

**SOCIAL SUPPORT, MARRIAGE AND  
PSYCHOBIOLOGICAL PATHWAYS TO  
ADJUSTMENT FOLLOWING ACUTE  
CORONARY SYNDROME**

**Gemma Hutton**

**Department of Epidemiology and Public Health**

**University College London**

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I, Gemma Hutton, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signature ..... Date .....

## ABSTRACT

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The key aims of this thesis were to investigate the role of social support and marriage in adjustment and recovery in coronary heart disease (CHD). Declining death rates in CHD due to medical and surgical advances combined with increasing prevalence rates have contributed to a large and steadily growing population of chronic CHD patients, many of whom have suffered an acute cardiac event. In the context of this population, there is considerable need to determine factors that improve both adjustment and prognosis. Aspects of social support and marriage have been robustly associated with morbidity and mortality in CHD. Exploration of the potential psychological and biological pathways that link these factors forms the core of this thesis. Data from two separate studies are presented with the majority of analyses originating from data gathered in the Tracking Recovery after Acute Coronary Events (TRACE) study, a longitudinal study exploring diverse correlates of adjustment and recovery in 298 ACS patients. Associations between social support, marital satisfaction, distress, quality of life and HRV among ACS patients followed up from hospital admission to 12 months following discharge are presented. Data were also derived from a second study which explored psychobiological factors in a sample of 88 suspected coronary artery disease (CAD) patients and the analysis focused on marital influence on HRV. The overall thesis objective was to identify significant relationships between social and marital support, and various psychobiological factors that may contribute to adjustment and, ultimately, influence CHD prognosis.

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## PUBLICATIONS

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Some of the research described in this thesis has been published and/or presented at conferences. Some of the publications listed here are not related to the content of this thesis but to data derived from the studies utilised by this thesis. All have been published under my previous maiden name (Randall).

### **Journal publications:**

**Randall, G.**, Molloy, G.J., & Steptoe, A. (2009). The impact of an acute cardiac event on the partners of patients: a systematic review. *Health Psychology Review*, 3, 1 – 84.

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Messerli-Burgy, N., Molloy, G. J., Wikman, A., Perkins-Porras, L., **Randall, G.**, & Steptoe, A. (2012). Cortisol levels and history of depression in acute coronary syndrome patients. *Psychological Medicine*, 42, 1815-1823.

Molloy, G. J., **Randall, G.**, Wikman, A., Perkins-Porras, L., Messerli-Burgy, N., & Steptoe, A. (2012). Type D personality, self-efficacy, and medication adherence following an acute coronary syndrome. *Psychosomatic Medicine*, 74, 100-106.

Molloy, G.J., Stamatakis, E., **Randall, G.**, & Hamer, M. (2009). Marital status, gender and cardiovascular mortality: behavioural, psychological distress and metabolic explanations. *Social Science and Medicine*, 69, 223-228.

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Steptoe, A., Molloy, G. J., Messerli-Burgy, N., Wikman, A., **Randall, G.**, Perkins-Porras, L. et al. (2011a). Fear of dying and inflammation following acute coronary syndrome. *European Heart Journal*, *32*, 2405-2411.

Steptoe, A., Molloy, G. J., Messerly-Burgy, N., Wikman, A., **Randall, G.**, Perkins-Porras, L. et al. (2011b). Emotional triggering and low socio-economic status as determinants of depression following acute coronary syndrome. *Psychological Medicine*, *41*, 1857-1866.

Wikman, A., Messerli-Burgy, N., Molloy, G. J., **Randall, G.**, Perkins-Porras, L., & Steptoe, A. (2012). Symptom experience during acute coronary syndrome and the development of posttraumatic stress symptoms. *Journal of Behavioral Medicine*, *35*, 420-430.

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# CHAPTER 1 LITERATURE REVIEW

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## Cardiovascular disease and psychological risk factors

### 1.1 Overview of Cardiovascular disease

Cardiovascular Disease (CVD) is the most common cause of death and premature death in the UK accounting for over a third of all deaths. Almost half of the deaths from CVD are from coronary heart disease (CHD) with one in five men and one in seven women dying from the disease. Death rates from CHD have been in decline since the 1970's with a 45% reduction among under 65's in the last ten years. However, about thirty five people aged under 65 still die every day from CHD. Declining CHD death rates have been attributed to both medical and surgical advancement, and to reductions in major risk factors (British Heart Foundation, 2010). In contrast to the declining death rates, prevalence rates for CHD are increasing particularly among older age groups. Prevalence rates for CHD are approximately 6.5% in men and 4% in women and it is estimated that there are 2.7 million people living with CHD in the UK (British Heart Foundation, 2010). The combination of increasing prevalence and declining death rates from CHD has led to a growing population of chronic CHD sufferers. CHD is a huge burden on both the individual and society costing the UK approximately £9 billion every year. The causes of CHD are relatively well understood and primary prevention is crucial to reducing escalating prevalence and cost. Numerous risk factors that contribute to CHD genesis have been identified including family history, age, gender, smoking, physical inactivity, diabetes, obesity, hypertension and hypercholesterolaemia. Psychosocial factors have also been associated with CHD development including depression and anxiety, work stress, personality factors and lack of social support (Hemingway & Marmot, 1999)

CHD is the end product of coronary artery atherosclerosis, resulting from the progressive accumulation of cholesterol-rich plaque and subsequent narrowing of one or more of the coronary arteries. A common primary symptom of CHD is angina which refers to chest pain during exertion caused by transient ischemia. As atheromatous plaque accumulates, plaque

disruption through rupture or erosion may occur resulting in an Acute Coronary Syndrome (ACS). ACS is an umbrella term referring to a range of acute myocardial ischaemic states that may result from plaque disruption and thrombus formation. These states include unstable angina, ST-elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI). Unstable angina refers to chest pain caused by ischemia that suddenly worsens, occurs at rest or lasts for more than 15 minutes. A STEMI is considered the most severe type of MI and reflects total occlusion of the coronary artery. This type of MI is characterised by a particular pattern of elevation in the ST segment of an ECG (electrocardiogram). A NSTEMI results from partial occlusion of the coronary artery and is a milder form of MI that does not produce changes in the ST segment on an ECG. Differential diagnosis in ACS is based on ECG changes and cardiac enzyme assessment which indicate the extent of damage to the heart (Arbab-Zadeh, Nakano, Virmani, & Fuster, 2012).

There are approximately 124, 000 myocardial infarctions every year and about 4.1% of men and 1.7% of women have had a myocardial infarction (MI) in the UK (British Heart Foundation, 2010). Immediate treatment requires hospitalisation and is aimed at destroying occluding blood clots and revascularisation of the coronary arteries. Treatment of ACS is dependent upon differential diagnosis of myocardial ischaemic state and may include medication, thrombolytics, percutaneous coronary interventions (PCI) (angioplasty, stenting) or coronary artery bypass surgery (CABG). Survival after MI is continuously improving leading to an increasing population of post ACS survivors coping with the aftermath of their MI (Fox et al., 2007; Radovanovic et al., 2007). Most patients spend between 24-48 hours in the Coronary Care unit before being transferred to a general or cardiac hospital ward. Most patients are discharged after 4 – 7 days; however, length of stay is dictated by numerous clinical factors. After discharge, patients are encouraged to gradually increase their physical activity and begin implementing lifestyle changes immediately. Based on the National Service Framework for Coronary Heart Disease, within four weeks of discharge patients

should expect to undergo assessment and receive information about completing a programme of cardiac rehabilitation (NSF, 2000).

Acute cardiac events are often highly traumatic experiences for the patient and their family (Bennett & Connell, 1999). The experience of an acute cardiac event has been equated to the “*medical equivalent of a ride down the turbulent and dangerous white-water rapids portion of a river*” (Rosamond et al., 2007) which highlights the life threatening and uncontrollable nature of such an event. The acute event also signifies the beginning of a long period of physical rehabilitation, psychological adaption and lifestyle change. Successful recovery and long term prognosis have been found to be contingent upon numerous factors including demographic factors, clinical management, disease severity, and psychosocial factors (Bhattacharyya, Perkins-Porras, Whitehead, & Steptoe, 2007; Yap et al., 2008; Jaffe et al., 2006).

## **1.2 Psychosocial factors and CHD**

A psychosocial factor refers to a “*measurement that potentially relates psychological phenomena to the social environment and to pathophysiological changes*” (Hemingway & Marmot, 1999). Numerous psychosocial factors have been implicated in both the development of CHD and the prognosis of ACS including depression, anxiety, hostility, psychosocial work characteristics, socio economic status, stress, personality and social support (Hemingway & Marmot, 1999; Albus, 2010; Steptoe & Kivimaki, 2012). Depression, anxiety and social support are the central constructs underlying the hypotheses in this thesis and will form the focus of my literature review; the role of depression and anxiety in the development and progression of CHD are discussed in the following sections and the role of social support in the following chapter.

### **1.2.1 The role of depression and anxiety in the aetiology of CHD**

There is a long history of research documenting the increased risk of developing CHD associated with depression. In a meta-analysis of 11 studies incorporating 36,000 patients,

Rugulies, (2002) identified an overall relative risk of 1.64 (95% CI: 1.29 – 2.08) for the development of CHD in depressed but otherwise healthy individuals. They also identified that clinical depression was a stronger predictor than depressed mood suggesting a dose-response relationship. Likewise, in a systematic review of 10 studies, Wulsin & Singal, (2003) found an overall risk of depression for the onset of CHD of 1.64 (95% CI: 1.41 – 1.90). Nicholson, Kuper, & Hemingway, (2006) conducted a meta-analysis of 21 aetiological cohort studies including 124, 509 individuals with a mean follow up of 10.8 years. They identified a pooled relative risk of developing CHD associated with depression of 1.81 (95% CI: 1.53 – 2.1). However, they concluded that no firm conclusions could be drawn from this finding due to the methodological limitations of the research. These included publication bias, lack of adjustment for cardiovascular risk factors, confounding from cardiovascular risk factors that are also associated with depression (for example, smoking) and the inclusion at baseline of participants who had symptoms which may indicate subclinical or undiagnosed CHD (for example, chest pain). Further results supporting an association were identified in a meta-analysis of 28 studies conducted by Van der Kooy et al., (2007). They found an overall risk of 1.60 (95% CI: 1.34 – 1.92) for myocardial infarction in depressed individuals. However, they also highlighted the heterogeneous nature of the research body. They also noted that the strongest relationship was found between MI and clinically diagnosed Major Depressive Disorder (MDD) which is again indicative of a dose response relationship.

Studies that have been conducted since these reviews continue to support a predictive effect of depression. In a prospective cohort study utilising a sample of 2728 adults aged 60+ followed up for 15 years, Brown, Stewart, Stump, & Callahan, (2011) found that elevated symptoms of depression assessed using the Center for Epidemiological Studies Depression Scale (CES-D) were significantly predictive of CHD events [MI or cardiovascular mortality] (RR: 1.46, 95% CI: 1.20 – 1.77) and all-cause mortality (RR: 1.16, 95% CI: 1.01 – 1.33). The study is particularly noteworthy for its inclusion of a significant proportion of women and ethnic minority adults (whom have been previously under-represented in this research), and

the long follow up period. Similarly, Shah, Veledar, & Hong, (2011) found that in a sample of 7641 adults aged 17 – 40 followed up for nearly 15 years depression and history of attempted suicide were associated with cardiovascular mortality with an adjusted hazard ratio of 3.70 (95% CI: 1.32 – 10.35) for depression and 7.12 (95% CI: 2.67 – 18.98) for attempted suicide history.

There are numerous emerging research issues within this field including identifying the most cardiotoxic aspects of depression (Stewart et al., 2012) and the role of distress in the aetiology of CHD precursors (for example, subclinical atherosclerosis). The presence of gender differences in the CHD risk imposed by depression also remains controversial (Sevick, Rolih, & Pahor, 2000). There is also dispute regarding the age group most at risk of CHD from psychological distress. Most studies identifying positive prospective associations have utilised middle aged and older populations, however, a recent study by Shah et al., (2011) identified a robust link in a younger population (aged under 40). There are mixed findings regarding the risk of depression in elderly adults (80+). Rapp, Gerstorf, Helmchen, & Smith (2008) found that depression-CVD mortality association was present in the 70 – 84 year age group but not in the 85+ age group. However, this is not a consistent finding with other studies investigating the 85+ age group identifying depression as a significant predictor of CVD mortality (Vinkers, Stek, Gussekloo, Van Der Mast, & Westendorp, 2004). Despite methodological issues and unanswered questions, the strength and consistency of the evidence strongly supports an aetiological role for depression in the development of depression. Depression is now formally recognised as a risk factor for CHD with effects comparable to smoking (Charlson, Stapelberg, Baxter, & Whiteford, 2011).

The research on anxiety is less prolific and the findings are less consistent than for depression with some studies detecting an independent aetiological role for anxiety and others identifying limited association. In an attempt to clarify the findings, Roest, Martens, de Jonge, & Denollet, (2010) conducted a meta-analysis of 21 studies incorporating over 250,000 participants followed up for up to 20 years and identified a pooled HR for incident

CHD of 1.26 (95% CI: 1.15 – 1.38) and for cardiac mortality of 1.48 (95% CI:1.14 – 1.92). These findings are even more striking on consideration that the analysis excluded studies utilising a psychiatric cohort (whom would generally have had more severe symptoms). This meta-analysis also identified that the anxiety research was more methodologically robust than the depression research with nearly all studies including adjustment for confounders; however, there was considerable heterogeneity between studies. It should be noted that the analysis also excluded studies focusing on populations aged over 75 and it would be useful to determine the anxiety-CHD risk in an older population.

Anxiety and depression share numerous facets and exhibit substantial comorbidity. Recent psychiatric research has identified that around two thirds of patients presenting with either current anxiety or depression also have current comorbid depression or anxiety (Lamers et al., 2011). There has even been suggestion that both disorders should be considered as a single, broader construct of negative affect (Suls & Bunde, 2005; Bleil, Gianaros, Jennings, Flory, & Manuck, 2008). Emerging evidence suggests that comorbid anxiety and depression pose the greatest CHD risk compared to anxiety or depression. Vogelzangs et al., (2010b) recently reported that in a sample of 2315 individuals recruited from community, primary care and secondary mental health service, current comorbid anxiety and depression was associated with a 3.5 fold risk of CHD (OR: 3.54, 95% CI: 1.79 – 6.98). This compared to no significant impact of depression and a 2.70 fold increased risk of CHD associated with anxiety. However, the results were cross-sectional in nature and CHD risk was based on self-report rather than clinical diagnosis so the results should be interpreted with caution. In a sample of 11, 828 women who did not report heart disease at baseline, Berecki-Gisolf, McKenzie, Dobson, McFarlane, & McLaughlin, (2012) found that comorbid depression and anxiety were significantly associated with a 1.78 (95% CI: 1.41 – 2.24) increased risk of new onset heart disease during the 15 year follow up independent of hypertension, diabetes, menopausal status, physical activity, smoking status, BMI, age, marital status, education, area of residence and deprivation. The adjusted OR's for anxiety or depression alone were

not significant. These findings do suggest that comorbid depression and anxiety may pose the greatest cardiovascular risk compared to depression or anxiety alone. However, more longitudinal research utilising clinically diagnosed CHD and standardised measures of anxiety and depression is needed to determine the impact of comorbidity on CHD risk. The role of comorbidity is further discussed in Section 1.2.2 in the context of increasing risk of negative outcomes in existing CHD patients where there is a greater body of longitudinal research.

There are numerous explanatory mechanisms for the aetiological role of depression and anxiety in the development of CHD including behavioural factors and biological effects, and it has also been hypothesised that treating depression/anxiety may help to reduce cardiac risk (Nemeroff & Goldschmidt-Clermont, 2012; de Jonge et al., 2010). There is a greater research literature focusing on these issues within existing CHD patients and subsequently pathways and treatment effects will be discussed in the following sections within the context of post ACS patients.

### **1.2.2 The role of depression in recovery and prognosis after ACS**

ACS patients are particularly vulnerable to developing depression following ACS with approximately one third of patients experiencing clinically relevant depressive symptoms during hospitalisation (Thombs et al., 2006; Smolderen et al., 2009). Following discharge, approximately 20% of patients experience severe depression and a further 25% experience mild to moderate depressive symptomatology (Amin, Jones, Nugent, Rumsfeld, & Spertus, 2006; Lett et al., 2005). Rates of depression are substantially higher in cardiac patients compared with general population (<4%) and primary care population rates (<10%) (Glassman, Bigger, Gaffney, Shapiro, & Swenson, 2006; Lesperance et al., 2007). Depression in ACS patients is also persistent; rates remain stable over the first year with low symptom attenuation in patients who do not seek treatment (Martens, Smith, Winter, Denollet, & Pedersen, 2008; Frasure-Smith, Lesperance, Juneau, Talajic, & Bourassa, 1999;

Kaptein, de, van den Brink, & Korf, 2006). Risk of suicide, particularly during the month following discharge, has also been found to be significantly elevated with a three-fold risk recorded for patients with no prior psychiatric history and even greater risk among younger patients and patients with prior psychiatric history (Larsen, Agerbo, Christensen, Søndergaard, & Vestergaard, 2010). Despite these findings, depression in this population may be under recognised by primary care practitioners (Haws, Ramjeet, & Gray, 2011) with low rates of treatment uptake (Huffman et al., 2008) and uncertainty regarding how to address depression amongst CHD patients who often present with psychosocial issues (Barley, Walters, Tylee & Murray, 2012).

There is considerable research highlighting a robust prospective association between depression and post ACS outcomes. An early study by Frasure-Smith, Lesperance, & Talajic, (1993) found that depression identified using the BDI at 5 – 15 days post MI was a significant predictor of mortality at 6 months (OR: 5.74, 95% CI: 4.61 – 6.87). This effect remained after control for predictors of mortality (left ventricular dysfunction and previous MI) identified in the dataset (OR; 4.29, 95% CI: 3.14 – 5.44). A follow up of this study at 18 months post MI, Frasure-Smith, Lesperance, & Talajic, (1995) revealed that the association between depression and cardiac mortality persisted at 18 months even with adjustment for other predictors of mortality (OR: 6.64, 95% CI: 1.76 – 25.09).

Since these initial findings, a large number of studies have been conducted whose findings have been summarised in four key meta-analyses (Meijer et al., 2011; Barth, Schumacher, & Herrmann-Lingen, 2004; van Melle et al., 2004; Nicholson et al., 2006). The results indicate that depression is consistently associated with clinical recovery following ACS and is a significant predictor of cardiac and all-cause mortality with even mild depression conferring increased mortality risk. The meta-analyses identified a 2.0 – 2.7 increased risk of adverse outcomes in depressed ACS patients compared to non-depressed ACS patients. The most recent meta-analysis conducted by Meijer et al., (2011) identified 29 relevant studies which included 16, 889 MI patients followed up for an average of 16 months. Post MI depression

was associated with increased risk of all-cause mortality (OR: 2.25, 95% CI: 1.73 – 2.93), cardiac mortality (OR: 2.71, 95% CI: 1.68 – 4.36), and cardiac events (OR: 2.25, 95% CI: 1.37 – 1.85). It should be noted that a small number of studies have not found a depression-mortality association in ACS patients (Kornerup, Zwisler, & Prescott, 2011; Lane, Carroll, Ring, Beevers, & Lip, 2001a). However, the vast majority of studies have identified a robust effect. Research has also identified that those patients with more severe levels of depression occurring during hospitalisation, patients with treatment resistant depression, patients who experience worsening depressive symptomatology during the first year post infarction, and patients who have co-existing diabetes may be particularly vulnerable to the effects of depression on mortality and recurrent cardiac events (Lesperance, Frasura-Smith, Talajic, & Bourassa, 2002; Kaptein et al., 2006; Bot, Pouwer, Zuidersma, van Melle, & de Jonge, 2012; Glassman & Bigger, 2009).

There has been considerable debate regarding the differential prognostic impact of depression experienced for the first time after MI versus that of continued pre-existing depression as it is estimated that around half of patients experiencing depression will have had previous episodes of depression (Freedland, Carney, Lustman, Rich, & Jaffe, 1992; Spijkerman et al., 2005). Some research suggests that depression experienced for the first time following MI may be particularly pathogenic (Grace et al., 2005; Dickens et al., 2008; Spijkerman et al., 2005; de Jonge, van den Brink, Spijkerman, & Ormel, 2006). Recently, in a sample of 1328 MI patients from the Enhancing Recovery In Coronary Heart Disease (ENRICHD) clinical trial, Carney et al., (2009) found that patients experiencing a first episode of Major Depression (MD) after a MI had poorer survival than those experiencing recurrent MD (Hazard Ratio (HR): 1.4, 95% CI: 1.02). However, other studies have found pre-existing depression to be particularly damaging to recovery (Huffman et al., 2008; Lesperance, Frasura-Smith, & Talajic, 1996). One large study found baseline MD during the early weeks following ACS was the strongest depression related predictor of long term mortality (Glassman & Bigger, 2009). A recent systematic review of 6 studies highlighted the

inconsistency within the research and suggested that conclusions cannot yet be drawn regarding the most pathological form of depression in ACS patients (Zuidersma, Thombs, & de Jonge, 2011). The debate has also centred upon the importance of the timing of the depressive episode relative to the acute cardiac event rather than whether it was a first or recurrent episode. The research suggests that depression occurring within one month of the acute cardiac event has the greatest association with mortality and adverse outcomes, regardless of whether this episode was the first, a continuation of prior depression or a recurrent episode in those with a previous history of depression (Parker et al., 2008; Parker et al., 2011b). This effect was found to persist at both one and five year follow up.

Depression experienced by cardiac patients has also been found to be characterised by a different configuration of symptoms compared with other depressed patients, with cardiac patients more likely to report autonomic nervous system symptoms, cardiac symptoms and early morning insomnia (Fraguas, Jr. et al., 2007). Depression in cardiac patients, and more severe depression in non-cardiac patients, is often characterised by a preponderance of somatic symptoms (Lesperance et al., 2002; Hoen et al., 2010) whereas depression in stable CHD patients tends to be typified by more cognitive symptoms (Spijkerman et al., 2005; Martens et al., 2006). There have been considerably mixed findings regarding the differential prognostic impact of depression characterised by somatic symptomatology or depression characterised by cognitive symptomatology in cardiac patients, with a tendency towards a greater impact of somatic symptoms. For example, de Jonge et al., (2006) investigated the predictive role of symptoms of depression assessed using the BDI with regard to mortality and cardiac events in over 2000 patients and found that somatic/affective symptoms were significantly predictive of mortality and cardiac events independent of cardiac clinical risk factors. Appetitive and cognitive/ affective symptoms did not show this effect. Other studies have shown that anhedonia is the symptom of depression that is most predictive of cardiac prognosis (Doyle, 2010; Davidson et al., 2010)

In a recent comprehensive review of 14 studies, Carney & Freedland, (2012) confirmed these mixed findings and suggested a number of issues within the research that may be contributing to these inconsistent findings including the considerable variation in the definition of somatic and cognitive symptoms and the presence of a potential reporting bias whereby cardiac patients may be less willing to report cognitive symptoms of depression. They also suggested that the greater preponderance of somatic symptoms among cardiac and also among severely depressed patients may account for the more significant role of somatic symptoms in mortality prediction in cardiac patients. An overlap between somatic symptoms and vital exhaustion (defined as extreme fatigue, demoralisation and increased irritability) has also been suggested as a pathway through which somatic depression symptoms predict cardiac prognosis (Carney & Freedland, 2012; Poole, Dickens, & Steptoe, 2011; Vroege, Zuidersma, & de Jonge, 2012) as vital exhaustion has also been found to predict cardiac prognosis (Williams et al., 2010).

As a result of the overwhelming evidence indicating the profound impact of depression on cardiac outcome, it has been hypothesised that treating depression in cardiac patients may improve both depressive symptoms and cardiac prognosis. A number of research trials and reviews have been conducted investigating the impact of various forms of depression treatment including cognitive behavioural therapy and pharmacological treatments. In general, treatment had limited clinically relevant impact on depression symptomatology (Glassman et al., 2002; Berkman et al., 2003; van Melle et al., 2007; Lesperance et al., 2007; Thombs et al., 2008). Furthermore, treatment generally did not significantly improve short or long term physical outcomes for ACS patients (Berkman et al., 2003), although more recent analyses have indicated an impact of individual and group therapy on event free survival in MI patients (Saab et al., 2009). However, it should be noted that there is debate regarding the efficacy of depression treatment in general and psychiatric populations. Meta-analysis findings indicate only modest benefits of antidepressant treatment over placebo treatment (Turner, Matthews, Linardatos, Tell, & Rosenthal, 2008; Kirsch, Moore, Scoboria,

& Nichols, 2002), but recent review findings suggest a greater efficacy for antidepressant combined with psychotherapy treatment (Khan, Faucett, Lichtenberg, Kirsch, & Brown, 2012). The low to modest effectiveness of antidepressant therapy in general is highlighted as an important factor in a critique of this research literature by Carney & Freedland., (2007). They highlight that caution should be taken when interpreting the findings regarding the cardiac efficacy of treating depression because, as previously stated, treatment for depression in any population is not particularly effective. They also state that depression treatment may impact cardiac outcomes but not necessarily impact depression, for example, certain antidepressant medications can have cardiotoxic effects, and others have more cardioprotective effects. They also highlight that the key clinical trials were underpowered and considerably larger sample size are required. Recommendations for future research include identifying better treatments for depression in general, and also identifying the specific aspects of depression that are related to specific aspects of cardiac outcome to strengthen the association between the two factors (Carney & Freedland, 2007; de Jonge & van Melle, 2007). A study examining the efficacy of a personalised primary care intervention aimed at improving both physical and mental health outcomes among depressed symptomatic CAD patients is currently underway and will provide greater insight (Tylee et al., 2012). Depression after ACS does not just have a clinical impact but has also been found to significantly reduce physical health quality of life after ACS (Dickens, Cherrington, & McGowan, 2012).

### **1.2.3 The role of anxiety in recovery and prognosis after ACS**

Anxiety is also extremely prevalent among post ACS patients with around 70-80% of patients experiencing anxiety in the immediate aftermath, up to 50% reporting significant anxiety during hospitalisation and 20-25% of patients reporting persistent anxiety at one year post ACS (Januzzi, Stern, Pasternak, & DeSanctis, 2000; Moser & Dracup, 1996; Crowe, Runions, Ebbesen, Oldridge, & Streiner, 1996). Similar to depression, anxiety is significantly under recognised and under treated in this population (Januzzi et al., 2000). Clinical anxiety

disorder prevalence is also elevated within CHD populations with around 26 - 36% of patients meeting diagnostic criteria for a current anxiety disorder and a 42 - 46% lifetime prevalence of anxiety disorder with a higher prevalence among female compared to male cardiac patients (Todaro, Shen, Raffa, Tilkemeier, & Niaura, 2007).

There have been mixed findings regarding the impact of anxiety symptomatology on mortality and adverse cardiac events in CHD populations with some studies reporting a positive association (Rothenbacher, Hahmann, Wusten, Koenig, & Brenner, 2007; Moser et al., 2007; Moser et al., 2011; Frasure-Smith & Lesperance, 2008; Martens et al., 2010), other studies finding no predictive impact (Mayou et al., 2000; Lane, Carroll, Ring, Beevers, & Lip, 2002a) and a few studies reporting a positive impact of anxiety on cardiac prognosis (Meyer, Buss, & Herrmann-Lingen, 2010; Herrmann, Brand-Driehorst, Buss, & Ruger, 2000). A recent review by Roest, Martens, Denollet, & de Jonge, (2010) of 12 studies investigating the role of anxiety symptomatology on mortality and morbidity in MI patients found a strong impact of anxiety. Anxiety (measured within 3 months of hospitalisation using standardised measures) was associated with elevated risk of adverse cardiac events (OR: 1.36, 95% CI: 1.18 – 1.56), all-cause mortality (OR: 1.47, 95% CI: 1.02 – 2.13), cardiac mortality (OR: 1.23, 95% CI: 1.03 – 1.47) and new cardiac events (OR: 1.71, 95% CI: 1.31 – 1.23). The authors concluded that anxiety was consistently associated with mortality and adverse cardiac events; however, they stated that the current research base is small and the degree to which this association is independent of other disease and psychological factors remains unclear. Moser et al., (2011) investigated the role of anxiety in CHD prognosis in a sample of over 3000 CHD patients and found that persistent anxiety (defined as anxiety during hospitalisation and at 3 months post discharge) predicted outcomes independent of age, gender, ethnicity, living alone, marital status, education, previous MI, diabetes, hypertension, BMI, sedentary lifestyle, smoking and depressive symptoms. A dose response relationship between anxiety and adverse outcome was observed whereby the highest risk was conferred by persistent anxiety, intermediate risk associated with anxiety at one time point

only and lowest risk associated with no anxiety. Anxiety has been also found to reduce quality of life in post ACS patient (Dickens et al., 2006; Lane et al., 2001a).

Emerging evidence also suggests that different anxiety disorders may be associated with different prognostic trajectories in CHD patients with some anxiety disorders conferring greater risk than others. Generalised Anxiety Disorder has been the focus of most research and has been associated with both a significant increased risk of adverse outcome (Roest, Zuidersma, & de Jonge, 2012; Martens et al., 2010) and a decreased risk of adverse outcome (a protective effect) compared with other anxiety disorders (Parker, Hyett, Hadzi-Pavlovic, Brotchie, & Walsh, 2011a) in ACS and CHD patients. Phobic anxiety has been associated with a 1.6 fold increased risk of cardiac mortality and a two-fold increased risk of sudden cardiac death in female CHD patients but not in male patients (Watkins et al., 2010). Elevated mortality risk in ACS patients has also been associated with lifetime diagnosis of agoraphobia (Parker et al., 2010). Thus, it appears that anxiety as a symptom and anxiety as a clinical disorder poses considerable risk to ACS and CHD patients. More research is needed to investigate potential protective effects of GAD.

As discussed in the previous section, comorbidity of anxiety and depression is extremely common and may potentially represent a single construct of negative affect. Research suggests that anxiety symptomatology may form an integral part of depression after ACS. In a sample of 176 post MI patients, Denollet, Strik, Lousberg, & Honig, (2006) identified that mixed anxiety-depressive symptomatology assessed using the Symptoms of Anxiety-Depression Index was present in 90% of depressed and 100% of severely depressed post ACS patients. Similarly, Frasure-Smith & Lesperance, (2008) found that in a sample of over 800 post ACS patients 77% of post ACS patients with high depression scores had high anxiety scores. In stable CHD populations, some research suggests no increased mortality risk associated with comorbid anxiety and depression (Frasure-Smith & Lesperance, 2008), whereas other studies have found increased risk of adverse cardiac events associated with composite depression-anxiety scores (Watkins et al., 2006). Recently, Rutledge et al.,

(2009) assessed the independent and combined impact of depression and anxiety in a sample of 489 female suspected MI patients followed up for a median of 5.9 years. Their findings reveal that depression was significantly predictive of adverse cardiac events in patients with low anxiety scores (HR: 2.3; 95% CI: 1.3 – 3.9) but not in patients with elevated anxiety scores (HR: 0.99, 95% CI: 0.70 – 1.4) suggesting the expediency of considering the synergistic effects of depression and anxiety on prognostic outcome. Recently, Celano et al., (2012) found that elevated baseline anxiety was independently associated with less improvement in depression symptoms and persistence of depression at 6 month follow up in a cohort of depressed cardiac patients. Further research is needed to clarify the extent to which depression and anxiety are independent or shared disorders following ACS, and to identify the different prognostic trajectories associated with independent or shared effects.

There are few studies investigating the impact of treating anxiety on anxiety remission and reduction of cardiac risk. One randomised controlled trial was identified that investigated the role of a 6 month psychotherapy treatment on a sample of CHD patients with elevated anxiety. Anxiety was found to significantly reduce over time, but no treatment effect was observed (Merswolken, Siebenhuener, Orth-Gomer, Zimmermann-Viehoff, & Deter, 2011). A Cochrane review found limited support for music based intervention to reduce anxiety and distress in CHD patients (Bradt & Dileo, 2009). However, a telephone based counselling intervention for post ACS patients did illustrate improvements in anxiety (as well as depression) at 6 month follow up (McLaughlin et al., 2005). More research is needed to establish the impact of different types of treatment on anxiety and how this may impact upon cardiac morbidity and mortality.

