>69.9</td>
<td>88</td>
</tr>
<tr>
<td>Raised fasting plasma glucose</td>
<td>136</td>
<td>38.1</td>
<td>76</td>
<td>39.4</td>
<td>60</td>
</tr>
</tbody>
</table>

**6.4 Prevalence of Metabolic Syndrome**

At the commencement of the SFF program (Time One), 139 (38.9%) of the 357 participants were identified as meeting the IDF criteria for metabolic syndrome; that is, central obesity (as measured by waist circumference) plus at least two of the four remaining factors. A two independent proportion z-test found that the prevalence rate in the current study was significantly greater than that found by Cameron et al. (2007) in the Australian population (n = 3453, N = 11,247), $z = 3.31$, $p = <0.001$, two-tailed.

Amongst participants in the current study, 35.8% (n = 69) of males and 42.7% (n = 70) of females met the criteria for metabolic syndrome. A Chi-square test for independence (with Yates Continuity Correction) indicated no significant
association between gender and metabolic syndrome ($\chi^2(1, 357) = 1.51, p = 0.22, phi = 0.7$).

Using results from previous research by Janus et al. (2007) on metabolic syndrome among rural men and women (without age adjustment), a two independent proportion z-test found that the prevalence rate amongst females was significantly greater in the current study than that found by Janus et al. (2007), in which IDF criteria prevalence 30.1% amongst females ($n = 127, N = 423$), $z = 2.914, p = 0.004$, two-tailed. A two independent proportion z-test found no significant difference between the prevalence rate amongst males in the current study and that of Janus et al. (2007), ($n = 129, N = 383$), $z = 0.49, p = 0.62$, two-tailed.

The association between metabolic syndrome and age group was also assessed. Participants were allocated to an age group according to their age at Time One. Table 9 below presents the prevalence data of metabolic syndrome by age group. A Chi-square test for independence indicated a significant association between metabolic syndrome status and age group ($\chi^2(5, 357) = 23.45, p < 0.001, phi = 0.26$). Results indicate that the prevalence of metabolic syndrome increased progressively with age.
Table 9.

*Prevalence of Metabolic Syndrome by Age Group Time One*

<table>
<thead>
<tr>
<th>Age Group (Years)</th>
<th>No. of participants ($N = 357$)</th>
<th>Prevalence ($n = 137$)</th>
<th>Prevalence within Age Group</th>
<th>% of Total Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-34</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>25-34</td>
<td>35</td>
<td>6</td>
<td>17.1</td>
<td>1.7</td>
</tr>
<tr>
<td>35-44</td>
<td>63</td>
<td>17</td>
<td>27.0</td>
<td>4.8</td>
</tr>
<tr>
<td>45-54</td>
<td>100</td>
<td>39</td>
<td>39.0</td>
<td>10.9</td>
</tr>
<tr>
<td>55-64</td>
<td>110</td>
<td>49</td>
<td>44.5</td>
<td>13.7</td>
</tr>
<tr>
<td>65+</td>
<td>46</td>
<td>28</td>
<td>60.9</td>
<td>7.8</td>
</tr>
</tbody>
</table>

These findings are consistent with those of previous research by Cameron et al. (2007) and Janus et al. (2007). Figure 6 (below) displays a comparison of previous research findings with the current results.

Figure 6. *Metabolic Syndrome Prevalence Data Comparison at Time One (%)*
As central obesity is central to the IDF definition of metabolic syndrome, all 139 participants who were identified as meeting the criteria for metabolic syndrome diagnosis met this criteria. Amongst the other criteria, 86.3% of participants met criteria for raised blood pressure, 66.5% met the criteria for raised fasting plasma glucose, 58.3% met the criteria for reduced HDL cholesterol, and 34.5% met the criteria for raised triglycerides. No gender differences were found between males and females with metabolic syndrome amongst these criteria. Table 10 below outlines the frequency and percentages of metabolic syndrome criteria met by those with metabolic syndrome by gender.

Table 10.

*Frequency of Factors for Participants with Metabolic Syndrome by Gender (n=139)*

<table>
<thead>
<tr>
<th>IDF Metabolic Syndrome criteria</th>
<th>Male (n=69)</th>
<th>Female (n=70)</th>
<th>Chi square</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td>square</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>69</td>
<td>100</td>
<td>70</td>
<td>100</td>
</tr>
<tr>
<td>Raised triglycerides</td>
<td>20</td>
<td>29</td>
<td>28</td>
<td>40</td>
</tr>
<tr>
<td>Reduced HDL cholesterol</td>
<td>35</td>
<td>50.7</td>
<td>46</td>
<td>65.7</td>
</tr>
<tr>
<td>Raised blood pressure</td>
<td>63</td>
<td>91.3</td>
<td>57</td>
<td>81.4</td>
</tr>
<tr>
<td>Raised fasting plasma glucose</td>
<td>48</td>
<td>69.6</td>
<td>44</td>
<td>62.9</td>
</tr>
</tbody>
</table>
6.5 Depression, Anxiety and Stress

The DASS21 yields separate scores for Depression, Anxiety, and Stress. Obtained scores are then multiplied by two to provide a means of comparing with subscale clinical cut-off scores provided for the full 42-item DASS (Lovibond & Lovibond, 1995). Table 11 (below) summarises the range of scores, means and standard deviations for each of the constructs for participants at Time One by gender.

6.5.1 Gender, Age Group and DASS Scores

Independent-samples t-tests were conducted to compare the Depression, Anxiety and Stress scores for males and females. There was a significant difference in Stress scores, with females ($M = 8.78, SD = 6.69$) scoring higher than males ($M = 7.32, SD = 6.33, t (355) = -2.12, p = 0.03$, two-tailed). The magnitude of the differences in the means (mean difference = -.95, 95% CI: -2.82 to -0.10) was small (eta squared = .01). There was no significant difference between the Depression scores for males ($M = 3.99, SD = 5.23$) and females ($M = 4.94, SD = 5.62, t (355) = -1.65, p = 0.10$, two-tailed). Likewise, there was no significant difference between Anxiety scores for males ($M = 2.55, SD = 4.05$) and females ($M = 3.27, SD = 4.04, t (355) = -1.69, p = 0.09$, two-tailed). Table 11 (below) summarises these analyses. According to Stevens (1996), when the sample size is large (e.g. 100 or more participants), power is ‘not an issue’. Therefore, it is unlikely that non-significant gender differences were due to sample size.
Table 11.

**DASS Subscale Range, Mean and Standard Deviations by Gender**

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Male (n = 193)</th>
<th>Female (n = 164)</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Depression</td>
<td>0-28</td>
<td>3.99</td>
<td>5.23</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0-30</td>
<td>2.55</td>
<td>4.05</td>
</tr>
<tr>
<td>Stress</td>
<td>0-28</td>
<td>7.32</td>
<td>6.33</td>
</tr>
</tbody>
</table>

It was hypothesised that levels of Depression, Anxiety and Stress would be higher amongst farm men and women when compared with the general population norms. However, when compared with DASS results from an Australian general adult population sample (Crawford et al., 2011), the means and standard deviations were marginally lower for Depression ($M = 5.02, SD = 7.54$), Anxiety ($M = 3.36, SD = 5.07$) and Stress ($M = 8.10, SD = 8.40$). A further review of the article was undertaken and it was found that Crawford et al., (2011) did not use age-adjustment in their results and therefore, the comparison appears valid.

One-way between-groups analyses of variance were conducted to explore the relationship between participant age group and DASS subscale scores. Participants were allocated into an age group according to their age at Time One (see Table 7). The models were not significant for Depression ($F(5, 356) = 1.92, p = 0.09$), Anxiety ($F(5, 356) = 0.16, p = 0.98$), or Stress ($F(5, 356) = 1.27, p = 0.28$). Table 12 below summarises these results.
Table 12.

DASS Subscale Range, Mean, Standard Deviation by Age Group (N = 357).

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Range</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-24</td>
<td>3</td>
<td>2-20</td>
<td>8.67</td>
<td>9.87</td>
<td>0-4</td>
<td>2.00</td>
<td>2.00</td>
<td>2-16</td>
<td>6.67</td>
<td>8.08</td>
</tr>
<tr>
<td>25-34</td>
<td>35</td>
<td>0-22</td>
<td>5.67</td>
<td>5.03</td>
<td>0-10</td>
<td>2.51</td>
<td>2.58</td>
<td>0-22</td>
<td>9.71</td>
<td>6.78</td>
</tr>
<tr>
<td>35-44</td>
<td>63</td>
<td>0-24</td>
<td>3.40</td>
<td>4.48</td>
<td>0-14</td>
<td>2.83</td>
<td>3.29</td>
<td>0-24</td>
<td>8.06</td>
<td>6.00</td>
</tr>
<tr>
<td>45-54</td>
<td>100</td>
<td>0-28</td>
<td>5.14</td>
<td>6.05</td>
<td>0-28</td>
<td>3.12</td>
<td>4.39</td>
<td>0-28</td>
<td>8.41</td>
<td>7.13</td>
</tr>
<tr>
<td>55-64</td>
<td>110</td>
<td>0-28</td>
<td>4.31</td>
<td>5.63</td>
<td>0-30</td>
<td>2.86</td>
<td>4.58</td>
<td>0-28</td>
<td>7.79</td>
<td>6.25</td>
</tr>
<tr>
<td>65+</td>
<td>46</td>
<td>0-18</td>
<td>3.35</td>
<td>4.30</td>
<td>0-20</td>
<td>2.83</td>
<td>4.08</td>
<td>0-20</td>
<td>6.26</td>
<td>6.15</td>
</tr>
</tbody>
</table>

6.6 Metabolic Syndrome and DASS Subscale Scores

Three independent-samples t-tests were conducted to compare the Depression, Anxiety and Stress scores for those with metabolic syndrome and those without. A summary of these results can be found in Table 13 below. There was a significant difference between the Anxiety scores of those with metabolic syndrome (M = 3.44, SD = 4.66) and those without (M = 2.53, SD = 3.58; t (239.81) = -1.96, p = 0.05, two-tailed), with those meeting metabolic syndrome diagnostic criteria scoring higher. The magnitude of the differences in the means (mean difference = -0.91, 95% CI: -1.83 to 0.003) was small (eta squared = .01). There was no significant difference between the Depression scores of those with metabolic syndrome (M = 4.52, SD = 6.0) and those without (M = 4.37, SD =
5.04; \( t(256.7) = -0.25, p = 0.81, \) two-tailed). Likewise, there was no significant difference in the Stress scores for those with metabolic syndrome (\( M = 8.22, SD = 6.73 \)) and those without (\( M = 7.85, SD = 6.41; \ t(355) = -0.52, p = 0.61, \) two-tailed).

Table 13.

*Metabolic Syndrome Status and DASS Subscale Scores.*

<table>
<thead>
<tr>
<th>Subscale</th>
<th>With Metabolic Syndrome (( n = 139 ))</th>
<th>Without Metabolic Syndrome (( n = 218 ))</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>4.52, SD 6.01</td>
<td>4.37, SD 5.04</td>
<td>-0.25 0.81</td>
</tr>
<tr>
<td>Anxiety</td>
<td>3.44, SD 4.66</td>
<td>2.53, SD 3.58</td>
<td>-1.96 0.05</td>
</tr>
<tr>
<td>Stress</td>
<td>8.22, SD 6.73</td>
<td>7.85, SD 6.41</td>
<td>-0.52 0.61</td>
</tr>
</tbody>
</table>

Three further independent-samples t-tests were conducted to compare the DASS subscale scores for males and females with metabolic syndrome. There was no significant differences found between Depression, Anxiety or Stress for males and females. Table 14 displays these results.
Table 14.

*DASS Subscale Scores for those with Metabolic Syndrome by Gender.*

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Males (n = 69)</th>
<th>Females (n = 70)</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Depression</td>
<td>4.20</td>
<td>6.29</td>
<td>4.83</td>
</tr>
<tr>
<td>Anxiety</td>
<td>3.99</td>
<td>5.03</td>
<td>3.49</td>
</tr>
<tr>
<td>Stress</td>
<td>8.14</td>
<td>6.97</td>
<td>8.29</td>
</tr>
</tbody>
</table>

6.7. Relationship Between DASS Subscale Scores and Metabolic Syndrome Factors

The HPA Axis hypothesis posits that cortisol levels, as regulated by the HPA axis, play an important role in lipid and glucose metabolism, with evidence of prolonged elevated levels leading to Type II diabetes, a redistribution of body fat (as characterised by central adiposity) and hypertension (Rosemond & Bjorntorp, 2000). Psychological characteristics such as anger, hostility, Depression and Anxiety also influence the risk of developing CVD and diabetes (Goldbacher & Matthews, 2007; Weber-Hamann et al., 2002). Therefore, it could be expected that all of the metabolic syndrome factors would be significantly related to levels of Depression, Stress and Anxiety.

In line with this, previous studies by Akbaraly et al. (2009), Dunbar et al. (2008), Miettola et al. (2008), Muhntz et al. (2009), Richter et al. (2009), Skilton et al. (2007), Vanhala et al. (2008), and Vogelzangs et al. (2007) have found relationships between various metabolic syndrome factors and Depression.
Therefore, correlation and multiple regression analyses were conducted to examine the relationship between the each of the Depression, Anxiety and Stress scores and the individual factors of metabolic syndrome criteria for both the total sample \( (N = 357) \) and the sample with metabolic syndrome \( (n = 139) \). The metabolic syndrome factor of raised blood pressure is separated into Systolic blood pressure (Systolic BP) and Diastolic blood pressure (Diastolic BP) as these are two separate physiological readings that are required to be separated for the purpose of analysis (as continuous variables). Tests for outliers, normality, linearity, homoscedasticity and independence of residuals indicated no violations of assumptions for each of the analyses.

6.7.1 Depression Subscale

Correlation and multiple regression analyses were conducted to examine the relationship between Depression and metabolic syndrome factors for both the total sample and for the sample with metabolic syndrome. Table 15 displays the correlations between the variables for the total sample. Within the total sample, waist circumference and triglycerides are positively and significantly correlated with Depression. Raised fasting blood glucose is also positively, but not significantly, correlated with Depression. Reduced (HDL) cholesterol and both Systolic and Diastolic blood pressure are negatively, but not significantly correlated with Depression. These results indicate that, within the total sample, those participants experiencing higher levels of Depression tend to have a larger waist circumference and higher triglyceride levels.
Table 15.

*Pearson Correlations between Depression Scores and Metabolic Syndrome Factors for the Total Sample.*

<table>
<thead>
<tr>
<th>Factor</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Depression Score</td>
<td>-0.15**</td>
<td>0.09*</td>
<td>-0.03</td>
<td>-0.04</td>
<td>0.03</td>
<td>0.07</td>
<td></td>
</tr>
<tr>
<td>2. Waist Circumference</td>
<td>-0.09*</td>
<td>0.09*</td>
<td>-0.29**</td>
<td>0.30**</td>
<td>0.34**</td>
<td>0.22**</td>
<td></td>
</tr>
<tr>
<td>3. Triglycerides</td>
<td>-0.00</td>
<td>0.00</td>
<td>0.08</td>
<td>0.12*</td>
<td>0.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. HDL Cholesterol</td>
<td>-0.08</td>
<td>-0.08</td>
<td>-0.10*</td>
<td>-0.06</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Systolic Blood Pressure</td>
<td>-0.70**</td>
<td>0.70**</td>
<td>0.15*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Diastolic Blood Pressure</td>
<td>-0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Blood Glucose</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* *p < 0.05  ** p < 0.01

The multiple regression model for the total sample, with all six predictors, produced $R^2 = 0.043$, $F(6, 350) = 2.59$, $p = 0.018$, explaining only 4.3% of the variance in Depression scores. Table 16 below summarises the descriptive statistics and analysis results by subscale. As can been seen in Table 16, waist circumference had a significant positive regression weight (1.9% of the variance, $sr^2 = 0.019$, $p = 0.008$), whilst Systolic blood pressure had a significant negative regression weight (1.1% of the variance, $sr^2 = -0.011$, $p = 0.04$), indicating that participants with higher waist circumference and lower Systolic blood pressure were more likely to have higher levels of Depression. Triglycerides, HDL cholesterol, Diastolic blood pressure and blood glucose did not contribute significantly to Depression scores within the total sample.
Table 16.

**Summary Statistics, Correlations and Results from the Depression Subscale Regression Analysis for the Total Sample**

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
<th>Correlation with subscale</th>
<th>Multiple regression weights</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression (total sample)</td>
<td>Depression</td>
<td>4.43</td>
<td>5.43</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Waist</td>
<td>94.77</td>
<td>12.30</td>
<td>0.15**</td>
<td>0.16**</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>1.28</td>
<td>1.06</td>
<td>0.09*</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>HDL Cholesterol</td>
<td>1.14</td>
<td>0.42</td>
<td>-0.03</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>132.72</td>
<td>16.78</td>
<td>-0.04</td>
<td>-0.15*</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP</td>
<td>87.46</td>
<td>9.83</td>
<td>-0.03</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>Blood Glucose</td>
<td>5.40</td>
<td>0.78</td>
<td>0.07</td>
<td>0.05</td>
</tr>
</tbody>
</table>

* $p < 0.05$ ** $p < 0.01$

Correlation and multiple regression analyses were then conducted to examine the relationship between Depression and metabolic syndrome factors in the sample with metabolic syndrome. Table 17 below displays the correlations between variables for the metabolic syndrome sample.
Table 17.

Pearson Correlations between Depression Scores and Metabolic Syndrome Factors for Sample with Metabolic Syndrome.

<table>
<thead>
<tr>
<th>Factor</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Depression Score</td>
<td>-</td>
<td>0.25**</td>
<td>0.11</td>
<td>-0.00</td>
<td>-0.07</td>
<td>0.07</td>
<td>0.17*</td>
</tr>
<tr>
<td>2. Waist Circumference</td>
<td>-</td>
<td>0.05</td>
<td>-0.34**</td>
<td>0.08</td>
<td>0.24*</td>
<td>0.15*</td>
<td></td>
</tr>
<tr>
<td>3. Triglycerides</td>
<td>-</td>
<td>-0.06</td>
<td>-0.05</td>
<td>-0.02</td>
<td>-0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. HDL Cholesterol</td>
<td>-</td>
<td>-0.07</td>
<td>-0.04</td>
<td>-0.18*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Systolic Blood Pressure</td>
<td>-</td>
<td>0.60**</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Diastolic Blood Pressure</td>
<td>-</td>
<td>0.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Blood Glucose</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* $p < 0.05$  ** $p < 0.01$

Unlike the total sample, which indicated that participants with higher waist circumference and lower Systolic blood pressure were more likely to have higher levels of Depression, amongst the sample of participants with metabolic syndrome, waist circumference and fasting blood glucose were positively and significantly correlated with Depression scores. Triglycerides and Diastolic blood pressure were also positively, but not significantly, correlated with Depression. Reduced (HDL) cholesterol and Systolic blood pressure were negatively, but not significantly correlated with Depression.

The multiple regression model for the metabolic syndrome sample, with all six predictors, produced $R^2 = 0.121$, $F(6, 132) = 3.03$, $p = 0.008$, explaining 12.1% of the variance in the model; almost three times more than that of the total sample (4.3%). As can be seen in Table 18, waist circumference (4.7% of the variance,
$sr^2 = 0.047$, $p = 0.008$) and fasting plasma glucose (2.8% of the variance, $sr^2 = 0.03$, $p = 0.04$) had significant positive regression weights, indicating that, amongst participants with metabolic syndrome, those who have higher waist circumference and blood glucose levels were likely to have higher levels of Depression. Triglycerides, HDL cholesterol, Systolic and Diastolic blood pressure did not contribute to Depression amongst those with metabolic syndrome.

Table 18.

Summary Statistics, Correlations and Results from the Depression Subscale Regression Analysis for the Metabolic Syndrome Sample

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>Correlation with subscale</th>
<th>Multiple regression weights</th>
<th>$sr^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Depression</td>
<td>4.52</td>
<td>6.01</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Metabolic Syndrome Sample)</td>
<td>Waist</td>
<td>101.56</td>
<td>11.28</td>
<td>0.25**</td>
<td>0.24**</td>
<td>0.13</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>1.59</td>
<td>1.18</td>
<td>0.11</td>
<td>0.11</td>
<td>0.55</td>
</tr>
<tr>
<td></td>
<td>HDL Cholesterol</td>
<td>1.20</td>
<td>0.43</td>
<td>&lt;0.01</td>
<td>-0.11</td>
<td>1.53</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>139.99</td>
<td>16.72</td>
<td>-0.07</td>
<td>-0.16</td>
<td>-0.06</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP</td>
<td>87.98</td>
<td>9.21</td>
<td>0.07</td>
<td>0.12</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>Blood Glucose</td>
<td>5.76</td>
<td>0.88</td>
<td>0.17*</td>
<td>0.18*</td>
<td>1.20</td>
</tr>
</tbody>
</table>

* $p < 0.05$  ** $p < 0.01$
6.7.2 Anxiety Subscale

Correlation and multiple regression analyses were also conducted to examine
the relationship between Anxiety scores and the individual factors of metabolic
syndrome criteria for both the total sample and also for the sample with metabolic
syndrome. Table 19 displays the correlations between the variables for the total
sample.

Within the total sample, waist circumference, Systolic and Diastolic blood
pressure and blood glucose were positively and significantly correlated with
Anxiety. Triglycerides were also positively, but not significantly, correlated with
Anxiety. Reduced (HDL) cholesterol was negatively, but not significantly
correlated with Anxiety in the total sample.

Table 19.

Pearson Correlations between Anxiety Scores and Metabolic Syndrome Factors
for the Total Sample

<table>
<thead>
<tr>
<th>Factor</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Anxiety Score</td>
<td>-</td>
<td>0.17**</td>
<td>0.05</td>
<td>-0.07</td>
<td>0.13*</td>
<td>0.14*</td>
<td>0.12*</td>
</tr>
<tr>
<td>2. Waist Circumference</td>
<td>-</td>
<td>0.09*</td>
<td>-0.29**</td>
<td>0.30**</td>
<td>0.34**</td>
<td>0.22**</td>
<td></td>
</tr>
<tr>
<td>3. Triglycerides</td>
<td>-</td>
<td>0.00</td>
<td>0.08</td>
<td>0.12*</td>
<td>0.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. HDL Cholesterol</td>
<td>-</td>
<td>-0.08</td>
<td>-0.10*</td>
<td>-0.06</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Systolic Blood Pressure</td>
<td>-</td>
<td>0.70**</td>
<td>0.15*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Diastolic Blood Pressure</td>
<td>-</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Blood Glucose</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < 0.05   ** p < 0.01
The multiple regression model for the total sample, with all six predictors, produced $R^2 = 0.048$, $F(6, 350) = 2.96$, $p = 0.008$, explaining only 4.8% of the variance in the model. As can been seen in Table 20, waist circumference accounted for a significant proportion of variance in Anxiety scores (1.4% of the variance, $sr^2 = 0.014$, $p = 0.025$), indicating that participants with higher waist circumference had higher levels of Anxiety. Triglycerides, HDL cholesterol, Systolic and Diastolic blood pressure and blood glucose did not contribute to Anxiety within the total sample.

Table 20.

Summary Statistics, Correlations and Results from the Anxiety Subscale Regression Analysis for the Total Sample

<table>
<thead>
<tr>
<th>Subscale (total sample)</th>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
<th>Correlation with subscale</th>
<th>Multiple regression weights $b$</th>
<th>$\beta$</th>
<th>$sr^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety</td>
<td>Anxiety</td>
<td>2.88</td>
<td>4.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Waist</td>
<td>94.77</td>
<td>12.3</td>
<td>0.17**</td>
<td>0.13**</td>
<td>0.04</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>1.28</td>
<td>1.06</td>
<td>0.46</td>
<td>0.02</td>
<td>0.06</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td>HDL Cholesterol</td>
<td>1.14</td>
<td>0.42</td>
<td>&lt;-0.01</td>
<td>0.05</td>
<td>0.46</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>132.72</td>
<td>16.7</td>
<td>0.13*</td>
<td>0.02</td>
<td>&lt;0.01</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP</td>
<td>87.46</td>
<td>9.83</td>
<td>0.14*</td>
<td>0.08</td>
<td>0.03</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td>Blood Glucose</td>
<td>5.40</td>
<td>0.78</td>
<td>0.17</td>
<td>0.43</td>
<td>0.08</td>
<td>0.00</td>
</tr>
</tbody>
</table>

* $p < 0.05$  ** $p < 0.01$
Correlation and multiple regression analyses were then conducted to examine the relationship between Anxiety and metabolic syndrome factors in the sample with metabolic syndrome. Table 21 below displays the correlations between variables for the metabolic syndrome sample.

Table 21.

_Pearson Correlations between Anxiety Scores and Metabolic Syndrome Factors for Sample with Metabolic Syndrome_

<table>
<thead>
<tr>
<th>Factor</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Anxiety Score</td>
<td>-</td>
<td>0.25*</td>
<td>-0.01</td>
<td>0.03</td>
<td>0.16*</td>
<td>0.13</td>
<td>0.17*</td>
</tr>
<tr>
<td>2. Waist Circumference</td>
<td>-</td>
<td></td>
<td>0.05</td>
<td>-0.34**</td>
<td>0.08</td>
<td>0.24*</td>
<td>0.15*</td>
</tr>
<tr>
<td>3. Triglycerides</td>
<td>0.05</td>
<td>-0.06</td>
<td>-0.05</td>
<td>-0.02</td>
<td>-0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. HDL Cholesterol</td>
<td>-</td>
<td>-0.07</td>
<td>-0.04</td>
<td>-0.18*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Systolic Blood Pressure</td>
<td>-</td>
<td>0.60**</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Diastolic Blood Pressure</td>
<td>-</td>
<td>0.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Blood Glucose</td>
<td>0.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < 0.05  ** p < 0.01

Similar to the total sample, in which waist circumference, Systolic and Diastolic blood pressure and blood glucose were positively and significantly correlated with Anxiety, amongst the sample of participants with metabolic syndrome, waist circumference, Systolic blood pressure and fasting blood glucose were positively and significantly correlated with Anxiety. Reduced (HDL) cholesterol and Diastolic blood pressure were also positively, but not significantly, correlated with Anxiety. Triglyceride levels are negatively, but not significantly correlated with Anxiety.
The multiple regression model for the metabolic syndrome sample, with all six predictors, produced $R^2 = 0.114$, $F(6, 132) = 2.83$, $p = 0.013$, explaining 11.4% of the variance in Anxiety scores; almost two and a half times more than that of the total sample (4.8%). As can be seen in Table 22 (below), waist circumference (5% of the variance, $sr^2 = 0.05$, $p = 0.005$) was the only factor to account for a significant proportion of the variance, indicating that, amongst participants with metabolic syndrome, those who have a higher waist circumference are more likely to experience higher levels of Anxiety. Triglycerides, HDL cholesterol, Systolic and Diastolic blood pressure and blood glucose levels did not contribute to Anxiety for those with metabolic syndrome.

Table 22.

Summary Statistics, Correlations and Results from the Anxiety Subscale Regression Analysis for the Metabolic Syndrome Sample

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
<th>Correlation with subscale</th>
<th>Multiple regression weights</th>
<th>$sr^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety (Metabolic Syndrome Sample)</td>
<td>Anxiety</td>
<td>3.44</td>
<td>4.66</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waist</td>
<td>101.56</td>
<td>11.2</td>
<td></td>
<td>0.25**</td>
<td>0.26**</td>
<td>0.11</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.59</td>
<td>1.18</td>
<td></td>
<td>-0.01</td>
<td>&lt;0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>1.20</td>
<td>0.43</td>
<td></td>
<td>&lt;0.01</td>
<td>0.13</td>
<td>1.38</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>139.99</td>
<td>16.7</td>
<td></td>
<td>0.16</td>
<td>0.04</td>
<td>0.15</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>87.98</td>
<td>9.21</td>
<td></td>
<td>0.13</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Blood Glucose</td>
<td>5.76</td>
<td>0.88</td>
<td></td>
<td>0.17*</td>
<td>0.15</td>
<td>0.82</td>
</tr>
</tbody>
</table>

* $p < 0.05$  ** $p < 0.01$
6.7.3. Stress Subscale

Correlation and multiple regression analyses were also conducted to examine the relationship between Stress subscale scores and the individual factors of metabolic syndrome criteria for both the total sample and also for the sample with metabolic syndrome. Table 23 displays the correlations between the variables for the total sample.

Within the total sample, there were no significant correlations found between the individual factors and Stress. Waist circumference, triglycerides and blood glucose levels were positively, but not significantly, correlated with Stress. Reduced (HDL) cholesterol and both Systolic and Diastolic blood pressure were negatively, but not significantly correlated with Stress in the total sample.

Table 23.

*Pearson Correlations between Stress Scores and Metabolic Syndrome Factors for the Total Sample.*

<table>
<thead>
<tr>
<th>Factor</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Stress Score</td>
<td>-0.04</td>
<td>0.05</td>
<td>-0.00</td>
<td>-0.08</td>
<td>-0.02</td>
<td>0.07</td>
<td></td>
</tr>
<tr>
<td>2. Waist Circumference</td>
<td>-</td>
<td>0.09*</td>
<td>-0.29**</td>
<td>0.30**</td>
<td>0.34**</td>
<td>0.22**</td>
<td></td>
</tr>
<tr>
<td>3. Triglycerides</td>
<td>-</td>
<td>0.00</td>
<td>0.08</td>
<td>0.12*</td>
<td>0.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. HDL Cholesterol</td>
<td>-</td>
<td>-0.08</td>
<td>-0.10*</td>
<td>-0.06</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Systolic Blood Pressure</td>
<td>-</td>
<td>0.70**</td>
<td>0.15*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Diastolic Blood Pressure</td>
<td>-</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Blood Glucose</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < 0.05   ** p < 0.01
The multiple regression model for the total sample, with all six predictors, produced $R^2 = 0.021$, $F(6, 350) = 1.25$, $p = 0.28$, explaining only 2.1% of the variance in the Stress scores. As can been seen in Table 24 (below), Systolic blood pressure contributed significant unique variance (1.1% of the variance, $sr^2 = -0.107$, $p = 0.04$), indicating that participants with lower Systolic blood pressure were likely to have higher levels of Stress. This result is counter-intuitive to the medical literature and may be due to biased self-reporting of stress, or the suppression effect of another unknown variable. Waist circumference, triglycerides, HDL cholesterol, Diastolic blood pressure and blood glucose did not contribute to Stress scores for the total sample.