### **1.3 Pathways between psychological factors and recovery and prognosis after ACS**

In this section, the potential pathways that link the depression and anxiety to post ACS prognosis are described. Pathways linking social support and post ACS prognosis will be discussed in detail in Chapter 2. It is possible that patients who experience a severe myocardial infarction are more vulnerable to depression and/or anxiety and would

subsequently have a worse prognosis. In order to control for such a confounding effect, the vast majority of studies investigating the prognostic impact of distress include measures of disease severity within their multivariate predictive models and distress has frequently emerged as independent from disease severity. However, it should be noted that the typical measures of disease severity have been criticised as being heterogeneous, inaccurate and exclusive of patients own perceptions of the severity of their condition suggesting that studies linking distress to prognosis may be vulnerable to residual confounding by disease severity (Lane, Ring, Lip, & Carroll, 2005). Importantly, disease severity has not been found to be substantially correlated with depression or anxiety in post ACS patients (Doyle, McGee, Conroy, & Delaney, 2011; Carney, Freedland, Miller, & Jaffe, 2002; Denollet & Brutsaert, 1998). In addition, studies using comprehensive indices such as the Global Registry of Acute Cardiac Events (GRACE) risk score have shown that associations between depression and later cardiac morbidity are independent of clinical cardiac risk (Kronish, Rieckmann, Schwartz, Schwartz, & Davidson, 2009). Further research is required to explore the most accurate way in which to assess cardiac severity and to utilise this to determine the independence of the distress-prognosis link from cardiac severity. However, even if there were a relationship between cardiac severity and distress, it is unlikely that cardiac severity would account for all of the variance in depression and anxiety symptoms.

The key pathways that have been proposed to link distress and poor prognosis following ACS tend to be related either to increased cardiac risk factors or to biological mechanisms which will be discussed in the following sections.

### **1.3.1 Distress and increased cardiac risk factors**

Health behaviour modification following ACS is an essential component for recovery and has a strong influence on prognosis. Patients are encouraged to engage in regular exercise, to give up smoking, to eat a healthy, low fat diet and to reduce stress. However, the experience of distress, in particular depression, is associated with poorer health behaviour among

cardiac patients including reduced smoking cessation (Dawood et al., 2008; Gerber et al., 2011; Kuhl, Fauerbach, Bush, & Ziegelstein, 2009), increased smoking cessation relapse (Busch, Borrelli, & Leventhal, 2012), lower adherence to a low fat diet (Romanelli, Fauerbach, Bush, & Ziegelstein, 2002; Ziegelstein et al., 2000; Bonnet et al., 2005; Murphy et al., 2012), and less engagement in regular physical activity (Romanelli et al., 2002; Ziegelstein et al., 2000; Blumenthal et al., 2004; Bonnet et al., 2005; Murphy et al., 2012). Depression is also associated with reduced exercise tolerance (Marchionni et al., 2000) and less stress management (Romanelli et al., 2002; Ziegelstein et al., 2000) in post ACS patients. Further evidence for a health behavioural pathway between distress and prognosis comes from studies that have found interaction effects between health behaviour and depression on prognosis. Chrysohoou et al., (2011) found that depression was related to significantly worse 30 day prognosis in a consecutive sample of 277 ACS patients aged >65. However, this relationship was mediated by adherence to a Mediterranean diet. Furthermore, a recent prospective cohort study by Whooley et al., (2008) identified that behavioural factors (in particular, physical inactivity) were the most significant factors in explaining increased risk in mortality associated with depressive symptomatology in a sample of 1017 stable CHD patients. Other cardiac risk factors are also elevated in depressed and anxious patients and may be accounted for by behavioural, genetic factors and environmental features. Increased prevalence of hypertension among depressed individuals has been noted (Adamis & Ball, 2000). There is also research to suggest that depression and anxiety may be risk factors for development of hypertension (Meng, Chen, Yang, Zheng, & Hui, 2012), obesity (Blaine, 2008) and diabetes (Knol et al., 2006). Depression is considerably more prevalent in individuals with diabetes, and diabetes has been associated with a 2 – 3 fold increased risk of cardiac mortality (Anderson, Freedland, Clouse, & Lustman, 2001). Comorbid diabetes and depression are also associated with increased numbers of other cardiac risk factors (Katon et al., 2004) and also with significantly higher mortality after ACS (Bot et al., 2012).

Following an ACS, patients are also encouraged to follow a medication regime which may include a combination of aspirin, a beta-blocker, an ACE (angiotensin-converting enzyme) inhibitor and a statin that would need to be taken every day for life. Non-adherence to this medication regime has been found to significantly increase the risk of adverse cardiac events and mortality in ACS patients (Choudhry & Winkelmayr, 2008; Horwitz et al., 1990). The experience of depression after an ACS has been strongly associated with poorer medication adherence with depressed patients 3 times more likely to be non-adherent than patients who are not depressed (DiMatteo, Lepper, & Croghan, 2000). A dose-response relationship between depression severity and adherence has been noted with more severe depression associated with greater non-adherence (Rieckmann et al., 2006). There is less evidence indicating an association between anxiety and adherence. In a meta-analysis, DiMatteo et al., (2000) found limited evidence of an association between anxiety and adherence in ACS patients.

Cardiac rehabilitation is also an important facet of current ACS patient care which is associated with better prognosis and reduced risk of adverse cardiac events (Jolliffe et al., 2001; Taylor et al., 2004; Dalal, Zawada, Jolly, Moxham, & Taylor, 2010). Depression and anxiety following ACS have been associated with poorer rates of attendance and higher rates of drop out from cardiac rehabilitation, with major depressive disorder associated with a 2.5 fold increased risk of non-attendance (Glazer, Emery, Frid, & Banyasz, 2002; Swardfager et al., 2011; Lane, Carroll, Ring, Beevers, & Lip, 2001b; McGrady, McGinnis, Badenhop, Bentle, & Rajput, 2009; Casey, Hughes, Waechter, Josephson, & Rosneck, 2008).

Overall, there is a significant relationship between increased distress after ACS and elevated cardiac risk factors which increases risk of morbidity and mortality. Distressed patients also tend to be less adherent to medication regimes and are less likely to attend and complete cardiac rehabilitation which further compounds risk. It should be noted that the majority of the research discussed here has focused primarily upon depression and cardiac risk factors,

with less research focusing on the role of anxiety. More research is required to determine the extent of association between anxiety and cardiac risk factors in post ACS patients. Increased levels of cardiac risk may provide partial explanation for distress differentials in post ACS prognosis. However, these factors do not account for all the variance and the relationship between distress and prognosis is far more complex and multifactorial.

### **1.3.2 Distress and biological mechanisms of risk**

Psychological distress has been found to have an extensive effect on a diverse range of biological cardiovascular mechanisms. Inflammation plays a key role in the development of atherosclerosis and post ACS prognosis. An ACS is itself associated with a huge inflammatory response and high levels of inflammatory markers have been found to be significantly predictive of post ACS mortality (Mulvihill & Foley, 2002). Both depression and anxiety have also been associated with higher levels of these inflammatory markers in clinical and community samples (Pitsavos et al., 2006; Maes, 2011). A recent meta-analysis found that the relationship between depression and various inflammatory markers has a dose-response nature whereby more severely depressed individuals have the highest levels of inflammation but even those with low levels of depression exhibited elevated inflammation (Howren, Lamkin, & Suls, 2009). In a recent review of the role of inflammation in CHD and depression, Poole et al., (2011) propose an acute inflammation model that emphasises inflammation as a common causal pathway in both the development of depression and the development or worsening of CHD. They argue that the depression observed in CHD patients is qualitatively different to depression identified in psychiatric patients, and that acute inflammation is a critical component in both the genesis of depression and adverse outcomes in CHD patients.

Elevated inflammatory markers have also been significantly associated with reduced heart rate variability (HRV) (Frasure-Smith, Lesperance, Irwin, Talajic, & Pollock, 2009; Steptoe et al., 2011). Reduced HRV reflects excessive sympathetic and/or insufficient parasympathetic

tone suggesting a lack of cardiac responsiveness to situational and emotional demands and reflects dysregulation of the autonomic nervous system (ANS). A more detailed description of HRV is provided in Chapter 3. Low HRV is a robust independent predictor of mortality in both stable CHD patients and post ACS patients, and has also been associated with depression. Kemp et al., (2010) conducted a meta-analysis on the relationship between depression and HRV in physically healthy individuals. They identified a significant association between depression and reduced HRV, with depression severity negatively correlated with HRV. They also found that antidepressant treatment had limited impact on HRV which remained reduced even when the depression symptoms had remitted. A similar relationship between depression and HRV has also been found in both stable CHD and post ACS patients (Stapelberg, Hamilton-Craig, Neumann, Shum, & McConnell, 2012; Carney & Freedland, 2009). There is less research explicitly exploring anxiety-HRV pathways. Kemp, Quintana, Felmingham, Matthews, & Jelinek (2012) found that in a physically healthy population, MDD was associated with reduced HRV. In those with comorbid GAD, HRV was further reduced indicating that comorbid anxiety and depression have particularly negative impact. Licht, de Geus, van Dyck, & Penninx, (2009) identified significantly lower HRV in patients with diagnosed anxiety disorders (panic disorder, social phobia, GAD) compared to non-anxious controls; however, the association was found to be the result of antidepressant use.

Anti-depressant use is a problematic confounding variable in research investigating distress-HRV links as current use of anti-depressants in physically healthy populations reduces HRV (Licht, de Geus, van Dyck, & Penninx, 2010). However, anti-depressant use has also been found to alleviate HRV impairment in treated depressed post ACS patients (Glassman, Bigger, Gaffney, & van Zyl, 2007). Another consequence of dysregulated ANS activity is increased levels of catecholamines which have been identified in both depression and anxiety (Ressler & Nemeroff, 2000), and are associated with numerous cardiotoxic states

including vasoconstriction, arrhythmia and high blood pressure which may impair post ACS recovery (Amadi, Ponikowski, & Coats, 1995).

A further biological mechanism concerns the dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis which has been extensively documented as a factor in the pathophysiology of depression. Research has identified elevated daytime cortisol levels, blunted cortisol awakening response and greater corticotropin-releasing and adrenocorticotropin hormone levels in depressed physically healthy individuals (Vreeburg et al., 2009; Nemeroff & Vale, 2005; Broadley et al., 2006) and in depressed CHD patients (Bhattacharyya, Molloy, & Steptoe, 2008; Messerli-Burgy et al., 2012). Although the results are not completely consistent with some studies identifying no relationship between HPA axis indicators and depression (Stetler & Miller, 2011). Emerging evidence suggests similar patterns of HPA disruption in anxiety and anxiety disorders (Vreeburg et al., 2010), although the research is less extensive. Various manifestations of HPA dysfunction has been identified in the aetiology and progression of CHD. Elevated 24 hour urinary cortisol was independently associated with increased cardiovascular mortality in a 6 year follow up study of older adults (Vogelzangs et al., 2010a). Nijm, Kristenson, Olsson, & Jonasson, (2007) found higher 24 hour cortisol secretion and a flattened diurnal slope in CAD patients due to significantly elevated evening cortisol, compared to healthy controls. Evening cortisol was significantly associated with increased inflammation which, as previously discussed, is also a risk factor for poor ACS outcome. Elevated cortisol levels and increased cortisol reactivity to stress have been implicated in the development of sub-clinical atherosclerosis which increase the risk of ACS (Hamer, O'Donnell, Lahiri, & Steptoe, 2010; Hamer, Endrighi, Venuraju, Lahiri, & Steptoe, 2012). Thus, the shared HPA axis dysfunction associated with both distress and CHD aetiology and progression presents a potential causal pathway.

## 1.4 Chapter Summary

There is strong evidence to suggest that depression and anxiety independently contribute to both the genesis of CHD and to the prognosis of patients with diagnosed CHD and ACS. Following an ACS, patients who experience distress are more likely to have reduced quality of life and are at considerably higher risk of morbidity and mortality in the short and long term. Comorbid depression and anxiety appears to pose the greatest risk; although more research is needed to understand the specific and synergistic aspects of depression and anxiety that are particularly cardiotoxic. Disappointingly, pharmacological treatment interventions have not been particularly successful in reducing the prognostic burden of distress. However, this is most likely a reflection of the lack of efficacious treatments for distress in general. Furthermore, recent research has begun to identify a positive prognostic impact of psychological treatment for distressed cardiac patients and further studies are currently underway. Numerous pathways have been proposed to explain the link between distress and cardiac outcome including adverse health behaviours, increased cardiac risk factors, and biological mechanisms including HPA axis dysfunction and ANS disturbance. Thus, it is clear that the psychological response of a patient following ACS significantly contributes to their outcome. This response rarely evolves in isolation. Instead it is influenced and compounded by the responses of those around the patient and the support they receive. In the next chapter, I will examine in more detail the role of the social support in recovery after ACS.

## CHAPTER 2 LITERATURE REVIEW

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### **Social support, marriage and CHD**

#### **2.1 Social support**

Social support refers to the support an individual has or believes they have from other people. Social support is a notoriously difficult concept to define because it consists of many different components and levels, and has been described as a “meta-construct” because of its composition from sub-constructs (Heller & Swindle, 1983). The interdisciplinary nature and wide proliferation of social support research has also led to variation and lack of consensus regarding the definition and operationalisation of social support (Thoits, 1982). However, the general unanimity is that social support can be delineated into two categories: (1) structural or network social support which refers to the type, size, density and frequency of contact within an individual’s social network, and (2) functional social support which refers to the types support available from an individual’s social network (Lett et al., 2005). This dual categorisation will form the basis of my further discussion of social support.

##### **2.1.1 Structural social support**

Structural social support refers to the degree to which an individual is integrated into a social network and can be established via exploration of the composition and interconnectedness of this social network. The concept of structural social support and social integration is derived from Durkheim’s (1951) early exploration of suicide and the social environment which identified a greater prevalence of suicide among unmarried and socially isolated individuals. Durkheim proposed that lack of, or weak social bonds may cause suicide suggesting that social bonds are important to social integration (attachment to others within society) and social regulation (attachment to society’s norms) which were suggested as central to mental health and wellbeing. More recent theories expand this concept and propose that our social connections and roles provide structure, norms, purpose and meaning to life that govern and shape our behaviour, emotions and relationships over the lifespan (Thoits, 2011; Stryker & Burke, 2000; Thoits, 1983). There are many aspects of

structural social support that can be assessed, from the basic composition of an individual's social network and the degree of contact with each member to more complex examination of the centrality (the importance of each member based on the extent of interconnectedness) and density (interconnectedness of each member of the network) of the social network. In response to the multidimensional nature of structural social support, a diversity of different measures have been developed to capture the numerous aspects of structural social support (Brissette, Cohen, & Seeman, 2000). A key facet of many of these measures is the assessment of marital or partner status which is the most commonly used method of assessing structural social support within research. The characteristics of social networks tend to vary with age. Younger people tend to have networks populated more by friends whereas older people tend to report more family members within their network (Levitt, Weber, & Guacci, 1993). A recent meta-analysis by Wrzus, Hanel, Wagner, & Neyer, (2012) investigating the typical size and composition of social networks across the life span found that social network size tended to increase in size until young adulthood and then decreased over the rest of the lifespan. Family network size remained stable from adolescence until old age. Interestingly, they also found that average personal and friendship network size has decreased steadily over the last 35 years. The age related decline in structural social support (which is not accompanied by a reduction in functional social support) has been explained by Socioemotional Selectivity theory. This theory provides an explanation for motivation over the life course and proposes that as individuals age, they become much more selective about emotional resources and tends to focus more upon personally satisfying and meaningful activities. Applying this theory to social network, a reducing social network over time may be driven by a need to main only emotionally satisfying relationships (Carstensen, Isaacowitz, & Charles, 1999).

There have been mixed research findings with regard to gender differences in social network size and composition although current research suggests that women tend to have larger social networks across the lifespan (McLaughlin, Vagenas, Pachana, Begum, & Dobson,

2010; Ajrouch, Blandon, & Antonucci, 2005). Gender differences in network composition have also been noted. Women's social networks tend to be characterised by more kin and friendship ties whereas men's networks are more dominated by employment ties (Moore, 1990). Race differences in network composition have also been noted with minority groups reporting smaller networks composed of more family members with whom there is higher contact, and fewer non-family members compared to white groups (Ajrouch, Antonucci, & Janevic, 2001; Small, 2007). Social networks are dynamic and transform in size and composition according to numerous life course transitions including marital transition, parenthood and employment alterations (Song, 2012; Kalmijn, 2003; Bost, Cox, Burchinal, & Payne, 2002; Szinovacz & Davey, 2001; Kalmijn, 2012). The emerging field of network analysis which analyses the dynamics between ties in a single network has revealed how behaviours and emotions (including smoking, alcohol consumption, obesity, happiness and cooperative behaviour) can be transmitted through individuals within a single social network (Christakis & Fowler, 2007; Christakis & Fowler, 2008; Fowler & Christakis, 2010; Fowler & Christakis, 2008; Rosenquist, Murabito, Fowler, & Christakis, 2010).

A key issue within social network research is the idea of homophily within networks which alludes that an individual's network will tend to consist primarily of individuals who are similar to them and each other. The findings regarding the presence of network homophily are robust and appear to apply across a wide range of network types (marital, friendship, workplace). However, the extent of homophily varies according to the social dimension examined. For example, network homophily according to race tends to be fairly consistent across relationship types whereas homophily based on age is more variable according to the type of social tie. In general, there is strong evidence supporting various manifestations of homophily according to race, age, religion, gender, education, occupation and even personal values (McPherson, Smith-Lovin, & Cook, 2001).

There are limitations to purely focusing on social networks. Structural measures do not acknowledge that some social roles may be health impairing. For example, some social

roles may expose an individual to risky health behaviours, or may be sources of stress themselves (Burg & Seeman, 1994; Rook, 1984). Most structural measures do not enable “weighting” of relationships within a social network whereby certain relationships are likely to be more salient than others in different circumstances, and therefore exert greater influence over health and behaviour (Norton, Stephens, Martire, Townsend, & Gupta, 2002; Styker, 1987). There is also emerging evidence suggesting a differentiation between voluntary social roles and obligatory social roles whereby voluntary social roles appear to afford most health and wellbeing benefit (Berbrier & Schulte, 2000).

### **2.1.2 Functional social support**

Functional social support refers to the type of support available from an individual’s network. The most common types of social support include emotional support, informational support, belonging support, and tangible or practical support (Lett et al., 2005; Barrera, 2000). Emotional support refers to affection, care and concern. Informational support refers to advice and guidance. Belonging support refers to having others to engage in shared social activities with. Finally, tangible support refers to practical and material help. Based on the matching hypothesis (Cutrona & Russell, 1990; Dunkel-Schetter & Bennett, 1990), the efficacy of each support type may depend upon the extent to which that support matches the situational demands suggesting that different support types may be more beneficial in some circumstances and less effective in others (Lindorff, 2005; Pennix et al., 1998).

Functional support is usually further disaggregated into two main categories: (1) received functional support which describes the support an individual actually receives, and (2) perceived functional support which describes an individual’s perception of available support (Lett et al., 2005). Measures of received functional support are considered more accurate appraisals of support because of the requirement to recall specific examples of support (Barrera, 1986) whereas measures of perceived support are more subjective and vulnerable to cognitive distortion (Lakey & Drew, 1997). Measures of perceived social support are

robustly and consistently related to morbidity and mortality (Barrera, 2000; Wills & Shinar, 2000; Uchino, 2009). However, the findings for received support are more complex with a number of studies even suggesting a negative impact of received support on mortality (for example, Kaplan et al., 1994; Krause, 1997). Uchino (2009) highlights the possibility that these findings may be due to poorer health status among individuals reporting higher received support but also states that most studies control for initial health status reducing the possibility of such confounding. In a review of functional support, Uchino, (2004b) proposed a number of explanations to account for the differential effect of perceived versus received support including poor quality of received support, the potentially detrimental effect on self-esteem of actually receiving help and finally, that individuals who receive most help may be those who are under most stress. Received and perceived functional support are separate constructs that are not closely correlated with research suggesting that received support does not account for the relationship between perceived support and health (Haber, Cohen, Lucas, & Baltes, 2007; Kaul & Lakey, 2003; Lakey et al., 2002). Perceived support is typically more stable over time and it has been suggested is rooted in early childhood and familial processes whereas received support is more mutable varying by situation and less influenced by early life experiences (Uchino, 2009).

Research has also found that sociodemographic factors influence the way functional social support is perceived and received, although the research is currently limited. There is evidence to suggest that women perceive, receive and provide greater emotional social support than men (Matthews, Stansfeld, & Power, 1999; Fuhrer & Stansfeld, 2002; Flaherty & Richman, 1989). Men are significantly more likely than women to nominate their spouse as their closest person and women report having more close persons within their network indicative of greater availability of emotional support (Fuhrer & Stansfeld, 2002). Culture has also been found to be important to the perception of social support (Glazer, 2006). Social relationships are strongly governed by social norms and these vary considerably according to culture. For example, independence and autonomy are greatly valued social norms

amongst North American and European cultures which contrasts with the greater value attached to collectivism and interdependency encouraged by Eastern cultures. Thus, these types of social and cultural norms influence how individuals seek and perceive social support. A recent study by (Kim, Sherman, & Taylor, 2008) found that Asian American groups are less likely to disclose stressful personal events to garner support and are generally less likely to ask for support due to concerns about how this may be interpreted compared to European Americans. Seeking social support has been associated with greater problem resolution among European Americans and less problem resolution and greater stress among Asian Americans (Kim, Sherman, Ko, & Taylor, 2006; Wang, Shih, Hu, Louie, & Lau, 2010). Other studies have highlighted the tendency for ethnic minorities to have greater reliance on familial ties for social support and lower perceived support from friends compared to white groups (Almeida, Molnar, Kawachi, & Subramanian, 2009; Ajrouch et al., 2001). However, these findings have not been consistent and many studies have not adjusted for SES which often ameliorates racial differences in support (Griffin, Amodeo, Clay, Fassler, & Ellis, 2006). Both gender and cultural differences in social support are intrinsically associated with socioeconomic status and a number of studies have highlighted a complex interplay between these factors (for example, Bartley, Martikainen, Shipley, & Marmot, 2004). Age has also been found to influence how functional social support is perceived and utilised. In a study of 1103 older individuals Shaw, Krause, Liang, & Bennett, (2007) found that perceptions of emotional support remained stable with increasing age; however, practical support increased with age. They also found declining levels of contact with friends, as well as decreasing ability to provide practical support to others. Over the lifespan, individuals encounter numerous life transitions and critical events (e.g. marriage, retirement) and these factors have been found to influence perceptions and sources of social support (Kalmijn, 2012; Bost et al., 2002). Overall, although there are considerable gaps in the literature, functional social support can be observed as dynamic varying according to numerous sociodemographic and lifespan factors.

A final distinction within functional support refers to an individual's general perception of the overall support available to them versus the specific sources of support and the centrality of these sources (Uchino, 2004c). Friends, close and distant relatives, work colleagues, neighbours, health care providers, children and partners are all potential sources of support. It is intuitive to suggest that certain support sources are likely to be more effective or important than others contingent on numerous individual, relationship, sociocultural and lifespan factors. The marital relationship is the most common adult relationship and an individual's spouse is regarded as a particularly central and potent source of support, particularly among middle age groups (for example, Kirkevold, Gortner, Berg, & Saltvold, 1996). Marriage and marital status will be discussed in the following section.

### **2.1.3 Marital status and satisfaction**

Marriage has been defined as a social institution that serves a multitude of functions ranging from child rearing to sharing resources (Waite, 1995). Marriage may provide a fulfilling intimate relationship that affords significant social support, intimacy and a sense of purpose in life (House, 1988). Married people made up 48.2% of the adult population in 2010 in England and Wales, with 35.5% being single, 9.3% being divorced and 7% being widowed. Marriage has been in decline since 1970 which has been attributed to more people delaying marriage and more people choosing to cohabit. It is estimated that 90% of men and 94% of women born in 1930 had ever married by age 40 which compares with 63% of men and 71% of women born in 1970 (Office of National Statistics, 2010). Thus, a greater proportion of middle aged to older people are married compared to younger generations.

Marital status is one of the most commonly used assessments of basic structural social support. However, marriages vary considerably in quality and assessment of marital quality in terms of both marital satisfaction and marital conflict provides greater insight into the qualitative elements of marital relationships. Both marital satisfaction and conflict have been the focus of a large research literature over the last 30 years. Kamp Dush & Taylor, (2012)

describe how marital quality and conflict are associated with each other, but are separate constructs that are not necessarily polar opposites. They also point out that there is limited longitudinal research on marital conflict compared to marital satisfaction and suggest that this may be due to difficulties in assessing marital conflict over a long period and also the overlap of aspects of conflict within measures of marital satisfaction.

Marital satisfaction has also been found to be affected by numerous life events including parenthood, traumatic events and economic difficulties (Bradbury, Fincham, & Beach, 2000; Twenge, Campbell, & Foster, 2003). Age has also been found to be important with recent longitudinal research suggesting a slightly curvilinear relationship between age and mean marital satisfaction with significant declines noted in the early and late marital years (VanLaningham, Johnson, & Amato, 2001). Research has also begun to identify particular types of married couples based on levels of marital satisfaction who each have different marital satisfaction trajectories (for example, Beach, Fincham, Amir, & Leonard, 2005; Kamp Dush, Taylor, & Kroeger, 2008).

As mentioned, there are limited longitudinal studies investigating trajectories of marital conflict; however, Umberson, Williams, Powers, Liu, & Needham, (2006) found a general increase in marital conflict and negative interaction over an 8 year follow up of 1, 049 married couples. Kamp Dush & Taylor, (2012) documented a more stable trajectory for marital conflict over a 20 year follow up of 2031 married individual. However, using latent class analysis they identified three key conflict trajectories. High conflict couples exhibited a gradual increase in conflict over the first 8 years, followed by a steady decline. Medium conflict couples showed a fairly stable conflict trajectory with a slight increase in conflict toward the end of the study. Low conflict couples displayed stable levels of low conflict throughout the follow up.

High conflict alone may not be a predictor of low marital satisfaction as Fincham & Beach, (2010) point out in their recent review of marital processes including marital conflict. They

note that studies have found that it is the interaction between marital conflict and positive marital behaviours that is important. For example, Janicki, Kamarck, Shiffman, & Gwaltney, (2006) found that high levels of conflict combined with low levels of positive affect predicted rapid declines in marital satisfaction whereas high levels of positive affect appeared to shield the negative satisfaction effects of high conflict. Fincham & Beach (2010) also emphasise the importance of considering the external context of the couple as research has found that both early familial interaction and current situational factors have profound influence on levels of conflict and satisfaction within marriage.

Marital satisfaction has been found to vary according to various sociodemographic factors. In general, research has indicated greater marital happiness in women compared to men, although the findings are not completely consistent with some studies suggesting no gender differences and others noting a recent decline in the gap between male and female marital satisfaction (Corra, Carter, Carter, & Knox, 2009). Lower marital satisfaction has also been noted among ethnic minorities (Corra et al., 2009). Poorer marital satisfaction has also been robustly associated with lower SES assessed in a variety of different ways including income, educational attainment and financial stability (Conger, Conger, & Martin, 2010). The division of household labour and perceived equity within the marital relationship has also been found to be a significant predictor of marital satisfaction (Stohs, 2000; Blair, 1998; Mikula, 1998). The importance of considering the interaction between race, gender, SES and division of household labour in predicting marital satisfaction has also been highlighted (Dillaway & Broman, 2001).

Marriage has been associated with numerous socioeconomic and psychological benefits including increased wealth over time (Zagorsky, 2005), higher family income (Waite & Gallagher, 2000), higher earnings for men (Pollmann-Schult, 2011), greater happiness ((Waite & Gallagher, 2000) and improved health and wellbeing in children born into marriage (Brown, 2010). The greatest and most robust benefit of marriage is improved physical health, mental health and longevity which will be discussed in more detail in Section 2.2.

However, the extent to which these benefits are a direct outcome of marriage or whether they are the products of the type of person selected into marriage is controversial and will be further discussed in Section 2.4. Conversely, poor marital quality has been associated with negative social and health outcomes including with increased atherosclerosis (Gallo et al., 2003), increased risk of psychiatric disorder (Whisman & Uebelacker, 2006), greater functional limitation and distress associated with poor vision (Bookwala, 2011) and poorer outcomes for adolescent children of poor quality married couples (Hair et al., 2009). There is an emerging research investigating the difference in these benefits between cohabitation and marriage; however, this research will not be discussed within my thesis as the population affected by ACS tends to be aged 50+ for whom cohabitation is less prominent.

## **2.2 Social support, marriage and health**

The relationship between social relationships and health has been a feature of scientific research for many decades. In 1988, House, Landis, & Umberson published a pivotal review of 5 prospective studies that indicated consistent prospective evidence that social relationships predicted mortality, independent of gender and various biomedical risk factors. Since this review, a substantial literature of large population-based prospective studies utilising measures of structural and functional social support has identified a robust relationship between social support and all-cause mortality whereby individuals with low levels of social support (both structural and functional) have between two-three times greater risk of mortality from a diverse range of diseases compared to individuals with high levels of social support (for example, Berkman, Leo-Summers, & Horwitz, 1992; Brummett et al., 2001; Cohen & Janicki-Deverts, 2009). This association has been found to be independent of age, initial health status, gender and socioeconomic factors. A recent comprehensive meta-analysis by Holt-Lunstad, Smith, & Layton, (2010) detected 148 studies incorporating 308, 849 participants followed up for an average of 7.5 years (range: 3 months – 58 years) investigating the relationship between social support and mortality. They concluded that individuals reporting stronger social relationships had a 50% increased likelihood of survival

compared to participants reporting poor social relationships (OR =1.50, 95% CI: 1.42 – 1.59). They also noted considerable variation according to the type of social support assessed and the specific constructs measured. Low structural social support (Study N =63) was associated with relative risk of 1.57 (95%CI: 1.46 – 1.70) of all-cause mortality. However, there was considerable heterogeneity in effect size across the different measures of structural social support ranging from greater effects noted for complex measures of social integration (OR: 1.91, 95% CI:1.63 – 2.23) to much lower effects observed for binary measures of living alone (OR: 1.19, 95% CI: 0.99 – 1.44). Low functional social support (N=24) was associated with a relative risk of 1.46 (95% CI: 1.28 – 1.66) of all-cause mortality with moderate heterogeneity according to measure. Combined low structural and functional social support (N=61) predicted an increased risk of 1.47 (95% CI: 1.34 – 1.60) of all-cause mortality. These findings were adjusted for age, gender, initial health status, length of follow up and cause of death. The authors conclude that the strength of this evidence robustly ratifies social support as an independent risk for mortality that is comparable to traditional risk factors (smoking, drinking excessive alcohol, obesity).

Social support enhances recovery from illness and is associated with reduced morbidity in individuals suffering from a range of diseases including coronary artery disease, rheumatoid arthritis and stroke (for example, Berkman et al., 1992; Ikeda et al., 2008; Kulik & Mahler, 1993; Morris, Yelin, Wong, & Katz, 2008). Social isolation has been associated with greater health risk behaviour among older adults (Shankar, McMunn, Banks & Steptoe., 2011). Research has also found that social support contributes to reduced psychological distress and improved adjustment during periods of chronic and acute stress (Cohen & Wills, 1985; Kawachi & Berkman, 2001). Low social support has also been associated with worse mental health in community samples (for example, Kawachi & Berkman, 2001; Stansfeld, Bosma, Hemingway, & Marmot, 1998; Cohen & Wills, 1985) and also a higher prevalence of diagnosed psychiatric disorder in diverse clinical populations (for example, Torgrud et al., 2004; Bruce, 2002; Huang, Yen, & Lung, 2010; Lancaster et al., 2010; Gandy, Sharpe, &

Perry, 2012). Further evidence for the health protective impact of social support comes from intervention based research whereby some interventions aimed at changing the social environment and facilitating social support have proved successful in reducing mortality and morbidity risk (for example, Anderson, 1992; Mendes de Leon et al., 2006; Hogan, Linden, & Najarian, 2002).

There has been a growing research emphasis on the specific impact of marital relationships on health. Simply being married appears to be significantly health protective and has consistently been associated with increased longevity and significantly reduced risk of mortality and morbidity from a wide range of diseases (Goldman, Korenman, & Weinstein, 1995; Goldman, 1993; House et al., 1988; Johnson, Backlund, Sorlie, & Loveless, 2000; Manzoli, Villari, Pirone, & Boccia, 2007; Murray, 2000; Scafato et al., 2008; Fors, Lennartsson, & Lundberg, 2011; Ben-Shlomo, Smith, Shipley, & Marmot, 1993; Shor, Roelfs, Bugyi, & Schwartz, 2012; Murphy, Grundy, & Kalogirou, 2007). In the most recent review of marital status and mortality, Shor et al., (2012) conducted a meta-analysis of 104 studies examining differences in mortality between married and unmarried adults who have experienced marital dissolution. They showed an overall elevated mortality risk in individuals with a marital dissolution compared to married individuals which was greater for men (HR=1.37, 95% CI: 1.27 – 1.49) than for women (HR=1.22, 95% CI: 1.13 – 1.32) and was independent of age and numerous other sociodemographic and methodological covariates. They also revealed a strong effect of age with significantly more elevated mortality risk noted in younger age groups compared to older age groups. They also found that the magnitude of mortality risk declined more rapidly by age for men than for women. However, it is important to note that this meta-analysis did not examine differences between married and never married populations.