Table 24.

Summary Statistics, Correlations and Results from the Stress Subscale Regression Analysis for the Total Sample

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
<th>Correlation with subscale</th>
<th>Multiple regression weights</th>
<th>$sr^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress (total sample)</td>
<td>Stress</td>
<td>7.99</td>
<td>6.53</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Waist</td>
<td>94.77</td>
<td>12.30</td>
<td>0.04</td>
<td>0.05</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>Triglycerides</td>
<td>1.28</td>
<td>1.06</td>
<td>0.05</td>
<td>0.05</td>
<td>0.30</td>
</tr>
<tr>
<td></td>
<td>HDL Cholesterol</td>
<td>1.14</td>
<td>0.42</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>0.19</td>
</tr>
<tr>
<td></td>
<td>Systolic BP</td>
<td>132.72</td>
<td>16.78</td>
<td>-0.07</td>
<td>-0.15*</td>
<td>-0.06</td>
</tr>
<tr>
<td></td>
<td>Diastolic BP</td>
<td>87.46</td>
<td>9.83</td>
<td>-0.02</td>
<td>0.07</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>Blood Glucose</td>
<td>5.40</td>
<td>0.78</td>
<td>0.07</td>
<td>0.08</td>
<td>0.65</td>
</tr>
</tbody>
</table>

* $p < 0.05$
Correlation and multiple regression analyses were then conducted on the metabolic syndrome sample to examine the relationship between Stress and individual factors of metabolic syndrome. Table 25 displays the correlations between variables for those participants with metabolic syndrome.

Table 25.

Pearson Correlations between Stress Scores and Metabolic Syndrome Factors for Sample with Metabolic Syndrome

<table>
<thead>
<tr>
<th>Factor</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Stress Score</td>
<td>-</td>
<td>0.10</td>
<td>-0.01</td>
<td>0.01</td>
<td>-0.08</td>
<td>0.02</td>
<td>0.06</td>
</tr>
<tr>
<td>2. Waist Circumference</td>
<td>-</td>
<td>0.05</td>
<td>-0.34**</td>
<td>0.08</td>
<td>0.24*</td>
<td>0.15*</td>
<td></td>
</tr>
<tr>
<td>3. Triglycerides</td>
<td>-</td>
<td>-0.06</td>
<td>-0.05</td>
<td>-0.02</td>
<td>-0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. HDL Cholesterol</td>
<td>-</td>
<td>-0.07</td>
<td>-0.04</td>
<td>-0.18*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Systolic Blood Pressure</td>
<td>-</td>
<td>0.60**</td>
<td>0.03</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Diastolic Blood Pressure</td>
<td>-</td>
<td>0.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Blood Glucose</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < 0.05   ** p < 0.01

As with the total sample, there were no significant correlations within the sample of participants with metabolic syndrome between Stress and any of the metabolic syndrome factors. Waist circumference, reduced (HDL) cholesterol, Diastolic blood pressure and fasting blood glucose were positively, but not significantly, correlated with Stress. Triglycerides and Systolic blood pressure were negatively, but not significantly, correlated with Stress.
The multiple regression model for the metabolic syndrome sample, with all six predictors, produced $R^2 = 0.027$, $F(6, 132) = 0.60$, $p = 0.73$, explaining only 2.7% of the variance in the model. This is a very similar result to that of the total sample (2.1%). As can be seen in Table 26 (below), metabolic syndrome factors did not contribute to Stress.

Table 26.

*Summary Statistics, Correlations and Results from the Stress Subscale Regression Analysis for Metabolic Syndrome Sample*

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
<th>Correlation with subscale</th>
<th>Multiple regression weights</th>
<th>$sr^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress (Metabolic Syndrome Sample)</td>
<td>Stress</td>
<td>8.22</td>
<td>6.73</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Metabolic Syndrome Sample)</td>
<td>Waist</td>
<td>101.56</td>
<td>11.28</td>
<td>0.10</td>
<td>0.09</td>
<td>0.06</td>
</tr>
<tr>
<td>(Metabolic Syndrome Sample)</td>
<td>Triglycerides</td>
<td>1.59</td>
<td>1.18</td>
<td>-0.01</td>
<td>-0.02</td>
<td>-0.09</td>
</tr>
<tr>
<td>(Metabolic Syndrome Sample)</td>
<td>HDL Cholesterol</td>
<td>1.20</td>
<td>0.43</td>
<td>0.01</td>
<td>0.05</td>
<td>0.69</td>
</tr>
<tr>
<td>(Metabolic Syndrome Sample)</td>
<td>Systolic BP</td>
<td>139.99</td>
<td>16.72</td>
<td>-0.08</td>
<td>-0.14</td>
<td>-0.06</td>
</tr>
<tr>
<td>(Metabolic Syndrome Sample)</td>
<td>Diastolic BP</td>
<td>87.98</td>
<td>9.21</td>
<td>0.02</td>
<td>0.09</td>
<td>0.07</td>
</tr>
<tr>
<td>(Metabolic Syndrome Sample)</td>
<td>Blood Glucose</td>
<td>5.76</td>
<td>0.88</td>
<td>0.06</td>
<td>0.06</td>
<td>0.47</td>
</tr>
</tbody>
</table>

* $p < 0.05$
6.8 Metabolic Syndrome and DASS Severity

As previous research by Richter et al. (2007) has found a relationship between the severity of Depression and metabolic syndrome status, analyses were conducted to explore any relationships. Mann-Whitney U Tests found that there was no significant difference in the Depression, Anxiety or Stress severity ratings between those who had metabolic syndrome and those who did not. Table 27 summarises the descriptive statistics and analysis results for the above.

Table 27.

*Differences between those with Metabolic Syndrome and those without on DASS Subscale Severity Ratings*

<table>
<thead>
<tr>
<th>Subscale</th>
<th>With Metabolic Syndrome (n = 139)</th>
<th>Without Metabolic Syndrome (n = 218)</th>
<th>U</th>
<th>z</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression Severity</td>
<td>2</td>
<td>2</td>
<td>14382</td>
<td>-1.21</td>
<td>0.23</td>
</tr>
<tr>
<td>Anxiety Severity</td>
<td>2</td>
<td>2</td>
<td>14173</td>
<td>-1.88</td>
<td>0.60</td>
</tr>
<tr>
<td>Stress Severity</td>
<td>8</td>
<td>6</td>
<td>14056</td>
<td>-1.46</td>
<td>0.14</td>
</tr>
</tbody>
</table>

6.9 Summary of Study One Results

Results from this study show that in relation to metabolic syndrome factors, a significantly higher proportion of females met the IDF central obesity criteria than males, whilst a significantly higher proportion of males met the IDF blood pressure level criteria. The prevalence of metabolic syndrome in the current study
was significantly greater than previously reported in the Australian population, as hypothesised. When compared to previous research results from rural Australian men and women, a significantly higher prevalence of metabolic syndrome was found amongst females but not males. Within the whole sample females reported higher levels of Stress than males, however there were no significant differences between genders on DASS subscale scores for participants with metabolic syndrome. Larger waist circumference accounted for significant amount of variance in Depression and Anxiety for both the total sample and for those with metabolic syndrome. Raised fasting plasma glucose was also significantly related to Depression in the metabolic syndrome sub-sample.

In summary, it appears that those with metabolic syndrome have higher levels of Anxiety than those who do not, and that both waist circumference and raised fasting plasma glucose are related to Depression in those with metabolic syndrome. These findings present a mixed picture of the relationship between metabolic syndrome and Depression and Anxiety and may be due to issues surrounding psychological self-report measures, particularly within a cohort of farm men and women for reasons discussed earlier. However, following the participants over time, as has been done in Study Two, and also analysing the relationships between the continuity of an individual’s metabolic syndrome status and Depression and Anxiety scores, may assist in providing a better understanding of relationships between metabolic syndrome and Depression, Anxiety and Stress.
Chapter 7: Results – Study Two

The HPA axis hypothesis posits that there is a relationship between psychological distress and metabolic syndrome. However, due to a lack of longitudinal research, it is unclear as to whether Depression, Anxiety and/or Stress will influence metabolic syndrome status over time, or if metabolic syndrome status influences levels of Depression, Anxiety and/or Stress over time. As Sustainable Farm Families Program participants are health screened on an annual basis for three years as a part of the program, this regime provides an ideal opportunity to determine the direction of the relationship between Depression, Stress and Anxiety and metabolic syndrome.

7.1 Participants

Of the total sample from Time One of 357 participants, 250 (70%) returned at Time Two to undertake the program, thirteen participants (3.6%) returned paperwork but did not attend the program, and 94 participants (26.3%) did not return paperwork or participate. Of the 250 participants who did return, 15 cases were removed from the analyses due to incomplete data and a further case was removed due to pregnancy interfering with valid data (i.e. waist measurement), leaving a total of 234 cases with valid data. Demographic information for this reduced sample is provided in Table 5 (Chapter 5) for those 234 cases identified as “Sample Time Two”.

A Mann-Whitney U Test revealed no significant difference at Time One between those who returned to the program and those who did not on self-reported overall general health ($U = 13194, z = -0.22, p = 0.83$), and self-reported
health interference with normal activities during the past four weeks ($U = 12186, \ z = -1.40, \ p = 0.16$). A Mann-Whitney U Test also revealed there were no significant differences between those who returned at Time Two and those who did not, on Time One data relating to Weight ($U = 13018, \ z = -0.4, \ p = 0.69$), waist measurement ($U = 13365, \ z = -0.01, \ p = 0.99$), Blood Glucose Level ($U = 12839, \ z = -0.60, \ p = 0.55$), HDL Cholesterol level ($U = 11923, \ z = -1.63, \ p = 0.10$), Triglyceride level ($U = 12117, \ z = -1.43, \ p = 0.15$), Systolic blood pressure ($U = 12439, \ z = -1.05, \ p = 0.29$) and Diastolic blood pressure ($U = 13320, \ z = -0.06, \ p = 0.95$). A Chi-square test for independence (with Yates Continuity Correction) indicated no significant association between gender and attrition, $\chi^2(1, \ 357) = 1.02, \ p = 0.31, \ phi = 0.26$.

7.2 Demographics

The sample for analysis at Time Two ($n = 234$) had a mean age of 52.3 years ($SD = 11.8$) and ranged between 24 and 80 years. There was no significant gender difference in mean age ($t(232) = 0.91, \ p = 0.24$).

7.3 Metabolic Syndrome

At Time Two, 103 (44%) of the total 234 participants (43.2% of males, 45.1% of females) were identified as meeting the criteria for metabolic syndrome (versus 38.9% in Study One). A two independent proportion z-test found that the prevalence rate in the current study was significantly greater than that found by Cameron et al. (2007) in the Australian population ($n = 3453, \ N = 11,247$), $z = 4.36, \ p < 0.001$, two-tailed.
Of those who met the criteria for metabolic syndrome in the current study, 55.3% were male, and 44.7% were female. A Chi-square test for independence (with Yates Continuity Correction) indicated that this gender difference was not significant ($\chi^2(1, n=234) = 0.03, p = 0.87, \phi = 0.02$). At Time One these figures were 49.6% and 50.4% for males and females respectively. Using results from previous research by Janus et al. (2007) on metabolic syndrome among rural men and women, a two independent proportion z-test found that prevalence rates were significantly greater in the current study than those found by Janus et al. (2007) among males ($z = 1.96, p = 0.05$, two-tailed) and females ($z = 2.91, p = 0.003$, two-tailed).

7.3.1 Metabolic Syndrome and Age

The association between metabolic syndrome and age group was also assessed. Participants were allocated into an age group based on their age at Time Two. Table 28 below presents the prevalence of those with metabolic syndrome by age group. Results indicate that the prevalence of metabolic syndrome progressively increases with age.
Table 28.

*Prevalence of Metabolic Syndrome by Age Group at Time Two*

<table>
<thead>
<tr>
<th>Age Group (Years)</th>
<th>No. of participants (N=234)</th>
<th>Prevalence (n=103)</th>
<th>Prevalence within Age Group %</th>
<th>% of Total Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-34</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>25-34</td>
<td>23</td>
<td>8</td>
<td>34.8</td>
<td>3.4</td>
</tr>
<tr>
<td>35-44</td>
<td>38</td>
<td>11</td>
<td>28.9</td>
<td>4.7</td>
</tr>
<tr>
<td>45-54</td>
<td>61</td>
<td>25</td>
<td>41.0</td>
<td>10.7</td>
</tr>
<tr>
<td>55-64</td>
<td>78</td>
<td>39</td>
<td>50.0</td>
<td>16.7</td>
</tr>
<tr>
<td>65+</td>
<td>33</td>
<td>20</td>
<td>60.6</td>
<td>8.5</td>
</tr>
</tbody>
</table>

The above results are similar in trend to those found in Time One, as well as those of previous research discussed in Chapter 1. A greater proportion of participants in the 25-34 year old age group had metabolic syndrome in the Time Two sample, however cannot be judged as significant as the number of participants is too small. Figure 7 below is a visual comparison of the prevalence results of both Times One and Two of the current study and those of Cameron et al. (2007) and Janus et al. (2007), however it should be noted that the number of participants between Times One and Two in the current study varied greatly.
7.4 Depression, Anxiety and Stress Scale Scores

A summary of the range of scores, means, and standard deviations for each of the mood constructs for participants at Times One and Two by gender, can be seen in Table 29 below.

An independent-samples t-test was conducted to compare the Depression, Anxiety and Stress scores for males and females at Time Two. There was a significant difference in Depression scores for males \((M = 3.68, SD = 3.84)\) and females \((M = 5.22, SD = 6.22; t(158.54) = -2.19, p = 0.03, \text{two-tailed})\), with females scoring higher than males. The magnitude of the differences in the means (mean difference = -1.53, 95%CI: -2.92 to -0.15) was small (eta squared = 0.02).

There was also a significant difference in Stress scores for males \((M = 6.55, SD = 6.08)\) and females \((M = 8.84, SD = 6.87; t(232) = -2.71, p = 0.007, \text{two-tailed})\), with females again scoring higher than males. The magnitude of the differences in
the means (mean difference = -2.30, 95%CI: -3.97 to -0.63) was small (eta squared = 0.03). There was no significant gender difference in Anxiety scores.

These results were very different to those at Time One (N = 357) (see Table 29 below) in both the range of the scores, and gender differences, as at Time One only Stress was found to be significantly different between genders. Therefore, paired-samples (repeated) t-tests were conducted on the Time Two sample, by gender, to evaluate any differences in the means on DASS subscale scores between Times One and Two. No statistically significant differences were found for males. However, it was found that the females within the Time Two sample had a statistically significant decrease in Anxiety from Time One (M = 3.42, SD = 3.67) to Time Two (M = 2.53, SD = 3.47), t (101) = 2.69, p = 0.008 (two tailed). The mean decrease in Anxiety score was 0.89 with a 95% confidence interval ranging from 0.23 to 1.55. The Eta squared statistic (0.06) indicated a moderate effect size.

7.4.1 Metabolic Syndrome and DASS Scores

Independent-samples t-tests were conducted to compare the Depression, Anxiety and Stress scores at Time Two for those with metabolic syndrome and those without. In this cross-sectional analysis, those meeting metabolic syndrome diagnostic criteria scored higher on Depression (M = 5.42, SD = 6.35) than those without the syndrome (M = 3.51, SD = 3.56; t(151.47) = -2.73, p = 0.007, two-tailed). The magnitude of the differences in the means (mean difference = -1.91, 95%CI: -3.29 to -0.53) was small (Eta squared = 0.03). There was no significant difference in Anxiety scores for those with metabolic syndrome (M = 2.72, SD =
3.45) and those without (M = 2.18, SD = 2.92; t (199.50) = -0.34, p = 0.21, two-tailed). Likewise, where was no significant difference in Stress scores for those with metabolic syndrome (M = 8.10, SD = 7.33) and those without (M = 7.11, SD = 5.81; t (232) = -1.14, p = 0.25, two-tailed). These results were different from those of Time One, at which time those with metabolic syndrome scored higher on Anxiety.

Table 29.

Summary Statistics of DASS Subscale Scores at Times One and Two by Gender and Differences over Time (N = 234)

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Time One</th>
<th>Time Two</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>M</td>
</tr>
<tr>
<td><strong>Males</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>0-24</td>
<td>3.89</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0-12</td>
<td>2.32</td>
</tr>
<tr>
<td>Stress</td>
<td>0-28</td>
<td>7.07</td>
</tr>
<tr>
<td><strong>Females</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>0-28</td>
<td>4.96</td>
</tr>
<tr>
<td>Anxiety</td>
<td>0-20</td>
<td>3.42</td>
</tr>
<tr>
<td>Stress</td>
<td>0-26</td>
<td>9.10</td>
</tr>
</tbody>
</table>

7.5 Relationships between Metabolic Syndrome Status and DASS Scores Over Time

Analyses were then conducted to determine if a predictive relationship could be found between Time One metabolic syndrome status and Time Two DASS
subscale scores, or Time One DASS subscale scores and Time Two metabolic syndrome status.

7.5.1 Relationship between Time One Metabolic Syndrome Status and Time Two DASS Scores

Three separate correlations and hierarchical multiple regression analyses were conducted to examine the relationship between Time One metabolic syndrome status and Time Two DASS subscale scores, whilst controlling for the relevant Time One DASS subscale scores. The dependent variable in each analysis was Time Two Depression score, Anxiety score or Stress score. The independent variables were the corresponding Time One DASS subscale score and Time One metabolic syndrome status. Preliminary analyses were conducted to ensure no violation of the assumptions of normality, linearity, multicollinearity and homoscedasticity. Table 30 below displays the partial correlations. As to be expected, there were strong, positive partial correlations between Time One and Time Two Depression scores ($r = 0.54, p < 0.001$), Anxiety scores ($r = 0.57, p < 0.001$) and Stress scores ($r = 0.62, p < 0.001$). However, there were no significant correlations between either Time One or Time Two DASS subscale scores and Time One metabolic syndrome.

In the regression analysis for Depression, Time One Depression score was entered at Step One, explaining 29% of the variance in Time Two Depression. After entry of Time One metabolic syndrome status at Step Two, the total variance explained by the model as a whole was 29.1%, $F(2,231) = 47.47, p < 0.001$. Time One metabolic syndrome status explained less that 0.01% of the
Table 30.

*Partial Correlations between Time One Metabolic Syndrome Status and Times One and Two DASS Subscale Scores*

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Time One Depression</td>
<td>-</td>
<td>0.54</td>
<td>0.01</td>
</tr>
<tr>
<td>2. Time Two Depression</td>
<td>-</td>
<td>-</td>
<td>-0.02</td>
</tr>
<tr>
<td>3. Time One Metabolic Syndrome Status</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1. Time One Anxiety</td>
<td>-</td>
<td>0.57</td>
<td>0.08</td>
</tr>
<tr>
<td>2. Time Two Anxiety</td>
<td>-</td>
<td>-</td>
<td>0.01</td>
</tr>
<tr>
<td>3. Time One Metabolic Syndrome Status</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>1. Time One Stress</td>
<td>-</td>
<td>0.62</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>2. Time Two Stress</td>
<td>-</td>
<td>-</td>
<td>-0.03</td>
</tr>
<tr>
<td>3. Time One Metabolic Syndrome Status</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Variance in Depression scores at Time Two, after controlling for Time One Depression scores, $R$ squared change = 0.001, $F$ change (1, 231) = 0.25, $p = 0.62$. In the final model, Time One Depression was statistically significant, ($beta = 0.54, p < 0.001$) whilst Time One metabolic syndrome status was not ($beta = -0.03, p = 0.62$).
In the regression analysis for Anxiety, Time One Anxiety score was entered at Step One, explaining 32.4% of the variance in Time Two Anxiety. After entry of Time One metabolic syndrome status at Step Two, the total variance explained by the model as a whole was 32.5%, $F(2,231) = 55.52, p < 0.001$. Time One metabolic syndrome status explained less that 0.01% of the variance in Anxiety scores at Time Two, after controlling for Time One Anxiety scores, $R^2$ change = 0.001, $F$ change (1, 231) = 0.39, $p = 0.54$. In the final model, Time One Anxiety was statistically significant, ($\beta = 0.57, p < 0.001$) whilst Time One metabolic syndrome status was not ($\beta = -0.03, p = 0.54$).

In the regression analysis for Stress, Time One Stress score was entered at Step One, explaining 38.2% of the variance in Time Two Stress. After entry of Time One metabolic syndrome status at Step Two, the total variance explained by the model as a whole was 38.3%, $F(2,231) = 71.69, p < 0.001$. Time One metabolic syndrome status explained less that 0.01% of the variance in Stress scores at Time Two, after controlling for Time One Stress scores, $R^2$ change = 0.001, $F$ change (1, 231) = 0.29, $p = 0.59$. In the final model, Time One Stress was statistically significant, ($\beta = 0.62, p < 0.001$) whilst Time One metabolic syndrome status was not ($\beta = -0.03, p = 0.59$).

Overall, these results indicate that metabolic syndrome status at Time One did not make a unique contribution to Depression, Stress or Anxiety at Time Two, once the relevant Time One mood state had been accounted for. Thus, the causal relationship between mood and metabolic syndrome is not in this direction.
7.5.2 Relationship between Time One DASS Scores and Time Two Metabolic Syndrome Status

A one-way between-groups multivariate analysis of variance was performed to investigate whether Time Two metabolic syndrome status was associated with DASS subtest scores at Time One. Three dependent variables were used: Depression score, Anxiety score and Stress score. The independent variable was Time Two metabolic syndrome status. Preliminary assumption testing was conducted, to check for normality, linearity, univariate and multivariate outliers, homogeneity of variance-covariance matrices, and multicollinearity, with a violation of homogeneity of variance noted in Levene’s test of equality of error variances. As recommended by Howell (2007), although this was significant, an inspection of the variance for each of the groups found that the largest variance was no more than four times the smallest, and therefore the results were likely to be valid.

There was a statistically significant difference between those who had metabolic syndrome at Time Two and those who did not on the combined dependent variables, $F(3, 230) = 3.318, p = 0.021$; Wilks’ Lambda = 0.96; partial eta squared = 0.04. When the results for the dependent variables were considered separately, the two variables to reach statistical significance were Anxiety ($F(1, 232) = 7.21, p = 0.008$, partial Eta squared = 0.03) and Depression ($F(1,232) = 6.59, p = 0.01$, partial Eta squared = 0.27). An inspection of the mean scores indicated that those with metabolic syndrome at Time Two reported higher Time One levels of Anxiety ($M = 3.45$ vs 2.29) and Depression ($M = 5.32$ vs 3.60).
These results indicate that participants with metabolic syndrome at Time Two were more likely to have had higher levels of Depression and Anxiety at Time One, and suggest a causal contribution in this direction.

7.6 Continuity of Metabolic Syndrome Status and DASS scores

A perusal of the results at Time One when compared to those at Time Two indicates some noticeable differences in the relationship between metabolic syndrome status and Depression and Anxiety. At Time One, participants with metabolic syndrome were significantly higher on Anxiety only, whereas at Time Two, participants with metabolic syndrome were significantly higher on Depression. There was also a relationship found between metabolic syndrome status at Time Two and higher levels of Depression and Anxiety at Time One.

Given the above results, analyses were conducted to investigate how the DASS variables were associated with the continuity of participants’ metabolic syndrome status over time. The sample was divided into four groups according their metabolic syndrome status at both Time One and Time Two. Table 31 below outlines these groups and the number of participants who either maintained or changed their metabolic syndrome status over time. There is a limitation to using categorical variables as individuals who are on the borderline can easily tip either way, however in the treatment setting, practitioners would be using such categories to assist in determining metabolic syndrome status.
7.6.1 Time One DASS scores and Continuity of Metabolic Syndrome Status

A one-way between-groups multivariate analysis of variance was performed to investigate Time One DASS subtest scores in relation to the continuity of metabolic syndrome status. Three dependent variables were used: Depression score, Anxiety score and Stress score at Time One. The independent variable was continuity of metabolic syndrome status. Preliminary assumption testing was conducted to check for normality, linearity, univariate and multivariate outliers, homogeneity of variance-covariance matrices, and multicollinearity, with a violation of homogeneity of variance noted in Levene’s test of equality of error variances. As recommended by Howell (2007), although this was significant, an inspection of the variance for each of the groups found that the largest variance was no more than four times the smallest, and therefore the results were likely to be valid.

Table 31.

Continuity of Metabolic Syndrome Status over Time (N = 234)

<table>
<thead>
<tr>
<th>Metabolic Syndrome Status</th>
<th>Time Two Positive</th>
<th>Time Two Negative</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time One Positive</td>
<td>58</td>
<td>28</td>
<td>86</td>
</tr>
<tr>
<td>Time One Negative</td>
<td>45</td>
<td>103</td>
<td>148</td>
</tr>
<tr>
<td>Total</td>
<td>103</td>
<td>131</td>
<td>234</td>
</tr>
</tbody>
</table>

There was no statistically significant difference between metabolic syndrome status groups on the combined dependent variables of Depression, Stress and Anxiety at Time One, $F(9, 555) = 1.42, p = 0.18$; Wilks’ Lambda = 0.95; partial
Eta squared = 0.02. However, when the results for the dependent variables were considered separately, there was a statistically significant difference in Anxiety, $F(3, 555) = 3.01$, $p = 0.03$, partial Eta squared = 0.4. An inspection of the mean scores indicated that those participants with metabolic syndrome at both Time One and Two reported slightly higher levels of Anxiety at Time One ($M = 3.76$, $SD = 3.62$) than those who did not have metabolic syndrome at Time One but developed it by Time Two ($M = 3.04$, $SD = 4.08$); those who had metabolic syndrome at Time One, but not Time Two ($M = 1.86$, $SD = 2.61$); and those who did not have metabolic syndrome at either Time One or Time Two ($M = 2.41$, $SD = 2.76$). Therefore, those participants who maintained a positive metabolic syndrome status over time had higher levels of Anxiety at Time One when compared with the other three continuity of metabolic syndrome groups.

7.6.2 Continuity of Metabolic Syndrome Status and DASS scores at Time Two

A one-way between-groups analysis of variance was conducted to explore the relationship between continuity of metabolic syndrome and Depression at Time Two (see Table 32, Figure 8). There was a statistically significant effect: $F(3, 230) = 3.55$, $p = 0.02$. The effect size, calculated using eta squared, was 0.04 was small to medium. Post-hoc comparisons using the Tukey HSD test indicated that the mean score for those who had no metabolic syndrome at Time One, but developed metabolic syndrome at Time Two ($M = 6.09$, $SD = 6.84$) was significantly higher than those who had metabolic syndrome at Time One, and no metabolic syndrome at Time Two ($M = 2.79$, $SD = 3.19$), and those who did not have metabolic syndrome at either time point ($M = 3.71$, $SD = 3.65$).
To investigate whether group's scores changed differentially, a one-way analysis of covariance (ANCOVA) was then conducted to compare scores on the Depression subscale of the DASS21 at Time Two for the different metabolic syndrome status groups, whilst controlling for Depression subscale scores at Time One. A significant effect was found, $F (3, 230) = 3.84, p = 0.01$, partial $\eta^2$ 0.05).

Table 32.

Descriptive Statistics for Continuity of Metabolic Syndrome Status and Depression Scores

<table>
<thead>
<tr>
<th>Metabolic Syndrome Status</th>
<th>$n$</th>
<th>$M$</th>
<th>$SD$</th>
<th>$M$</th>
<th>$SD$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Syndrome positive at Times One and Two</td>
<td>58</td>
<td>5.28</td>
<td>6.41</td>
<td>4.90</td>
<td>5.95</td>
</tr>
<tr>
<td>No Metabolic Syndrome Time One, Metabolic Syndrome Time Two</td>
<td>45</td>
<td>5.38</td>
<td>6.44</td>
<td>6.09</td>
<td>6.83</td>
</tr>
<tr>
<td>Metabolic Syndrome Time One, No Metabolic Syndrome Time Two</td>
<td>28</td>
<td>2.71</td>
<td>3.90</td>
<td>2.79</td>
<td>3.19</td>
</tr>
<tr>
<td>No Metabolic Syndrome at Times One or Two</td>
<td>103</td>
<td>3.84</td>
<td>3.72</td>
<td>3.71</td>
<td>3.65</td>
</tr>
</tbody>
</table>

Post-Hoc comparisons using the Tukey HSD test indicated that, there was a significant difference in the changes in Depression scores of those who maintained their positive metabolic syndrome status over time and those who had metabolic syndrome at Time One then no metabolic syndrome at Time Two (mean difference = 2.34, $p = 0.02$, 95% CI 0.34 to 4.33). In looking at Table 32
and Figure 8 it appears that the change in those who maintained metabolic syndrome was toward becoming less depressed, whereas those whose metabolic syndrome resolved changed minimally.

![Figure 8. Continuity of Metabolic Syndrome Group and Mean Depression Scores at Times One and Two](image)

Similarly, after controlling for Time One Depression scores, those who did not have metabolic syndrome at Time One but developed it by Time Two had a greater change in Depression scores than those who had metabolic syndrome at Time One then no metabolic syndrome at Time Two (mean difference = 2.98, \( p = 0.005 \), 95% CI 0.90 to 5.07), and those who did not have metabolic syndrome at either time. The magnitude of the changes in the scores of the Time Two positive metabolic status groups was not different, although in opposite directions.