Other recent studies examining marital status (including never married status) and mortality associations have also confirmed the protective impact of marriage. Rendall, Weden, Favreault, & Waldron, (2011) conducted a large panel survey based study (n=582,211) in

the US and identified a robust and significant association between unmarried status at 50 and mortality in the following year for both men (OR=1.72, 95% CI not given) and women (OR=1.38) which was independent of age, year, sociodemographic factors and time varying variables (disability, employment, income). This association was found to significantly decrease with increasing age and was found to be significantly larger for men compared to women. They also found no mortality differences between the different unmarried groups. The study was particularly robust due to a large sample size and the use of linked panel and administrative data that enable marital status to be observed in the year preceding mortality rather than as well as a baseline covariate. In a longitudinal study using large scale Census data from both England & Wales, and Finland, Blomgren, Martikainen, Grundy, & Koskinen, (2010) documented a significant marriage advantage. Never married men had a 20% (RR=1.20, 95% CI: 1.12 – 1.28) increased mortality risk in the English population and a 39% (RR=1.39, 95% CI: 1.33 – 1.44) increased mortality risk in the Finnish population. Never married women had a 34% (RR=1.34, 95% CI: 1.24 – 1.44) increased mortality risk in the English population and a 32% (RR=1.32, 95% CI: 1.27 – 1.38) increased mortality risk in the Finnish population. These are interesting findings as they suggest a marriage premium effect for women which contrast with the marriage premium effect for men noted by Rendell et al (2011) and numerous other studies.

Stahelin, Schindler, Spoerri, Zemp Stutz, & for the Swiss National Cohort Study Group, (2011) explored mortality differentials by marital status in a large census based study in Switzerland with a sample size in excess of 2.4 million people. They identified a protective effect of marriage for men and women. Unmarried men (aged 45 – 49) had an 87% elevated mortality risk (HR=1.87, 95% CI: 1.80 – 1.92) and unmarried women had a 65% higher mortality risk (HR = 1.65, 95% CI = 1.57 – 1.72) compared to their married counterparts. These mortality differentials decreased with increasing age and disappeared for women aged older than 80 years. The mortality risk associated with being unmarried was significantly higher for men than for women (except for those aged 90+) indicative of a male

marital premium. An interesting facet of this study was the identification that living arrangement was an important covariate with the highest mortality risks among divorced and single men who lived alone, and among single women who were living with a partner. Overall, the research presents a robust association between marriage and mortality, although the extent to which this is a genuine benefit of marriage or the product of the types of individuals selected into marriage is not known. The findings suggest that this marriage mortality effect may reduce with age, may be more pronounced for men and may be influenced by living arrangements; however, more research is required to confirm these variations.

Marital status is not a binary concept, as being unmarried encompasses a range of different marital states including being single and never married, widowed, divorced or separated. Mortality differentials among the different non married states have been found, although the findings are inconsistent (Dupre, Beck, & Meadows, 2009; Kaplan & Kronick, 2006; Manzoli et al., 2007; Staehelin et al., 2011). It is clear, however, that being married offers a clear survival advantage over all the unmarried groups and research suggests that the mortality gap between married and unmarried is increasing over time (Murphy et al., 2007). Furthermore, an individual's marital status may change a number of times over the lifespan and this concept of marital history has become an area of emerging research interest. Current research suggests that being continuously married with no or few marital disruptions confers the greatest health benefit and mortality reduction, particularly for low SES men (Dupre & Meadows, 2007; Choi & Marks, 2011; Blomgren et al., 2010). These findings are consistent with research highlighting that marital disruption has a negative impact on physical health, mental health and longevity (Hemström, 1996; Martikainen & Valkonen, 1996; Ebrahim, Wannamethee, McCallum, Walker, & Shaper, 1995; Pienta, Hayward, & Jenkins, 2000; Hughes & Waite, 2009). Other factors including timing of first and subsequent marriages, duration of marriage and timing of marital dissolution have also been found to impact upon health and mortality outcomes (Dupre & Meadows, 2007).

It is also important to consider that not all marriages are good and a poor quality marriage may actually represent a risk to health. Poor quality marriages tend to be characterised by increased conflict and reduced social support (Coyne & Anderson, 1999). There has been considerably less research examining the health correlates of marital satisfaction. In a review, Kiecolt-Glaser & Newton, (2001) revealed that better marital functioning was associated with better health outcomes, self-reported health and less pain and pain disability. The review findings suggest that marital functioning has a wide impact on health reflected by the diversity of biological systems that are implicated (e.g. immunological, cardiovascular) and also by the different aspects of disease trajectory that are influenced (e.g. aetiology, prognosis). Robles & Kiecolt-Glaser, (2003) also conducted a review of marital strain and mortality and morbidity and located four longitudinal prospective studies linking increased marital strain to increased risk of mortality in both community and clinical populations.

Poor quality marriages may reduce the mortality benefits normally associated with marriage and have also been associated with greater health risks than being single (Williams, 2003; Holt-Lunstad, Birmingham, & Jones, 2008; Gallo et al., 2003). Specific aspects of marital quality may also be important to morbidity and mortality. In an early study, Hibbard & Pope, (1993) found that equality in decision making and companionship were specifically protective against mortality for women. A more recent longitudinal study by Birditt & Antonucci, (2008) examined the impact of relationship quality on mortality in a sample of 2098 married individuals aged 40 years and older who were followed up for 19 years. Although baseline relationship quality was not significantly associated with mortality, they found that certain patterns of marital relationship quality were predictive of mortality, independent of demographic, health and chronic illness factors. Higher mortality was associated with increased spousal criticism (HR=1.44, 95% CI: 0.99-2.09) and surprisingly increased spousal love over time (HR=1.74, 95% CI: 1.05–2.87). They also found that consistently low spousal listening posed an increased mortality risk. These findings suggest that poor quality

marriages characterised by criticism and lack of listening may increase mortality risk thereby negating the benefits normally associated with marriage. It is interesting that increased spousal love was associated with elevated mortality as this seems counterintuitive; the authors suggest that this may reflect increases in positive relationship aspects prior to death that have been noted in other studies, and may also indicate increased overprotectiveness within the relationship which has been associated with increased risk to health. In another longitudinal study examining the impact of marital quality on health over the lifespan, Umberson et al., (2006) demonstrated that poor quality marital relationships accelerated the decline in self-reported health associated with increasing age with negative marital experience having the most significant impact on health at older ages. Marital quality has been the focus of a significant body of laboratory research with particular emphasis on the physiological correlates of marital conflict and strain. This research will be discussed in more detail in Section 2.6.4 of this chapter within specific relevance to ACS.

Poor marital quality has also been associated with worse mental health (elevated anxiety, depression, substance misuse) in non-psychiatric community populations (Whisman, 1999; Plaisier et al., 2008; Holt-Lunstad et al., 2008; Whisman & Uebelacker, 2006; Whisman & Uebelacker, 2009), and also with increased prevalence of psychiatric disorders (Whisman, 1999; Whisman, 2007; Whisman & Baucom, 2012). In a recent population based study of 2213 married adults followed up for 12 months, Whisman et al., (2007) examined the association between marital distress (assessed using a composite standardised measure) and psychiatric disorders (assessed using the DSM-IV). They found that marital distress was significantly associated with increased risk of any anxiety disorder (OR=1.69, 95% CI: 1.36-2.10), any mood disorder (OR=2.25, 95% CI: 1.80-2.81) and any substance use disorder (OR=2.34, 95% CI: 1.48-3.69). They noted the strongest specific relationships between marital distress and alcohol disorder, GAD and bipolar disorder. They found no evidence of gender moderation but found increased association strength between MDD and marital distress with age. There is growing evidence to support a causal relationship where marital

distress precedes psychiatric disorder and psychological distress and also for a dyadic interaction between individual distress and partner marital satisfaction (Whisman & Baucom, 2012). Interestingly, gender has not been found to be a robust moderator of this relationship (Whisman & Uebelacker, 2009; Whisman, 2007), although some specificity of disorder risk by gender has been noted. For example, Whisman, (1999) reported elevated rates of MDD and PTSD among women, and elevated rates of dysthymia among men reporting low marital dissatisfaction. Recent research has also begun to explore the dyadic elements of the marital distress-psychopathology association with growing focus upon the bidirectional impact of marital distress and psychiatric disorders on the individual and their partner (Whisman & Baucom, 2012).

Marital satisfaction has also been related to wellbeing. A meta-analysis of 93 studies identified that higher levels of marital quality were associated with greater personal wellbeing in both cross sectional and longitudinal analyses (Proulx, Helms, & Buehler, 2007). More recently, Holt-Lunstad et al, (2008) found that higher marital satisfaction was associated with greater life satisfaction, and lower risk of depression in a community sample of 303 adults. The importance of the temporal trajectory of marital satisfaction and the cumulative effects of long term marital unhappiness was highlighted by Hawkins & Booth, (2005) in a large study of married individuals. They found that being continuously unhappily married (assessed using a detailed composite assessment at four follow up points) over the 12 year follow up was associated with significantly lower happiness, life satisfaction, self-esteem and overall health, as well as elevated levels of psychological distress compared to individuals in continuously happy or varying marriages. This relationship has also been found to be independent of personality and gender, both factors that have been associated with wellbeing, suggesting a specific effect (Whisman, Uebelacker, Tolejko, Chatav, & McKelvie, 2006).

Overall, there is robust research evidence that functional and structural social support (including marital status and quality) are associated with mental and physical health in

diverse ways. The following sections (section 2.3 and 2.4) will explore the theoretical perspectives that have been proposed to explain these social support and marital differentials in health.

## **2.3 Theoretical models of social support**

It is clear that social support and health are intrinsically linked and numerous explanations have been proposed to explain why. Theories of social support tend to fall within the remit of two different but not mutually exclusive theoretical models; the stress buffering model or the direct (or main) effect model. The key facets of each of these models will be discussed in the following sections.

### **2.3.1 Stress buffering model of social support**

The buffering model of social support proposes that social support is beneficial because it buffers the well acknowledged pathogenic effects of stress on health (Cohen & Wills, 1985). Thus, social support is most beneficial to individuals experiencing stress. This perspective originates from the cognitive transactional model of stress and coping which suggests that the impact of a stressor is a function of two concurrent cognitive processes: primary appraisal and secondary appraisal. Primary appraisal refers to the evaluation of a stressor as either a threat, as harmful or as a challenge. Secondary appraisal refers to an individual's evaluation of their available coping resources and the likelihood that these resources will be sufficient to deal with the stressor. Negative primary and secondary appraisals are proposed to contribute to increased emotional distress (Lazarus & Folkman, 1984). Social support is suggested to impact both at the appraisal level and at the coping resources level. Individuals with greater perceptions of social support are less likely to appraise a stressor as threatening or harmful. Furthermore, individuals with greater perceptions of social support are more likely to feel capable of coping with a stressor because they perceive greater coping resources. Received social support may also buffer the impact of stress by facilitating adaptive coping and preventing maladaptive coping (Cohen & Wills, 1985). There is

significant research supporting the stress buffering model of social support. The presence of social support has been found to be significantly protective against the deleterious consequences of a diverse range of stressful life events (Cutrona & Russell, 1990). Research suggests that personality plays a role in determining the buffering impact of social support by influencing an individual's ability to foster and elicit support from social relationships, as well as affecting their perceptions of interpersonal interactions (Cohen, Sherrod, & Clark, 1986; Pierce, Lakey, Sarason, Sarason, & Joseph, 1997). Certain facets of the stressor may also impact upon the buffering impact of social support, for example, the burden of illness or a chronic stressor on an individual's social network may gradually erode support (Johnson, 1991; Lepore, Evans, & Schneider, 1991). Temporal factors may also contribute whereby a crisis may initially elicit support from others but as time passes this support is reduced. There may also be a differential buffering effect of social support contingent upon who the support provider is and the quality of the relationship (Norton et al., 2002; Styker, 1987).

### **2.3.2 Direct effects model of social support**

The direct effects model (also known as the main effects model) proposes that significant and direct benefits can be derived from being socially integrated irrespective of stress levels (Berkman, 1985; Cohen & Wills, 1985). From this perspective, social isolation is conceptualised as being particularly and directly damaging to wellbeing and health which is supported by considerable research revealing the pathogenic impact of social isolation on health (House et al., 1988). Numerous theories have been proposed to explain how greater social integration may improve health. Socially integrated individuals have greater exposure and access to information and resources which improve quality of life and facilitate health and wellbeing (Berkman, 1985). Thoits, (2011) provide a comprehensive review of the possible pathways between social networks and health suggesting seven key trajectories of influence; social influence, social control, meaning and purpose of life, self-esteem, sense of control/mastery, belonging and companionship, and finally perceived social support.

Socially integrated individuals are exposed to more social influence and norms regarding their behaviour and health (Uchino, 2006; Thoits, 2011). Members of a social network can directly facilitate health behaviours through endorsing health-promoting practices and disapproving health risk behaviour (Uchino, 2006; Umberson, 1987). Thoits (2011) describe social control as a more active form of social influence whereby an individual directly influences another's behaviour (e.g. reminding them, encouraging them to perform certain health behaviours). It is important to note that social influence and control have a positive health enhancing impact based on the assumption that all social ties are beneficial to health, however, some interpersonal relationships may be sources of stress, perceived as overly controlling or may encourage or normalise risky or health impairing behaviours (Burg & Seeman, 1994; Rook, 1984; Christakis & Fowler, 2007). Furthermore, there is a tendency towards homophily within the social network whereby an individual's social network is most likely to comprise of individuals who are similar to them in terms of sociodemographic, interpersonal and behavioural aspects (McPherson et al., 2001). Research indicates that this homophily principle extends to health behaviour and lifestyle, particularly with regard to smoking, obesity and physical activity (Flatt, Agimi, & Albert, 2012; Christakis & Fowler, 2007). Thus, individuals who engage in unhealthy behaviours may be more likely to have a social network comprising of similarly unhealthy counterparts. Interestingly, a recent new study conducted by Centola, (2011) illustrated that the adoption of a new health behaviour (using a diet diary) occurred more rapidly and was more diffuse within individuals randomised to a homophilous social network (defined by similarity of age, gender and BMI) compared to those allocated to an unstructured social network. This emerging evidence suggests that homophily within social networks may help to facilitate health behaviour change among network members. Thus, the tendency for homophily within social networks may be both an advantage with regard to health behaviour change, and a disadvantage with regard to shared health impairing behaviours.

Being socially integrated has also been proposed to have significant benefits for self-esteem, sense of identity and existential purpose which influence health and wellbeing via modulation of the neuroendocrine response to stress and through greater self-care behaviour due to attaching greater value on health (Kawachi & Berkman, 2001; Thoits, 2011; Thoits, 1983). Having social ties dictate certain social roles (partner, mother, friend) which are defined by particular role expectations and form a central part of one's identity and purpose. Individuals have to self-regulate their behaviour to conform to these expectations and their identity. Greater self-regulation has been associated with better health behaviour and various health outcomes (Shepperd, Rothman, & Klein, 2011). Thoits et al., (2011) describe how self-esteem and a sense of control are also important corollaries of an individual's ability to fulfil their social role expectations. An individual who feels efficacious in their ability to fulfil their social role by regulating their behaviour would be predicted to have higher self-esteem. Higher levels of self-esteem are associated with a multitude of physical and mental health benefits including better health protection behaviour (Marmot, 2003; McGee & Williams, 2000; Mann, Hosman, Schaalma, & de Vries, 2004). Similarly, performing certain social role behaviours aligned to expectation fosters a stronger sense of personal control. A greater sense of control has been well established as a significant influence on health, health behaviour and mortality (Chipperfield et al., 2012; Steptoe & Wardle, 2001). Furthermore, control beliefs form a central component of numerous theories of health behaviour (for example, Theory of Planned Behaviour, Health Locus of Control) which have been successfully applied to the prediction of health behaviour for many decades.

The importance of belonging and companionship is also highlighted by Thoits (2011). Loneliness or a lack of companionship has been well established as a risk to mental and physical health (Loboprabhu & Molinari, 2012; Luanaigh & Lawlor, 2008), and has also been significantly associated with elevated mortality, particularly among older people (Patterson & Veenstra, 2010; Shiovitz-Ezra & Ayalon, 2010; Perissinotto, Stijacic & Covinsky, 2012). Relatedly companionship and sense of belonging have been identified as contributing to

better mental and physical health, and to improved health behaviour change (Hagerty, Lynch-Sauer, Patusky, Bouwsema, & Collier, 1992; Turagabeci, Nakamura, Kizuki, & Takano, 2007; Hystad & Carpiano, 2012; Ross, 2002). Increasing companionship has also been successfully utilised in social support interventions to improve social outcomes, particularly within aspects of maternal health (for example, Small, Taft, & Brown, 2011; Khresheh, 2010).

Perceived social support is the final mechanism suggested by Thoits (2011) which is controversial as it contrasts with research suggesting that the health benefits from perceived social support originate from different mechanisms to those derived from social ties (Cohen & Wills, 1985; House et al., 1988). From this dualistic perspective, perceived social support is beneficial to health only when levels of stress are high (and thus can only be used to explain health effects within the context of buffering models), whereas social ties are beneficial to health all the time irrespective of stress levels derived from different mechanisms. However, Thoits (2011) argues that numerous reviews have highlighted a direct link between perceived social support and various outcomes suggesting that this type of social support is of benefit to health in all situations, not just those that are difficult or stressful. The author also proposes that the nature of perceived social support is different in everyday versus crisis situations. Everyday support provides constant low level support that facilitates daily problem solving, increases sense of control and provides minor practical assistance that all contribute to an easier daily life which in turn has implications for health and wellbeing. Perceived support does appear to contribute to the links between social integration and health which highlights the complex interplay between structural and functional aspects of social support.

There is research support for many of the different pathways proposed by the direct effects model, although more research is required to explore how certain facets of the model operate in the form of both longitudinal and experimental research (Thoits., 2011). There is also significant variation in how social integration and social ties are operationalized and

measured between different studies (Glass, Mendes de Leon, Seeman, & Berkman, 1997). Measures range from simple assessment of marital status to composite measures of social capital. Thus, with such variation it is difficult to extricate the specific aspects of social integration that are most beneficial to health and wellbeing.

Each model proposes a distinct set of processes linking social support and health and there is considerable research support for both direct and buffering effects of social support on health. However, there are research gaps and, in particular, there has been limited research investigating links between these two theoretical models which has limited progression towards a more integrated model (Uchino, 2004c).

## **2.4 Theoretical models of marriage and health**

Two main theories have been proposed to account for marital status differentials in health; selection effects and protection effects (Joung, van de Mheen, Stronks, van Poppel, & Mackenbach, 1998). These theories are not mutually exclusive and can be assessed through well controlled longitudinal research design.

### **2.4.1 Selection effects**

The selection effects theory states that healthier people are more likely to get married because they are perceived as more desirable partners and more likely to have better health over the lifespan (Goldman, 1993). Allied to the concept of selection effects is the concept of assortative mating whereby individuals are more likely to mate with individuals who have a similar genetic composition. Numerous studies have observed significant spousal homogeneity for diverse factors including educational attainment (Blossfeld, 2009), anti-social behaviour (Zwirs et al., 2011), psychiatric disorders (MAES et al., 1998) and BMI (Silventoinen, Kaprio, Lahelma, Viken, & Rose, 2003). Thus, individuals who are healthier, wealthier and more educated are more likely to be selected into marriage and are more likely to be attracted to similarly bestowed individuals which in turn would contribute to better health outcomes for the couple. There is mixed evidence based on longitudinal research regarding the role of

selection effects in explaining marital differentials in health. Some studies have identified selection effects evidenced by lower likelihood of marriage among individuals reporting previous chronic illness, medical problems or activity restricting illness (Pless, Cripps, Davies, & Wadsworth, 1989; Mastekaasa, 1992; Cheung & Sloggett, 1998). Waldron, Hughes, & Brooks, (1996) demonstrated that women with better health were more likely to marry and less likely to divorce or separate, but only if they were not in fulltime employment. However, other studies have not found an association between ill health and future marriage (Gortmaker, Must, Perrin, Sobol, & Dietz, 1993; Fu & Goldman, 1994). Research has also observed a relationship between unhealthy behaviours (e.g. smoking) and characteristics (e.g. obesity) and lower likelihood of marriage (Fu & Goldman, 1994). Similarly, risk taking behaviours (smoking and drug use) have been related to increased risk of marital dissolution (Fu & Goldman, 2000) An interesting study by Murray, (2000) used a historical dataset of nearly 2000 men from age 18 until death to examine the role of various anthropometric factors in influencing selection into marriage in a cohort assessed from 1832 - 1879. The findings indicated that underweight and very short men were less likely ever to marry whereas overweight and very tall men were more likely to marry. The authors note that at the time of the men's lives (19<sup>th</sup> Century) being overweight signalled health and prosperity whereas low weight suggested poverty and ill health. This study is particularly fascinating because it highlights how social norms regarding health change over time and subsequently influence the way in which individuals may be selected into marriage. In a more recent cross sectional study of 1175 middle aged Danish twin pairs, Osler, McGue, Lund, & Christensen, (2008) examined physical and psychological health differences in twin pairs who had different current marital status. They found evidence for marital selection effects with regard to level of physical activity, BMI and depression with individuals reporting lower physical activity, higher BMI and greater depression more likely to report being "never married". Finally, in a longitudinal panel study, Lillard & Panis, (1996) identified that health related factors rather than measures of general health were predictive of marriage in a sample of 4092 men assessed over 22 years. Thus, it may be that selection into marriage occurs at a

more specific level than general health; rather marriage may be more associated with indicators of health that can be appraised by potential partners. It is often difficult to make an accurate appraisal of an individual's overall health directly but it can be inferred from other indicators such as body shape, health and risk taking behaviours.

There is also some research supporting selection effects with regard to wellbeing and mental health. Evidence from the German Socio-Economic Panel Study spanning 17 years and 15,262 participants suggests that happier individuals are more likely to get married than their less happy counterparts independent of numerous sociodemographic factors. Interestingly, they found a significant impact of age with these effects observed in those who marry young (pre 18) and those who marry after 30 (Stutzer & Frey, 2005). Similarly, Mastekaasa, (1992) reported a predictive effect of life satisfaction on likelihood of marriage in a large population study of a 9000 unmarried individuals residing in a single county in Norway. This effect was significant among women aged 20 – 39 and among men aged 26 – 39 but not among the youngest category for men (20 – 25). Associations between psychiatric disorders and likelihood of marriage have also been found. A multi-national population study conducted by Breslau et al, (2011) examining 14,128 individuals from 19 countries found significantly lower likelihood of marriage related to fourteen of the eighteen psychiatric disorders assessed (including all anxiety, mood and substance abuse disorders). Reduced likelihood of marriage was not associated with externalising disorders. Interestingly, the authors also examined the association between psychiatric disorders and the timing of marriage (early, on time or late). They found that GAD, specific phobia, depression, bipolar disorder and drug dependence were also related to greater likelihood of early marriage. Similarly, Forthofer, Kessler, Story, & Gotlib, (1996) found that the presence of psychiatric disorder was associated with increased likelihood of early first marriage and decreased likelihood of “on-time” and late first marriage. They argue that early first marriage does not confer the financial and supportive benefits of marriage at a later stage providing further evidence of the complexity of selection effects. There is evidence to suggest that early marriage (before

18) is associated with fewer of the benefits normally associated with marriage (for example, greater financial stability) (Dahl, 2010). Thus, the role of selection effects with regard to psychological factors may be influenced by both age and timing of marriage, although research is limited and more longitudinal studies are required to greater understand the role of selection effects in marital health differentials.

#### **2.4.2 Protection effects**

Marriage is also proposed to directly confer numerous social, mental and physical health benefits which serve to protect health and promote longevity. Researchers have argued that a primary mechanism through which marriage protects health is due to greater financial resources. As previously discussed, research has demonstrated that marriage increases wealth with married individuals having higher income, greater probability of affluence over the life course, greater family income, and more financial assets (Waite & Gallagher, 2000; Hirschl, Altobelli, & Rank, 2003; Waite, 1995; Waite & Lehrer, 2003; Zagorsky, 2005). The wealth premium described here has often been found to be significantly greater for men than women, and also for whites compared to ethnic minority groups (Waite & Lehrer, 2003). Numerous explanations for the wealth premium bestowed by marriage have been proposed. The role of specialisation within household labour has been suggested whereby individuals can perform certain tasks and leave other tasks to their spouse leaving greater time and energy to devote to work or other earning pursuits compared to unmarried individuals (Stutzer & Frey, 2005). Couples are also able to share the cost of household goods, homes and cars which reduces expenditure. Furthermore, the responsibilities and norms governing married life tend towards more economic restraint, savings and investment. Research has also found evidence of longer working hours and positive discrimination towards married men within work organisations (Waite, 1995; Waite & Gallagher, 2000; Chun & Lee, 2001). This association between marriage and increased wealth is important because of the robust and well-established association between wealth, health and mortality (Marmot & Smith, 1997). However, most longitudinal studies of marital and health control for socioeconomic

status using standardised and reliable composite measures of income and wealth suggesting that other factors play a role.

Married individuals also tend to engage in less health risk behaviours and more health promoting behaviours compared to their unmarried counterparts. Being married has been associated with healthier diet (Haapala et al., 2012; Harrington et al., 2011; Thompson et al., 1999), better physical functioning and greater physical activity (Guralnik, Butterworth, Patel, Mishra, & Kuh, 2009; Osler et al., 2008), lower general alcohol consumption and rates of heavy and binge drinking (Power, Rodgers, & Hope, 1999; Temple et al., 1991) and less illicit drug use (Duncan, Wilkerson, & England, 2006). Marriage has also been associated with greater probability of smoking cessation (Broms, Silventoinen, Lahelma, Koskenvuo, & Kaprio, 2004) and lower likelihood of relapse (Miller, Ratner, & Johnson, 2003). There has been less consistent evidence supporting a marital protection effect with regard to weight gain and obesity with entry into marriage associated with weight gain, and higher prevalence of overweight and obesity noted in married compared to unmarried populations (Jeffery & Rick, 2002; Sobal & Hanson, 2011). Substantial review evidence also highlights significant spousal concordance in health behaviours and health behaviour change (Meyler, Stimpson, & Peek, 2007; Falba & Sindelar, 2008) indicating that the shared spousal environment may be an important determinant of individual health behaviour. This may be particularly beneficial to married men as research suggests that women generally have healthier lifestyles than men (Liang, Shediak-Rizkallah, Celentano, & Rohde, 1999; Ford et al., 2010) which may contribute to the greater beneficial health impact of marriage noted for men compared for women (Rendall et al., 2011).

Numerous studies have also observed that married people are more likely to attend recommended preventative screening including colorectal endoscopy, cervical screening, mammography and prostate screening (Burns, Walsh, O'Neill, & O'Neill, 2012; El-Haddad, Ablah, Dong, & Salyers, 2012; Stimpson, Wilson, Watanabe-Galloway, & Peek, 2012; Sutton

& Rutherford, 2005; Bulliard, de Landtsheer, & Levi, 2004; Sutton, Bickler, Sancho-Aldridge, & Saidi, 1994). Most of these studies have noted a robust effect of marital status within multivariate predictive models. A number have noted socioeconomic and gender influences on these marital status differentials in screening uptake with greater marital premium noted in men and those with higher SES. Research findings also highlight that married individuals experience better adjustment, adaptation and management of chronic illness (Elliott, Charyton, McAuley, & Shneker, 2011; Elliott, Charyton, Sprangers, Lu, & Moore, 2011; Berg & Upchurch, 2007). Medication and treatment adherence has also been found to be higher among married compared to unmarried populations with various chronic conditions (Wu et al., 2012; Trivedi, Ayotte, Edelman, & Bosworth, 2008; Gagnadoux et al., 2011; Molloy, Hamer, Randall, & Chida, 2008a). Better prognosis, recovery trajectories and adjustment have frequently been found among married populations with a diversity of serious chronic and acute diseases including cancer and coronary heart disease (for example, Wang, Wilson, Stewart, & Hollenbeak, 2011; Mahdi et al., 2011; Hadi Khafaji et al., 2012; Chung et al., 2009; Gerward, Tyden, Engstrom, & Hedblad, 2010). Furthermore, marriage has been found to have a protective effect on mental health and wellbeing which correspondingly influences physical health. Psychiatric disorders (including depression, anxiety and substance use disorders) have been found to be significantly less prevalent in married compared to unmarried populations (Inaba et al., 2005) with psychological wellbeing correspondingly higher in married compared to unmarried individuals (Kim & McKenry, 2002; Hughes & Waite, 2009; Murray, 2000).

Marriage appears to protect health because it improves and supports health promoting behaviour over the life course and within the context of chronic illness. This is most likely due to a combination of diverse factors. Most prominent is the role of increased social support in married compared to unmarried individuals. As I have previously detailed, elevated social support is robustly associated with better health and wellbeing. Furthermore, the type of social support garnered from a spouse (in contrast to other social ties) is likely to

occur on a daily basis providing continuous availability of emotional and instrumental support occurring within the context of everyday life which may have great influence on behaviour and functioning (Ross, 1995; Waite & Gallagher, 2000). It is important to consider that research has shown that men rely more heavily on their spouses for social support whereas women rely more on other sources of support (Reevy & Maslach, 2001; Olson & Shultz, 1994). Thus, it may be that there is greater availability of the type of social support most suited to supporting health and health behaviour for men compared to women which may contribute to gender differentials in the marriage-health premium. Other important processes include increased social control and influence (again predominantly provided by female spouses to male spouses (August & Sorkin, 2010)), shared environmental factors and greater motivation to self-care and health promotion due to the social norms governing married life (Waite, 1995; Umberson & Montez, 2010; Umberson, Crosnoe, & Reczek, 2010). Ultimately, all of the mechanisms discussed above represent conduits between marriage and individual physiology. Physiological mechanisms represent the concluding pathway through which marriage may protect health. Evidence for the final physiological pathways linking marriage to health has been established and illustrates a role for a diversity of biological processes (Robles & Kiecolt-Glaser, 2003; Umberson & Montez, 2010). This research will be discussed in more detail in the context of recovery from ACS later in this Chapter.

### **2.4.3 Summary of theoretical models**

The positive impact of social support on health and longevity is well established and two central explanations have been proposed to explain this association. It is most likely that both these explanations contribute to social support health differentials. Direct effect theories state that there are numerous benefits to social support that occur consistently over the lifespan whereas buffering effect theories state that social support primarily protects health during periods of stress and crisis helping to negate the adverse health implications of such periods. There is substantial evidence supporting a role for both direct and buffering effects

of social support and it is most likely that both contribute to the health benefiting impact of social support. Marriage is one form of social support that has been particularly well explored in the literature and there is a robust marital advantage in health and longevity. These marital status differentials in health and mortality may be accounted for by two key effects: selection and protection. These two effects are not mutually exclusive and are both supported by substantial research evidence supporting a role for both factors.

## **2.5 Social support and ACS**

Social support including marriage has been found to be a strong predictor of CHD mortality with measures of structural and functional support associated with between a 2-4 fold risk of cardiac mortality in patients with CHD (for reviews, Kuper, Marmot, & Hemingway, 2005; Lett et al., 2005; Uchino, 2004a). However, it has been suggested that the differential role of structural measures versus functional social support measures needs to be more clearly established in future cardiovascular research (Hemingway & Marmot, 1999). Uchino, (2004c) suggested that there are two main ways that social support may influence CHD mortality: (1) an aetiological role and (2) a prognostic role. The influence of social support on the aetiology of CVD will be briefly discussed before the research exploring the impact of social support on recovery following ACS is explored in more detail.