Similarly, an ANOVA was conducted to explore the relationship between metabolic syndrome status and Anxiety scores at Time Two (see Table 33, Figure
9 below). There was no statistically significant effect: $F(3, 230) = 0.62, p = 0.60$.

An ANCOVA controlling for Anxiety scores at Time One also found no significant effect, $F(3, 230) = 2.0, p = 0.12$; partial Eta squared = 0.025, and indicate that any changes in Anxiety score were consistent across groups.

Table 33.

Descriptive Statistics for Continuity of Metabolic Syndrome Status and Anxiety Scores

<table>
<thead>
<tr>
<th>Metabolic Syndrome Status</th>
<th>n</th>
<th>M</th>
<th>SD</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Syndrome positive at Times One and Two</td>
<td>58</td>
<td>3.76</td>
<td>3.62</td>
<td>2.72</td>
<td>3.49</td>
</tr>
<tr>
<td>No Metabolic Syndrome Time One, Metabolic Syndrome Time Two</td>
<td>45</td>
<td>3.04</td>
<td>4.08</td>
<td>2.71</td>
<td>3.44</td>
</tr>
<tr>
<td>Metabolic Syndrome Time One, No Metabolic Syndrome Time Two</td>
<td>28</td>
<td>1.86</td>
<td>2.61</td>
<td>1.93</td>
<td>2.34</td>
</tr>
<tr>
<td>No Metabolic Syndrome at Times One or Two</td>
<td>103</td>
<td>2.41</td>
<td>2.76</td>
<td>2.25</td>
<td>3.06</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Metabolic Syndrome Status</th>
<th>n</th>
<th>M</th>
<th>SD</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Syndrome positive at Times One and Two</td>
<td>58</td>
<td>3.76</td>
<td>3.62</td>
<td>2.72</td>
<td>3.49</td>
</tr>
<tr>
<td>No Metabolic Syndrome Time One, Metabolic Syndrome Time Two</td>
<td>45</td>
<td>3.04</td>
<td>4.08</td>
<td>2.71</td>
<td>3.44</td>
</tr>
<tr>
<td>Metabolic Syndrome Time One, No Metabolic Syndrome Time Two</td>
<td>28</td>
<td>1.86</td>
<td>2.61</td>
<td>1.93</td>
<td>2.34</td>
</tr>
<tr>
<td>No Metabolic Syndrome at Times One or Two</td>
<td>103</td>
<td>2.41</td>
<td>2.76</td>
<td>2.25</td>
<td>3.06</td>
</tr>
</tbody>
</table>

One or Two
Finally, an ANOVA was conducted to explore the relationship between metabolic syndrome status and Stress scores at Time Two (see Table 34, Figure 10 below). There was no statistically significant effect: $F(3, 230) = 0.69$, $p = 0.56$. An ANCOVA controlling for Stress scores at Time One also found no significant effect, $F(3, 230) = 0.77$, $p = 0.51$; partial Eta squared = 0.01, and indicate that any changes in Stress score were consistent across groups.
Table 34.  
*Descriptive Statistics for Continuity of Metabolic Syndrome Status and Stress Scores*

<table>
<thead>
<tr>
<th>Metabolic Syndrome Status</th>
<th>$n$</th>
<th>$M$</th>
<th>$SD$</th>
<th>$M$</th>
<th>$SD$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic Syndrome positive at Times One and Two</td>
<td>58</td>
<td>8.66</td>
<td>6.52</td>
<td>7.69</td>
<td>6.87</td>
</tr>
<tr>
<td>No Metabolic Syndrome Time One, Metabolic Syndrome Time Two</td>
<td>45</td>
<td>8.31</td>
<td>7.63</td>
<td>8.62</td>
<td>7.93</td>
</tr>
<tr>
<td>Metabolic Syndrome Time One, No Metabolic Syndrome Time Two</td>
<td>28</td>
<td>6.57</td>
<td>6.23</td>
<td>6.57</td>
<td>4.83</td>
</tr>
<tr>
<td>No Metabolic Syndrome at Times One or Two</td>
<td>103</td>
<td>7.78</td>
<td>5.70</td>
<td>7.26</td>
<td>6.06</td>
</tr>
</tbody>
</table>

Figure 10. Continuity of Metabolic Syndrome Group and Mean Stress Scores at Times One and Two
7.7 Summary of Study Two Results

Results from this study indicate that the prevalence of metabolic syndrome progressively increases with age, which supports both the findings at Time One and that of previous research.

Cross-sectional analyses at Time Two found that, in relation to DASS subscale scores and gender, females scored significantly higher on both Depression and Stress than males at Time Two, whilst the Anxiety levels of females had also significantly decreased from Time One to Time Two. Those participants with metabolic syndrome at Time Two scored higher on Depression at Time Two than those without metabolic syndrome.

Longitudinal analyses found that Time One metabolic syndrome status was not associated with Depression, Anxiety or Stress at Time Two, however higher levels of Anxiety and Depression at Time One were associated with a positive metabolic syndrome status at Time Two. Analyses of the continuity of participants’ metabolic syndrome over time found that the development of a positive metabolic syndrome status over time was associated with higher levels of Depression, and that those participants who maintained a positive metabolic syndrome status over time had higher levels of Anxiety at Time One than the other three groups.
Chapter 8: Discussion

This thesis was concerned with the relationship between metabolic syndrome and mental health. As discussed in the introductory chapters, metabolic syndrome is a clustering of metabolic, anthropometric and haemodynamic abnormalities that, when occurring together, significantly increase the risk of morbidity and mortality from Type II diabetes and CVD. It appears that the aetiology and pathogenesis of metabolic syndrome is an interaction between a number of factors including increased adipose fatty tissue, higher caloric diet, sedentary lifestyle and a dysfunctional endocrine system; possibly due to dysregulation of the HPA axis brought on by chronic stressors.

The differences in intervention results may be explained by the HPA axis pathology conceptualisation, and suggests that psychological factors may be placing additional stress on an endocrine system that is shared with the biological indicators of metabolic syndrome. A greater understanding of the interrelationship between psychological and biological risk factors in the aetiology and pathogenesis of metabolic syndrome may inform interventions, which are currently aimed at reducing only physical symptomatology.

The studies reported in this thesis were conducted with farm men and women, who generally have poorer physical health and health outcomes than their urban counterparts due to a number of environmental factors and barriers to health care and help seeking behaviours. Due to the chronic nature of the stressors they endure, it could be expected that farm men and women would also be experiencing higher levels of psychological distress than the general population.
Consequently, if the HPA axis hypothesis is correct, it would also be expected that the prevalence of metabolic syndrome amongst this cohort would be higher than the general population and possibly related to the level of psychological distress they are experiencing. Therefore, studying the physical and mental health interactions can provide direction for future research, health promotion and disease prevention interventions for this particularly under researched, at risk population.

Whilst it would be pragmatic to suggest a causal relationship between metabolic syndrome and mood disorders, the evidence at present suggests that, whilst there are associations between these factors, there is currently no consensus on direction or causation. However, awareness of symptoms of psychological distress could be important in the clinical management of metabolic syndrome, and vice versa.

At Time One, based on the evidence of poorer health outcomes in rural areas, it was hypothesised that the prevalence of metabolic syndrome amongst farm men and women would be higher than that of the general population. Furthermore, based on the research findings related to unique environmental stressors, it was hypothesised that levels of Depression, Anxiety and Stress would be higher amongst farm men and women when compared with the general population norms. Based on previous research findings and the HPA axis dysregulation hypothesis, it was hypothesised that elevated levels of Depression and Stress would be significantly associated with metabolic syndrome, but that Anxiety would not. Based on previous research findings it was hypothesised that waist
circumference and elevated levels of triglycerides would be significantly associated with Depression, but that no factors would be significantly associated with Anxiety or Stress. Based on previous research findings suggesting those with metabolic syndrome are more likely to have moderate to severe Depression, it was also hypothesised that there would be a significant association between Depression severity classification and the presence of metabolic syndrome. Due to conflicting evidence surrounding gender differences, no hypotheses relating to gender differences in metabolic syndrome were proposed.

At Time Two, based on previous research findings, it was hypothesised that those participants with metabolic syndrome at Time One would display increased Depression scores at Time Two, when compared to those without metabolic syndrome. Due to the inconsistency of the research findings, no predictions were made in relation to gender difference. It was also hypothesised that higher levels of Depression at Time One would be associated with a positive metabolic syndrome status at Time Two. Furthermore, based on previous research findings and the HPA Axis hypothesis on the effects of chronic Stress, it was hypothesised that higher levels of Stress at Time One would be associated with a positive metabolic syndrome status at Time Two. Due to the inconsistency of research findings, no predictions were made in relation to gender.

Finally, the continuity of participants’ metabolic syndrome over time and possible relationships between Depression, Anxiety and Stress were also explored both prospectively and retrospectively. It was hypothesised that the maintenance or development of a positive metabolic syndrome status over time would be
associated with higher levels of Depression at Time One. Furthermore, it was hypothesised that those who maintained or developed metabolic syndrome would have higher levels of Depression at Time Two due to the cumulative effect of metabolic syndrome factors on Depression. Due to the unavailability of previous research findings, no predictions were made in relation to Anxiety or Stress.

These hypotheses will be discussed in this chapter in relation to the analyses.

8.1 Mental Health of Participants

When comparing the baseline DASS results from the current study at Time One with results from an Australian general adult population sample (Crawford et al., 2011), the means and standard deviations were similar for Depression. In fact, the mean scores were marginally lower in the current study than those previously reported by Crawford et al. (2011) from the general population. This does not support the hypothesis that farm men and farm women experience elevated levels of Depression, Anxiety and Stress due to prolonged stressors.

Previous research on Australian farm men and farm women that has shown a higher incidence of psychological distress (Brumby et al., 2011) has used the Kessler 10 as the measure. The K10 is particularly designed to monitor population prevalence and trends and to identify community cases of non-specific psychological distress, whilst the DASS has been designed to measure the three related negative emotional states of Depression, Anxiety and Tension/Stress (Kessler et al., 2002; Lovibond & Lovibond, 1995). Other research has used such measures as the Hospital Anxiety and Depression Scale (HADS), which was
developed for, and validated on, medical patients and excludes many of the somatic symptoms of Depression that overlap with physical problems such as fatigue, sleep disturbance, psychomotor changes and loss of appetite and weight. Amongst the literature, this is one of the first studies to use the DASS21 specifically on farm men and farm women.

Lovibond and Lovibond (1995) also note that the DASS, like other self-report measures, is transparent and it is possible for respondents to disguise their symptoms. When the DASS has been used in previous research on regional and rural Australian communities, it has not distinguished between farmers and non-farmers. As discussed in Chapter 3, previous research by Thomas et al. (2003) has found that whilst farmers self-report a lower prevalence of psychiatric morbidity, they were more likely to report thinking that life is not worth living; a question that is not asked on the DASS21. A culture of stoicism and perceived stigma associated with mental illness amongst farm men and women may also lead to a lower self-reporting of symptoms. Alternatively, farm men and women, whilst experiencing many environmental and economic stressors, may enjoy a high level of social support which acts as a buffer to mental health problems (Johnson & Booth, 1990). Future research could perhaps explore differences in reporting by farm men and women based on self-report questionnaires and the questions asked to determine which self-report scale is more able to capture Depression, Anxiety and related constructs, and validated against a measure such as the Structured Clinical Interview for DSM Disorders.
As the study was conducted over two time periods in consecutive years, ‘vulnerable episodes’ of psychological distress in life course epidemiology were neither discussed or able to be measured given the measures used and the data provided. Future longitudinal research should consider this inclusion to determine the effects of these episodes on results.

8.2 Physical Health of Participants

In the current studies, 38.9% of participants met the criteria for metabolic syndrome at Time One, and 44% met the criteria at Time Two. These prevalence rates were significantly higher at both times than Australian prevalence rates reported by Cameron et al. (2007) (30.7%). For females, the prevalence rate at each time point was higher than that for females from a rural population in South Australia reported by Janus et al. (2007). For males, the prevalence rate was higher than that reported by Janus et al. only at Time Two. As the measures used were objective, it can confidently be stated that Australian men and women in farming communities are at higher risk of developing metabolic syndrome. Whilst 28 participants reduced their symptoms between Times One and Two so they no longer had metabolic syndrome, 45 new incidences of metabolic syndrome were seen at Time Two.

The apparently higher rates of metabolic syndrome amongst farm men and women may reflect the reduction in physical activity levels due to increased mechanisation, less livestock, decreased farming production, and fewer recreational activities. Indeed, farm men and women anecdotally report that their level of physical activity is seasonal, spasmodic, unplanned or non-existent
(Brumby, Willder & Martin, 2010). However, each of the metabolic syndrome factors is responsive to lifestyle changes (Riediger & Clara, 2011). Given the abundance of evidence on the benefits of physical activity in improving physical health outcomes, interventions aimed at increasing physical activity to at least the minimum current recommended levels should reduce the burden of disease associated with metabolic syndrome in farming communities. However, the challenge will be in developing novel interventions to increase physical activity. As a part of their program, the SFF facilitators work in conjunction with their participants to develop ways of increasing incidental and physical activity as a part of their farming family life. The program also now offers short video clips via its internet site to demonstrate ways in which farm men and women are able to incorporate these activities into their daily lives. However, as these interventions are currently limited to program participants, it may also be possible to promote such activities through local newspapers (including agricultural specific newspapers), rural television stations or agricultural shows to capture a wider audience.

When prevalence rates were broken down by age group, the findings from the current studies were consistent with previous research, with metabolic syndrome prevalence steadily increasing by age group at a rate of 10 to 15% with each increasing 10 year age bracket. One difference was observed at Time Two in the current study, with a greater proportion of those participants in the 25-34 year age bracket having metabolic syndrome that the 35-44 year age group. It is not clear why this result occurred, although one possible explanation is that the number of participants in that age group decreased from 35 at Time One to 23 at Time Two,
whilst the number of participants with metabolic syndrome increased from six to eight between Times One and Two, thereby increasing the prevalence proportion. Therefore, caution should be taken when drawing any conclusions from this result. Interestingly, in regards to metabolic syndrome prevalence rates increasing by age group, amongst the literature reviewed, no study provided a conclusive explanation as to why this occurs. However, obesity prevalence increases with age, whilst basal metabolic rate as well as lean body mass decreases with age. Increasing prevalence rates with age may also be due to secondary ageing; that is, the preventable changes that occur in some people that are the result of health habits, disease or environmental influences that have a cumulative effect over time (Bee & Bjorklund, 2004). For example, risk factors for Coronary heart disease (CHD) such as high-fat diets, tobacco use, physical inactivity, obesity and high blood pressure, are cumulative in nature and the more risk factors one has, the higher the chance of developing CHD through adulthood (Bee & Bjorklund, 2004). Further research is required to determine factors that may influence age related prevalence rates in metabolic syndrome.