### **2.5.1 Social support, marriage and aetiology of ACS**

In a review of the aetiological role of social support in CHD incidence and development, Barth, Schneider, & von Kanel, (2010) identified five prospective studies examining the role of social support in CHD incidence in previously healthy populations utilising a range of different measures. Sample sizes ranged between 500 and 45,414 participants who were followed up over a period of between 4 and 10.3 years. Social support level was assessed by either self-report questionnaire or census data. The type of social support measured was variable including functional and structural measures. Meta-analysis was not possible on such a small number of papers but quantitative analysis revealed that there was some

evidence supporting a significant protective role for functional social support in the development of CHD with two of the three papers assessing functional support identifying a significant effect (unadjusted HR's ranged between 1.53–2.23). However, there was no evidence to suggest a protective impact of structural social support on CHD development. It is important to note that although most of the included studies controlled for social and biological confounders, none of the studies controlled for psychological factors which reduces the validity of these findings (Low, Thurston, & Matthews, 2010). The conclusions of this review contrast with the findings of a previous review by Lett et al., (2005) which examined eight prospective studies and found a stronger association between structural social support and CHD development than functional social support. The adjusted risk values ranged from 1.19 to 3.1 for measures of structural social support indicative that healthy individuals with low structural social support considerably elevated risk of developing CHD than those reporting higher social support. These mixed findings are similar to other older reviews highlighting inconsistent research support for an aetiological role of social support which requires further clarification (Kuper et al., 2005; Hemingway & Marmot, 1999).

Another recent review explored psychosocial risk factors in the development of CHD exclusively in women. Low et al., (2010) found 12 studies exploring the role of social support in incident CHD and revealed a positive impact of social support (HR range: 1.81 – 2.72) in protecting against the development of CHD. However, only 2 of the 12 studies utilised validated measures of social support reducing the methodological reliability of these findings. In light of the methodological diversity in the assessment of social support, the lack of control for psychological confounding and the inconsistent limited findings, more research elucidating the form of social support most significant for CHD development is required.

There are few studies explicitly investigating the role of marital status in CHD incidence and no review evidence could be found. In a recent case control study investigating the role of marital status and education in predicting MI in a symptom and history free Chinese population, Hu et al., (2012) found a 51% increased risk of incident MI in single individuals

(OR=1.51, 95% CI: 1.18-1.93) independent of age, gender, BMI, psychosocial and lifestyle factors. The risk was significantly higher amongst single women (OR=2.00, 95% CI: 1.39-2.86) compared to single men (OR=1.19, 95% CI: 0.84–1.68). They also identified a particularly high risk of incident MI in single women with low education (OR= 2.95, 95% CI: 1.99-4.37). In a population cohort study of 33, 224 individuals without MI history, Gerward et al., (2010) found that being never married, divorced or widowed in males but not females was significantly associated with between 10 – 77% increased risk of a first coronary event independent of numerous lifestyle, biological and occupational factors. In particular, being widowed was associated with the highest risk (OR=1.77, 95% CI: 1.31-2.40). In another large prospective cohort study of 138 260 participants aged 30 - 69, Nielsen, Faergeman, Larsen, & Foldspang, (2006) observed that single living (single and living alone) was associated with over a two-fold increased risk (OR=2.3, 95%:1.7-3.0) of incident ACS in women and almost a three-fold increased risk in men (OR=2.9, 95% CI: 2.4-3.5) independent of age, family type, citizenship, education, economy, SES and occupation. These studies suggest that being unmarried may pose an increased risk of developing CHD although some studies have found no marital differences in CHD incidence (Eaker, Sullivan, Kelly-Hayes, D'Agostino, Sr., & Benjamin, 2007).

A further body of research has found an association between atherosclerosis progression (indicative of future CHD) and marital quality. In a recent study of marital discord and coronary artery calcification (an indicator of atherosclerosis) in 150 healthy couples, Smith, Uchino, Berg, & Florsheim, (2012) found that coronary artery calcification was significantly greater in discordant couples compared to non-discordant couples independent of behavioural and biomedical risk factors. The study is interesting because the authors utilised categorical definition of marital quality (discordant versus non discordant) based on a marital disagreement discussion, self-report of anxiety and anger during the disagreement as well as self-report measures of marital adjustment providing a more holistic assessment of marital quality. Similarly, a number of other previous studies have identified an association

between poor marital quality (assessed by self-report) and various biomedical measures of atherosclerosis (Gallo et al., 2003; Gallo, Troxel, Matthews, & Kuller, 2003; Janicki, Kamarck, Shiffman, Sutton-Tyrrell, & Gwaltney, 2005).

There is also an emerging research identifying an association between marital status, marital quality and various early physiological markers of CHD risk including blood pressure, heart rate variability and markers of inflammation which have been found to have an aetiological capacity in asymptomatic individuals (for example, Liao et al., 1997; Danesh, Collins, Appleby, & Peto, 1998). Associations between these factors and marital parameters have been identified and continue to emerge (Smith et al., 2011; Sbarra, 2009). These associations will not be discussed in detail here as they are addressed within Section 2.6.4 in the context of physiological pathways that may contribute to the prognosis of ACS.

Overall, the research investigating the aetiological role of social support and marital status in incident CHD is limited, methodologically diverse with inconsistent findings. In particular, the differential role of functional versus structural social support is not known. Emerging research illustrates a burgeoning relationship between marital quality and atherosclerosis progression. However, the diversity of measures used to assess marital quality limits amalgamation of these findings. More research engaging consistent methodological approaches and specific aspects of social support may further elucidate the significance of specific aspects of social support and marital relationships in the development of CHD and ACS.

### **2.5.2 Social support and prognosis after ACS**

A prognostic association between social support and ACS was first identified in the Beta Blocker Heart Attack trial (Ruberman, Weinblatt, Goldberg, & Chaudhary, 1984) where post MI patients who were socially isolated and reported high levels of stress had a 4-fold increased risk of cardiac mortality compared to patients with high social support or low stress levels. In a recent systematic review, Barth et al., (2010) located twenty separate prognostic

studies incorporating sample sizes between 194 and 13,240 individuals, follow up periods ranging between 6 months and 14.5 years and measures of functional and structural support. Patients were diagnosed with either MI, existing CHD or had undergone CABG or angioplasty. Low functional social support was significantly associated with elevated all-cause mortality (HR=1.59, 95% CI: 1.21-2.08) independent of other risk factors. There was a less clear prognostic association with structural social support with an insignificant association when other risk factors were controlled. The authors also noted considerable heterogeneity between all the studies.

The lack of impact of structural social support contrasts with previous review findings. A prior review conducted by Lett et al., (2005) identified nineteen prospective studies of patients with existing CHD. Patients were followed up between 6 months and 10 years post diagnosis of CHD, ACS, chronic heart failure or CABG surgery. The results indicated that both structural and functional measures showed considerable predictive utility with regard to both morbidity and cardiac mortality. Low social support (structural or functional) was associated with a 2 – 4 fold increased risk of cardiac mortality. In another systematic review, Mookadam & Arthur, (2004) explored the role of social support in mortality and morbidity following an acute myocardial infarction and described five relevant studies with a mean sample size of 687. These studies incorporated both functional and structural measures of social support including social network size, social network constitution, living alone, disrupted marriage and perceived social support. The review findings suggested that social isolation or lack of a social support network was associated with a 2-3 fold increased risk of mortality and morbidity independent of traditional post AMI mortality predictors. The reviewers state that this is equivalent to the risk conferred from other factors indicating that low social support is a predictor of mortality after AMI. These results are analogous to the conclusions of other older reviews of the prognostic value of social support (for example, Hemingway & Marmot, 1999; Uchino, 2004a). Furthermore, recent research published after these reviews also consistently supports the notion that lack of social support confers a greater risk of mortality

and morbidity among acute cardiac patients in both the short and long term (Heffner, Waring, Roberts, Eaton, & Gramling, 2011; Roohafza, Talaei, Pourmoghaddas, Rajabi, & Sadeghi, 2012). Thus, these findings indicate a significant role for both functional and structural social support in prognosis following ACS. There is recent inconsistency within the findings regarding structural social support (Barth et al., 2010), however, this may be a product of the heterogeneity within the research as more recent studies continue to illustrate a predictive effect of structural social support (Heffner et al., 2011).

### **2.5.3 Marital status, marital satisfaction and ACS prognosis**

One area of structural social support that has been well explored is the role of marital status in post ACS survival. Numerous early studies identified significantly better survival prospects after MI in married compared to unmarried individuals (for example, Wiklund et al., 1988; Chandra, Szklo, Goldberd, & Tonascia, 1983; Case, Moss, Case, McDermott, & Eberly, 1992). More recent studies have continued to confirm a marriage survival premium. In a sample of 225 CABG patients, King & Reis, (2012) noted that married CABG patients were 2.5 times less likely to die during the 15 year follow up than unmarried patients independent of age. In a previously discussed large cohort study of 33,224 individuals, Gerward et al., (2010), found that being never married (OR=2.14, 95% CI: 1.63-2.81), divorced (OR=1.91, 95% CI: 1.50-2.43) or widowed (OR=1.49, 95% CI: 0.77-2.89) was associated with between 49%-214% increased risk of mortality during the first day following a coronary event. Another recent prospective cohort study of 242 patients followed up for 16 years after their first MI. They found that patients living alone had more than a two-fold increased risk of death during the follow up period (HR=2.55, 95% CI:1.52-4.50) compared to patients living with a partner adjusting for various confounders (Nielsen & Mard, 2010).

An investigation of 10 year mortality rates in 3682 coronary patients by Eaker et al., (2007) identified that married men were half as likely to die during the follow up period compared to unmarried men (HR=0.54, 95% CI: 0.34-0.83) adjusting for age, blood pressure, BMI,

smoking, diabetes and cholesterol. No significant differences were observed in married compared to unmarried women. Numerous other studies have also confirmed that being unmarried or living alone increases risk of mortality following ACS independent of established confounders with a particularly pronounced effect amongst men compared to women (Hadi Khafaji et al., 2012; Schmaltz et al., 2007; Kandler, Meisinger, Baumert, Lowel, & the KORA Study Group, 2007; Malyutina et al., 2004; Pfiffner & Hoffman, 2004).

The growing interest in more functional aspects of marriage has fostered research investigating the role of marital quality in ACS prognosis. In a sample of 225 CABG patients, King & Reis, (2012) found that patients reporting higher marital satisfaction were over three times less likely to die during the 15 year follow up compared to those in low satisfaction marriages independent of age. In a larger consecutive sample of 292 30 – 65 year old women recruited while hospitalised for ACS and followed up for five years, Orth-Gomer et al., (2000) found that married or cohabiting women who reported severe marital stress had nearly a three-fold increased risk of a new coronary event (HR=2.92, 95% CI: 1.3-6.5) compared to women reporting no or mild marital stress independent of sociodemographic and clinical confounders. They also noted similar patterns associated with marital stress for cardiovascular mortality and MI. However, these were non-significant which may be attributed to the small number of such events (n=14). No prognostic relationship was identified with work related stress or marital status suggesting a specific effect of marital stress.

Eaker et al., (2007) that female participants who reported “self-silencing” during marital conflict had a four-fold (HR=4.01, 95% CI: 1.75-9.20) increased risk of mortality during the 10 year follow up compared to women who did not use “self-silencing”. Interestingly they found that measures of marital happiness, satisfaction and disagreements were not related to mortality. Furthermore, in a sample of 296 CABG patients, Kulik & Mahler, (2006) found that better marital quality (assessed using the Dyadic Adjustment Scale) was associated with a significantly shorter post-operative hospital stay in female but not male patients. There is

also substantial research linking marital status and quality to certain physiological processes that may contribute to ACS prognosis. These processes represent one potential pathway through which marital status and quality influence coronary outcomes in ACS patients and will be discussed in more detail in Section 2.6.4.

Both marital status and marital quality appear to influence mortality and morbidity amongst ACS patients. In particular, there is a substantial research indicating that unmarried ACS patients have significantly elevated risks of mortality compared to married patients, an effect which is particularly pronounced in men. The research on marital quality is less developed and robust but suggests that poorer marital quality and marital communication may impair prognosis particularly amongst female patients.

## **2.6 Pathways between social support and prognosis after ACS**

The research presented within this literature review strongly suggests that social support, marital status and marital satisfaction represent important influences on recovery following ACS. The next step, which is central to this thesis, is gaining understanding of how these social and marital factors exert such a profound influence. Numerous pathways have been proposed to explain these relationships and primarily include biological, behavioural and psychological mechanisms. These pathways are proposed to be highly interdependent and overlapping. It should be noted that the predominant focus of this thesis is establishing whether there are social support and marital differentials in psychobiological factors. The identification of such differentials would provide the basis for further exploration of the role of these factors in determining clinical outcome. Thus, behavioural factors are not central to my thesis although a brief discussion of the key behavioural pathways will be provided because aspects of behaviour are related to both psychological and biological factors, and behaviour is included in some of the analysis within this thesis.

## **2.6.1 Behavioural pathways**

The behavioural pathway proposes that social and spousal support facilitate secondary prevention health promoting behaviours, encourages adherence to medication and health promoting regimes, and also promotes cardiac rehabilitation attendance which all contribute to successful recovery.

### **2.6.1.1 Health behaviour and lifestyle change**

Following ACS, patients are encouraged to facilitate their recovery by engaging in diverse lifestyle modifications including smoking cessation, dietary change, weight loss and increased exercise. Extensive lifestyle change exhibits substantial positive prognostic value for recovery following ACS highlighting the importance of secondary prevention (Daubenmier et al., 2007; Pischke, Scherwitz, Weidner, & Ornish, 2008). Lack of social support in coronary patients has been related to lack of exercise (Aggarwal, Brooke, Liao, & Mosca, 2008; Brummett et al., 2005), reduced likelihood of smoking cessation (Allen, Markovitz, Jacobs, Jr., & Knox, 2001) and reduced adherence to a low fat diet (Bovbjerg et al., 1995; Sayers, Riegel, Pawlowski, Coyne, & Samaha, 2008; Aggarwal, Liao, Allegrante, & Mosca, 2010). There is less consistent research illustrating a link between social support and weight loss following ACS (Lopez-Jimenez et al., 2008), however, social support is associated with weight loss and maintenance in general population samples (Wing & Phelan, 2005).

Research suggests a particularly prominent effect of support from a patients' spouse on patient health behaviour post ACS (Bovbjerg et al., 1995; Franks et al., 2006; Kulik & Mahler, 1989). Furthermore, general population research has identified that marriage is consistently associated with better health behaviour and less health risk behaviour which has been discussed in detail earlier in this chapter. Being married is also associated with earlier presentation for care among men experiencing chest pain (Atzema et al., 2011) which is associated with decreased ACS mortality (De Luca G., Suryapranata, Ottervanger, & Antman, 2004).

An emerging literature highlights an association between marital quality and health behaviour with poor marital quality associated with greater health risk and less health promoting behaviour (Robles & Kiecolt-Glaser, 2003; Gallo et al., 2003). Although the research is limited and predominantly based on self-report measures of health behaviour rather than more reliable objective measures (Uchino, 2004b), the findings suggest that patients with greater social support, married patients and in particular highly satisfied married patients are more likely to engage in health promoting behaviours and less likely to participate in health impairing practices following an ACS. This would have a profound influence on ACS prognosis.

### **2.6.1.2 Adherence to medication**

Following ACS, a long-term regime of cardioprotective medications is prescribed to facilitate recovery and this is highly effective in reducing risk of mortality and reinfarction (Mukherjee et al., 2004). Cardioprotective medication non-adherence is associated with significantly increased risk of post ACS mortality and morbidity (Ho et al., 2006; Ho et al., 2008). The substantial mortality risk conferred by medication non-adherence has been explained in terms of a direct lack of benefit from medications, the relationship between medication and lifestyle change non-adherence, and the association with psychosocial factors that contribute to poor prognosis e.g. depression (Ho et al, 2006).

Social support has been found to contribute to medication adherence. In a comprehensive meta-analysis of social support and medication adherence in a wide range of diseases, DiMatteo, (2004) identified that practical support, emotional support, being married, having a close and cohesive family and living with at least one other person were all significantly associated with increased adherence. Most significantly, practical support yielded the strongest relationship, with the odds of adherence being 3.6 times higher in individuals receiving practical support. Emerging research in ACS patients suggests that social support,

particularly practical support, contributes to adherence to cardiac medication in ACS patients (Molloy, Perkins-Porras, Bhattacharyya, Strike, & Steptoe, 2008).

The support of a spouse may also be particularly valuable to medication adherence in cardiac patients and there is significant research illustrating that married cardiac patients tend to be more adherent to medication than unmarried patients (Kulkarni, Alexander, Lytle, Heiss, & Peterson, 2006; Trivedi, Ayotte, Edelman, & Bosworth, 2008; Doherty, Schrott, Metcalf, & Iasiello-Vailas, 1983). Although the role of marital quality in coronary medication adherence has not been explicitly explored, a number of studies have identified that negative aspects of social relationships may contribute to non-adherence. Di Matteo (2004) found that family conflict was associated with increased risk of medication non-adherence. Partner stress has been associated with medication non-adherence in post ACS patients (Molloy, Perkins-Porras, Strike, & Steptoe, 2008a; Trivedi et al., 2008).

#### **2.6.1.3 Adherence to cardiac rehabilitation**

Cardiac rehabilitation (CR) is an integral part of standard post ACS care (Leon et al., 2005). Research suggests that attending CR substantially reduces risk of all-cause mortality, cardiac mortality and reinfarction after ACS (Jolliffe et al., 2001; Taylor et al., 2004). CR attendance rates are typically low particularly among female, ethnic minority and elderly populations with approximately 33% of MI and CABG patients attending (Nielsen et al., 2008; Williams et al., 2004; Witt et al., 2004). In response to this deficit, research has identified numerous factors that predict attendance including demographic, physician, clinical and psychosocial factors (Jackson et al., 2005; Shanks et al., 2007).

There is increasing evidence suggesting that various measures of social support may also be associated with CR attendance. Living alone (Nielsen, Faergeman, Foldspang, & Larsen, 2008; Ramm, Robinson, & Sharpe, 2001) and having a smaller social network (Molloy, Perkins-Porras, Strike, & Steptoe, 2008a) have been associated with non-attendance whereas greater practical support (Molloy et al., 2008b) and higher perceived social support

(Daly et al., 2002) have been noted as predictors of attendance. Marital status has also been found to be an important predictor of CR attendance. As part of my PhD work, I contributed to a meta-analysis which investigated the predictive utility of marital status with regard to CR attendance (Molloy, Hamer, Randall, & Chida, 2008b). Based on the findings of eight studies which included 6984 CHD patients, the results indicated that married patients were between 1.5 and 2 times more likely to attend CR which is similar to the findings of a previous review (Jackson, Leclerc, Erskine, & Linden, 2005). No studies were identified that directly explore the role of marital quality in predicting CR attendance.

Overall there is clear evidence supporting a role for social support and marriage in behaviour, medication adherence and cardiac rehabilitation attendance. Emerging evidence suggests that marital quality may also be important. Behaviour clearly has an important role on prognosis following ACS, however, behaviour does not completely account for social support and marital differentials in prognosis indicative that other factors contribute.

## **2.6.2 Psychological pathways**

Psychological pathways between social support and recovery after ACS are emphasised as centrally important within the stress buffering model of social support. As previously discussed, this model proposes that social support reduces the appraisal of stress and subsequently reduces maladaptive psychological reactions e.g. anxiety, depression (Cohen, 1988). A number of psychological factors have been proposed; however, depression and anxiety have been found to have the greatest prognostic value in ACS patients and will be discussed below.

### **2.6.2.1 Depression**

As discussed in the Chapter 1, depression is very common among ACS patients, imposes significant risk for morbidity and is an established risk factor for mortality. Depression has also been associated with adverse health behaviours, medication non-adherence and less participation in cardiac rehabilitation which, as discussed, can significantly impede recovery

and may contribute to increased risk of mortality (DiMatteo, Lepper, & Croghan, 2000; Kronish et al., 2006; Ziegelstein et al., 2000; Glazer, Emery, Frid, & Banyasz, 2002; Myers, Gerber, Benyamini, Goldbourt, & Drory, 2012). Depression is also associated with reduced return to work (Soderman, Lisspers, & Sundin, 2003), poorer health behaviour (Whooley et al., 2008) and psychophysiological correlates of poor prognosis (Carney & Freedland, 2009).

There is a sizeable literature examining the association between social support and depression. In the most recent review, Lett et al (2005) describes how lack of social support, assessed using diverse structural and functional measures, has been found to be a significant risk factor for the development or exacerbation of depression in both general population studies and in studies of recovering ACS patients measured both cross sectionally and longitudinally. They note that measures of perceived social support were more consistently associated with depression than structural measures suggesting a buffering effect of social support against post ACS depression genesis. They acknowledge the possibility of reverse causality but highlight how the vast majority of research supports an anterior role for social support in depression. This causal direction is also supported by the wider social support and depression literature utilising general population and psychiatric populations (Patten, Williams, Lavorato, & Bulloch, 2010).

More recent studies have continued to confirm a prospective association between low social support and increased risk of depression following ACS. In a longitudinal study of 2411 patients hospitalised for MI, Leifheit-Limson et al., (2010) found that low social support (assessed using the ENRICH social support inventory during hospitalisation) was associated with higher mean depressive symptoms (assessed using the Primary Care Evaluation of Mental Disorders Patient Health Questionnaire PHQ-9) at 12 month follow up Independent of sociodemographic, clinical and baseline factors. This effect was particularly pronounced for female patients. Recent studies using general population samples continue to support an association between functional social support and depression (Grav, Hellzèn, Romild, & Stordal, 2012).

High levels of social support have also been found to protect against the adverse consequences of depression on post ACS mortality. For example, Frasure-Smith et al., (2000) revealed that depressed ACS patients who reported high levels of perceived social support were not at increased risk of mortality at 1 year post MI compared to depressed patients who reported low social support at baseline. There has also been emerging evidence suggesting that the association between depression and social support may actually reflect a single shared construct. Potential propositions for this shared construct include negative affect, personality influences and genetic factors (Raynor, Pogue-Geile, Kamarck, McCaffery, & Manuck, 2002; Wade & Kendler, 2000; Lara, Leader, & Klein, 1997).

The combination of lack of social support and depressive symptomatology may have particularly adverse consequences in CHD patients. In a sample of 292 female ACS patients, Horsten, Mittleman, Wamala, Schenck-Gustafsson, & Orth-Gomer, (2000) found that 35% of patients who lacked social integration (assessed in terms of both structural and functional characteristics) and reported the presence of two or more depressive symptoms at between 3-6 months post ACS had suffered a coronary relapse during the subsequent 5 years compared with 9% of patients who did not experience depression and were socially integrated. This association was independent of a wide range of established risk factors including age. A similar synergistic association between depression and social support was observed for risk of atherosclerotic progression in female ACS patients (Wang, Mittleman, Leineweber, & Orth-Gomer, 2006).

The evidence supporting an association between global measures of structural social support and depression in ACS patients is less substantial and the findings are inconsistent (Lett et al., 2005; Lin, Ye, & Ensel, 1999), although there is support for a relationship between smaller social networks and increased risk of depression within general population research (Kawachi & Berkman, 2001; Lin et al., 1999). Network analysis studies in the general population have illustrated that negative emotion and depression can spread within a social network indicating that the individuals in a social network can have a profound

influence on the mental health of other members (Rosenquist, Fowler, & Christakis, 2011; Hill, Rand, Nowak, & Christakis, 2010). Although no network analysis studies of depression effects in ACS patients have been conducted, this type of analysis may be particularly relevant in the context of increased prevalence of distress, depression and anti-depressant use in the spouses of MI patients (Fosbél et al., 2012).

The relationship between marriage and lower prevalence of depression in the general population, particularly among men, is well established and has been discussed in section 2.4.2. As depression poses considerable prognostic risk in ACS, being married may improve prognosis due to its buffering and protective effects against depression. Few studies were identified that explicitly explored marital status differentials in depression in ACS patients and the findings are mixed. In a sample of 2172 ACS survivors, Panagiotakos et al., (2008) documented a non-significant trend towards higher depression scores (assessed using Centre of Epidemiological Studies Depression Scale) in never married compared to married patients suggesting limited impact of marital status on depression risk. However, it is important to note that depression was assessed in hospital and related to symptoms over the preceding month. It may be that this study is capturing pre-existing depression rather than depression occurring after the ACS. In a sample of 105 female CAD patients, Blom et al., (2007) found no association between marital status and depressive symptoms. However, the cross sectional nature of the study does not illuminate any possible longitudinal associations. Similarly, Astin, Jones, & Thompson, (2005) found no relationship between marital status and depressive symptoms (assessed using the Cardiac Depression Inventory) in a sample of 141 CHD patients undergoing PTCA followed up for 6-8 months. In contrast, Cheok, Schrader, Banham, Marker, & Hordacre, (2003) found that 61% of divorced or separated patient's experienced higher levels of depression (assessed using the CES-D and HADS) compared with 43.1% married or 42.9% never married patients in a sample of 1455 hospitalised ACS patients. Other studies have identified an increased risk of in hospital post ACS depression in patients who live alone (Spijkerman, van den Brink, Jansen, Crijns, &

Ormel, 2005). No longitudinal, mixed gender studies were found that explore marital status differentials in depression in ACS patients. Poor marital quality and low marital satisfaction have also been found to be strongly associated with risk of depression in the general population, particularly amongst women, as discussed in Section 2.2. Although research is limited, low marital dissatisfaction and poor marital functioning have also been found to increase risk of depression following ACS, particularly in female patients (Balog et al., 2003; Burg & Seeman, 1994; Kiecolt-Glaser & Newton, 2001).

The combination of the robust prognostic impact of depression on cardiac outcomes in ACS patients, and the associations between social support, marital status, marital quality and depression in the general population suggests that these factors may form a causal pathway. Although there is research support for an association between functional social support and depression following ACS, there is a lack of longitudinal research exploring structural support, marital status and marital quality influences on depression development. In order to address this gap, I conducted a study to explore the relationships between social support, marital status, marital satisfaction and depression in a large mixed gender sample of ACS patients assessed at 2 weeks, 6 months and 12 months post ACS. The results of this part of the study are presented in Chapter 6.

#### **2.6.2.2 Anxiety**

Rates of anxiety symptomatology and anxiety disorders are significantly elevated in CHD and ACS populations. As noted in Chapter 1, there have been mixed findings regarding the impact of anxiety on mortality in CHD populations although a recent review found that anxiety was associated with significantly elevated rates of all-cause mortality, cardiac mortality and adverse cardiac events (Roest, Martens, Denollet, & de Jonge, 2010). Anxiety has also been associated with unhealthy behaviours in ACS patients and physiological correlates of poorer prognosis in ACS including reduced HRV (Bonnet et al., 2005; Martens, Nyklicek, Szabo, & Kupper, 2008; Kuhl, Fauerbach, Bush, & Ziegelstein, 2009).

Lack of social support has also been implicated in the genesis of some clinical anxiety disorders (Furmark et al., 1999; Guay, Billette, & Marchand, 2006) as well as the experience of anxiety in various clinical populations (Hipkins, Whitworth, Tarrier, & Jayson, 2004; Korostil & Feinstein, 2007). There have been relatively few studies examining the relationship between social support and anxiety in ACS patients with mixed results. In 114 consecutively recruited MI patients assessed at 4-6 weeks post MI, Pedersen, Middel, & Larsen, (2002) found that patients reporting low crisis social support had over a three-fold elevated risk of PTSD (OR=3.10, 95% CI: 1.08-9.20) and significantly higher mean depression score. No significant difference was noted in mean anxiety scores. They also found that lower satisfaction with social support (measured using a single item) was significantly associated with increased risk of PTSD (OR=4.35, 95% CI: 1.50-12.98), increased mean depression and anxiety scores. However, adjustment for multiple confounding variables eliminated the significant associations with anxiety implying no independent role for social support or satisfaction with support in anxiety. In a study of 226 consecutive ACS patients, Murphy et al, (2008) examined trajectories of anxiety over the year following ACS. They found no association between living alone or social interaction and risk for high and worsening anxiety during the year following ACS. Thus, the relationship between social support and anxiety in ACS is not clear and has not been differentially examined with regard structural and functional aspects.

The relationship between marital status and anxiety in ACS is similarly poorly understood. As detailed in section 2.2 of this Chapter, there is general population and psychiatric research associating marital status and marital quality with the development of anxiety symptomatology and clinical anxiety disorders with unmarried and those in poor quality marriages at greater risk of anxiety compared to married and happily married populations (Whisman, 2007; Holt-Lunstad et al., 2008; Priest, 2012; Leach, Butterworth, Olesen, & Mackinnon, 2012; Scott et al., 2010). There have been less consistent findings regarding

the role of marriage and marital quality in clinical populations with serious or chronic illness (Collins, Corcoran, & Perry, 2009; Fafouti et al., 2010).

A few studies have explicitly explored the relationship between marital status, marital quality and the development of anxiety in ACS patients. Kim et al., (2000) explored anxiety (measured with the State Anxiety Inventory and Brief Symptom Inventory) occurring within 72 hours of hospital admission within a sample of 424 MI patients. They found that women experienced significantly elevated anxiety compared with men. Married women had significantly elevated anxiety compared to single and widowed women whereas married men had significantly lower anxiety compared to single men. However, an international study of 912 MI patients assessed within 72 hours of hospital admission found that marital status did not predict anxiety levels nor did it interact with gender (Moser et al., 2003). Other studies of MI and CHD patients have found similar null effects of marital status (Akhtar, Malik, & Ahmed, 2004; Astin et al., 2005). All these studies are limited by their cross sectional focus on assessment of anxiety during hospitalisation. Assessment of anxiety levels during hospitalisation may be particularly unreliable as research suggests that anxiety levels vary considerably during the first 72 hours following MI with peak anxiety levels occurring at 12 hours post MI (An et al., 2004). A number of these studies utilised small samples which may reduce power to detect differences. Finally, there is considerable heterogeneity of both measure and type of ACS population examined reducing cross comparison.

No longitudinal studies specifically exploring marital status influence on anxiety occurring later in recovery were identified. However, a number of longitudinal studies of anxiety and cardiac outcome have examined marital status as a covariate. Moser et al., (2011) reported no effect of marital status on anxiety levels occurring over the two years following ACS in a sample of 3522 CHD patients. Similar findings have been found in other older studies although these studies only explored the covariate relationship between marital status and baseline anxiety and therefore do not provide longitudinal insight (Moser & Dracup, 1996; Moser et al., 2007; Welin, Lappas, & Wilhelmsen, 2000).

In the light of the limited research, inconclusive findings and methodological deficiencies, there is a clear need for more research to determine the longitudinal associations between social support, marriage and anxiety in ACS. In order to address this gap, I conducted a study to explore the relationships between social support, marital status, marital satisfaction and anxiety in a large mixed gender sample of ACS patients assessed at 2 weeks, 6 months and 12 months post ACS. The results of this part of the study are presented in Chapter 6.

### **2.6.3 Quality of life**

In the context of the increasing survival rates among ACS patients and the chronic nature of CHD, it is important to explore the impact of social support on prognostic factors beyond mortality and physical morbidity. Quality of life (QoL) is a well-established endpoint within medical and psychosocial research (Editorial, 1995). Although the construct of QoL has been operationalized in many different ways with continuing controversy regarding its definition, it is generally accepted that quality of life is a multidimensional construct that encompasses the physical, functional, psychological and social functioning of an individual (Smith, Avis, & Assmann, 1999). Quality of life is often conceptualised as an outcome in social support research but can also be understood as a pathway between social support factors and ACS outcome because impaired quality of life following ACS has been associated with increased risk of mortality and morbidity (Rumsfeld et al., 1999; Spertus, Jones, McDonell, Fan, & Fihn, 2002; Westin, Nilstun, Carlsson, & Erhardt, 2005).

Research suggests that quality of life is impaired following ACS but tends to improve over time with some persisting residual impairment among certain individuals. Factors increasing the risk of poor quality of life included being female, older, impaired cardiac functioning, having a prior history of MI, as well as the presence of comorbid physical and psychological disorders (Uchmanowicz, Loboż-Grudzien, Jankowska-Polanska, & Sokalski, 2011; Dias et al., 2005; Emery et al., 2004; Simpson & Pilote, 2003; Schweikert et al., 2009; Pettersen, Kvan, Rollag, Stavem, & Reikvam, 2008; Norris et al., 2008). A recent review confirmed that

depression has a particularly negative effect on quality of life in CHD patients (Dickens, Cherrington, & McGowan, 2012).

There is a large literature illustrating that social support may play an important contributory role in determining quality of life and functioning following ACS. Much of the quality of life research has examined cross sectional associations between various measures of social support and different aspects of quality of life in patients with either diagnosed CHD or following acute MI. In a consecutive sample of 560 CAD patients attending cardiac rehabilitation, Staniute, Brozaitiene, & Bunevicius, (2011) found that functional perceived social support was significantly associated with health related life in both male and female patients. However, they found that among male patients perceived social support was related to psychological aspects of quality of life but not to physical aspects of quality of life whereas among female patients perceived social support was significantly related to both psychological and physical aspects of quality of life. In a recent study of 84 patients awaiting coronary artery bypass grafting, Thomson, Molloy, & Chung, (2012) found an association between higher patient perceived informational and emotional social support (assessed using the Medical Outcomes Study Social support survey) and better mental health quality of life. They found no effect of affectionate support, tangible support and positive social interaction. Previous studies have also confirmed the cross sectional impact of various aspects functional social support on quality of life, although most of these studies did not control for confounding clinical factors (Wingate, 1995; Woloshin et al., 1997; Bosworth et al., 2000; Perez-Garcia, Ruiz, Sanjuán, & Rueda, 2011).

Longitudinal studies have provided more robust evidence for social support differentials in quality of life. In a large prospective study of 2411 MI patients, Leifheit-Limson et al., (2010) examined the longitudinal relationship between perceived social support (assessed using the ENRICH Social support inventory) and quality of life (measured using the MOS SF-12 and the Seattle Angina Questionnaire (SAQ) over a 12 month period. They found that perceived social support assessed at baseline was significantly predictive of disease specific quality of

life and mental health related quality of life in a stringently risk adjusted model that adjusted for a vast array of clinical, sociodemographic and psychological factors. They also found that quality of life was poorer among female patients and this gender difference was particularly pronounced among women reporting low social support at baseline. In another longitudinal study investigating the association between social support (assessed using the Interpersonal Support Evaluation List) and quality of life (assessed using the MOS-SF36), Emery et al., (2004) followed 536 cardiac patients from hospitalisation to 12 months. They found that social support, particularly a sense of belonging or companionship, was significantly associated with mental health quality of life over the follow up period independent of depression, optimism and perceived stress. Similarly, an 18 month longitudinal study of women with CHD by Janevic et al., (2004) investigated the relationship between social support (assessed using the MOS social support inventory, number of close ties and marital status) and a variety of outcomes including depression, and self-rated health. They found that higher total social support was associated with less depression and symptom impact, as well as better self-rated health. They also found that different aspects of social support were associated with different long term outcomes. However, the sample were recruited from a disease management intervention and may have been more likely to be in better health with less impairment and distress than others who may be too unwell to participate in such a programme. This is further compounded by the lack of control for disease characteristics which have been found to predict quality of life.

In another longitudinal study of 288 MI patients, Lane et al (2001) found that living alone was a significant predictor of poor quality of life at 12 months post MI. A similar predictive role for social support has also been noted among other longitudinal studies (Rankin & Fukuoka, 2003; Rankin, 2002; Barry, Kasl, Lichtman, Vaccarino, & Krumholz, 2006; Yates, 1995; Barefoot et al., 2000). However, the findings are not entirely consistent as not all longitudinal studies have illustrated an impact of functional social support on quality of life outcomes in cardiac patients (Elizur & Hirsh, 1999; Hamalainen et al., 2000).

A small number of studies have shown that being unmarried may be associated with worse quality of life following ACS compared to being married (Badura & Waltz, 1984; Janevic et al., 2004; Dias et al., 2005; Waltz, 1986). There have been exceptions with one study identifying no impact of marital status on quality of life in female patients (Wingate, 1995). I could not find any studies that explored marital quality influence on post ACS quality of life specifically although some studies have explored marital quality effects on psychosocial adjustment. In a sample of CABG patients, Elizur & Hirsh, (1999) found that marital satisfaction, support and adaptability were significant predictors of post CABG psychosocial adjustment. Brecht, Dracup, Moser, & Riegel, (1994) examined psychosocial adjustment in 198 men recently diagnosed with CHD and found that better adjustment was associated with better marital quality and less emotional distress.

The findings suggest that social support may contribute to quality of life outcomes among ACS patients, particularly among women. However a substantial number of studies were cross sectional preventing causal direction to be established with many of the longitudinal studies incorporating a single follow up preventing observation of the relationship trajectory. Many have not controlled for important confounding factors (for example, depression, clinical factors) and thus it is difficult to elucidate the influence of social support independent of factors that have been shown to influence quality of life. The presence of substantial heterogeneity in how social support and quality of life are assessed, as well as in the cardiac populations explored, makes it difficult to integrate the findings into a coherent framework. There is a need to clarify the specific role of structural social support in influencing post ACS quality of life as few studies have explicitly assessed this relationship using standardised measures. Similarly, little is known regarding the differential quality of life trajectories of married compared to unmarried patients nor the potential influence of marital quality. To gain greater insight into social support influences on quality of life, I conducted a study examining the relationship between social support, marital status, marital quality and quality of life

assessed at two weeks, 6 months and 12 months post ACS utilising standard measures and controlling for numerous confounding variables. The results are presented in Chapter 7.

#### **2.6.4 Biological pathways**

Biological pathways are crucial to fully understanding how social, psychological and behavioural factors can be “translated” into actual health outcomes. Uchino, (2004a) describes how the physiological systems of the body constitute the final universal pathway between social support and health. There is growing research suggesting that diverse physiological systems are involved in mediating the impact of social support on health and a comprehensive review of all systems is provided by Uchino, (2006). The key physiological pathways implicated in the genesis and worsening of ACS include cardiovascular functioning, neuroendocrine effects and immune-mediated inflammatory factors. The detailed evaluation of all these potential physiological pathways is beyond the scope of this thesis. Thus, this section will begin with a brief description of the role of cardiovascular functioning as this is my primary focus because it is the mechanism most relevant to cardiac populations and forms a central part of my thesis. A brief summary of the research exploring social support influences on the other physiological pathways will follow.

##### **2.6.4.1 Cardiovascular functioning and ACS prognosis**

Exaggerated cardiovascular reactivity to stress has been consistently associated with increased risk for CHD development and progression (Chida & Steptoe, 2010). Laboratory manipulated social support in stressor situations have been found to predict cardiovascular reactivity with social support associated with decreased cardiovascular reactivity (Phillips, Gallagher, & Carroll, 2009; Christian & Stoney, 2006; Linden, Chambers, Maurice, & Lenz, 1993; O'Donovan & Hughes, 2008; Schwerdtfeger & Schlagert, 2011). The use of laboratory manipulated social support during a stressor (for example, the presence of a companion during a stressor) rather than assessments of global functional or structural social support potentially limits generalizability to more naturalistic settings. However, recent research has

demonstrated the ecological validity of these techniques and highlights that effects actually tend to be greater in real life compared to laboratory paradigms (Zanstra & Johnston, 2011). I could not identify any studies that explored the differential impact of marital status on cardiovascular reactivity. However, aspects of the marital relationship and quality have been associated with cardiovascular reactivity. In a sample of healthy adults, Phillips, Carroll, Hunt, & Der, (2006) found that during an acute psychological stressor task female participants who had their spouse/partner present exhibited lower cardiovascular reactivity than those without their spouse/partner present. This effect was not found for male participants. Similarly, warm partner contact prior to a lab based stressor has been found to reduce cardiovascular reactivity during the stressor (Grewen, Anderson, Girdler, & Light, 2003).

Marital conflict has been associated with elevated cardiovascular reactivity (Smith et al., 2009; Newton & Sanford, 2003; Kiecolt-Glaser & Newton, 2001; Robles & Kiecolt-Glaser, 2003). Marital dissatisfaction has been related to increased physiological reactivity during marital conflict (Smith et al., 2009; Heffner, Kiecolt-Glaser, Loving, Glaser, & Malarkey, 2004; Smith, Gallo, Goble, Ngu, & Stark, 1998). Similarly particular patterns of behaviour during marital conflict have been found to further increase cardiovascular reactivity including negative and hostile behaviour (Smith et al., 2009; Newton & Sanford, 2003; Denton, Burleson, Hobbs, Von, & Rodriguez, 2001). Evidence suggests that women may experience greater cardiovascular reactivity to marital conflict than men (Robles & Kiecolt-Glaser, 2003; Kiecolt-Glaser & Newton, 2001).

Social support effects have also been observed in various measures of ambulatory blood pressure (ABP). Lower ABP is an important prognostic indicator and has been associated with reduced incidence of cardiac events, reduced mortality and reinfarction risk after ACS (Clement et al., 2003; Kario & Pickering, 2000). Various measures of functional and structural social support has also been associated with lower resting blood pressure, lower ambulatory blood pressure (ABP) and greater nocturnal blood pressure dipping, particularly

among older individuals, in healthy and hypertensive populations (Uchino et al., 1996; Uchino et al., 1999; Uchino, 2006(Stepptoe, 2000; Gump, Polk, Kamarck, & Shiffman, 2001; Clays et al., 2012; Routledge & McFetridge-Durdle, 2007). In a sample of 97 healthy couples, Bowen et al., (2012) observed no significant association between global functional social support (assessed using the Interpersonal Support Evaluation List) and 12 hour ABP. However, they also explored relationships between ABP and specific aspects of social support and found that emotional social support was significantly associated with ABP in women. Thus there may be specific aspects of social support that have a greater effect on ABP than other aspects. Studies have also identified that social support may buffer the negative ABP effects of stressful acute and chronic life events (Gallagher & Whiteley, 2012).

Poor marital quality has also been associated with elevated ambulatory blood pressure in both community and hypertensive populations (Holt-Lunstad et al., 2008; Holt-Lunstad, Jones, & Birmingham, 2009; Baker et al., 2000; Heffner et al., 2004). These studies have used a variety of measures to assess marital quality including standardised questionnaires, specific measures of spousal support and discussion of spousal conflict. This heterogeneity of measures makes it difficult to amalgamate the findings; however, the general theme is that higher marital quality and better marital interaction is associated with lower blood pressure. For example, Holt-Lunstad et al., (2008) found that marital satisfaction and adjustment (assessed using Marital Adjustment Test and the Dyadic Adjustment Scale) predicted 24 hour ambulatory blood pressure in a sample of 303 healthy adults. They also found that unhappily married participants had significantly higher 24 hour and waking ambulatory blood pressure compared to single individuals highlighting that single individuals had better cardiovascular functioning compared to unhappily married individuals. In a sample of 120 healthy adults, Gump et al.,(2001) found that ambulatory blood pressure was significantly lower during partner interactions compared to interactions with other social ties. There is also research suggesting a buffering effect of marital quality against the negative effects of stress on ABP. Tobe et al., (2005) found that higher marital cohesion was

associated with reduced 24 hour ABP in individuals reporting higher job strain suggesting a buffering effect of marital cohesion. The relationship between marital status and blood pressure is less clear with some studies illustrating elevated blood pressure in unmarried compared to married individuals (Lipowicz & Lopuszanska, 2005), and others finding the reverse (Blumenthal, Thyrum, & Siegel, 1995).

A final way in which social support may influence cardiovascular function is by facilitating the maintenance of normal heart rate variability. As previously discussed in Chapter 1, reduced HRV indicates increased cardiac sympathetic and/or reduced parasympathetic modulation and has been proposed as a significant marker for disease. Reduced HRV is often conceptualised as a lack of physiological flexibility with chronically reduced HRV proposed to reflect physiological vulnerability to the negative impact of stress (Porges, 1995). Impaired heart rate variability has been associated with all-cause mortality (Gerritsen et al., 2001; Kikuya et al., 2008), atherosclerotic progression (Huikuri et al., 1999), sudden cardiac death (Bigger et al., 1992) and CHD incidence (Liao et al., 1997). In patients recovering after ACS, HRV may be reduced which is a robust predictor of post myocardial infarction mortality (Bigger et al., 1992; La Rovere, Bigger, Jr., Marcus, Mortara, & Schwartz, 1998; Nakagawa, Saikawa, & Ito, 1994).

There are two main ways in which social support and marital factors may influence HRV - indirectly or directly. Reduced heart rate variability has been associated with both modifiable (e.g. smoking) and non-modifiable (e.g. family history) CHD risk behaviours with reduction in risk behaviour associated with concomitant improvement in HRV (Thayer & Lane, 2007; Thayer, Yamamoto, & Brosschot, 2010). As previously discussed, social support has been found to contribute to increased engagement in health behaviours and reduction in risk behaviour following ACS suggesting an indirect link between social support and HRV. Furthermore, HRV has been associated with both the development of metabolic syndrome and hypertension (Liao et al., 1998; Liao et al., 1996; Thayer et al., 2010) which have also been associated with social support and may worsen post ACS prognosis. Psychological

distress has also been found to influence HRV with both depression and anxiety, as well as antidepressant use associated with reduced HRV and other markers of autonomic imbalance (Rottenberg, 2007; Kemp et al., 2010; Gorman & Sloan, 2000; Licht, de Geus, van Dyck, & Penninx, 2009). As previously discussed, ACS populations are at increased risk of depression and anxiety, with lack of social support, unmarried status and low marital satisfaction identified as potential risk factors. Thus, social support may indirectly influence cardiac outcomes via HRV consequences of increased psychological distress.

Emerging evidence suggests that lack of social support may be a risk factor for reduced HRV although the current research base is small. Horsten et al., (1999) showed in a sample of 300 healthy women that social isolation and the inability to relieve anger were associated with significantly reduced HRV independent of traditional correlates and depressive symptoms. More recently, in a sample of 1727 healthy adults aged over 40 years, Shin et al., (2012) found that higher social support (assessed using Medical Outcomes Study-Social Support Survey) was significantly associated with reduced frequency and time domain measures of HRV independent of age and gender. Although the extent to which these findings represent a direct effect or an indirect effect of other psychological or behavioural factors is not clear as these factors were not controlled for. There is also evidence from animal research supporting an association between social isolation and reduced HRV (Grippe, Lamb, Carter, & Porges, 2007). No studies have explored the relationship between marital status and HRV in general or clinical populations. The association between marital quality and HRV was recently explored by Smith et al., (2011) in a sample of 114 young married couples who identified an association between resting high frequency HRV and marital quality. This is an interesting finding as the authors point out that high frequency HRV is an indicator of self-regulatory capacity and marital functioning requires considerable self-regulation of emotions and behaviour. They also found that a laboratory manipulated negative marital interaction was associated with a reduction in female (but not male) participants resting high frequency HRV indicating a greater physiological cost for women

during marital conflict. The current findings examining social support and marital influences on HRV effects are limited which is surprising in the context of HRV robust relationship with post ACS mortality. In order to address this deficit in the research, I conducted a study to determine whether marital status influences HRV in a sample of patients with suspected coronary artery disease. These findings are reported in Chapter 3. I also completed a further study investigating social support (functional and structural), marital status and marital satisfaction differences in HRV in a large sample of ACS patients assessed two weeks after their ACS. These results are described in Chapter 8.

#### **2.6.4.2 Other potential biological pathways between social support and prognosis in ACS patients**

Inflammation has also been found to be an important biological antecedent to CHD (Pearson et al., 2003; Ridker, 2009) and a significant prognostic indicator following ACS (Hatmi, Saeid, Broumand, Khoshkar, & Danesh, 2010). A number of studies have documented significant associations between markers of inflammation (including C-reactive protein, interleukin-6 and fibrinogen) and both structural and functional social support (Steptoe et al., 2003; Heffner et al., 2011; Ford, Loucks, & Berkman, 2006; Loucks, Berkman, Gruenewald, & Seeman, 2006; Loucks et al., 2006; Gleit, Goldman, Ryff, Lin, & Weinstein, 2012). Emerging research indicates a potential buffering effect of functional social support on the inflammatory impact of stress; however this effect was only identified in middle aged women (Mezuk, Diez Roux, & Seeman, 2010). A few studies have also explored the impact of marital status and quality on measures of inflammation. The findings suggest that being unmarried is associated with elevated inflammation (Sbarra, 2009; Engström, Hedblad, Rosvall, Janzon, & Lindgärde, 2006) with greatest effects noted for men compared to women. Poor marital quality and elevated marital conflict have also been associated with elevated inflammatory markers (Whisman & Sbarra, 2012; Kiecolt-Glaser et al., 2005). The research also suggests a greater inflammatory effect of poor marital quality and conflict for women compared to men (Whisman & Sbarra, 2012).

Another proposed pathway between social support and prognosis refers to neuroendocrine processes and dysregulation of the hypothalamic pituitary adrenal (HPA) axis dysregulation. With normal regulation of the HPA axis, cortisol levels following a diurnal pattern with peak cortisol levels occurring in the early morning with subsequent decline throughout the day reaching a low at about 2-3am. Short term increases in cortisol typically occur during the first hour post awakening and also following meals or stressors (Van Cauter, Leproult, & Kupfer, 1996). Dysregulation of this pattern may be observed in terms of alteration of the overall cortisol levels or by a smaller cortisol decline throughout the day and evening i.e. a flatter slope. There is evidence suggesting that HPA axis dysregulation is associated with many risk factors for cardiovascular disease including obesity, increased blood pressure, hypercholesterolemia and atherosclerosis (Dekker et al., 2008; Matthews, Schwartz, Cohen, & Seeman, 2006; Rosmond & Bjorntorp, 2000). HPA axis dysregulation is also common after ACS with elevated cortisol levels observed in response to MI which decline within 28 – 72 hours (Bain et al., 1992; Donald et al., 1994). Elevated acute cortisol levels have been found to predict adverse cardiac outcomes and mortality in post ACS patients (Guder et al., 2007; Tenerz et al., 2003). Poorer structural and functional social support have both been associated with elevated cortisol levels and attenuated cortisol response in laboratory and large community studies, which is particularly pronounced in men compared to women (Seeman, Berkman, Blazer, & Rowe, 1994; Lederbogen et al., 2010; Cohen et al., 2006; Kirschbaum, Klauer, Filipp, & Hellhammer, 1995; Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). Marital status has also been found to influence cortisol response with unmarried individuals exhibiting reduced cortisol levels compared to married individuals (Englert et al., 2008). An interesting study by Saxbe & Repetti, (2010) identified significant co-regulation of cortisol level and negative mood in a sample of married couples when the couple were in a shared environment (e.g. at home in the early morning or evening). They also found that marital satisfaction was an important moderator with highly satisfied couples showing greater cortisol co-regulation than unsatisfied couples. There has also been significant laboratory and naturalistic research demonstrating an association between marital

quality and HPA axis dysfunction. Marital conflict appears to be associated with increased HPA dysfunction whereas marital warmth, positive marital interaction and marital satisfaction have all been associated with reduced HPA dysfunction (Slatcher, Robles, Repetti, & Fellows, 2010; Ditzen et al., 2007; Holt-Lunstad et al., 2008; Kiecolt-Glaser, Bane, Glaser, & Malarkey, 2003; Kiecolt-Glaser et al., 1997; Robles & Kiecolt-Glaser, 2003). These effects have been found to persist over the long term and are also particularly pronounced among women compared to men (Robles & Kiecolt-Glaser, 2003).

## **2.6 Chapter summary**

Both functional and structural aspects of social support have been established as significant predictors of various health outcomes. Theoretical perspectives posit a role for both direct and buffering effects. Being married has also been associated with numerous health outcomes and both selection and protection effects have been proposed as explanatory mechanisms. There is also an emerging research highlighting the importance of marital quality in many health processes and outcomes. Social and marital factors have an important prognostic influence in CHD and ACS. Behavioural, psychological, quality of life and biological factors have all been found to play an important and interactional mediating role between these social factors and post ACS prognosis. In order to address the gaps identified in the research that have been highlighted throughout this Chapter, I conducted a multidimensional study that simultaneously investigates psychological (depression, anxiety and quality of life) as well as biological (HRV) correlates of structural and functional social support, as well as marital status and marital satisfaction, in a large sample of ACS patients assessed at 2 weeks, 6 months and 12 months post ACS. These results are presented in Chapters 5, 6, 7 and 8. I also contributed to a study investigating the role of marital status in HRV in a sample of suspected CHD patients, a previously un-researched relationship. The findings are described in Chapter 3.

# CHAPTER 3 HEART RATE VARIABILITY AND MARITAL STATUS STUDY

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## 3.1 Introduction

As discussed in the preceding two chapters, there is substantial evidence indicative of marital status differentials in health, whereby married individuals have higher subjective health, lower incidence of physical and mental health conditions and lower rates of mortality and morbidity (Johnson, Backlund, Sorlie, & Loveless, 2000; Joung, 2007). In particular there is a pronounced association between marital status and coronary heart disease (CHD) morbidity and mortality (Lett et al., 2005; Horsten, Mittleman, Wamala, Schenck-Gustafsson, & Orth-Gomer, 2000; Rosengren, Wilhelmsen, & Orth-Gomer, 2004). Research investigating marital status differentials in CHD mortality and morbidity has identified a number of these contributing factors which have been discussed in detail within Chapter 2. Briefly recapping these findings, being married has been associated with better health behaviour, greater health behaviour change and less health risk behaviour in both general population and cardiac populations. Marriage also appears to provide protection against negative affective states like depression and anxiety which are well substantiated risk factors for both the development and worsening prognosis of CHD providing further explanation for the greater CHD mortality and morbidity experienced by unmarried individuals. Finally, a number of physiological systems have been implicated in the association between social support and health outcomes including neuroendocrine, immune and cardiovascular pathophysiological processes (Uchino, 2006). In particular, cardiovascular pathophysiological processes represent the most explored physiological connections between social support and health outcomes.

An area of growing interest within the cardiovascular pathophysiological pathway is heart rate variability (HRV). In Chapter 2, I presented the research indicating associations between social support, marital factors and HRV. As previously defined in Chapter 1 & 2, HRV refers

to the short-term oscillation in the intervals between consecutive heartbeats and provides an important index of autonomic nervous system function. Heart rate variability can be evaluated in two key ways; through time domain measures or frequency domain measures. Time domain measures such as the square root of the mean of the sum of the squares of successive normal to normal (NN) differences (RMSSD) are calculated from the heart period series and are thought to mainly reflect cardiac parasympathetic activity (Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology, 1996). Frequency domain measures, assessed through power spectral density analysis of ECG results, distinguish three main spectral components; very low frequency (VLF), low frequency (LF) and high frequency (HF) HRV. The high frequency component is activated by respiration and reflects cardiac parasympathetic activity (Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology, 1996)). There is disagreement regarding the physiological correlates of LF-HRV with some evidence suggesting that it reflects only cardiac sympathetic modulation and other research indicating derivation from both sympathetic and parasympathetic influences (Houle & Billman, 1999). The physiological origin of VLF-HRV is not well understood, but is thought to be primarily determined by parasympathetic activity (Taylor, Carr, Myers, & Eckberg, 1998). In healthy individuals, both LF-HRV and HF-HRV exhibit a circadian pattern with greater LF modulation during the day and greater HF modulation during the night, reflecting elevated sympathetic activity during the day and raised parasympathetic activity during the night. Impaired HRV reflects excessive cardiac sympathetic and/or reduced parasympathetic modulation, indicating physiological rigidity, and has been established as a significant marker for disease. Impaired HRV (indexed using both time and frequency domain measures) has been found to predict all-cause mortality (Gerritsen et al., 2001; Tsuji et al., 1994), atherosclerotic progression (Huikuri et al., 1999), post-myocardial infarction (MI) mortality (La Rovere, Bigger, Jr., Marcus, Mortara, & Schwartz, 1998), sudden cardiac death (Bigger et al., 1992) and CHD incidence (Liao et al., 1997). Reduced VLF-HRV has been found to have particular prognostic value with regard to

post-MI mortality (Bigger et al., 1992). Furthermore, impaired HRV has been associated with both modifiable (e.g. smoking) and non-modifiable (e.g. family history) risk factors for the development of CHD (Thayer & Lane, 2007) suggesting a role for cardiac autonomic regulation in both the development and progression of CHD.

There are a number of psychosocial correlates of CHD which may also be associated with reduced HRV. Depression has been associated with modified HRV in some studies of physically healthy populations, CHD and post MI populations (Rottenberg, 2007). Other psychological states implicated in the development and worsening of CHD have also been associated with impaired HRV including anxiety and emotional stress (Dishman et al., 2000; Friedman & Thayer, 1998). In a previous analysis from the study described in this chapter, there was no association between depression assessed with the Beck Depression Inventory (BDI) and HRV, although relationships were observed when depressed mood over the sampling period were measured using the Day Reconstruction Method (Bhattacharyya, Whitehead, Rakhit, & Steptoe, 2008).

As highlighted in Chapter 2, aspects of the social environment may also be important with social isolation and low social support associated with reduced HRV (Grippe, Lamb, Carter, & Porges, 2007; Horsten et al., 1999; Shin et al., 2011). Marital quality has also recently been associated with resting high frequency HRV (Smith et al., 2011). However, no studies were identified that explore the possibility of marital status differentials in HRV suggesting a substantial gap within the research that merits exploration. In order to identify potential marital status differentials in HRV, we analysed marital status and 24-hour HRV in patients with suspected coronary artery disease (CAD) who had been referred to a chest pain clinic. Experiencing chest pain and being investigated for a potential diagnosis of CAD is a distressing occurrence which may lead to increased cardiovascular reactivity. Consequently, we investigated patients prior to definitive diagnosis of CAD with both researchers and patients unaware of CAD status. This allowed assessment of the relationship between

marital status and HRV in patients both with and without CAD who shared a comparable clinical experience. We hypothesised that unmarried patients would have significantly impaired HRV compared with married patients. In view of the differences in dominance of sympathetic and parasympathetic processes in the day and night, the two periods were separated in the analysis of 24-hour records.

## **3.2 Methodology**

### **3.2.1 Participants**

The sample consisted of 88 patients (28 women, 60 men, mean age 61.6, 60% married) recruited from three London hospital based Rapid Access Chest Pain clinics between June 2006 and December 2007. All patients had been referred by their GP or hospital doctor on the basis of new onset chest pain and either positive exercise tests or positive myocardial perfusion scans with evidence of myocardial ischemia. All patients were being investigated for suspected CAD and anticipated a cardiac diagnosis. Patients participated in the study before undergoing coronary angiography to confirm the presence and severity of CAD. Exclusion criteria included being on antidepressant medication, inability to speak English, suffering from a significant non-cardiac disease or other cardiac disorder (heart failure, valvular disease, major arrhythmia). The response rate was 61% from a total sample of 144 eligible patients. Patients who took part in the study were significantly younger ( $M$  61.1,  $SD$  9.8 years,  $t=2.16$ ,  $p=0.032$ ) than patients who declined ( $M$  64.9,  $SD$  10.2 years). No other significant differences were found between completers and non-completers.

### **3.2.2 Procedure**

Patients were recruited during attendance at Rapid Access Chest Pain clinics where they provided signed consent and scheduled a convenient appointment for the study at the University College London research laboratory. During this appointment, demographic, health behaviour, psychological, clinical and anthropometric data was collected. Participants were also fitted with a 24hour digital Holter monitor (Lifecard CF, Del Mar Reynolds,

Hertford, UK) to obtain measures of heart rate variability. The patient returned at the same time on the following day to complete other study measures and to remove the Holter monitor. All sessions began between 9:00 and 13:00hr on one day, and finished at the same time on the following day.

### **3.2.3 Measures**

#### **3.2.3.1 Demographic and anthropometric measures**

Information regarding age, marital status, whether the patient lived alone, ethnicity, education, employment, household income, medication use, smoking status, sleep quality, alcohol consumption and habitual physical activity were assessed through interview. Anthropometric measures were also recorded to assess body mass index (BMI). Patients were classified as either married or unmarried, with participants who were currently legally married or living as married being defined as married, while the unmarried group included patients who were never married, separated, divorced or widowed.

#### **3.2.3.2 Psychological measures**

Patients completed a number of psychological measures to identify levels of depression and anxiety, and to evaluate health status and health quality of life. The measures are described in detail below.

##### **3.2.3.2.1 Beck Depression Inventory (BDI)**

Patients completed the Beck Depression Interview (BDI) to assess levels of depression (37). The BDI comprises of 21 self-report questions and scores can range from 0 – 63 with higher values representing greater depressive affect. A score of 10 or above is classified as subclinical indicating symptoms of mild to moderate depression. Reliability and validity have been established for this measure (Ziegelstein et al., 2000).

#### *3.2.3.2.2 Hospital Anxiety and Depression Scale (HADS)*

Patients completed the anxiety scale of the Hospital Anxiety and Depression Scale (HADS) (Zigmond & Snaith, 1983). The anxiety scale consists of 7 self-report questions and total scores can range from 0 – 21. Higher scores indicate greater anxiety and the threshold for subclinical anxiety is defined as a score exceeding or equal to 8. Reliability and validity for this measure has been established (Herrmann, 1997).

#### *3.2.3.2.3 Medical Outcome Short Form-36 (SF-36)*

Patients also completed the MOS 36 item Short Form Health Survey (SF-36) (Ware, Jr. & Sherbourne, 1992) to evaluate health status and quality of life. This consists of eight subscales: limitations in physical activities because of health problems, limitations in social activities because of physical or emotional problems, limitations in usual role activities because of physical health problems, bodily pain, general mental health, limitations in usual role activities because of emotional problems, vitality and general health perceptions. A summary score for physical health status was calculated by averaging scores from the physical health subscales and a summary score for mental health status was calculated by averaging scores from the mental and social subscales (Ware, Kosinski, & Keller, 1994). Scores can range from 0 – 100 with 0 representing the lowest and 100 the highest level of functioning. Reliability and validity has been established for this measure (Brown et al., 1999).

#### **3.2.3.3 Clinical measures**

Clinical notes were consulted to identify any prior history of CHD and after the patient had completed the study, the findings from the coronary angiography were consulted by a physician (MB) to identify the presence or absence of definite CAD (defined as significant stenosis in at least one major vessel (Bhattacharyya et al., 2008)).

#### **3.2.3.4 Heart Rate Variability measurement**

Heart Rate Variability data was obtained by fitting the patient with a 24hour digital Holter monitor (Lifecard CF, Del Mar Reynolds, Hertford, UK) which was worn for between 22 and 26 consecutive hours. Data was recorded in 3 channels with a 6 electrode array and was digitised at 125Hz. Data was excluded after screening the QRS sequences if the current beat and the two beats before the current beat were not in sinus rhythm, and the normal to normal (NN) interval sequence was derived. Both frequency and time domain measures were calculated for up to 6 periods of five minutes within each 30 minute segment during the 24 hour monitoring. Each 5 minute period was assessed to identify any arrhythmia abnormalities and any periods with less than 80% valid data were excluded. VLF estimates were based on the complete 30 minute segments. The following frequency domain variables were computed: very low frequency (VLF) power between the limits 0.003 Hz and 0.04 Hz ( $\text{ms}^2$ ), low frequency (LF) power in the range 0.04 Hz to 0.15 Hz ( $\text{ms}^2$ ), and high frequency (HF) power in the range 0.15 Hz to 0.40 Hz ( $\text{ms}^2$ ). The following time domain variables were also computed: RMSSD (Square root of the mean of the sum of the squares of successive NN differences), pNN50 (Number of pairs of adjacent NN intervals differing by more than 50ms) and mean NN interval, as recommended by the Task Force (Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology, 1996). Higher values of each of these frequency and time domain HRV indices signify greater HRV whereas lower values of each of these indices indicates reduced HRV. The 30 minute means for both frequency and time domain measures were aggregated into a single day (start of monitoring until bedtime) and a single night value (bedtime until waking) for each measure of HRV. Bedtime and time of waking were identified by reference to participants' sleep logs in which the times of going to bed and waking up were recorded.

### **3.3 Statistical analyses**

Data were analysed using SPSS version 14. Married and unmarried groups were compared using t-tests for continuous variables, and  $\chi^2$  tests for categorical variables. Mean values of HRV measures in the day and night were compared using paired t-tests. The dependent variables were the computed HRV variables: NN interval, VLF, LF, HF, RMSSD and pNN50. All of the HRV measures were log transformed prior to analysis. The relationship between marital status and HRV was assessed using repeated measures analysis of covariance with marital status (married, unmarried) as the between-person factor and time of recording (day or night) as the within-person factor, and beta-blocker usage, BDI depression score, definitive diagnosis of CAD, age and gender as covariates, selected on the basis of previous literature associating them with HRV. *Post hoc* comparisons were carried out using Tukey's LSD test.

#### **3.3.1 My role in the study**

I was not directly involved with the data collection or initial processing of the data from this study. However, I completed the specific data analyses described here.