Previous research has reported inconsistent results regarding gender differences in the prevalence of metabolic syndrome; therefore gender differences were not hypothesised. A review of the literature also found that previous gender differences, if any, appear to be related to ethnicity, as discussed in Chapter 3. The current study found no difference in prevalence rates between genders at either Times One and Two. This suggests that both farm men and farm women are at equal risk of developing metabolic syndrome. It may be that both farm men and farm women are more likely to have a similar diet or physical activity level than
their rural or metropolitan counterparts. For example, those living and working on the farm together may eat the same meals at the same times and share the workload on the farm. One limitation of this study was the inability to pair participants to their partner or those living within the same residence due to de-identification of the database. Future research should include the ability to examine proximal relationships to determine similarities in metabolic syndrome status as well as environmental influences such as diet, physical activity levels, and socioeconomic status.

Study One also determined the most commonly occurring factors of metabolic syndrome amongst farm men and women. As central obesity is mandatory to the IDF criteria, all metabolic syndrome positive participants met this. It was found that the next most commonly occurring factor amongst both genders was raised blood pressure, with 91.3% and 81.4% of men and women meeting this respectively. The next most commonly occurring factor was raised fasting plasma glucose for males (69.6%) and reduced HDL cholesterol for females (65.7%). The reverse relationship occurred for the third most commonly occurring factor, with 62.9% of females meeting the criteria for raised fasting plasma glucose and 50.7% of males meeting the criteria for reduced HDL cholesterol. Raised triglyceride levels were found in 29% and 40% of men and women respectively.

These results are concerning for this population as it has been found that hypertensive patients with metabolic syndrome have a two-fold greater risk of cardiovascular incidents compared to those without metabolic syndrome (Vyssoulis et al., 2010). Likewise, reduced HDL cholesterol levels increase the
risk of CVD as the body is not able to as effectively clear the artery walls from cholesterol deposits and take them to the liver, where they are processed and then excreted from the body (Nevid et al., 1998). Raised triglyceride levels indicate a high level of dietary fat in the bloodstream, derived from carbohydrates and fats, and also increase the risk of heart disease as they are often connected with high blood cholesterol and central obesity. Raised fasting plasma glucose levels increase the risk of Type II diabetes as the pancreas is required to produce more insulin to assist in the circulation of glucose through the blood stream.

It has been suggested that patterns of environmental influences affecting hypertension, reduced levels of HDL cholesterol, raised fasting plasma glucose levels and raised triglyceride levels include a lack of regular exercise, lack of stress control, lack of proper diet, and smoking (Nevid et al., 1998). Although age can be a factor in the body’s ability to take up glucose from the bloodstream, these metabolic risk factors can be reduced by adopting healthier lifestyle habits including regular aerobic exercise, diets low in saturated fats, controlling stress, and avoiding smoking and excessive drinking (Nevid et al., 1998). Therefore, the prevalence of metabolic syndrome and individual risk within the current sample could be significantly reduced through relatively simple and inexpensive therapeutic lifestyle changes including diet and exercise.

8.3 Relationship Between Mental Health and Metabolic Syndrome

Over the past decade there has been a paradigm shift in research that has resulted in an increasing focus on associations between mental health problems (particularly Depression) and the component factors of metabolic syndrome.
Indeed, there is a growing body of evidence to suggest the co-occurrence of metabolic syndrome and Depression. Based on the HPA axis dysregulation hypothesis, metabolic networks mediate both homeostasis and allostasis. Disturbances within these networks have been implicated in the pathogenesis, and also recovery from, depressive disorders. When compared with other studies reviewed, research conducted in rural regions of Australia (Dunbar et al., 2008) suggests that those living within rural areas are at increased risk of the co-occurrence of metabolic syndrome and Depression.

The current research explored psychological factors associated with metabolic syndrome in two ways: firstly, through a cross-sectional study (Time One) on a total of 357 participants, 139 of which met the criteria of metabolic syndrome; and secondly through a longitudinal study (Time Two) on participants returning to the SFF program the following year ($N = 234$), 103 whom met the criteria for metabolic syndrome.

8.3.1 Study One

Based on previous research findings, it was hypothesised that the constructs of Depression and Stress would be related to the presence of metabolic syndrome, whilst Anxiety would not. However, contrary to these expectations, comparisons of DASS subscale scores amongst those with and without metabolic syndrome at Time One revealed that those with metabolic syndrome scored significantly higher on Anxiety, whilst Depression and Stress were not related. These findings are partially consistent with those of Raikkonen et al. (2002), who found that trait Anxiety was significantly related to metabolic syndrome amongst middle-aged
women. Yet Raikkonen et al. (2002) also found women with metabolic syndrome to be higher in Depression, tension and anger scores relative to those without metabolic syndrome. These findings are also inconsistent with previous research by Skilton et al. (2007), who found that Depression, but not Anxiety was related to metabolic syndrome amongst men and women when using the HADS to measure Anxiety and Depression. Likewise, research by Dunbar et al. (2008) also found that Depression, but not Anxiety was related to metabolic syndrome when using the HADS.

The results from the Study One in relation to Depression and metabolic syndrome were not consistent with previous research findings. However, as discussed in Chapter 2, much of the research reviewed used the HADS as a measure of Depression and excludes many of the somatic symptoms of Depression that overlap with physical problems such as fatigue, sleep disturbance, and psychomotor changes. A culture of stoicism and stigma related to mental illness may have also contributed to a reluctance to self-report higher scores on the Depression subscale of the DASS; the HADS questions may possess more face validity in rural populations due to more socially acceptable phrasing of the questions for this cohort. The DASS also fails to ask about suicidality, which has been shown to be more widely reported by farmers than other psychiatric symptoms (Thomas et al., 2003).

It was also hypothesised that amongst the individual factors of metabolic syndrome, higher waist circumference and reduced HDL Cholesterol would be independently associated with Depression (Dunbar et al., 2008). Analysis of the
individual factors in the current study at Time One found that only waist circumference and blood glucose levels were able to explain any unique variance in Depression, whilst no individual factors significantly contributed to Anxiety or Stress. These findings are partially consistent with those of Muhtz et al. (2009) in that whilst Depression was not related to metabolic syndrome as an entity amongst men or women separately, women with depressive symptoms had a larger waist circumference, higher fasting blood glucose, diastolic blood pressure and lower HDL cholesterol than those without Depressive symptoms. One possible explanation for the findings from the current study is the relationship between psychological distress and the subsequent detrimental effects on positive health behaviours. These detrimental effects include an increase in harmful behaviours and poor compliance to treatment regimens which, like metabolic syndrome, are also associated with insulin resistance and increased central adiposity (Goldbacher & Matthews, 2007; Reaven, 1993).

Based on previous findings by Viinamaki et al., (2009) and Vanhala et al., (2009) who found relationships between Depression severity and metabolic syndrome, the hypothesis that moderate to severe levels of Depression would be associated with the presence of metabolic syndrome was not supported in the current study. This again may have been due to the low self-reporting of depressive symptoms on the DASS21, in which the severity ranged between normal to moderate levels only at both Times.
8.3.2 Study Two

The main interest of Study Two was the relationships between Depression, Anxiety, and Stress, and metabolic syndrome over time. These relationships are particularly important in determining how psychological risk factors are related to metabolic syndrome, and assist in targeting interventions that expand on therapeutic changes in lifestyle. The data allowed the exploration of these relationships in three ways: firstly, prospective analyses of baseline metabolic status at Time One and follow-up mental health status at Time Two; secondly, retrospective analyses of Time Two metabolic status and possible mental health factor differences at baseline (Time One); and finally, analyses of whether mental health factors were associated with the emergence, resolution or continuation of metabolic syndrome over time.

8.3.2.1 Prospective Analyses for Time One Metabolic Syndrome

The hypothesis that those participants with metabolic syndrome at Time One would display an increase in Depression scores at Time Two when compared to those without metabolic syndrome was not supported. A review of the previous research finding that supports a predictive relationship (Akbaraly et al., 2009) shows that the follow-up studies were conducted at three and six year intervals. Thus, the one year period between Times One and Two in the current study may not have been a sufficient amount of time to explore this relationship and compare to these previous results. As they also found no bidirectional associations, Akbaraly et al. (2009) suggest that depressive symptoms may be a consequence rather than a cause of metabolic syndrome. This notion of Depression as a consequence of metabolic syndrome shall be discussed in further detail when exploring the
relationship between continuity of metabolic syndrome status and Depression amongst participants.

8.3.2.2 Retrospective Analyses from Time Two Metabolic Syndrome

The hypothesis that higher levels of Depression would be found at Time One for those with metabolic syndrome at Time Two than those without metabolic syndrome at Time Two was supported. This finding is consistent with those of Raikkonen et al. (2007), Vanhala et al. (2009), and Viinamaki et al. (2009). Thus, depression may be a precursor of metabolic syndrome as well as a comorbid condition, as found at Time Two.

The hypothesis that Time Two metabolic status would be associated with higher levels of Stress at Time One was not supported. This finding mirrors that of Raikkonen et al. (2002) who reported perceived stress was not associated with metabolic syndrome amongst middle-aged women at baseline or follow-up seven years later. However, previous research has reported that frequent feelings of anger, tension, chronic stress and psychological distress were predictive of later metabolic syndrome (Raikkonen et al., 2002; Vitaliano et al., 2002). This raises a question about the way Stress was assessed in the current study. The DASS21 measures perceived Stress within the past four weeks, and may not have been the most appropriate measure for examining the chronic stressors which farm men and farm women experience. Future research should contemplate using measures of chronic stress, along with personal and social resources. One possibility is the Perceived Stress Reactivity Scale (SRS), which measures general perceived stress reactivity across a number of life domains (Schlotz, Yim, Zoccola, Jansen &
Schulz, 2011). The use of an objective measurement of Stress, such as cortisol levels, should also be considered as another method of measuring stress in this population who are known to underreport psychological symptoms.

No prediction was made in relation to Anxiety due to the unavailability of previous research findings; however those with a positive metabolic syndrome status at Time Two also reported higher levels of Anxiety at Time One than those without metabolic syndrome. Interestingly, at Time Two Anxiety levels were no longer significantly different between those with metabolic syndrome and those without, as they had been at Time One. This may have been associated with a significant decrease in Anxiety scores amongst females between Times One and Two. This decrease may have been due, in part, to participants of the SFF program receiving an intervention in the form of psychoeducation about Depression, Anxiety and Stress, and learning skills to assist in the management of these conditions.

8.3.2.3 Continuity of Metabolic Syndrome and Mental Health at Time One and Time Two

The investigation of the relationships between continuity, resolution or emergence of metabolic syndrome and mental health at Time One and Time Two allowed the above findings to be unpacked further.

The initial analyses indicated that those with metabolic syndrome at Time One had higher Anxiety at that time, and that those with metabolic syndrome at Time Two had higher Anxiety at Time One than those without the syndrome at Time
Two. However, when continuity of metabolic syndrome was investigated, only those who had maintained a positive metabolic status had reported higher levels of Anxiety at Time One when compared to participants who did not have metabolic syndrome at both times, had developed metabolic syndrome at Time Two, or had metabolic syndrome at Time One but did not meet the criteria at Time Two. Importantly, the group that developed metabolic syndrome by Time Two did not have a higher Anxiety score than other groups at Time One, and did not show a significant relative increase in their Anxiety scores by Time Two. In fact the Anxiety levels of all groups did not differ at Time 2. Given that it is unknown how long the stable positive metabolic syndrome group had met criteria, it is possible that the elevated Anxiety at baseline for this group could have developed after the onset of the condition, and was yet to develop in the new metabolic syndrome group. Overall, there is no evidence that elevated Anxiety preceded the syndrome.

The initial analyses above showed no cross-sectional association between metabolic syndrome and Depression at Time One, nor a relationship between metabolic syndrome at Time One and Depression at Time Two. However, retrospective analysis found that those with metabolic syndrome at Time Two had higher levels of Depression at Time One than those without metabolic syndrome. They also had higher levels of Depression at Time Two than those without metabolic syndrome, suggesting a causal or maintaining role for depression could exist.
When stability of metabolic syndrome was investigated, it was found that compared with participants who did not have metabolic syndrome at both times or who had metabolic syndrome at Time One but did not meet the criteria at Time Two, those participants who either maintained or developed metabolic syndrome had elevated Depression scores at Time One. This is consistent with the above. Those who had developed metabolic syndrome now had higher scores than those who did not. However, those whose metabolic syndrome status was stable did not differ from the other groups. When investigating relative change in depression scores over time by controlling for Time One depression scores in the ANOVA, both Time Two positive metabolic syndrome groups had significantly higher changes in scores than the other groups. The stable group's scores decreased, while the emergent metabolic groups' scores increased. In combination, these findings suggest Depression is associated with the emergence of metabolic syndrome, with there being no evidence to the contrary. While it may be co-morbid, its association may weaken over time.

These findings support the HPA axis hypothesis that psychological distress, in particular the construct of Depression, is a plausible underlying factor in both the development and maintenance of metabolic syndrome. It is interesting to note that Depression was not associated with metabolic syndrome status at Time One, yet increased waist circumference and raised fasting plasma glucose were factors associated with increased levels of Depression at Time One. However, Time One elevated Depression scores were related to Time Two metabolic syndrome status, which in turn was related to Time Two elevated Depression scores for those who developed metabolic syndrome at Time Two.
It has been suggested by Akbaraly et al. (2009) that perhaps there is an obesity-depression relationship, in which abnormal lipids and central obesity constitute risk factors for Depression, as overweight and obese individuals experience stigma and devaluation which may cause them to suffer from lower self-esteem and higher levels of Depression. Depressive symptoms at Time One may also have affected participants’ health behaviours, such as weight management and physical activity levels, which appear to be the cornerstone in any attempt to reduce risk factors associated with metabolic syndrome.

8.4 Implications for Metabolic Syndrome Interventions

Overall, the results of the current study demonstrates that Australian farm men and women may be at greater risk of metabolic syndrome than both the general or rural populations. It also appears that Depression is associated in the development of metabolic syndrome, whilst both Depression and Anxiety are associated in the maintenance of metabolic syndrome amongst farm men and farm women. Previous research by McIntyre et al. (2007) found a number of shared abnormal metabolic process overlaps between Major Depressive Disorder and Type II diabetes and conceptualised a basis for testing metabolic influence and therapies for mood disorders. From the results of the current study, it would appear that there is an interrelationship between the two, which provides further support of the HPA axis dysregulation hypothesis and the need to assess both the mental and physical health of farm men and women for better health outcomes.

This interrelation also highlights the need to address both the physical and mental health of farm men and farm women for decreasing the risk of
cardiovascular disease, Type II diabetes and achieving optimal wellbeing. As Depression, Anxiety and metabolic syndrome are associated with an increased risk in developing a number of physical illnesses, early detection of depressive and anxious symptomatology in individuals is important in the clinical management of those who are at risk of developing or maintaining metabolic syndrome.