### **3.4 Results**

The demographic and clinical characteristics of the sample are summarised in Table 3.1. Patients were predominantly male, late middle-aged (mean age = 61.63, range = 37 - 82) and white. Fifty three (60%) participants were classified as married or living as married and 35 participants were classified as unmarried (40%). Of the 35 participants classified as unmarried, 11 were single never married, 13 were divorced, 7 were widowed and 4 were separated. Only 16 (18%) participants reported a prior history of CHD, and post-study examination of angiography results revealed a definite diagnosis of CAD in 58 participants (64%). Most participants were taking aspirin (88%) and statins (77%), and 67% were taking beta-blockers. Most participants were non-smokers (76%) and engaged in physical activity

more than once a week (84%). Most participants consumed alcohol (75%) with a small number drinking more than the recommended weekly amount (13%). Results from the psychological measures reveal that 39% of participants scored above the HADS anxiety threshold (>8) and 43% scored above the BDI threshold (>10), indicating moderately elevated levels of depression and anxiety within the sample. The results from the SF-36 reveal that physical and mental health status were moderately high on average, indicating limited impairment. Married participants were significantly younger and more likely to report a history of CHD compared with unmarried participants. No other significant differences between married and unmarried participants on demographic, clinical, psychological or health measures were observed, indicating that the two groups were comparable.

**Table 3.1 Demographic, clinical, health behaviour and psychological characteristics by marital status (N=88 unless otherwise stated)**

	<b>Total</b>	<b>Married</b>	<b>Unmarried</b>
<b><i>Demographic characteristics</i></b>			
Age in years	61.63 (9.54)	59.64* (9.16)	64.54* (9.48)
BMI in kg/m <sup>2</sup>	26.59 (3.96)	26.60 (4.04)	26.70 (3.89)
Ethnic minority patients, N (%)	25 (28%)	18 (34%)	7 (20%)
Female patients, N (%)	28 (32%)	13 (25%)	15 (43%)
Secondary education or greater, N (%)	47 (53%)	29 (55%)	18 (51%)
<b><i>Clinical characteristics</i></b>			
Chest pain, N (%)	77 (88%)	48 (91%)	29 (83%)
Number with CHD history, N (%)	16 (18%)	10 (29%)*	6 (11%)*
Number with definite CAD, N (%)	56 (64%)	31 (58%)	25 (71%)
Number taking beta blockers, N (%)	59 (67%)	36 (68%)	23 (66%)
Number taking nitrate N, (%)	37 (42%)	22 (41%)	15 (43%)
Number taking statins N, (%)	68 (77%)	46 (87%)	22 (63%)
Number taking aspirin N, (%)	77 (88%)	48 (91%)	29 (83%)
Number taking ACE inhibitors, N (%)	28 (32%)	15 (28%)	13 (37%)
<b><i>Health behaviours</i></b>			
Current smoker	21 (24%)	11 (29%)	10 (21%)
Drinks alcohol	66 (75%)	36 (68%)	30 (86%)
Drink more than recommended safe limits (>14 units female, >21 units male)	11 (13%)	6 (11%)	5 (14%)
Less than once per week physical activity in last 6 months	14 (16%)	7 (13%)	7 (20%)
Reported satisfactory sleep (n=86)	55 (64%)	34 (65%)	21 (62%)
<b><i>Psychological and health characteristics</i></b>			
BDI (n = 86)	10.20 (7.13)	10.36 (7.73)	9.97 (6.26)
HAD Anxiety Scale	6.54 (3.73)	6.38 (3.93)	6.80 (3.45)
SF- 36 physical health	62.53 (20.81)	61.63 (22.01)	63.89 (19.06)
SF-36 mental health (n=87)	65.03 (20.80)	64.99 (20.02)	65.09 (22.27)

\* Significant difference between married and unmarried participants ( $p < 0.05$ )

Means and standard deviations for each measure of HRV for the total sample and by marital status are displayed in Table 3. 2. Valid HRV data were obtained from 83 participants (53 married participants, 30 unmarried participants) and there were significant changes in all measures between day and night. NN interval was significantly longer during the night ( $t = 16.18$ ,  $p < 0.001$ ), and HF-HRV, LF-HRV, VLF-HRV, RMSSD and pNN50 were greater during the night compared to the daytime ( $t = 4.05$  to  $8.74$ , all  $p < 0.001$ ), reflecting a diurnal pattern of increased parasympathetic and reduced sympathetic activity at night.

**Table 3.2: Heart rate variability and NN interval means (SD) over study period (N=83)**

	<b>Total</b>	<b>Married</b>	<b>Unmarried</b>
NN interval (ms) Day	858.50 (124.50)	863.95 (110.12)	850.23 (145.02)
NN interval (ms) Night	1004.19 (153.70)	1015.51 (139.25)	987.03 (174.18)
High frequency ( $ms^2$ ) Day	4.87 (0.93)	4.98 (0.81)	4.70 (1.08)
High frequency ( $ms^2$ ) Night	5.57 (1.00)	5.75 (0.94)	5.29 (1.03)
Low frequency ( $ms^2$ ) Day	5.87 (0.79)	6.04 (0.71)	5.60 (0.84)
Low frequency ( $ms^2$ ) Night	6.29 (0.86)	6.50 (0.81)	5.96 (0.84)
Very low frequency ( $ms^2$ ) Day	7.18 (0.66)	7.28 (0.67)	7.02 (0.62)
Very low frequency ( $ms^2$ ) Night	7.40 (0.69)	7.60 (0.61)	7.11 (0.71)
RMSSD (ms) Day	3.00 (0.48)	3.08 (0.38)	2.88 (0.57)
RMSSD (ms) Night	3.30 (0.48)	3.41 (0.44)	3.13 (0.50)
pNN50 Day	0.97 (1.33)	1.18 (1.29)	0.65 (1.35)
pNN50 Night	1.61 (1.43)	1.96 (1.25)	1.08 (1.53)

*Abbreviations: RMSSD = square root of the mean of the sum of the squares of successive NN differences, pNN50 = the number of pairs of adjacent NN intervals differing by more than 50 ms, divided by the total number of NN intervals. All HRV measures are logged.*

Significant associations between marital status and HRV measures in both the frequency and time domains were observed. In the frequency domain measures, a significant main effect of marital status was found for LF-HRV ( $F(1, 75) = 4.80$ ,  $p = 0.032$ ,  $\eta^2 = 0.06$ ) and VLF-HRV ( $F(1, 75) = 4.74$ ,  $p = .0033$ ,  $\eta^2 = 0.06$ ). In each case, HRV was lower in

unmarried than married participants, independent of covariates. The interaction between marital status and time of recording was also significant for VLF-HRV ( $F(1, 79) = 4.15, p = 0.045$ ). *Post hoc* tests indicated that the difference between married and unmarried participants was significant in the night ( $p = 0.007$ ), but not the day ( $p = 0.13$ ). There were no significant effects of marital status on HF-HRV. For the time domain measures, a significant main effect of marital status was found for RMSSD ( $F(1, 75) = 5.70, p = 0.020, \eta^2 = 0.07$ ) and pNN50 ( $F(1, 75) = 5.32, p = 0.024, \eta^2 = 0.07$ ). RMSSD was significantly higher and the pNN50 was greater in married compared with unmarried participants. No significant main effects of marital status or interaction effects were found for NN interval.

### 3.5 Discussion

This study examined the association between HRV and marital status in patients with suspected CAD being investigated at Rapid Access Chest Pain clinics. A significant but modest relationship was observed between 24-hour HRV and marital status. Unmarried participants exhibited significantly reduced LF-HRV and VLF-HRV, RMSSD, and pNN50. These effects were independent of age, gender, beta-blocker usage and definite CAD diagnosis. There were no differences in depression, anxiety or quality of life between marital status groups and including depression score as a covariate did not modify the results. These findings indicate that both sympathetic (as reflected by LF-HRV) and parasympathetic (as reflected by VLF-HRV, RMSSD and pNN50) aspects of autonomic regulation are associated with marital status.

Our study results complement Horsten et al's finding that low social support and social isolation were associated with lower SDNN, LF-HRV and VLF-HRV which again suggests an impact on both sympathetic and parasympathetic modulation. However, Horsten et al also reported a robust association between social isolation and reduced HF-HRV which we did not detect within our sample. It is possible that our smaller sample size may have

contributed to lack of power to detect this, or it is also plausible that marital status effects on HRV are not the same as social isolation effects on HRV. Furthermore, measures of HF-HRV typically correlate with time domain measures of HRV as they are both thought to capture parasympathetic modulation. We did identify significantly lower RMSSD and pNN50 in the unmarried suggesting that parasympathetic activity was reduced even though this was not captured in the HF-HRV values. The reduction in both sympathetic and parasympathetic tone observed in our study and Horsten et al's is notable in the context of conventional conceptualisation of autonomic function where sympathetic and parasympathetic tone operate in opposing directions. This disturbance of autonomic balance may represent a lack of physiological responsivity which increases vulnerability to damage and disease. As discussed in the introduction, lack of physiological responsivity as reflected by reduced HRV has been associated with increased risk of CHD. These findings suggest that social isolation and being unmarried may be associated with a physiological rigidity that may contribute to the increased risk of CHD identified in these groups.

An interesting finding was the interaction between time of recording and marital status for VLF-HRV. VLF-HRV was significantly lower in unmarried compared with married participants during the night but not during the day. The reason is not clear, but one possibility is that the impact of marital status is more apparent during a phase of the diurnal cycle in which parasympathetic tone dominates. These circumstances may permit influences on VLF-HRV to emerge that are obscured by everyday activity. A second possibility is that night-time behavioral differences between married and unmarried participants may contribute. There could, for example, be differences in sleep quality, though the relationship between sleep and VLF-HRV is not well understood (Togo, Kiyono, Struzik, & Yamamoto, 2006). Research suggests that insomnia is higher among unmarried individuals compared to married individuals; in particular happily married women (Troxel, Buysse, Hall, & Matthews, 2009; Ohayon, 2002). However, in our sample there were no significant differences between

married and unmarried participants in self-reported sleep quality during the monitoring period. More objective and detailed measures of sleep quality, as well as greater exploration of the physiological correlates of VLF-HRV may provide further insight.

Overall our findings suggest that the unmarried participants have lower HRV compared with the married participants. As previously discussed, lower HRV is a predictor of CHD incidence and prognosis. Lower HRV has also been associated with risk factors for CHD development including hypertension, diabetes, elevated cholesterol levels and lifestyle risk factors, including smoking, physical inactivity and obesity (Thayer & Lane, 2007). The relationship between marital status and HRV observed in our sample may represent a potential physiological mechanism through which marriage exerts its beneficial impact. It is also possible that marriage or high social support may serve as a buffer against the detrimental consequences on HRV of negative psychological states. Our findings currently support a more direct impact as the associations between HRV and marital status were independent of negative psychological states.

There are a number of limitations to this study that should be acknowledged. The study was small and did not include a control group without cardiac symptoms or medication use. However, differences between marital status groups were sustained after medication and the presence of significant CAD were taken into account statistically. The size and constitution of the sample precluded any analysis of the interaction between gender, marital status and HRV, which may be significant in the context of research that has identified a more beneficial effect of marriage on health for men compared with women (Williams & Umberson, 2004; Hu & Goldman, 1990). The cardiovascular results are also limited by the absence of ejection fraction data which would have identified individuals with poor left ventricular function (Nolan et al., 1998). No measures of marital quality were collected. Not all marriages are good, and

poor marital quality has been identified as a risk factor for CHD (Eaker, Sullivan, Kelly-Hayes, D'Agostino, Sr., & Benjamin, 2007) and as previously discussed, marital conflict has been found to increase cardiovascular reactivity whereas positive marital interaction and physical contact has been found to reduce cardiovascular reactivity (Kiecolt-Glaser & Newton, 2001; Ditzen et al., 2007). Emerging evidence also suggests a marital quality influence on high frequency HRV (Smith et al., 2011). Married individuals in high quality marriages may have greater HRV than married individuals in low quality marriages indicating a gradient of physiological protection.

A further limitation of the study was the binary classification of participants as either married or unmarried, since the unmarried group consisted of widowed, separated, divorced and never married participants. The sample was not large enough to distinguish these groups, but other work suggests that there are significant mortality and health differentials among the various non-married states. International and large scale population research indicates that divorced individuals have the highest risk of all-cause mortality among unmarried individuals, followed by widowed individuals (Manzoli, Villari, Pirone, & Boccia, 2007; Hu & Goldman, 1990; Johnson et al., 2000; Sbarra & Nietert, 2009; Lund, Christensen, Holstein, Due, & Osler, 2006). Potential explanations for this excess mortality have been suggested to include the effects of selection (other characteristics that may increase ones chance of divorce which may also reduce health), loss of the protective effects of marriage and the physiological impact of the psychological stress associated with marital discord and loss (Sbarra, Law, Lee, & Mason, 2009). Being divorced or widowed has been found to increase risk of mental health problems, chronic health conditions and mobility problems (Hughes & Waite, 2009; Hewitt, Turrell, & Giskes, 2010), and has also been associated with poorer self-rated health and psychological health in women (Cheung, 1998). Divorced or widowed individuals tend to exhibit less health oriented behaviour and more risky behaviour and have been found to have higher alcohol and tobacco consumption, higher likelihood of smoking cessation relapse, greater sleep disruption, increased weight loss and poorer dietary quality

in men (Eng et al., 2005; Lee et al., 2005). Few studies have explored potential psychophysiological factors which may link marital dissolution to health; one study found increased blood pressure among recently divorced or separated individuals who exhibited greater general and task related emotional reactions to a task focusing on the recent marital dissolution (Sbarra et al., 2009) and another study identified markers of compromised immunity among recently divorced individuals (Kiecolt-Glaser et al., 1987). In our study, divorced or widowed participants constituted a moderate proportion of the unmarried sample (57%) which may have contributed to the strong association between unmarried status and impaired HRV. Further exploration of the association between different unmarried states, particularly divorced, and HRV is warranted as this may represent an important psychophysiological link.

Furthermore, this study focused solely on current marital status whereas, reflective of a recent shift in social support research towards a more lifespan perspective, the importance of both current marital status *and* marital history in predicting morbidity and mortality has recently emerged. Research suggests that being continuously married (i.e. married once with no marital disruptions) confers the greatest general health benefits and is associated with lower all-cause mortality in mid-life and beyond (Hughes & Waite, 2009; Blomgren, Martikainen, Grundy, & Koskinen, 2010). Multiple marital disruptions and shorter marital duration are associated with increased risk of all-cause mortality (Henretta, 2010; Dupre, Beck, & Meadows, 2009). Research has also identified a higher prevalence of cardiovascular disease amongst individuals who have experienced marital loss (whether through divorce, separation or widowhood) with shorter marital duration being positively associated with cardiovascular disease incidence particularly for men (Zhang & Hayward, 2006). Research is currently underway at the University of Texas examining the relationship between marital history and various biological markers which include blood pressure (McFarland & Hayward, 2010). A more detailed exploration of the impact of marital history on HRV may offer further insight into how marital status past and present may exert an

impact on CHD morbidity and mortality. This may be particularly germane in the context of modern societal shifts towards more heterogeneous marital experiences over the lifespan.

This study illustrates a marital status differential in 24-hour HRV and suggests that impaired autonomic cardiac control may be a biological trajectory through which unmarried marital status increases risk of disease supporting the notion of a biological influence of marriage on health (Kiecolt-Glaser & Newton, 2001). The findings contribute to our understanding of factors that influence HRV and provide insight into the psychophysiological mechanisms linking marital status and health. By investigating the mechanisms underlying marital status health differentials, research can provide support for risk stratification in clinical cardiovascular populations and identify factors that might be targeted by interventions. In the context of the high prevalence of CAD, issues surrounding risk stratification and tailored interventions are becoming increasingly salient.

# CHAPTER 4 METHODOLOGY TRACE STUDY

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## Tracking Recovery After Coronary Events (TRACE) Study

### 4.1 Design

The TRACE study is a prospective longitudinal study incorporating four assessment time points conducted over one year following admission for ACS (Figure 4.1). Time 1 assessment was conducted in-hospital within two days of admission, Time 2 was conducted approximately 10-14 days following hospital discharge, Time 3 follow up assessment was conducted at six months post admission and Time 4 follow up assessment was conducted 12 months post admission.

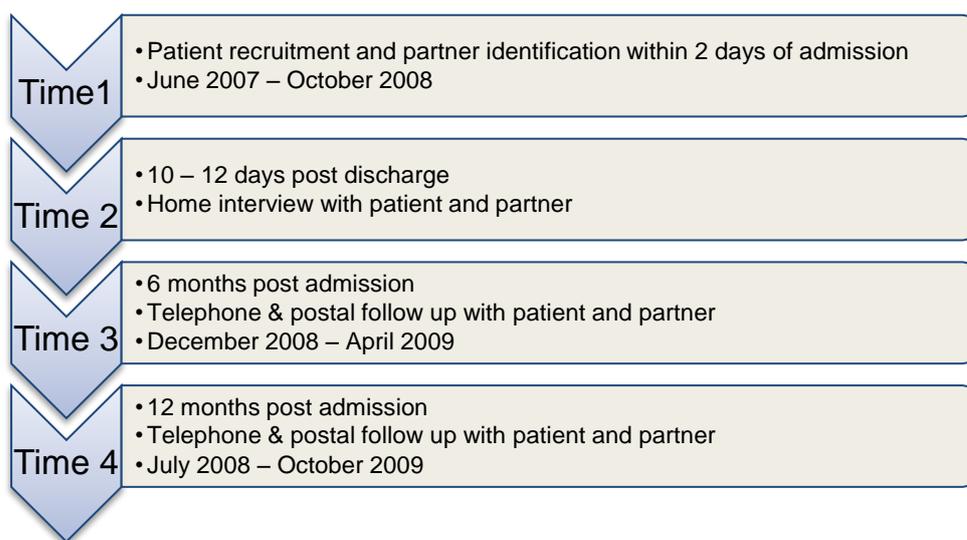


Figure 4.1 TRACE study design

### 4.2 Hypotheses

#### 4.2.1 Structural and functional social support as predictors of short and long term psychological response and adjustment following ACS

The most consistent research support illustrating an association between social support, mortality and morbidity has been found within the domain of cardiovascular disease

research. There is substantial evidence indicating a significant prognostic role for both structural and functional social support in recovery following CHD and ACS. Low social support has been associated with a 2 – 4 fold increased risk of cardiac mortality in patients with existing CHD (Lett et al., 2005). There are numerous pathways through which social support may exert an impact on prognosis and recovery. One of the key pathways may be via the psychological response of the patient following their ACS. Depression, anxiety and poor quality of life are strongly associated with social isolation and lack of social support, and have also been identified within post ACS populations (Moser & Dracup., 1996; Thombs et al., 2006). Psychological distress has also been found to increase mortality, morbidity and reduce engagement in recovery behaviours (Kaptein et al., 2006; Mayou et al., 2000). Socially isolated individuals who experience ACS may be more vulnerable to psychological distress which may increase their risk of a poor prognosis. In order to further explore the association between social support and psychological distress within cardiac populations, I intend to determine whether patient reported social support at Time 2 predicts the occurrence of depression, anxiety and quality of life at Time 2, Time 3 and Time 4.

Based on prior work within the social support research field, the following hypotheses will be addressed:

- i. Lower levels of functional and structural social support, assessed soon after hospital discharge for ACS will be associated with depression shortly after discharge (T2) and predictive of depression at six months (T3) and 12 months (T4).*
- ii. Lower levels of functional and structural social support, assessed soon after hospital discharge for ACS will be associated with anxiety shortly after discharge (T2) and predictive of anxiety at six months (T3) and 12 months (T4).*
- iii. Lower levels of functional and structural social support, assessed soon after hospital discharge for ACS will be associated with poorer quality of life shortly*

*after discharge (T2) and predictive of poorer quality of life at six months (T3) and 12 months (T4).*

#### **4.2.2 The relationship between social support and heart rate variability (HRV) in post ACS patients**

Numerous physiological pathways have been proposed to mediate the impact of social support on health outcomes (Uchino et al., 1996). One of the key physiological pathways may be via the dysregulation of the autonomic nervous system (ANS) and a critical indicator of autonomic nervous system regulation is heart rate variability (HRV). HRV. The emerging relationship between ACS, HRV and social isolation has already been described in detail in Chapter 3 where an association between marital status and HRV was noted. Building on these findings, I intend to explore whether lower HRV may represent a final physiological mechanism through which social isolation and lack of support may negatively impact recovery after ACS. Based on these previous research findings, I hypothesise that;

- iv. Lower levels of functional and structural social support, assessed at Time 2, will be predictive of reduced HRV at Time 2.*

#### **4.2.3 Marital status and satisfaction as predictors of short and long term psychological response and adjustment**

The social support of a partner has been found to be particularly valuable to recovering ACS patients, with married patients tending to have a better prognosis than unmarried patients (Randall et al., 2009). Furthermore, marital quality and functioning have also begun to emerge as important prognostic factors and have been associated with patient distress, adjustment, mortality and morbidity (Arefjord et al., 1998; Bennett & Connell, 1999; Coyne &

Smith, 1994; O'Farrell et al., 2000; Orth-Gomer et al., 2000). There is a need to further explore the role of marital status, satisfaction and functioning in influencing the biological and psychological response and adjustment of the patient. I intend to establish the role of marital status in predicting distress and quality of life at Time 2, 3 and 4. I also will explore the levels of marital satisfaction reported by patients at Time 2 and aim to determine whether patient reported marital satisfaction at Time 2 predicts the occurrence of depression, anxiety and quality of life at Time 2, Time 3 and Time 4.

Based on prior work within this area, the following hypotheses were addressed:

- v. *Married patients will be predicted to experience lower levels of anxiety and depression, and higher levels of quality of life shortly after discharge (T2), six months (T3) and 12 months (T4).*
- vi. *Lower levels of marital satisfaction, assessed soon after hospital discharge for ACS will be associated with depression shortly after discharge (T2), and predictive of depression at six months (T3) and 12 months (T4).*
- vii. *Lower levels of marital satisfaction, assessed soon after hospital discharge for ACS will be associated with anxiety shortly after discharge (T2), and predictive of anxiety at six months (T3) and 12 months (T4).*
- viii. *Lower levels of marital satisfaction, assessed soon after hospital discharge for ACS will be associated with poorer quality of life shortly after discharge (T2) and predictive of poorer quality of life at six months (T3) and 12 months (T4).*

#### **4.2.4 The relationship between marital status, marital satisfaction and heart rate variability (HRV)**

As previously described in hypothesis 4.2.2, there is growing evidence of an association between HRV and social isolation in ACS patients. A significant association between marital

status and HRV was noted in Chapter 3 whereby unmarried patients were found to have reduced HRV compared to married patients with suspected coronary artery disease. Research has also illustrated that marital satisfaction and interaction may influence aspects of cardiovascular reactivity including HRV (Kiecolt-Glaser & Newton, 2001). I aim to identify any relationships between marital status, marital satisfaction and HRV at Time 2. Based on these research findings, I hypothesise that;

- ix. *Unmarried patients will have lower HRV compared to married patients at Time 2.*
- x. *Low satisfied married patients will have lower HRV compared to high satisfied married patients at Time 2.*

#### **4.3 Participants**

Patients were recruited following admission into a South London Hospital for an ACS. Diagnostic criteria for confirmation of ACS were the presence of chest pain and verification by diagnostic ECG changes (new ST elevation  $>0.2\text{mV}$  in 2 contiguous leads in leads V1, V2 or V3 and  $>0.1\text{mV}$  in 2 contiguous other leads, ST depression  $>0.1\text{mV}$  in 2 contiguous leads in the absence of any QRS confounders, new left bundle branch block or dynamic T wave inversion in more than one lead), or troponin T measurement  $>0.1$  micro g/l and/or a creatine kinase measurement more than twice the upper range of normal for the measuring laboratory. Patient eligibility criteria for participation were admission for ACS, aged 18 years or over and ability to complete the interview and questionnaire measures in English. Exclusion criteria were the presence of co-morbid conditions which may have influenced either symptom presentation, mood state or contributed to false troponin positivity (such as severe psychiatric illness, unexplained anaemia, ongoing infection or inflammatory conditions, neoplasia and renal failure). Patients who were too unwell or clinically unstable (for example, patients experiencing continuing chest pain and critical ischaemia or

ventricular tachyarrhythmias) were also excluded. Partners of recruited patients were also invited to take part in the study. Partners were defined as legally married spouses or other long term cohabiting partner. Partner exclusion criteria were inability to complete the interview or questionnaires in English or aged less than 18.

Data collection was conducted in the hospital between June 2007 and October 2008. During this period, 693 potentially eligible patients were admitted on the days recruitment was conducted. Of these potentially eligible patients, a total of 395 patients were either excluded or refused to participate leaving a total sample size of 298 at Time 1. The primary reason for exclusion was the patient had been discharged or transferred to another hospital before the researcher was able to recruit them (n=125, 18%). Attrition rates and reasons for exclusion and attrition at Time 1 are detailed in Table 4.1. Of the 298 patients who completed the Time 1 interview, 222 completed that Time 2 home interview (74.5%). A further 4 patients completed a postal version of the Time 2 home interview making the total sample 226 (75.8%) at time 2. The main reason for attrition at Time 2 was patient refusal (n=40, 13.4%). Attrition rates and reasons for attrition at Time 2 are outlined in Table 4.1. Time 3 (6 month) follow up assessments were completed between December 2007 and March 2009. A total of 200 (67%) patients completed the Time 3 follow up assessment. The main reason for attrition was failure to contact the patient (n=63, 21.1%). Time 4 (12 month) follow up assessments were completed between June 2008 and October 2009. A total of 176 (59%) patients completed the Time 4 follow up assessment. The main reason for attrition was failure to contact the patient (n = 70, 35%). Attrition rates and reasons for attrition at Time 2, 3 and 4 are detailed in Table 4.1.

**TABLE 4.1 PATIENT ATTRITION RATES AND REASONS FOR EXCLUSION**

	n	%
<b>Eligible population</b>	<b>693</b>	
Discharged or transferred	125	18
Declined to participate	58	8.4
Deceased before inclusion	7	1
<b>Exclusions</b>	<b>395</b>	
Patient too unwell/unable to communicate	90	13
Not able to speak English	27	4
Recruitment break (Christmas)	24	3.5
Confused	23	3.3
Cardiac event occurred as in-patient	13	1.9
Serious psychiatric problem	10	1.4
Patient did not live within recruitment catchment area	9	1.3
Patient in isolation	5	0.7
Patients cardiac event not ACS	3	0.4
Adverse situation	1	0.1
<b>Total patients completing Time 1</b>	<b>298</b>	<b>75.4</b>
Declined to participate in interview	40	13.4
Failed to contact	17	7
Health reason (re-admissions, patient too unwell, memory problems)	12	4
Deceased	3	1
<b>Total patients completing Time 2 assessment</b>	<b>226*</b>	<b>75.8</b>
Failed to contact	63	28.4
Declined to participate further	32	14.4
Deceased	3	1.3
<b>Total patients completing Time 3 follow up assessment</b>	<b>200</b>	<b>67</b>
Failed to contact	70	35
Declined to participate further	39	19.5
Health reasons	7	3.5
Deceased	6	3
<b>Total patients completing Time 4 follow up assessment</b>	<b>176</b>	<b>59</b>

*\*This includes 9 patients who completed a home postal version of the interview and questionnaire assessment and 2 patients who completed a home postal version of the questionnaire assessment only.*

## **4.4 Procedure**

### *4.4.1 Time 1 (in hospital) assessment*

Patients were consecutively recruited, according to the exclusion criteria described previously, from the Coronary Care Unit at St. George's Hospital in London. As soon as practicable following admission for ACS, patients were approached by a researcher from the TRACE research team. The researcher provided full details of the study and gave the patient an information sheet for reference. Patients who decided to participate were asked to complete a consent form. A blood sample was taken within 24 hours for the assessment of CRP, neutrophil counts, tumour necrosis factor (TNF) alpha, and IL-10. The researcher conducted a detailed in-hospital interview which concentrated on the patients' acute fear response and distress during ACS as well as the circumstances surrounding symptom onset and hospital admission. Current mood state and quality of life prior to hospital admission was also obtained. The researcher reviewed the patient clinical notes to gather information regarding relevant clinical details including history of heart failure, arrhythmia on admission and prior ACS as well as the proposed treatment strategy for the patient. Angiography results were also collated where available. A clinical risk score was calculated using the composite measure developed in the Global Registry of Acute Coronary Events (GRACE).

### *4.4.2 Time 2 (post discharge) assessment*

Patient discharge dates were monitored by a researcher from the TRACE team and shortly after discharge, patients were contacted by telephone to organise the home based Time 2 assessment. The patient's spouse/partner was also invited to participate in this stage of the study. The home based Time 2 assessments were conducted an average of 21 days (SD 8.5 days) following admission for ACS, and the interval ranged from 8 and 51 days. Each home assessment was conducted by two researchers; one researcher conducted an assessment with the patient and the other simultaneously conducted an assessment with the patient's spouse/partner. The patient and spouse/partner assessments occurred in separate rooms wherever possible.

The patient home assessment consisted of a structured clinical interview (DISH) designed to evaluate depression and psychiatric history, and a number of self-complete and interview format psychosocial and health behaviour measures. The patients' heart rate and heart rate variability was also monitored for the duration of the home assessment using an Actiheart ambulatory device fitted by the researcher prior to the assessment. Salivary cortisol samples were collected at four points during the assessment: at the start of the assessment, prior to the DISH interview, at the end of the DISH interview and at the end of the assessment. Patients were also asked to complete a battery of questionnaires to be returned via a provided freepost envelope. The patients' spouse/partner home assessment consisted of an interview to assess the circumstances of the patients ACS, and a number of self-complete and interview format psychosocial and health behaviour measures. Salivary cortisol samples were collected at the start and at the end of the assessment. Both patients and spouses/partners were also asked to collect six salivary cortisol samples over the course of a single day (not on the same day as the home assessment) and tubes were to be returned via freepost.

#### *4.4.3 Time 3 (6 month) follow up assessment*

Patients were contacted by telephone at six months following their admission for ACS and a telephone assessment was conducted. This assessment involved a semi-structured interviewing assessing symptom recurrence, health problems, cardiac rehabilitation attendance, medication adherence, health behaviour and return to work. These assessments were conducted on average 193 days following the original admission date with a range between 137 and 281 days following admission. Both patients and their spouses/partners were sent a packet of self-complete questionnaires to return by post.

#### *4.4.4 Time 4 (12 month) follow up assessment*

Patients were contacted by telephone at six months following their admission for ACS and a telephone assessment was conducted. This assessment involved a semi-structured

interviewing assessing symptom recurrence, health problems, cardiac rehabilitation attendance, medication adherence, health behaviour and return to work. These assessments were conducted on average 387 days following the original admission date with a range between 345 and 765 days following admission. Both patients and their spouses/partners were sent a packet of self-complete questionnaires to return by post.

#### *4.4.5 My role in study design, data collection and analysis*

I had a number of key responsibilities within the TRACE study team of researchers. I contributed to study design through the identification and selection of questionnaires and discussion of procedural issues. I was significantly involved with data collection including organising and conducting patient and partner assessment at home. These home assessments included undertaking interviews, facilitating questionnaire completion and gathering biological data from patients and partners. I also completed 12 month follow up interviewing of patients and partners via telephone interviews and postal questionnaires. I contributed to 6 month follow up interviewing of patients and partners. I was also responsible for data entry of patient and partner data from Time 2, 3 and 4.

### **4.5 Measures**

The TRACE study utilised a diverse selection of measures which are described within this section. Questionnaire and interview measures used for patient assessment at Time 1, Time 2, Time 3 and Time 4 are provided in Appendices I, II, III and IV. Only those measures that were used for the purposes of this thesis are described in detail in this section. Table 4.2 provides a detailed depiction of all the measures that were administered to patients and/or partners and at which assessment point. A number of questionnaires were interview administered to improve data collection and aid completion, and most questionnaires had precedent in cardiac populations. Cronbach's alpha scores for each of the measures used are listed in Table 4.3.

#### **4.5.1 Time 1 Measures**

This section refers to measures administered to the patient during the Time 1 hospital interview.

##### *4.5.1.1 Socio-demographic information*

Socio-demographic information including age, marital status and duration, ethnicity, employment status at admission, educational qualifications and income were obtained at the Time 1 hospital interview. Patients were categorised as 'low', 'medium' or 'high' on a composite social deprivation index adapted from the Townsend Material Deprivation Index (1988). This index offers a comprehensive measure of social deprivation and has also been shown to be associated with increased cardiovascular risk factors (Sunquist et al., 1999). Social deprivation was evaluated based on the following four factors: home rental (rather than home ownership), living in a crowded household (defined as more than one person per room), not having access to a car or van and receiving state benefits. Scores on these items ranged from 0 to four, with four being the highest level of deprivation. Participants were classified into three categories; low deprivation (negative on all items), medium deprivation (one positive score) and high deprivation (two to four positive items). Socio-economic status (SES) was measured using patient income and educational qualifications. Educational attainment was selected as a gauge of socio-economic position due to ease of assessment and applicability to individuals both in stable employment and those outside active employment. Educational qualifications reported included none, school certificate, CSE's, GCSE's, A'level, Degree and Other. For statistical analyses, these qualifications were reclassified into four categories; 'none', 'basic', 'secondary' and 'degree'. Patients also indicated their gross personal yearly income and the total household income for the last year. Income was classified into 5 response categories: under £10,000, £10,000-20,000, £20,000-30,000, £30,000-40,000 or over £40,000.