8.5 Limitations and Recommendations for Future Research

The current research aimed to determine relationships between Depression, Anxiety and Stress in the pathogenesis of metabolic syndrome among farm men and women. It was designed to examine the current physical and psychological symptoms associated with metabolic syndrome and Depression, Anxiety and Stress and to examine predictive relationships between these constructs longitudinally to identify associations that could provide direction for future research, health promotion and disease prevention interventions for this particularly under researched, at risk cohort.

Prior to the current project, the prevalence of metabolic syndrome amongst farm men and women was unknown. Relationships between Depression, Anxiety and Stress and metabolic syndrome in this unique cohort had also not been examined. This is also the first study to measure the continuity of metabolic syndrome status over time in an attempt to determine prospective and retrospective associations. This study has been able to demonstrate farm men and women are at an apparently elevated risk of developing metabolic syndrome than the general population and that Depression is involved in the development of
metabolic syndrome, whilst both Depression and Anxiety are involved in the
maintenance of metabolic syndrome within this population. However, it should be
noted that the majority of participants did not report elevated levels of Depression,
Anxiety or Stress when compared with population norms. Despite efforts to
further the research knowledge in this area, there were shortcomings and
limitations to the current project. Combined with future recommendations, the
limitations of the current study are discussed below.

A major limitation of the current study was the intervention provided through
the Sustainable Farm Families program and environmental events that occurred in
between Times One and Two. This may have confounded results as both physical
and mental health issues were addressed by two ways. Firstly, education was
provided at the first workshop in relation to cardiovascular disease, nutrition, diet,
and stress. Participants also participated in exercises including stress management,
how to read food labels, supermarket tours and how to choose healthier options,
and exercise re-enforcement. At the end of each topic, participants were also
asked to develop an action plan and set goals for the following year to facilitate a
commitment to address identified priorities in the development and maintenance
of good health. Secondly, each participant’s physical evaluation results were
discussed during Individual Health Assessments to identify priority issues, and
referrals were made to the appropriate services if required. Whilst the objective
data in the current study was able to be adjusted to reflect IDF metabolic
syndrome criteria, including specific treatments for lipid abnormalities or previous
diagnosis of hypertension or Type II diabetes, external interventions relating to
mental health between the two time periods were unable to be controlled.
Another limitation of the study was the de-identification of the data used. This did not allow the current study to explore possible similarities between metabolic syndrome factors and status as well as Depression, Anxiety and Stress amongst participants who reside together. Both the research reviewed and the findings from the current study suggest the development of metabolic syndrome may be individualistic and dependent on a number of environmental and genetic influences. It could be expected that those participants living on and working the same property may have similar results on these measures due to a number of shared personal and environmental influences or experiences such as dietary intake, physical activity and the effects of economic, social or weather events on the farm. Furthermore, as twin studies have shown high concordance rates in relation to metabolic syndrome risk factors (Poulsen et al., 2001), genetic links between participants should also be considered. Future research should consider the ability to match participants based on their place of residence and also any genetic relationships to gain a greater understanding of the impacts of various influences in the pathogenesis of metabolic syndrome and its factors.

A further limitation of the current study includes the collection of biological samples across a number of locations by a number of Nurses using different equipment in each location, although this has been minimised by the use of the same equipment on each participant over the time periods.

The method of recruiting participants through media advertising and promotion of the program through various agriculture groups may also have resulted in a
sample that is not representative of the farming population. Future research needs to both determine what a representative farming population is, and conduct a study that matches these parameters.

A final limitation of the current study was the inability to measure the cortisol levels of participants. As the functions of cortisol include the regulation of blood sugar levels and stress, and levels can be affected by physical, emotional or environmental stressors, future research should consider measuring cortisol levels to further investigate the HPA axis dysregulation hypothesis.

8.6 Conclusion

Given the individual and societal burden of cardiovascular disease, Type II diabetes and mental illness, research efforts to reduce metabolic syndrome and its individual risk factors is important. The greater the effort to understand, intervene and improve the physical and mental health of those at risk of developing these, the better the outcomes for individuals and society. In particular, when considering both the determinants of poorer health outcomes and barriers to help-seeking for farm men and women, supporting efforts towards better health outcomes for this under-researched and under-serviced population has financial, emotional and social implications for individuals, families, populations and the Australian economy.

The elevated results from this study demonstrate that Australian farm men and women are at risk of developing metabolic syndrome and that Depression is associated with the development of metabolic syndrome, whilst both Depression
and Anxiety are associated with the maintenance of metabolic syndrome. These findings provide further support for the HPA axis dysregulation hypothesis of metabolic syndrome and suggest that psychological evaluation should be an integral part of assessment of metabolic syndrome. However, it appears that a multi-factorial approach to aetiology and pathogenesis is required for intervention and treatment. As both physical activity and good dietary habits are known to have positive effects on physical health and also mood states, farm men and women should continue to be evaluated, educated and encouraged to improve both physical and mental health and wellbeing.
References


Appendices
Appendix A: Consumer Information Form
### Consumer Information

**Consumer Details**

- **Family Name:**
- **Given Names:**
- **Date of Birth:**
- **Preferred Name:**

**Contact Details**

- **Contact Address:** (for correspondence, home visits, etc.)
  - (number)
  - (street)
  - (suburb/locality)
  - (postcode)

**Who the Agency Can Contact if Necessary**

- **Person 1 Name:**
- **Contact Details**
  - (number)
  - (street)
  - (suburb/locality)
  - (postcode)
- **Phone:**
- **Relationship to Client:**

**General Practitioner (If NA, write NA)**

- **Name:**
- **Address:**
- **Phone:**
- **Fax:**
- **Email:**

### Office Use Only

- **Name:**
- **Designation/Agency:**
- **Sign:**
- **Date:**
- **Contact Number:**
- **If information becomes superseded, indicate below and record updated information on new form**

**This Page Completed By:**
- [ ] The consumer or someone who represents the Consumer (care, parent or guardian)
- [ ] The agency (face-to-face with consumer)
- [ ] The agency (other, incl. telephone contact with consumer)

**Consumer privacy information brochure provided?**
- [ ] Yes
- [ ] No

---

- **Record Agency Assigned Consumer Identifier (key work agency):**
  - (if applicable)
  - (or affix label here)
Consumer Information
If question is irrelevant or information not known, write
Not Applicable or NA

Service Requested

Notes: (including alerts and comments on risks, urgency and access issues)

Source of Referral
Record: (1) Self, (2) Family, significant other, friend,
(3) GP/medical practitioner (community-based),
(4) Specialist aged or disability assess. team/service (e.g. AGAT)
(5) Comprehensive HACC assessment authority,
(6) Community nursing services, (7) Hospital (public),
(8) Psychiatric/Mental health service or facility,
(9) Extended care/rehabilitation facility,
(10) Palliative care facility/hospital,
(11) Government residential aged care facility,
(12) Aboriginal health service, (13) Carerlink centre,
(14) Other community-based government medical/health
Service, (15) Other government medical/health service,
(16) Other government community-based services agency,
(17) Hospital (private),
(18) Non-government residential aged care facility,
(19) Other non-government medical/health services,
(20) Other non-government community-based service,
(21) Law enforcement agency, (22) Other.

Source of Referral Contact Details:

Country of Birth
Record: (1) Australia, (2) Other.
If other, specify:

Indigenous Status
Record: (1) Aboriginal but not Torres Strait Islander origin,
(2) Torres Strait Islander but not Aboriginal origin,
(3) Both Aboriginal and Torres Strait Islander origin,
(4) Neither Aboriginal nor Torres Strait Islander origin.

Main Language Spoken at Home
Record: (1) English, (2) Other.
If other, specify:

Interpreter Required
Record: (1) Interpreter not needed, (2) Interpreter needed.

Preferred Language
(If most spoken English) include sign Language, and any required
communication devices or special interpreter needs:

Government Pensioner/Benefit Status
Record: (1) Aged Pension, (2) Veterans Affairs Pension,
(3) Disability Support Pension, (4) Carer Payment (pension),
(5) Unemployment-related benefits, (6) Other gov. pension or benefit,
(7) No gov. pension or benefit.

Card Number:

DVA Card Status
Record: (1) No DVA Card, (2) Yes Gold Card,
(3) Yes White Card, (4) Yes Other DVA Card.

DVA Card Number:

Insurance Status
Insurer Name and Card Number:

Medicare Number:

Health Care Card Number:

Office Use Only
Name: ____________________________
Designation/Agency: ____________________________
Sign: ____________________________
Date: ____________________________
Contact Number: ____________________________

If information becomes superseded, indicate below and record updated information on new form

The information on this form has been superseded
Date: ____________________________
Name: ____________________________
Sign: ____________________________
Appendix B: Physical Assessment Form
<table>
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<th>Recommended Values</th>
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<tr>
<td></td>
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<td>Kit..............</td>
<td>Kit..............</td>
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<tr>
<td>Weight and height</td>
<td>Per individual</td>
<td>Weight</td>
<td>Height</td>
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<td>Body mass index:</td>
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<tr>
<td></td>
<td>F 20-25</td>
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<tr>
<td>Oxygen Saturation</td>
<td>&gt;94% ideal</td>
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<td></td>
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<tr>
<td></td>
<td>&lt;94% w/ symptoms</td>
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<tr>
<td></td>
<td>&lt;94% w/ anemic/ref</td>
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<td>Percentage of Body Fat</td>
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<td>%</td>
<td>%</td>
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<tr>
<td></td>
<td>F 20-35%</td>
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<tr>
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<td>&gt; 5.6mmol/L</td>
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<td></td>
<td>&gt; 5.6mmol/L ref</td>
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<td>T.C, HDL, TRIG, LDL, T.C/HDL Ratio</td>
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<tr>
<td></td>
<td>≥ 140/90 refer</td>
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<td></td>
</tr>
<tr>
<td>Pulse Rate:</td>
<td>60-100 regular</td>
<td></td>
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</tr>
<tr>
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<td>Normal</td>
<td>L eye</td>
<td>L eye</td>
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<td>Without glasses</td>
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<td>With glasses or contact lenses</td>
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<td>Hip</td>
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<tr>
<td></td>
<td>F 0.8 to 1.0 ratio</td>
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<td></td>
</tr>
<tr>
<td>Respiratory Assessment</td>
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<tr>
<td>Signature of person completing this form</td>
<td>Date (Year 1)</td>
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<tr>
<td>Signature of person completing this form</td>
<td>Date (Year 2)</td>
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### Sustainable Farm Families – Physical Assessment Form

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<td>Body mass Index:</td>
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<tr>
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<td>6/6 standar</td>
<td>L eye</td>
<td>M eye</td>
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<td>With glasses or contact lenses</td>
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Signature of person completing this form__________________________ Date_______ (Year 4)

2009 V.13 Page 2 of 6 Sustainable Farm Families
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<table>
<thead>
<tr>
<th>Gastrointestinal Assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea/vomiting</td>
<td></td>
</tr>
<tr>
<td>Indigestion/reflux</td>
<td></td>
</tr>
<tr>
<td>Constipation/diarrhea</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Urological Assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress incontinence</td>
<td></td>
</tr>
<tr>
<td>Voiding &gt; 1 per night</td>
<td></td>
</tr>
<tr>
<td>Changes in voiding patterns</td>
<td></td>
</tr>
<tr>
<td>Due for prostate screening-yes or no</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sexual and Reproductive</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexually active - yes or no</td>
<td></td>
</tr>
<tr>
<td>Overdue pap smear/ mammography</td>
<td></td>
</tr>
<tr>
<td>Menstruating-yes or no</td>
<td></td>
</tr>
<tr>
<td>Erectile dysfunction</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Musculoskeletal Assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Joint or muscle pain (document where)</td>
<td></td>
</tr>
<tr>
<td>Other issues</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Psychosocial</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Living arrangements (care, partner, children)</td>
<td></td>
</tr>
<tr>
<td>Stress, anxiety or depression</td>
<td></td>
</tr>
</tbody>
</table>

Signature of person completing this form: ____________________________ Date: ______ (Year 1)
**Western District Health Service**

<table>
<thead>
<tr>
<th>Physical Assessment</th>
<th>Please comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Referral from Previous Year Actioned?</td>
<td>Yes ☐</td>
</tr>
<tr>
<td>General Appearance</td>
<td>☐ Allergies</td>
</tr>
<tr>
<td>Genetic Evaluation-Family History</td>
<td>☐ Cancer</td>
</tr>
<tr>
<td>Neuro assessment</td>
<td>☐ Visual impairments</td>
</tr>
<tr>
<td>Skin and mucous membranes</td>
<td>☐ Intact</td>
</tr>
<tr>
<td>Cardiovascular assessment</td>
<td>☐ Irregular pulse</td>
</tr>
<tr>
<td>Respiratory Assessment</td>
<td>☐ Cough/sputum</td>
</tr>
<tr>
<td>Gastrointestinal Assessment</td>
<td>☐ Nausea/vomiting</td>
</tr>
<tr>
<td>Urological Assessment</td>
<td>☐ Stress incontinence</td>
</tr>
<tr>
<td>Sexual and Reproductive</td>
<td>☐ Sexually active - yes or no</td>
</tr>
<tr>
<td>Musculoskeletal Assessment</td>
<td>☐ Joint or muscle pain (document where)</td>
</tr>
<tr>
<td>Psychosocial</td>
<td>☐ Living arrangements (care, partner, children)</td>
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</table>

Signature of person completing this form ________________ Date __________ (Year 2)

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2009 V.1.0 PAGE 4 OF 5 Sustainable Farm Futures ™
<table>
<thead>
<tr>
<th>General Appearance</th>
<th>General comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergies</td>
<td></td>
</tr>
<tr>
<td>List medications</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Genetic Evaluation - Family History</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cancer</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
</tr>
<tr>
<td>Other genetically linked disease</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Neuro assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual impairments</td>
<td></td>
</tr>
<tr>
<td>Frequent headaches</td>
<td></td>
</tr>
<tr>
<td>Hearing impairment</td>
<td></td>
</tr>
<tr>
<td>Other related disorders</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Skin and mucous membranes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Intact</td>
<td></td>
</tr>
<tr>
<td>Disorders noted</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cardiovascular assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Irregular pulse</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td>Elevated cholesterol</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Respiratory Assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough/sputum</td>
<td></td>
</tr>
<tr>
<td>Shortness of breath</td>
<td></td>
</tr>
<tr>
<td>Smoker number per day</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Gastrointestinal Assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Nausea/vomiting</td>
<td></td>
</tr>
<tr>
<td>Indigestion/ reflux</td>
<td></td>
</tr>
<tr>
<td>Constipation/diarrhoea</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Urological Assessment</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Stress incontinence</td>
<td></td>
</tr>
<tr>
<td>Voiding: &lt;1 per night</td>
<td></td>
</tr>
<tr>
<td>Changes in voiding patterns</td>
<td></td>
</tr>
<tr>
<td>Due for Prostate screening: yes or no</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sexual and Reproductive</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sexually active: yes or no</td>
<td></td>
</tr>
<tr>
<td>Ovulation pap smear/ mammography</td>
<td></td>
</tr>
<tr>
<td>Menstruating: yes or no</td>
<td></td>
</tr>
<tr>
<td>Erectile dysfunction</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
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<td>Joint or muscle pain (document where)</td>
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<tr>
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<table>
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<tr>
<th>Psychosocial</th>
<th></th>
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<tbody>
<tr>
<td>Living arrangements (carer, partner, children)</td>
<td></td>
</tr>
<tr>
<td>Stress, anxiety or depression</td>
<td></td>
</tr>
</tbody>
</table>