#### *4.5.1.2 Clinical data*

Clinical information was obtained from the hospital admission records. Information obtained included admission ECGs and troponin T or creatine kinase levels for review by a cardiologist in order to classify patients as presenting with ST-elevation myocardial infarction (STEMI), non ST- elevation myocardial infarction (NSTEMI) or unstable angina (UA). This information was subsequently categorised as a binary variable (STEMI vs NSTEMI/UA). Clinical risk indices used included the Global Registry of Acute Coronary Events (GRACE) index (Eagle et al., 2004). The GRACE index is a composite clinical algorithm which utilises nine criteria to estimate risk of six month post ACS discharge death. These criteria are age, history of congestive heart failure, history of MI, systolic blood pressure and heart rate on admission, ST segment depression, initial serum creatine, raised cardiac enzymes and no in-hospital percutaneous coronary intervention. The GRACE score was also transformed into a three category variable based on the cutoff points recommended by Elbarouni et al, (2009). The three categories are low risk ( $\leq 125$ ), moderate risk (126 – 154) and high risk ( $\geq 155$ ).

#### **4.5.2 Time 2 Measures**

This section refers to measures administered to patient during the Time 2 home interview.

##### ***4.5.2.1 Psychosocial measures***

###### *4.5.2.1.1 Beck Depression Inventory (BDI)*

The Beck Depression Inventory (Beck et al., 1988) was used to assess both patient and partner level of depression. The BDI is a well acknowledged standardised measure of depressive symptomatology that has been validated in cardiac populations (Buchanan et al., 1993; Crowe et al., 1996; Frasure-Smith et al., 1997). The BDI consists of 21 self-report items that assess the severity of depressive symptoms over the past week (this time period was adapted in the TRACE study to refer to the period following the patients ACS). Symptoms included sadness, anhedonia, guilt, crying, fatigue and lack of appetite. Patients

rate symptoms from *none (0)* to *severe (3)* and scores can range between 0 and 63. Higher scores indicate the presence of more severe depression. A series of standard cut off points can also be applied which are as follow; 0-9 suggests no indication of depression, 10 – 18 denotes mild to moderate depression, 19 – 20 suggests moderate to severe depression and 30 – 63 would denote severe depression.

#### *4.5.2.1.2 Hospital Anxiety and Depression Scale (HADS)*

The anxiety subscale from the Hospital Anxiety and Depression scale was utilised to assess anxiety in patients and partners. The HADS was developed to assess both anxiety and depression in medical patients suffering from a range of diseases, and is a well-regarded and prolific measure of distress in medical patients (Zigmond & Snaith, 1983). The HADS has demonstrated consistent good reliability and validity in medical, psychiatric and general populations (Bjalland et al., 2002). The HADS anxiety subscale consists of seven items (five items are reverse scored) which patients rate using a 4 point scale *from not at all anxious (0)* to *very often anxious (3)*. Total scores range from 0 to 21, with higher scores indicative of greater anxiety. A score of 8 or above is the established cut-off for moderate anxiety.

#### *4.5.2.1.3 Marital satisfaction*

Patient and partner marital satisfaction was assessed using a 7 item measure (Troxel et al., 2005). This measure assesses satisfaction with amount of time spent together, communication, sexual activity, agreement of financial matters as well as similarity of interests, lifestyle and temperament, as well as agreement on financial matters. All questions were scored using a 4 point Likert scale ranging from *not at all satisfied (0)* to *very satisfied (3)* with total scores ranging between 0 and 21. Higher scores indicated greater marital satisfaction. The measure has demonstrated good internal consistency and reliability in previous studies (Troxel et al., 2005).

#### *4.5.2.1.4 Social Network Index (SNI)*

The Social Network Index (SNI; Cohen et al., 1997) is a role based social integration measure that assesses the extent and diversity of the social network surrounding the individual. The scale measures participation in social relationships with 12 types of social contacts; partner, children, parents, parents-in-law, close relative, close friend, religious group contacts, educational group contacts, work colleagues, neighbours, volunteer group contacts and other group contacts. For each type of contact, participants indicate how often they speak to (in person or on the phone) that contact using a 5 item scale; never, once a month, once every two weeks, once a week or every day. A score of 1 is allocated for each type of contact (range 0 – 12) with whom the participant reports they speak to (in person or on the telephone) at least once every two weeks. Higher values indicate more diverse social networks. The SNI has been previously utilised with cardiac patient populations (Molloy et al., 2008).

#### *4.5.2.1.5 ENRICHD Social Support Inventory (ESSI)*

The ENRICHD social support inventory (ENRICHD Writing Committee, 2003) assesses the quality of available social support using six questions pertaining to the amount of instrumental and emotional support perceived to be available by the participant. Questions included “Is there someone available to you to give you good advice about a problem?” “Can you count on anyone to provide you with emotional support (talking over problems or helping you make a difficult decision)?” All questions are scored on a five point scale ranging from *none of the time (1)* to *all the time (5)*. A final question assessed marital status using a binary response (yes = 1, no = 0). The scores were totalled to provide an overall social support score between 7 and 34 whereby higher values indicate greater perceived social support. The ESSI has been used primarily as a screening tool to determine low perceived social support (LPSS) in CHD patients shortly after ACS and also for monitoring subsequent changes in levels of social support. LPSS scored in this way has been independently associated with increased risk of mortality and recurrent MI (Burg et al., 2005). The ESSI

provides detailed criteria for determining LPSS which is listed in Table Figure 4.2. These criteria were applied to our sample to determine the proportion of patients who would be classified as having LPSS.

Figure 4.2 ESSI Criteria for Low Perceived Social Support

***ESSI Low Perceived Social Support Criteria***

- a. Score  $\leq 2$  on at least two items of the ESSI (excluding item 4 – help with chores) **OR**
- b. Score  $\leq 3$  on at least two items (excluding item 4 – help with chores and assessment of marital status) **AND** a total score of 18 or less on items 1,2,3,5 and 6.

The ESSI offers a short, reliable and valid measure of social support and was specifically developed for cardiac patient populations (Vaglio et al., 2004).

*4.5.2.1.6 Medical Outcome Short Form 12 (SF-12) Quality of life*

The SF-12 measures perceived health related quality of life and is a 12 item abbreviated version of the SF-36 (Ware et al., 1996). The SF-36 consists of 36 items divided into eight subscales which assess three key domains which are: (1) functional status – physical functioning (limitations in physical activity due to physical problems), social functioning (interference with social activities due to physical and emotional health problems), role limitations due to physical problems (problems with work and daily activities due to physical health), role limitations due to emotional problems (problems with work and daily activities due to emotional problems); (2) well-being – mental health (anxiety and depression), vitality (energy and fatigue), bodily pain (severity); (3) overall evaluation of health – general health perception (evaluation of physical health and likelihood of improvement). Two summary score components can also be calculated from the scores on the eight subscales: physical health status and mental health status.

The SF-12 replicates the eight-scale profile of the SF-36 and the scores for each scale are coded, summarized and converted into a scale ranging from 0 (worse possible health) to 100 (best possible health) and total scores on the two summary components can also be calculated. The SF-12 offers a more concise and simple alternative to the SF-36 and has demonstrated reliable and robust assessment of health related quality of life of patients with coronary heart disease (e.g. Melville et al., 2003; Muller-Nordhorn et al., 2004).

#### **4.5.2.2 Health behaviours**

##### *4.5.2.2.1 Diet*

Patient and partner dietary intake was evaluated using two measures; the fruit and vegetable intake scale devised by Steptoe and Cappuccio, and a fat intake scale. The fruit and vegetable measure was developed for an intervention study on the effects of brief behavioural counselling for increasing fruit and vegetable intake (Steptoe et al, 2003). It was selected because it is short, and has been validated against biomarkers of fruit and vegetable intake (Cappuccio et al, 2003). Participants reported the average number of pieces of fruit consumed on a typical day and indicated how often they would consume less than this average figure reported per week (“How often do you eat less than this average figure?”) using a six point scale ranging from never to five or more times a week. The same questions were asked to determine average vegetable (excluding potatoes) intake. An average daily fruit and vegetable intake score was calculated based on participants responses regarding daily fruit and vegetable intake and the frequency of eating less than this amount per week. Dietary fat intake was assessed using nine questions that determined the frequency of consumption of high saturated fat foods including full fat milk, cheese, ready meals, take away food, cakes and biscuits. Higher scores indicated a diet higher in saturated fat.

##### *4.5.2.2.2 Physical activity*

Physical activity over the past week was assessed by asking patients about the number of minutes they spent walking and cycling on a weekday and on a weekend day. Patients were

also asked to report their average walking pace (slow, steady, brisk or fast). Patients and partners also reported the number of times per week they performed vigorous physical activity, enough to make them out of breath, prior to the patient's ACS. These measures have previously been used in the Whitehall II study, where it has been associated prospectively with the metabolic syndrome, impaired cognitive function, and cardiovascular morbidity (Rennie et al., 2003).

#### *4.5.2.2.3 Smoking*

Patients and partners were asked about their smoking status (current smoker, ex-smoker, never smoked) and, if applicable, the number of cigarettes/cigars/pipe smoked per day.

#### *4.5.2.2.4 Alcohol consumption*

Patient and partner weekly alcohol consumption was assessed using a measure adapted from one used in the Whitehall studies, to determine the number of units of alcohol consumed per week. A unit of alcohol was defined as one measure of spirit, a small glass of wine, or a half pint of beer.

#### *4.5.2.2.5 Medication adherence*

Patient self-reported adherence to medications was assessed using the Medication Adherence Report Scale (MARS; Horne & Weinman, 1999). Patients rate their adherence using five questions referring to forgetting medication, altering the dose of medication, stopping medication, deciding to miss a dose and taking less than instructed. The questions were scored on a 5-point Likert scale from *Never* (4) to *Always* (0), total scores range from 0 to 20 with higher scores suggesting greater adherence. The questionnaire was also adapted for partners to rate the patient's adherence.

#### 4.5.2 Heart Rate Variability

An Actiheart monitor (Cambridge Neuroscience Ltd) was attached to each patient at the beginning of the Time 2 patient assessment to record their heart rate and heart rate variability during this assessment. The Actiheart monitor is fixed to the chest and consists of two electrodes linked by a lead which clips onto two standard ECG pads. The Actiheart records heart rate, inter-beat interval and physical activity. On completion of the assessment, the Actiheart monitor was removed from the patient and the data was downloaded to a computer for storage and later analysis. The Actiheart converts the ECG signal to digital form and determines the inter-beat interval from the R-R interval. Heart rate and heart rate variability can then be calculated based on the inter-beat interval recording files. The Actiheart monitor has established reliability and validity for recording activity and heart rate (Brage, Brage, Franks, Ekelund, & Wareham, 2005).

The HRV sequences were screened for data quality, and NN (normal to normal) intervals were excluded if the current beat and the two beats before the current beat were not in sinus rhythm. Specifically, we excluded NN intervals  $<300$  ms or  $>3000$  ms, any NN intervals  $<80\%$  or  $>120\%$  of the previous NN, and any intervals  $>3$  times the SD of the preceding period. The interview recording sequence was analysed in 10-minute segments and any episodes with  $<80\%$  valid data were excluded. The following frequency domain variables were computed: very low frequency (VLF) power between the limits 0.003 Hz and 0.04 Hz ( $\text{ms}^2$ ), low frequency (LF) power in the range 0.04 Hz to 0.15 Hz ( $\text{ms}^2$ ), and high frequency (HF) power in the range 0.15 Hz to 0.40 Hz ( $\text{ms}^2$ ). The following time domain variables were also computed: RMSSD (Square root of the mean of the sum of the squares of successive NN differences), pNN50 (Number of pairs of adjacent NN intervals differing by more than 50ms) and mean heart rate, as recommended by the Task Force (Task Force of the European Society of Cardiology and the North American Society of Pacing Electrophysiology, 1996). Higher values of each of these frequency and time domain HRV

indices signify greater HRV whereas lower values of each of these indices indicates reduced HRV. All variables (excluding heart rate) were log transformed due to highly skewed data.

#### **4.7 Time 3 measures**

##### *4.7.1 Psychosocial measures*

The Time 3 assessment took place approximately 6 months following patient admission for ACS. Patients and partners completed the following measures; BDI, HADS-A, SF-12, ESSI, Marital satisfaction questionnaire.

##### *4.7.2 Health behaviour*

All health behaviour, including medication adherence, of both patient and partner were reassessed.

#### **4.8 Time 4 measures**

##### *4.8.1 Psychosocial measures*

The Time 4 assessment took place approximately 12 months following patient admission for ACS. Patients and partners completed the following measures; BDI, HADS-A, SF-12, ESSI, Marital satisfaction questionnaire.

##### *4.8.2 Health behaviour*

All health behaviour, including medication adherence, of both patient and partner were reassessed.

#### **4.9 Data Storage**

All data collected for this study was treated as strictly confidential and stored in locked filing cabinets with restricted access. Consent forms were stored separately from interview and questionnaire data, and all data entered into a database was anonymised with personal information stored separately.

#### **4.10 Statistical analyses**

All statistical analyses were performed using the statistical programme SPSS 17.0 (SPSS Inc). The statistical techniques used to analyse data from this study for the purposes of this thesis are described in detail in the relevant results section.

**Table 4.2 Measures administered in the TRACE study**

*Measures in Italics are those utilised by this thesis*

	Time 1	Time 2		Time 3		Time 4	
Place	Hospital	Home		Tel/post		Tel/post	
Time since admission	6-28 hrs	21 days		6 months		12 months	
MEASUREMENTS	Pt only	Pt	Part	Pt	Part	Pt	Part
<i>Socio-demographics</i>	<i>CN</i>		INT				
<i>Clinical ACS details</i>	<i>CN</i>						
Health details	<i>INT</i>						
Triggers (During 2 hours pre-ACS)	INT						
Triggers (During 2 hours previous day)	INT						
Acute Fear	INT						
Events surrounding heart problem / delay	INT		INT				
Cardiac Rehabilitation attendance				INT		INT	
<b>1. Emotional distress</b>							
<i>Hospital Anxiety and Depression Scale – Anxiety</i>		<i>INT</i>	SR	SR	SR	SR	SR
<i>Beck Depression Inventory</i>		<i>INT</i>	SR	SR	SR	SR	SR
PTSD/ Acute Stress		INT	SR	SR	SR	SR	SR
Depression Interview and Structured Hamilton		INT					
Profile of Mood states	INT						
<b>2. Behaviour</b>							
Medication Adherence Report Scale	<i>INT</i>	INT	INT	INT	SR	INT	SR
Physical Activity		INT	INT	SR	SR	SR	SR
Diet		INT	INT	SR	SR	SR	SR
Smoking / drinking		INT	INT	INT	SR	INT	SR
Jenkins Sleep Scale		INT	INT	SR	SR	SR	SR
<b>3. Health Status</b>							
<i>Quality of life - SF-12</i>		<i>INT</i>	SR	SR	SR	SR	SR
<b>4. Biological</b>							
Blood	INT						
Cortisol		INT/HM	INT/HM			HM	
<i>Heart rate and heart rate variability</i>		<i>INT</i>					
<b>6. Psychosocial measures</b>							
<i>Social Network Scale</i>		SR	SR				
<i>ENRICH Social support Inventory</i>		SR	SR	SR	SR	SR	SR
<i>Marital satisfaction</i>		SR	SR	SR	SR	SR	SR
Illness perceptions Questionnaire- Revised		SR		SR		SR	
Illness perceptions Questionnaire – Partner			SR		SR		SR
Causal attributions		SR	SR	SR	SR	SR	SR
Self-efficacy for recovery behaviour		SR	SR	SR	SR	SR	SR
Cardiac Denial of impact Scale		SR				SR	
Type D		SR		SR		SR	
Cook Medley Hostility Scale		SR		SR		SR	
Life Orientation Test – Optimism		SR	SR				
Coping Inventory of Stress Situations		SR				SR	
Benefit finding Scale		SR	SR	SR	SR		
Seattle Angina Questionnaire		SR		SR		SR	

Key: CN – taken from clinical notes, INT – Interview measure or questionnaire by interview, SR – Self-Report questionnaire, HM – home based collection

**Table 4.3 Cronbach's alpha for measures administered to TRACE patients**

<b>Measure</b>	<b>Time 2</b>	<b>Time 3</b>	<b>Time 4</b>
BDI	.86	.93	.93
HADS-A	.88	.91	.89
Marital satisfaction	.86	.86	.86
ESSI (social support)	.85	.92	.93

## CHAPTER 5 TRACE STUDY RESULTS PART 1

### *Exploring the relationship between social support, and psychological, quality of life and biological factors in ACS patients*

#### **Part 1: Sample characteristics, attrition analysis and descriptive examination of social support**

This chapter describes the results obtained from the Time 1, 2, 3 and 4 assessments of the patients in the TRACE study. The baseline sample characteristics and attrition analysis at each assessment point are summarised. The structural and functional social support reported by patients at Time 2, 3 and 4 are discussed.

#### **5.1 Patient characteristics Time 1 – Time 4**

693 potentially eligible patients were admitted on the days of recruitment. Of these, 125 patients (19%) had been discharged or transferred to a different hospital before they could be recruited into the study, 90 (14%) were too clinically fragile (e.g. critical ischemia, ventricular tachyarrhythmia) to take part, 58 patients (9%) declined to participate, 75 (11%) did not complete measures of depression 3 weeks after hospitalization, 27 (4%) could not speak English, 23 (3%) were in confusional states, 7 (1%) patients died in hospital, and a further 38 (6%) were excluded for other reasons.

##### **5.1.1 Baseline (Time 1) patient characteristics**

The baseline (Time 1) demographic and clinical characteristics of the total patient sample are depicted in Table 5.1. The majority of patients were white European married men with an average age of 60 years and an average marital duration of 31 years. The majority had elementary educational attainment (secondary school or below) and just over a half were

currently employed. Almost a quarter of patients were living alone at the time of their ACS and a third of the sample was classified as living in high or medium deprivation. The majority of patients had suffered a ST elevation MI. The mean GRACE score was 92.85 indicative of clinical risk ascribed as low in terms of mortality risk in hospital or within 6 months post discharge. Only a small proportion (7.4%) had suffered a cardiac arrest during their ACS, and few (13%) had experienced a previous MI. Over a third of patients reported suffering from an upper respiratory tract infection in the two months preceding their admission. A substantial proportion (63%) of patients reported a family history of CHD and just under a quarter had a personal history of CHD. The mean BMI of the sample fell within the overweight range, almost half the patients were current smokers and most reported drinking alcohol.

### **5.1.2 Time 2 Patient characteristics**

Of the 298 patients who completed the Time 1 in-hospital assessments, 226 (76%) also completed the Time 2 home assessment (including 11 patients who completed a postal version of the assessment). Of these, 166 patients had valid data for the measure of functional social support and 167 patients had valid data for the measure of structural support. The reason for the difference in number is that the support and network measures were part of the postal questionnaire, and not all the participants in the home visit returned this set of measures. The Time 2 patient sample was analogous to the Time 1 sample with regard to the demographic, clinical and psychosocial variables assessed at baseline. Those who did not complete Time 2 were more likely to be classified as moderate or high deprived ( $X^2 = 7.94$ ,  $p < 0.05$ ), more likely to be living alone ( $X^2 = 7.14$ ,  $p < 0.05$ ), less likely to be married or living as married ( $X^2 = 5.46$ ,  $p < 0.05$ ) and more likely to report previous heart disease ( $X^2 = 6.89$ ,  $p < 0.05$ ) than those who did complete Time 2.

The Time 2 home assessment comprised of an in-home interview and a postal questionnaire. Of the 226 patients who completed the Time 2 home assessment interview, 167 also completed and returned the Time 2 postal questionnaire. Analysis comparing those who completed both the time 2 interview and questionnaire (N=167) and those who completed only the time 2 interview (N=59) revealed that Time 2 interview and questionnaire completers were older ( $F(1, 223) = 21.08, p < 0.05$ ), had higher GRACE scores ( $F(1, 223) = 13.56, p < 0.05$ ) and a longer marital duration ( $F(1, 223) = 4.46, p < 0.05$ ). They were also more likely to be classified as living in low deprivation ( $X^2 = 25.17, p < 0.05$ ) and less likely to be a current smoker ( $X^2 = 9.79, p < 0.05$ ).

### **5.1.3 Time 3 Patient characteristics**

A total of 200 patients (67%) completed the Time 3 telephone assessment, of whom 160 patients also returned their completed postal questionnaire assessment. A total of 174 completed both the Time 2 and Time 3 assessments. Since the Time 2 assessment, 21 (10.9%) patients reported another major cardiac event and 18 (9.4%) described having recurrent cardiac symptoms. The mean age of the Time 3 sample was 60.85 and the majority of patients were male, white and married or living as married. 55% were employed and most had elementary educational attainment (secondary school or below). Just over a quarter of patients were classified as living in moderate or high deprivation and 22.5% reporting living alone. The mean GRACE score was 94.18; the vast majority of the patients had experienced a ST elevation MI and most had no prior history of MI. The Time 3 sample was comparable to the Time 2 sample in terms of the baseline characteristics. Time 3 completers were older ( $F(1, 224) = 10.71, p < 0.05$ ), more likely to be white ( $X^2 = 4.68, p < 0.05$ ), more likely to be classified as living in low social deprivation ( $X^2 = 17.83, p < 0.05$ ) and less likely to have diabetes ( $X^2 = 3.95, p < 0.05$ ) than those who did not complete Time 3. Time 3 completers also had higher GRACE scores ( $F(1, 224) = 5.26, p < 0.05$ ).

#### **5.1.4 Time 4 Patient characteristics**

A total of 176 patients (59%) completed the Time 4 assessment, of whom 94 also returned their postal questionnaire. A total of 138 patients completed the Time 2, 3 and 4 assessments. Since the Time 3 assessment, 27 (15.8%) patients reported another major cardiac event and 44 (25.6%) described having recurrent cardiac symptoms. The mean age of the Time 3 sample was 61.18 and the majority of patients were male, white and married or living as married. 55% were employed and most had elementary educational attainment (secondary school or below). Just over a quarter of patients were classified as living in moderate or high deprivation and 23% were living alone. The mean GRACE score was 95.09; the vast majority of the patients had experienced a ST elevation MI and most had no prior history of MI. The Time 4 sample was similar to the Time 3 sample with regard to the characteristics assessed at baseline. Completers at Time 4 were more likely to have had a previous heart condition ( $X^2 = 6.14, p < 0.05$ ), less likely to be diabetic ( $X^2 = 6.29, p < 0.05$ ), and were more likely to be white ( $X^2 = 5.06, p < 0.05$ )

**Table 5.1. Demographic and clinical characteristics of the total TRACE patient sample at Time 1, 2, 3, and 4.**

	Time 1		Time 2		Time 3		Time 4	
TOTAL N	298		226		200		176	
	Mean (SD) / N (%) Range	n	Mean (SD) / N (%) Range	n	Mean (SD) / N (%) Range	n	Mean (SD) / N (%) Range	n
<b>Demographic</b>								
Age	60.15 (11.57) 24-83	298	59.74 (11.74) 32-88	226	60.85 (10.89) 37-86	200	61.18 (11.11) 32-88	176
Gender		298		226		200		176
	<i>Female</i>	48 (16)	36 (16)		28 (14)		25 (14)	
	<i>Male</i>	250 (84)	190 (84)		172 (86)		151 (86)	
Marital status		298		226		200		176
	<i>Single</i>	31 (10)	21 (9)		20 (10.0)		21 (12)	
	<i>Cohabiting</i>	18 (18)	17 (8)		11 (6)		7 (4)	
	<i>Married</i>	185 (62)	145 (64)		130 (65)		112 (64)	
	<i>Divorced</i>	32 (11)	19 (8)		19 (10)		15 (8)	
	<i>Separated</i>	9 (3)	7 (3)		5 (2)		7 (4)	
	<i>Widowed</i>	22 (7)	17 (8)		15 (7)		14 (8)	
	<i>Other</i>	1 (0.3)	0		0		0	
Marital duration (years)	30.87 (14.40) 1.5 – 60	203	30.02 (14.67) 1.5 – 60	161	30.56 (14.03) 1.5 – 60	140	31.96 (13.55) 1.5-60	121
Lives alone	69 (23)	298	44 (20)	226	45 (23)	200	40 (23)	176
Ethnicity		298		226		200		175
	<i>White</i>	247 (83)	188 (83)		170 (85)		156 (89)	
	<i>Asian</i>	35 (12)	25 (11)		21 (10)		14 (8)	

	<i>Black</i>	10 (3)		9 (4.0)		7 (4)		3 (2)	
	<i>Other</i>	6 (2)		4 (2)		2 (1)		3 (2)	
Education			297		225		199		
	<i>None</i>	84 (28)		63 (28)		51 (26)		54 (31)	
	<i>Basic</i>	74 (25)		61 (27)		57 (29)		47 (27)	
	<i>Secondary</i>	93 (31)		68 (20)		62 (31)		50 (29)	
	<i>Degree</i>	36 (16)		33 (15)		29 (14)		24 (14)	
Employment			296		224		199		175
	<i>Unemployed</i>	127 (43)		97 (43)		89 (45)		79 (45)	
	<i>Employed</i>	169 (57)		127 (57)		110 (55)		96 (55)	
Deprivation			294		223		197		173
	<i>Low</i>	188 (64)		152 (68)		139 (71)		121 (70)	
	<i>Medium</i>	70 (24)		45 (20)		40 (20)		36 (21)	
	<i>High</i>	36 (12)		26 (12)		18 (9)		16 (9)	
<b>Clinical</b>									
GRACE score		92.85 (27.72)	298	91.81 (26.45)	226	94.18 (25.87)		95.09 (25.44)	
		33 - 179		33 - 166		37-166		37-166	
ACS type			298		226		200		176
	<i>STEMI</i>	260 (87)		199 (88)		175 (88)		155 (88)	
	<i>NSTEMI/UA</i>	38 (13)		27 (12)		25 (12)		21 (12)	
Cardiac arrest during ACS		22 (7)	298	16 (7)	226	13 (7)	200	14 (8)	176
Previous MI		39 (13)	297	28 (12)	226	31 (16)	200	24 (14)	176
Previous CHD		66 (22)	298	42 (19)	226	49 (25)	200	37 (21)	176
Family history CHD		189 (63)	298	141 (62)	226	129 (65)	200	112 (64)	176
URTI previous two mths		91 (34)	267	66 (33)	201	62 (35)	179	57 (36)	160
Diabetic		47 (16)	298	35 (16)	226	19 (12)	160	20 (11)	176
Current smoker		117 (39)	298	84 (37)	226	76 (38)	200	68 (39)	176

BMI	27.55 (4.65) 17.5 – 48.4	277	27.53 (4.70) 17.5 – 48.4	210	27.53 (4.62) 19.2 – 48.4	188	27.57 (4.52) 19.2 – 44.8	169
Drink alcohol	202 (69)	295	154 (69)	226	143 (72)	200	127 (73)	176

## **5.2 Social Support at Time 2, 3 and 4**

### **5.2.1 Analytic dataset**

Of the 226 patients completing the Time 2 assessment, 166 patients had valid data for the measure of functional social support and 167 patients had valid data for the measure of structural support at Time 2 (baseline social support). At Time 3, of the 200 patients completing the telephone interview, 155 had valid data for the social support measures. At Time 4, of the 176 who completed the telephone interview, 152 returned data for the social support measures. The reason for the difference in numbers is that the support and network measures were part of a postal questionnaire, and not all the participants in the home visit or telephone follow up interviews returned this set of measures despite repeated follow up attempts.

### **5.2.2 Social support measures**

The structural and functional social support of the patients was evaluated to attain an understanding of the amount and type of social support perceived by the patient and how this may change over time following the patient's ACS. Structural social support was assessed using the Social Network Index (Cohen et al, 1997) at Time 2 only. Scores could range between 0 – 12 whereby higher scores reflect a more diverse social network. Structural social support was only measured at Time 2 as social network size tends to remain fairly stable within this age demographic (Ajrouch, Blandon, & Antonucci, 2005). Functional social support was measured using the Enriched Social Support Inventory (ESSI) (Writing Committee for the ENRICHD Investigators, 2003) at Time 2, 3 and 4. Scores could range between 7 and 34 whereby higher values indicate greater perceived social support.

#### **5.2.2.1 Structural social support**

The mean Time 2 structural social support scores are displayed in Table 5.2 and indicate that the sample had a fairly small social network consisting of an average of four people.

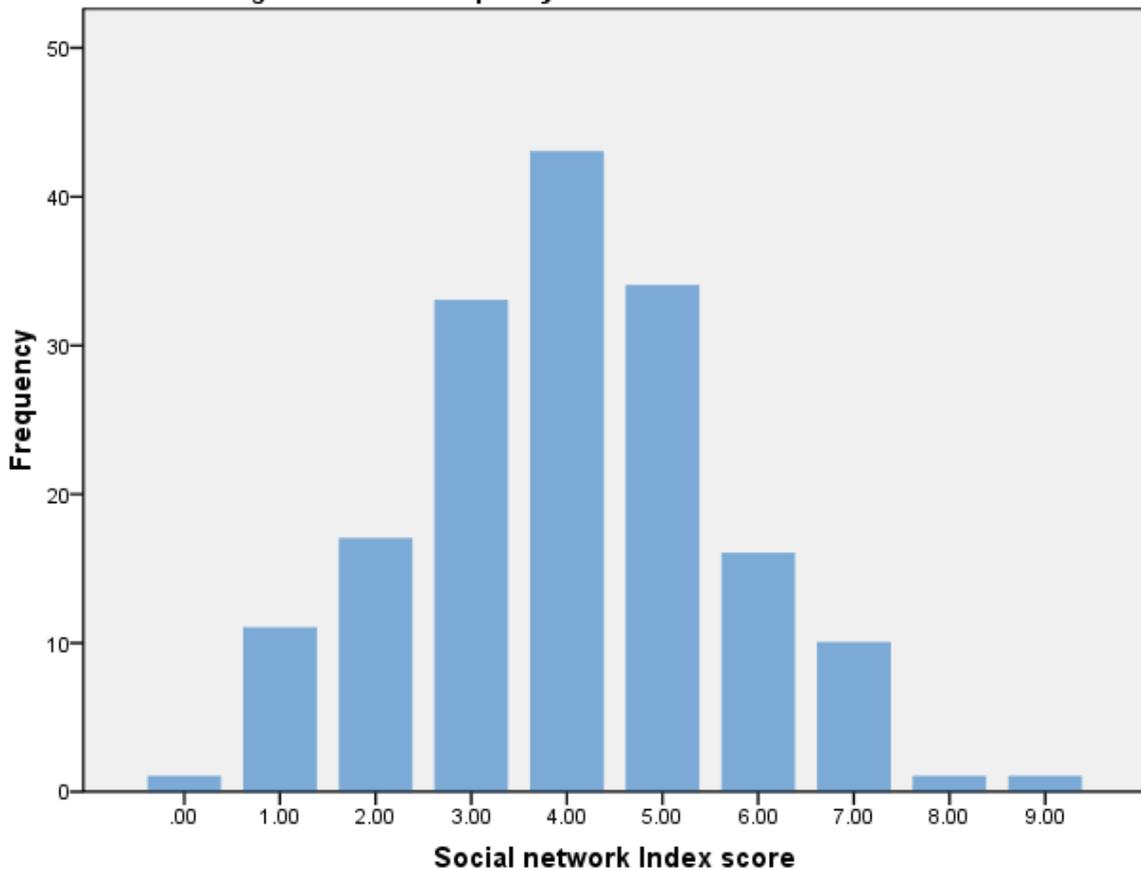
**Table 5.2 Patient structural social support (SNI) at Time 2**

	Time 2
<b><i>Structural social support</i></b>	
<b>Mean (SD)</b>	4.01 (1.64)
<b>Mode</b>	4
<b>Range</b>	0 – 9
<b>N</b>	167

The distribution of scores is depicted in Figure 5.1 and illustrates a modal response of 4 people within the social network. This is comparable to the structural support reported by other studies using the SNI in a similarly aged sample. For example, the Heart Scan study found a mean social network score of 4.19 (SD 1.6) in a sample of 543 men and women from the Whitehall II cohort with an average age of 62.9 (SD 5.7).