Signature of person completing this form: [Signature]
Date: [Date] (Year 2)
**SFF Physical Assessment**

<table>
<thead>
<tr>
<th>Referral from Previous Year Actioned?</th>
<th>Please comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Yes</td>
<td></td>
</tr>
<tr>
<td>☐ No</td>
<td></td>
</tr>
<tr>
<td>☐ N/A</td>
<td></td>
</tr>
</tbody>
</table>

### General Appearance
- Allergies
- List medications

### Genetic Evaluation - Family History
- Cancer
- Cardiovascular disease
- Diabetes
- Other genetically linked disease

<table>
<thead>
<tr>
<th>Neuro assessment</th>
<th>General comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>☐ Impairments</td>
<td>(review assessments)</td>
</tr>
<tr>
<td>☐ Headaches</td>
<td></td>
</tr>
<tr>
<td>☐ Hearing impairment</td>
<td></td>
</tr>
<tr>
<td>☐ Other related disorders</td>
<td></td>
</tr>
</tbody>
</table>

### Skin and Mucous Membranes
- Intact
- Disorders noted

### Cardiovascular Assessment
- Irregular pulse
- Hypertension
- Elevated cholesterol

### Respiratory Assessment
- Cough/sputum
- Shortness of breath
- Smoker number per day

| (review resp assessment questions) |

### Gastrointestinal Assessment
- Nausea/vomiting
- Indigestion/flatulence
- Constipation/diarrhea

### Urological Assessment
- Stress incontinence
- Voids/kg per night
- Changes in voiding patterns
- Due for prostate screening - yes or no

### Sexual and Reproductive
- Sexually active - yes or no
- Ovulation/pap smear/mammography
- Menstruation - yes or no
- Erectile dysfunction

### Musculoskeletal Assessment
- Joint or muscle pain (document where)
- Other issues

### Psychosocial
- Living arrangements (caregiver, partner, children)
- Stress, anxiety or depression

---

Signature of person completing this form: ____________________________ Date: ___________ (Year 4)
Appendix C: Plain Language Statement
Dear Participant,

We would like to invite you to participate in a project that has been designed to explore links between farming family health, farm related accidents and farm sustainability. The project also involves the delivery of a health education program to assist farmers and farming families to identify strategies to enhance individual and family health. We are particularly interested in working with farmers (male and female) who are currently farming and would like to be involved in this project over a 3 year period.

Before you decide whether to accept this invitation, it is important that you understand what the purpose of the project is and what is required of you. This information is provided below and if there is anything that is not clear, or if you would like more information, please contact us.

GENERAL PURPOSE OF THE PROJECT

The general purpose of this study is:

- For farmers to participate in a health education program that helps them recognise and identify factors that affect family farm health.
- For farmers to write up a health action plan and support their improvement in health.
- Undertake a health assessment of participating farmers initially and in 12 months and in 24 months, and to monitor health status over time.
- To interview through focus groups, farmers’ experiences of the project.
- To investigate the link between farming family health, farm accidents and farm sustainability.
- To roll out a training program that can be used across a range of farming industries in Victoria or Australia.

DO I HAVE TO TAKE PART?

You are under no obligation to participate in this project. Your decision to participate is entirely voluntary. Should you decide to take part and then change your mind, you are still free to withdraw without giving a reason.
AM I ELIGIBLE TO PARTICIPATE?

We would like farmers to participate and we would like some to be from the same family for example as spouses or children. You are eligible to participate if you:

- are over the age of 18 years and under the age of 75;
- are currently farming;
- speak English;
- are competent to decide and have capacity to consent to participate, attend health education program and health assessment, and be interviewed as a member of a focus group.

WHAT IS REQUIRED OF ME IF I PARTICIPATE?

If you participate, you will be required to:

- Sign the attached consent form (to attend health education, undergo health assessment, participate as a member of focus group and to have some comments documented – the comments will be de-identified);
- Attend health education sessions of approximately 2 days in the first year of 6 hours per day, 1 day in the second year and 1 day in the third year;
- Participate in focus group sessions for approximately one hour (45-90 minutes) about your thoughts and experiences as a participant in the health program;
- Keep (as in write and maintain) an action plan to improve health over the two years as a before, during and after participating in the sustainable farm families program;
- Undergo a physical nursing assessment which includes blood pressure, pulse, cholesterol, weight, blood glucose, height, waist to hip ratio measurement, % fat body mass, respiratory assessment and/or a venous blood sample.

WHAT ARE THE POSSIBLE COSTS, RISKS AND BENEFITS TO ME OF PARTICIPATING?

There may be some minor travelling costs associated with attending the venue for the health education sessions and focus groups. Unfortunately, our budget does not permit us to reimburse you for these. There are no readily foreseeable risks associated with the conduct of this project, however, should on physical assessment we find an indication of a illness or disease you will be referred to a practitioner of your choice and/or health service. You may also withdraw from the project altogether. Please note that nurses are mandated by law to report certain findings — such as child abuse, domestic violence.

There may be some benefits of participating in the project such as increasing your understanding of wellness, lifestyle factors, prevention of ill health, and a health assessment and report. You may decide to change personal behaviour to improve your health, well-being and safety practices.
WILL MY INFORMATION AND RESPONSES BE KEPT CONFIDENTIAL?

Your responses will be kept confidential and your anonymity assured by the following processes: all health information will remain confidential as a health record with Western District Health Service and / or key health services and will not be deleted until 7 years after the last occasion on which the health service provided a service to you. Should it be recommended that you seek further medical advice your information about you will only be passed on if you consent to the referral.

For the purposes of the project all health and general information will be de-identified for project purposes. False names will be used when reporting or quoting people. Transcripts of individual and focus group interviews will be identified by a number, to ensure that you cannot be identified. This means that the information collected can only be presented in a manner that would make personal identification of you impossible.

The findings of the sustainable farming families report will be presented in a final report and will also be made available through the publication of articles in professional journals and presentations at rural industry and health conferences. Neither of these works will contain personally identifying information.

WHO SHOULD I CONTACT IF I HAVE ANY QUESTIONS?

The best person to contact regarding queries on your physical assessment is Susan Brumby - a registered nurse (RN Div 1). Susan can be contacted directly on 5551 84560.

For any general queries, please contact Cate Mercer-Grant, who can be contacted on 5551 8508 or by email: Cate.Mercer-Grant@wdhs.net

Yours sincerely

Signature Redacted by Library

Susan Brumby
Principal Investigator
Western District Health Service
Appendix D: Consent Form
Sustainable Farm Families Consent Form

Project Title: Sustainable Farm Families – the human resource in the triple bottom line

Group Location: [Insert Location]

Name(s) of investigators: (1) Susan Brumby  Phone: (03) 55518460

1. I have received a statement explaining the interview/questionnaire/health assessments and education involved in this project.

2. I consent to participate in the above project, the particulars of which - including details of the interviews, questionnaires, health assessment and education have been explained to me.

3. I authorise the project manager or his or her assistant to interview me, administer a questionnaire, undertake a health assessment and deliver the education program.

4. I acknowledge that:
   (a) Having read Plain Language Statement, I agree to the general purpose, methods and demands of the project.
   (b) I have been informed that I am free to withdraw from the project at any time and to withdraw any unprocessed data previously supplied.
   (c) The project is for the purpose of raising awareness and/or teaching. It may not be of direct benefit to me.
   (d) The privacy of the information I provide will be safeguarded. However should information of a private nature need to be disclosed for moral, clinical or legal reasons, I will be given an opportunity to negotiate the terms of this disclosure.
   (e) The security of the research data is assured during and after completion of the study. The data collected during the study may be published (may include photographs), and a report of the project outcomes will be provided to the relevant health service. Any information which will identify me, will not be used, except for photographs.
   (f) I consent to receiving marketing messages from the SFF program.
   (g) I understand that I will be required to consent to a physical assessment and/or venous blood samples with a Western District Health Service consent form.

Participant Consent

Name: 

Address: 

(Please Print)

Telephone: Fax: 

Email: 

Mobile Number: Date of Birth: 

Signed: Date: 

Participates will be sent a photocopy of this consent form after it has been signed.

PLEASE FAX to 03 5551457
Appendix E: Profile Health Conditions
### Overall Health
In general, how would you say your health is?
- Excellent
- Very Good
- Good
- Fair
- Poor

How much bodily pain have you had during the past 4 weeks?
- None
- Very Mild
- Moderate
- Severe
- Very Severe

How much did your health interfere with your normal activities (outside and/or inside the home) during the past 4 weeks?
- Not at all
- Slightly
- Moderately
- Quite a bit

### Vision
How is your eyesight for reading?
- Excellent
- Good
- Fair
- Poor

How is your long distance eyesight?
- Excellent
- Good
- Fair
- Poor

Do you wear glasses?
- Yes
- No

### Hearing
How is your hearing?
- Good hearing both ears
- Difficulty hearing with one ear
- A little trouble hearing both ears
- A lot of trouble hearing both ears
- Deaf in both ears

Do you wear a hearing aid?
- Yes
- No

### Falls
Have you had a fall inside/outside the home in the past 6 months?
- Yes
- No

If yes, record number of falls ____

### Health Conditions
(include all issues and history eg. allergies, medical conditions, disabilities, conﬁdence, denial, developmental issues, anxiety and depression)

1. 
2. 
3. 
4. 
5. 

### Current Medications
(include prescriptions, over-the-counter, alternative products and pain killers)

1. 
2. 
3. 
4. 
5. 
6. 
7. 
8. 

Comments.

### Office Use Only
Name: ____________________________
Designation/Agency: ________________
Sign: ____________________________
Date: ____________________________
Contact Number: ____________________

IRI 2010 V.17
Profile: Health Behaviours

Smoking
- Never smoked
- Has quit smoking
- Currently smokes
  If quit, record when: Date/Year ______________

Alcohol
How often do you have a drink containing alcohol?
- Never - if never, proceed to Continence
- Monthly
- Once a week
- 2 to 4 times per week
- 5+ per week

How often do you have more than 6 standard drinks (males) or 4 standard drinks (females) on one occasion?
- Never
- Monthly
- Once a week
- 2 to 4 times per week
- 5+ per week

How many standard drinks do you have on a typical day when you are drinking?
- 1 to 2
- 3 to 4
- 5 to 6
- 7 to 8
- 9+ per day

Continence
My water works bother me
- Monthly
- Weekly
- Daily
- Never

Prostate Screening Test (50 and over)
- Yes
- No
- PSA Blood Test
  - Date ______________
- Digital Rectum Examination
  - Date ______________

Respiratory
Do you currently have a cold?
- Yes
- No

Over the last month I have had:
- Nasal congestion
- Frequent cough
- Wheezy breathing
- Morning cough
- None of the above

How often does shortness of breath prevent you from doing something that you think you ought to be able to do?
- A few times a year
- Monthly
- Weekly
- Daily
- Never

I sneeze at night:
- Every night
- Sometimes
- Rarely
- Never

When I wake in the morning I feel refreshed:
- Always
- Sometimes
- Rarely
- Never

I experience breathing difficulties, cough, wheezy breathing or chest tightness after working with livestock, dust, chemicals or grime:
- Always
- Sometimes
- Rarely
- Never

Over the last year I have had:
- Nasal congestion
- Frequent cough
- Wheezy breathing
- Morning cough
- None of the above

Physical Activity
Would you accumulate 30 minutes or more of moderate intensity physical activity on most days of the week?
- Yes
- No

Breast Screen
- Yes
- No
  If yes, record when: Date/Year ______________

Pap Smear
- Yes
- No
  If yes, record when: Date/Year ______________

Office Use Only

Name: __________________________
Designation/Agency: __________________________
Sign: __________________________
Date: __________________________
Contact Number: __________________________

W 2019 V 1.17
Appendix G: Short Form of the Depression, Anxiety and Stress Scale
<table>
<thead>
<tr>
<th>Serial No.</th>
<th>Statement</th>
<th>Tardiness</th>
<th>Sometimes</th>
<th>Frequently</th>
<th>Most of the time</th>
<th>Office use only</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>I found it hard to wind down</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>I was aware of dryness of my mouth</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>I couldn’t seem to experience any positive feeling at all</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>I found it difficult to work up the initiative to do things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>I tend to over-react to situations</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>I experienced trembling (e.g., in the hands)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>I felt that I was using a lot of nervous energy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>I was worried about situations in which I might panic and make a fool of myself</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>I felt that I had nothing to look forward to</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>I found myself getting agitated</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>I found it difficult to relax</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>I felt down-hearted and blue</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>I was intolerant of anything that kept me from getting on with what I was doing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>I felt I was close to panic</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>I was unable to become enthusiastic about anything</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>I felt I wasn’t worth much as a person</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>I felt that I was rather touchy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart flushing, a beat)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>I felt scared without good reason</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>I felt that life was meaningless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
</tbody>
</table>

**DASS**

Date: ____________________

SFF Code: ____________________

Please read each statement and circle a number 0, 1, 2, or 3, which indicates how much the statement applied to you OVER THE PAST WEEK. There is no right or wrong answer. Do not spend too much time on any statement.
Appendix H: South West Health Care SFF Ethics Letter
Ref: 010/928/11
JPKgke

2 September, 2003

Mrs. S. Brumby
Director Community Services
Western District Health Service
PO Box 283
HAMILTON VIC. 3300

Dear Mrs. Brumby,

RE: SUSTAINABLE FARM FAMILIES

I refer to your application to undertake the above study and wish to advise that the Multidisciplinary Ethics Committee granted approval at its meeting on Thursday, 25 August, 2003.

The approval was granted subject to a number of amendments to the questionnaires as outlined by Dr. Peter O'Brien, Director of Medical Services, on the attached documentation. Your advice in regard to these matters would be greatly appreciated.

As part of the approval to undertake this research there are a number of conditions with which you are required to comply.

It is a requirement of the National Health and Medical Research Council that researchers provide an annual report and that the report contain the following documentation:

(i) progress to date or outcome in the case of completed research;
(ii) maintenance and security of records;
(iii) compliance with the approved protocol; and
(iv) compliance with any conditions of approval.

It is also a condition of approval that researchers immediately report anything which might warrant review of ethical approval of the project including:

a) serious or unexpected adverse effects on participants;
b) proposed changes in the protocol; and
c) unforeseen events that might affect continued ethical acceptability of the project.

The researcher will also be required to inform the Multidisciplinary Ethics Committee and provide reasons if the research project is discontinued before the expected date of completion.

Yours sincerely,

JOHN F. KRYGGER
Chief Executive Officer

Address all correspondence to Chief Executive Officer
55 Street, Warrnambool Vic. 3280 Phone: 03 5563 1405 Fax: 03 5563 1620

A.B.N. 41 189 754 235

Signature Redacted by Library