Social network size was approximately normally distributed with just fewer than 20% of the sample reporting having two or less people within their network size and just over 40% reporting having five or more people within their network. Nearly three quarters of the sample were married or living as married (N=121, 72.9%). Only one patient (0.6%) reported having no-one within their social network.

**Figure 5.1 Score frequency for the Social Network Index**



### 5.2.2.2 Functional social support

The mean and modal functional support scores are displayed in Table 5.3 and suggest that the patients perceived a high level of support. There were no significant differences between functional social support scores at each assessment point and the scores were significantly highly correlated suggesting that functional social support was stable during the first twelve months following ACS. The mean ESSi scores were similar to other studies using this measure in a cardiac population (for example, Vaccarino et al., 2003; Mitchell et al., 2003).

**Table 5.3 Patient functional social support (ESSi) at Time 2, 3 and 4**

	<b>Time 2</b>	<b>Time 3</b>	<b>Time 4</b>
<b>Functional social support</b>			
<b>Mean (SD)</b>	27.71 (5.24)	27.73 (6.74)	27.14 (7.29)
<b>Mode</b>	32	34	34
<b>Range</b>	10 - 34	8 - 34	8 - 34
<b>N</b>	166	156	152

Examination of the ESSI score distribution revealed that the scores were highly positively skewed with the 25<sup>th</sup> percentile represented by a score of <23 at Time 2 and 3, and a score of <22 at Time 4. At each assessment, the mean score was close to the maximum score of 34. Therefore, scores were aggregated into three categories: low support (25<sup>th</sup> percentile score or below), moderate support (25<sup>th</sup> – 75<sup>th</sup> percentile score) and high support (75<sup>th</sup> percentile or above) according to the score parameters depicted in Figure 5.2. The frequency of scores within each social support category is displayed in Table 5.4.

**Table 5.4 Aggregated ESSI score frequency at Time 2, 3 and 4**

	<b>Time 2*</b>	<b>Time 3**</b>	<b>Time 4***</b>
	<i>N (%)</i>	<i>N (%)</i>	<i>N (%)</i>
<b>Total N</b>	166	155	152
<b>Low</b>	43 (25.9)	41 (26.5)	41 (27.0)
<b>Moderate</b>	66 (39.8)	71 (45.8)	67 (44.1)
<b>High</b>	57 (34.3)	43 (27.7)	44 (28.9)

\* T2 score parameters: Low≤23, Mod 24-31, High>32  
 \*\*T3 score parameters: Low≤23, Mod 24 – 33, High>34  
 \*\*\*T4 score parameters: Low≤22, Mod 23 – 33, High>34

As discussed in Chapter 4, the ESSI also provides criteria to determine low perceived social support in the period shortly following an MI which is described in Figure 4.2 in Chapter 4. This criterion was applied to the ESSI scores reported at Time 2 and revealed that 23 (13.9%) of patients fulfilled the criteria for low perceived social support indicating that approximately 1 in 8 patients would be classified as having low perceived social support. LPSS scored on the ESSI following MI has been independently associated with increased risk of mortality and recurrent MI (Burg et al., 2005).

### **5.3 The influence of demographic factors on levels of social support**

The beneficial effects of social support are not universally experienced and the identification of factors moderating the relationship between social support and CHD outcomes has been

highlighted as a priority for future research (Lett et al., 2005). The first step in this research is to decipher the factors that influence levels of social support. Although this remains an under-researched domain, a number of demographic factors have been proposed. These factors have been discussed in detail in Chapter 1 and include gender, age, marital status, ethnicity, SES, educational level and employment status. In order to explore the impact of these Time 1 demographic factors on social support and social network scores analysis, a series of one-way between-group analyses of covariance were conducted to identify any demographic or clinical factors that may influence functional social support at Time 2, 3 and 4, or structural social support at Time 2. Continuous ESSI and SNI scores were the dependent variables, age and gender were entered as covariates and the independent variables were ethnicity (white/non-white), marital status (married/unmarried), employment status (employed/not employed), educational status (basic/secondary/degree), deprivation index (low/moderate/high) and GRACE score (low/moderate/high).

### **5.3.1 Demographic influences on Time 2 functional and structural social support**

There was no significant variation in structural or functional social support scores by gender, age or educational level. Functional social support differed according to marital status with married patients reporting higher functional social support ( $F(1, 162) = 4.38, p < 0.05$ , partial  $\eta^2 = 0.03$ ). White patients also reported significantly higher functional social support compared to non-white patients ( $F(1, 162) = 5.62, p < 0.05$ , partial  $\eta^2 = 0.03$ ). Finally, female patients reported significantly lower functional social support compared to male patients ( $F(1, 163) = 3.96, p < 0.05$ , partial  $\eta^2 = 0.02$ ).

Based on the findings of these ANCOVA analyses, a multiple regression analysis was conducted using Time 2 ESSI score as the dependent variable and age, gender, ethnicity and marital status as the independent variables. The model explained a significant proportion of variance in social support scores ( $R^2 = 0.09, F(4, 165) = 12.21, p < 0.05$ ) with ethnicity being the only significant independent predictor (Table 5.5).

**Table 5.5 Demographic predictors of functional social support at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	27.70	22.83 – 32.57		11.23	0.001
Age	0.05	-0.02 - 0.12	0.10	1.30	0.195
Gender	-1.82	-4.16 – 0.51	-0.12	-1.54	0.125
Ethnicity*	-2.57	-4.85 – -0.28	-0.17	-2.22	0.028
Marital status	-1.76	-0.36 – 0.05	-0.15	-1.92	0.057

\*Significant independent predictor

ANCOVA analysis using structural social support (SNI score) as the dependent variable revealed that structural social support only significantly varied according to employment status. Employed patients reported significantly higher structural social support compared to non-employed patients ( $F(1, 162) = 5.23, p < 0.05, \text{partial } \eta^2 = 0.03$ ). Multiple regression analysis was conducted using Time 2 SNI score as the dependent variable and age, gender, and employment status as the independent variables. The model explained a significant proportion of variance in anxiety scores ( $R^2 = 0.06, F(3, 165) = 3.48, p < 0.05$ ) with employment status being the only independent predictor (Table 5.6). None of the variables included in either model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 5.6 Demographic predictors of structural social support at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	3.87	1.75 – 6.00		3.60	0.001
Age	0.01	-0.02 – 0.04	0.04	0.42	0.672
Gender	-0.57	-1.28 – 0.14	-0.12	-1.59	0.114
Employment status*	0.75	0.10– 1.40	0.23	2.29	0.024

\*Significant independent predictor

### 5.3.2 Demographic influences on Time 3 functional social support

ANCOVA analysis using Time 3 ESSI score as the dependent variable revealed that functional social support was higher among male patients ( $F(1, 153) = 10.38, p < 0.05, \text{partial } \eta^2 = 0.06$ ), married patients ( $F(1, 152) = 86.96, p < 0.05, \text{partial } \eta^2 = 0.36$ ) and those living

in low deprivation ( $F(2, 148) = 8.27, p < 0.05, \text{partial } \eta^2 = 0.10$ ). Multiple regression analysis was conducted using Time 3 ESSI score as the dependent variable with age, gender, deprivation and marital status as the independent variables. The model explained a significant proportion of variance in functional social support scores ( $R^2 = 0.45, F(4, 148) = 30.11, p < 0.05$ ) with marital status and age identified as significant independent predictors (Table 5.7).

**Table 5.7 Demographic predictors on functional social support at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	25.05	19.59 – 30.51		9.07	0.001
Age*	0.12	-0.05 – 0.20	0.19	3.13	0.002
Gender	-1.87	-4.37 – 0.63	-0.01	-1.48	0.141
Marital status*	-8.36	-10.32 – -6.40	-0.56	-8.44	0.001
Deprivation	-1.33	-2.79 – 0.14	-0.12	-1.79	0.075

\*Significant independent predictor

### 5.3.3 Demographic influences on Time 4 functional social support

ANCOVA analysis using Time 4 ESSI score as the dependent variable revealed that functional social support was higher among male patients ( $F(1, 149) = 7.78, p < 0.05, \text{partial } \eta^2 = 0.05$ ), married patients ( $F(1, 148) = 48.92, p < 0.05, \text{partial } \eta^2 = 0.25$ ) and those living in low deprivation ( $F(2, 144) = 3.26, p < 0.05, \text{partial } \eta^2 = 0.04$ ). Multiple regression analysis was conducted using Time 4 ESSI score as the dependent variable with age, gender, deprivation and marital status as the independent variables. The model explained a significant proportion of variance in functional social support scores ( $R^2 = 0.31, F(4, 148) = 16.16, p < 0.05$ ) with marital status and age identified as significant independent predictors (Table 5.8). None of the variables included in this model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 5.8 Demographic predictors of functional social support at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	26.13	19.94 – 32.31		8.35	0.001
Age*	0.10	0.01 – 0.19	0.15	2.14	0.034
Gender	-2.26	-5.18 – 0.66	-0.11	-1.53	0.128
Marital status*	-7.71	-10.04 - -5.38	-0.49	-6.55	0.001
Deprivation	-0.43	-2.13 – 1.27	-0.04	-0.50	0.619

\*Significant independent predictor

#### 5.4 Summary: Social support at Time 2, 3 and 4.

The patient sample reported a moderate social network and high functional social support at levels that were comparable to previous study findings using similar populations and measures. This combination of a moderately sized social network providing high levels of functional support is characteristic of middle aged, married men which made up the majority of our sample (Antonucci & Akiyama, 1987). Levels of structural social support varied according to employment status with employed patients reporting a larger social network compared to non-employed patients. Functional social support was highest among white, married, male, older and less deprived patients.

Social isolation and very low social support are the social support variables that have most robust associations with mortality, morbidity and post MI recovery, and consequently are of paramount interest within social support research because of this predictive efficacy. Within our sample, 12 (6.6%) patients reported having one or fewer people in their social network, 23 (13.9%) patients were classified as having low perceived social support using ESSI criteria and 25 (15%) patients fell into the lowest 25th percentile for both social network (3 or less people within the social network) and ESSI score (score $\leq$ 23) at Time 2 indicating social isolation and low levels of social support were present in this sample. In the following two chapters (Chapter 6&7) I will explore whether the structural and functional social support described here influence the short and long term psychological response and adjustment of the ACS patient, and also the heart rate variability of the patient shortly after their ACS.

## CHAPTER 6 TRACE STUDY RESULTS PART 2

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### **Part 2: Psychological distress after ACS and the relationship between social support and post ACS psychological distress**

The psychological response of patients at Time 2, 3 and 4 is described and is followed by an evaluation of the relationship between patient social support and their psychological health at Time 2, Time 3 and Time 4. The chapter closes with a discussion of the results presented.

#### **6.1 Psychological distress at Time 2, 3 and 4**

##### **6.1.1 Analytic dataset**

Of the 226 patients completing the Time 2 assessment, 223 patients had valid data for the measures of depression and anxiety, and 203 had valid data for the measure of quality of life at Time 2. At Time 3, of the 200 patients completing the telephone interview, 152 had valid data for the depression measure, 155 for the anxiety measure and 146 for the quality of life assessment. At Time 4, of the 176 who completed the telephone interview, 155 returned data for the depression measure, 154 for the anxiety measure and 147 for the quality of life measure. The difference in numbers is because the psychological response measures were part of a postal questionnaire, and not all the participants completing the telephone follow up interviews returned this set of measures despite repeated follow up attempts.

The psychological response of patients following ACS was assessed using measures of depression (BDI) and anxiety (HADS). These measures were assessed at Time 2, 3 and 4 and have been described in detail in Chapter 4.

##### **6.1.2 Psychological distress at Time 2**

The mean scores for the BDI and HADS scales at Time 2 are depicted in Table 6.1. The scores indicate low average levels of depression and anxiety among the patients. Examining the score frequency revealed that 43 (19.3%) patients exceeded the clinical threshold

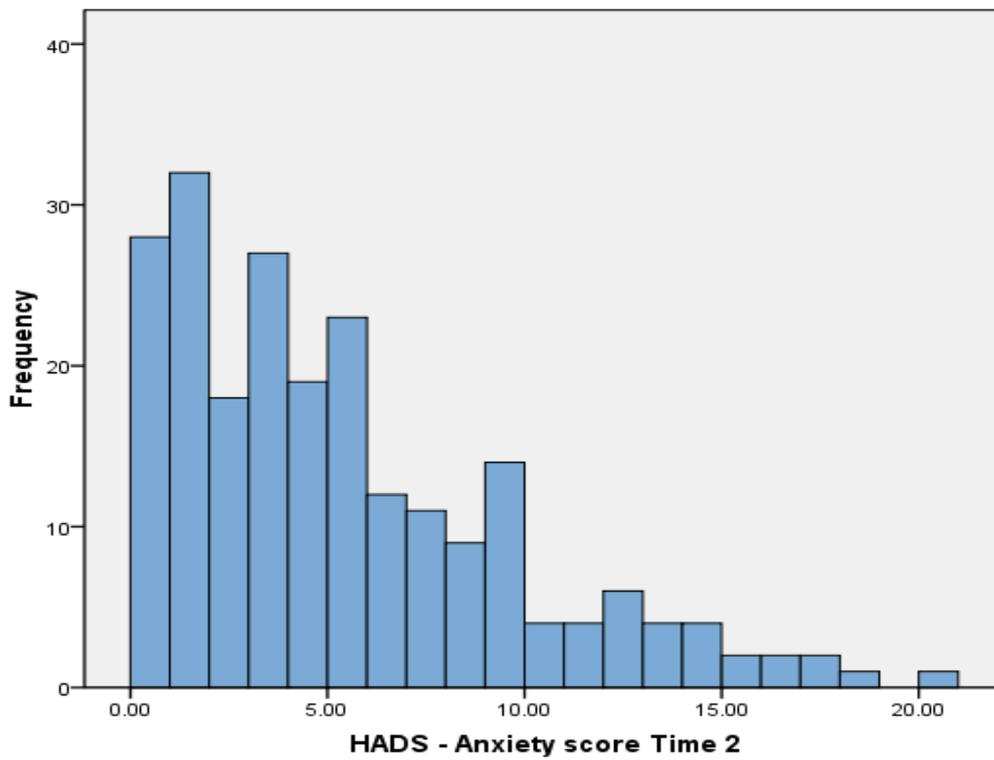
(score $\geq$ 10) for significant depressive symptomatology on the BDI and 55 (23.8%) patients exceeded the cut-off (score $\geq$ 8) for moderate anxiety on the HADS-A indicating that a significant proportion of patients were struggling with notable psychological disturbance in the period shortly following their ACS.

**Table 6.1 Mean depression (BDI) and anxiety (HADS) score at Time 2**

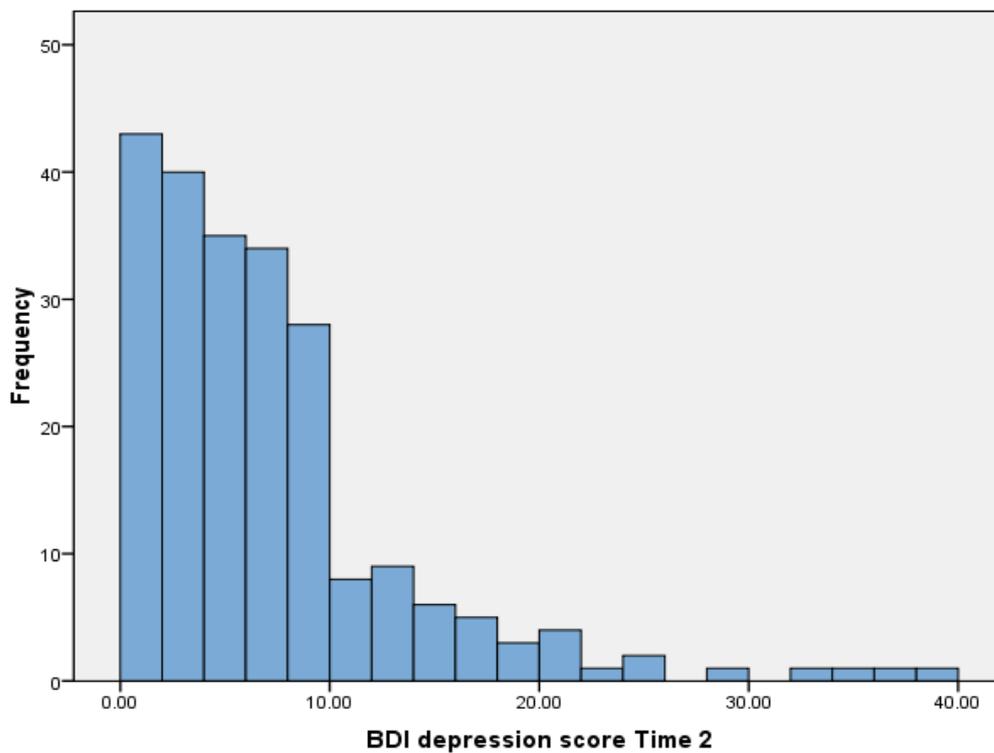
	<b>BDI</b>	<b>HADS</b>
<b>Mean (SD)</b>	6.69 (6.71)	4.87 (4.30)
<b>Range</b>	0 - 38	0 – 20
<b>N</b>	223	223

The score frequency and distribution of depression and anxiety scores at Time 2 are presented in Figures 6.1 and 6.2. The depression and anxiety scores were highly positively correlated suggesting significant comorbidity with 34 patients exceeding the threshold criteria for both significant anxiety and depression. Both score sets were positively skewed indicating that the majority of people scored fairly low on both scales which is typical of these types of clinical measures. Both distributions included a number of outliers including 4 extreme outliers for the depression scores. The 5% trimmed mean (5.88) is different from the mean (6.69) depression score indicating an influence of outliers on the mean score. The 5% trimmed mean (4.53) does not differ substantially from the mean (4.87) anxiety score suggesting no undue influence from the outliers.

**Figure 6.1 Score frequency for HADS-Anxiety scores Time 2**



**Figure 6.2 Score frequency for BDI depression scores at Time 2**



### **6.1.2.1 The influence of demographic and clinical variables on psychological distress at Time 2**

In order to determine the influence of demographic and clinical variables collected at Time 1 on depression and anxiety scores at Time 2, two different analyses were conducted using BDI depression scores and HADS-Anxiety scores as both continuous and categorical outcomes in order to ensure that the skewed nature of the anxiety and depression score distributions and the influence of outliers on depression scores was accounted for. A series of one way between group's analyses of covariance were conducted to identify any demographic or clinical factors that may influence depression and anxiety scores at Time 2. Continuous anxiety and depression scores were the dependent variables, age and gender were entered as covariates and the independent variables were ethnicity (white/non-white), marital status (married/unmarried), employment status (employed/not employed), educational status (basic/secondary/degree), deprivation index (low/moderate/high), history of depression (yes/no), the presence of diabetes (yes/no), prior heart disease (yes/no) and GRACE score (low/moderate/high). Depression at Time 2 did not significantly vary according to ethnicity, educational level or whether the patient reported diabetes or a previous heart condition. Younger patients were significantly more depressed compared to older patients ( $F(49, 172) = 1.46, p < 0.05, \text{partial } \eta^2 = 0.29$ ). Female patients were significantly more depressed than male patients ( $F(1, 220) = 3.98, p < 0.05, \text{partial } \eta^2 = 0.02$ ). After adjusting for age and gender, unmarried patients were significantly more depressed compared to married patients ( $F(1, 219) = 4.84, p < 0.05, \text{partial } \eta^2 = 0.02$ ), unemployed patients were significantly more depressed compared to employed patients ( $F(1, 219) = 4.84, p < 0.05, \text{partial } \eta^2 = 0.02$ ) and patients with a history of depression were significantly more depressed compared to patients with no history of depression ( $F(1, 219) = 6.39, p < 0.05, \text{partial } \eta^2 = 0.03$ ). Patients with a higher GRACE score were also significantly more depressed compared to patients with a lower GRACE score ( $F(2, 218) = 3.46, p < 0.05, \text{partial } \eta^2 = 0.03$ ). Finally, more deprived patients were more depressed than less deprived patients ( $F(2, 215) = 10.35, p < 0.05, \text{partial } \eta^2 = 0.09$ ).

A multiple regression analysis was conducted using depression scores as the dependent variable and age, gender, marital status, employment status, history of depression, GRACE score and deprivation as the independent variables. The model explained a significant proportion of variance in depression scores ( $R^2=0.17$ ,  $F(7, 205) = 5.86$ ,  $p<0.05$ ) with patient age and deprivation being the strongest independent predictors (Table 6.2). None of the variables included in this model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.2 Demographic and clinical predictors of depression at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	15.63	8.25 – 23.05		4.18	0.001
Age*	-0.17	-0.06 - -0.27	-0.31	-3.08	0.002
Gender	-0.26	-2.76 – 2.24	-0.01	-0.20	0.839
Marital status	0.86	-1.10 – 2.81	0.06	0.86	0.390
Employment status	-1.69	-3.88 - -0.13	-0.13	-1.52	0.130
GRACE score*	2.68	0.29 – 5.07	0.17	2.21	0.028
Deprivation*	2.17	0.86 – 3.49	0.23	3.27	0.001
Depression history*	1.96	0.15 – 3.77	0.14	2.13	0.034

\*Significant independent predictor

Anxiety at Time 2 did not significantly vary according to gender, ethnicity, marital status, educational level, employment status, GRACE score or whether the patient reported diabetes or a previous heart condition. Younger patients were significantly more anxious than older patients ( $F(49, 172) = 1.71$ ,  $p<0.05$ , partial  $\eta^2 = 0.33$ ). After adjusting for age, patients who reported a history of depression were more likely to be anxious than patients who did not report a history of depression ( $F(1,219) = 7.77$ ,  $p<0.05$ , partial  $\eta^2 = 0.03$ ) and greater deprivation was also predictive of greater anxiety ( $F(2, 219) = 15.58$ ,  $p<0.05$ , partial  $\eta^2 = 0.13$ ).

Based on the findings of these ANCOVA analyses, a multiple regression analysis was conducted using anxiety score as the dependent variable and age, gender, history of depression and deprivation as the independent variables. The model explained a significant proportion of variance in anxiety scores ( $R^2 = 0.19$ ,  $F(4, 203) = 12.21$ ,  $p < 0.05$ ) with patient age and deprivation being the strongest independent predictors (Table 6.3). None of the variables included in this model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.3 Demographic and clinical predictors of anxiety at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	15.63	4.75 – 10.99		4.18	0.001
Age*	-0.07	-0.02 - -0.12	-0.19	-2.82	0.005
Gender	-0.12	-1.77 – 1.54	-0.01	-0.14	0.888
Deprivation*	1.96	1.14 – 2.78	0.31	4.71	0.001
Depression history*	1.51	0.32 – 2.70	0.16	2.50	0.013

\*Significant independent predictor

Binary variables were also created for both the depression and anxiety scores based on the cut off threshold ( $\geq 10$  BDI,  $\geq 8$  HADS) to create two status categories for each scale: non-depressed versus depressed, non-anxious versus anxious. Mean scores, sample sizes and % for non-depressed/depressed and non-anxious/anxious groups for depression and anxiety are described in Table 6.4.

**Table 6.4 Mean anxiety and depression scores by depression and anxiety status at Time 2**

	Mean (SD)	N	%N
<b>BDI Depression</b>			
Non-depressed	4.14 (2.91)	180	81
Depressed	17.36 (7.56)	43	19
Total	6.69 (6.71)	223	100
<b>HADS Anxiety</b>			
Non-anxious	2.88 (2.16)	170	76
Anxious	11.26 (3.05)	53	24
Total	4.87 (4.30)	223	100

Logistic regression was performed to assess the influence of demographic and clinical factors on the likelihood that patients would report depression above the cut off threshold at Time 2. The model contained nine categorical independent variables (gender, ethnicity, marital status, educational level, employment status, depression history, deprivation level, previous CHD, and presence of diabetes) and two continuous independent variables (age and GRACE score). The full model containing all the predictors was statistically significant ( $X^2 (13, 217) = 39.03, p < 0.05$ ) indicating that the model was able to distinguish between patients who did and did not report depression and the model is depicted in Table 6.5. The full model explained 26.3% of the variance in depression status and correctly classified 82.9% of cases. The strongest independent predictor of depression status was deprivation level recording an odds ratio of 5.79 indicating that patients living in high deprivation were almost 6 times more likely to report depression compared to those who were living in low deprivation. Age was also found to predict depression status with an odds ratio of 0.92 suggesting that the older a patient is, the less likely they are to report depression. Employment status was the only other significant predictor in the model recording an inverted odds ratio of 3.45 suggesting that non-employed patients were over three times more likely to report depression compared to employed patients.

**Table 6.5 Logistic regression predicting likelihood of depression at Time 2**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
Age*	<i>Annual increase</i>	0.92	0.86 to 0.99	0.026
Gender	<i>Male</i>	1		
	<i>Female</i>	2.08	0.63 to 6.67	0.23
Social deprivation*	<i>Low</i>	1		
	<i>Intermediate</i>	1.87	0.73 to 4.76	0.19
	<i>High</i>	5.79	1.88 to 17.80	0.002
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.02	0.42 to 2.30	0.93
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	2.01	0.78 – 5.18	0.15
Education	<i>Basic</i>	1		
	<i>Secondary</i>	0.62	0.25 – 1.53	0.30
	<i>Degree</i>	0.48	0.14 – 1.65	0.25
Employment*	<i>Employed</i>	1		
	<i>Not employed</i>	3.45	1.20 – 10.00	0.021
Previous CHD	<i>No</i>	1		
	<i>Yes</i>	1.14	0.42 – 3.13	0.57
Diabetes	<i>No</i>	1		
	<i>Yes</i>	0.72	0.23 – 2.23	0.47
Depression history	<i>No</i>	1		
	<i>Yes</i>	2.02	0.91 – 4.50	0.09
GRACE score	<i>Score increase</i>	1.00	0.98 – 1.04	0.67

\* Significant independent predictor

Logistic regression was also performed to assess the influence of demographic and clinical factors on the likelihood that patients would report anxiety above the cut off threshold at Time 2. The model contained nine categorical independent variables (gender, ethnicity, marital status, educational level, employment status, depression history, deprivation level, previous CHD, and presence of diabetes) and two continuous independent variables (age and GRACE score). The full model containing all the predictors was statistically significant ( $X^2 (13, 217) = 38.72, p < 0.05$ ) indicating that the model was able to distinguish between patients who did and did not report anxiety and the model is depicted in Table 6.6. The model explained 24.5% of the variance in anxiety status and correctly classified 79.3% of cases. Deprivation level was a significant independent predictor in the model with an odds

ratio of 6.91 indicating that patients living in high deprivation are almost 7 times more likely to report significant anxiety compared to those living in low deprivation. The only other significant independent predictor was educational level with an inverted odds ratio of 3.85 indicating that individuals with basic level education were almost 4 times more likely to report high anxiety compared to those high educational attainment (degree level or above).

**Table 6.6 Logistic regression predicting likelihood of anxiety at Time 2**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
Age	<i>Annual increase</i>	0.98	0.92 to 1.04	0.53
Gender	<i>Male</i>	1		
	<i>Female</i>	1.37	0.48 to 3.85	0.56
Social deprivation*	<i>Low</i>	1		
	<i>Intermediate</i>	1.20	0.49 to 2.95	0.68
	<i>High</i>	6.91	2.33 to 20.48	0.001
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.09	0.40 to 2.11	0.85
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	1.74	0.71 – 4.22	0.22
Education*	<i>Basic</i>	1		
	<i>Secondary</i>	0.71	0.32 – 1.56	0.39
	<i>Degree</i>	0.26	0.07 – 0.93	0.037
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.08	0.42 – 2.71	0.88
Previous CHD	<i>No</i>	1		
	<i>Yes</i>	1.62	0.65 – 4.04	0.31
Diabetes	<i>No</i>	1		
	<i>Yes</i>	0.90	0.33 – 2.41	0.83
Depression history	<i>No</i>	1		
	<i>Yes</i>	1.17	0.54 – 2.53	0.68
GRACE score	<i>Score increase</i>	0.98	0.96 – 1.01	0.18

\* Significant independent predictor

### 6.1.2.2 Summary: Psychological distress at Time 2

The findings from the ANCOVA, linear regression and logistic regression analyses indicate that the some of the selected demographic and clinical variables predicted the occurrence of

psychological distress at Time 2. In particular, age and deprivation level appear to offer the greatest predictive efficacy with regard to both depression and anxiety. Age, marital status, employment status, history of depression, educational level and GRACE score also contributed to depression and anxiety scores. However, these demographic and clinical variables only accounted for approximately a quarter of the variance in anxiety and depression scores and subsequently much of the variation in distress remains unaccounted for. These findings do suggest that younger patients and patients living in high deprivation should be monitored more closely for symptoms of psychological distress in the immediate weeks following ACS.

### **6.1.3 Psychological distress after ACS at Time 3**

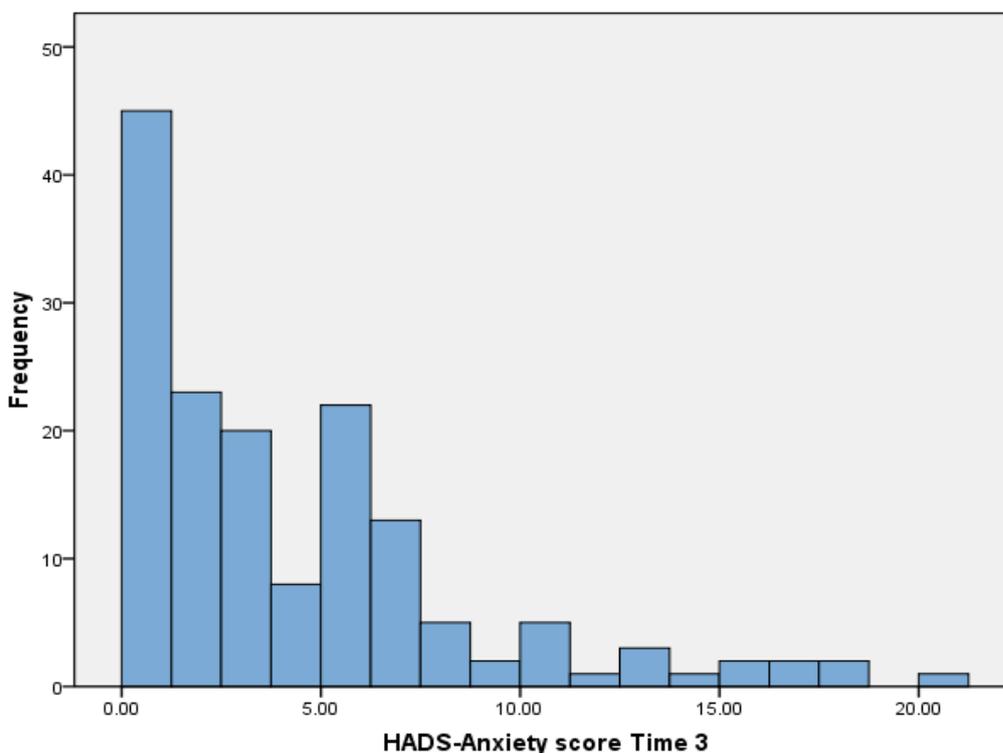
The mean scores for the BDI and HADS-anxiety scale at Time 3 are depicted in Table 6.7. All Time 2 psychological response scores were highly correlated with the corresponding Time 3 scores (Anxiety:  $r(139) = 0.72$ ,  $p < 0.05$ , Depression:  $r(136) = 0.76$ ,  $p < 0.05$ ). Examining the score frequency revealed that 36 (23.5%) patients exceeded the clinical threshold (score  $\geq 10$ ) for significant depressive symptomatology on the BDI and 24 (15.4%) patients exceeded the cut-off (score  $\geq 8$ ) for moderate anxiety on the HADS-A. There were no significant differences between Time 2 and 3 anxiety scores; however, there was a significant increase in mean BDI scores ( $t(135) = -2.31$ ,  $p < 0.05$ ). The number of patients scoring above the clinical threshold for depression at Time 2 was similar to Time 3 (19.3% T2 v 23.5% T3). However, there is a noticeable drop in the number of patients scoring above the clinical threshold for anxiety at Time 3 compared to Time 2 (23.7% T2 v 15.4% T3) indicating a reduction over time in the number of patients experiencing significant anxiety symptomatology.

**Table 6.7 Mean depression (BDI) and anxiety (HADS) score at Time 3**

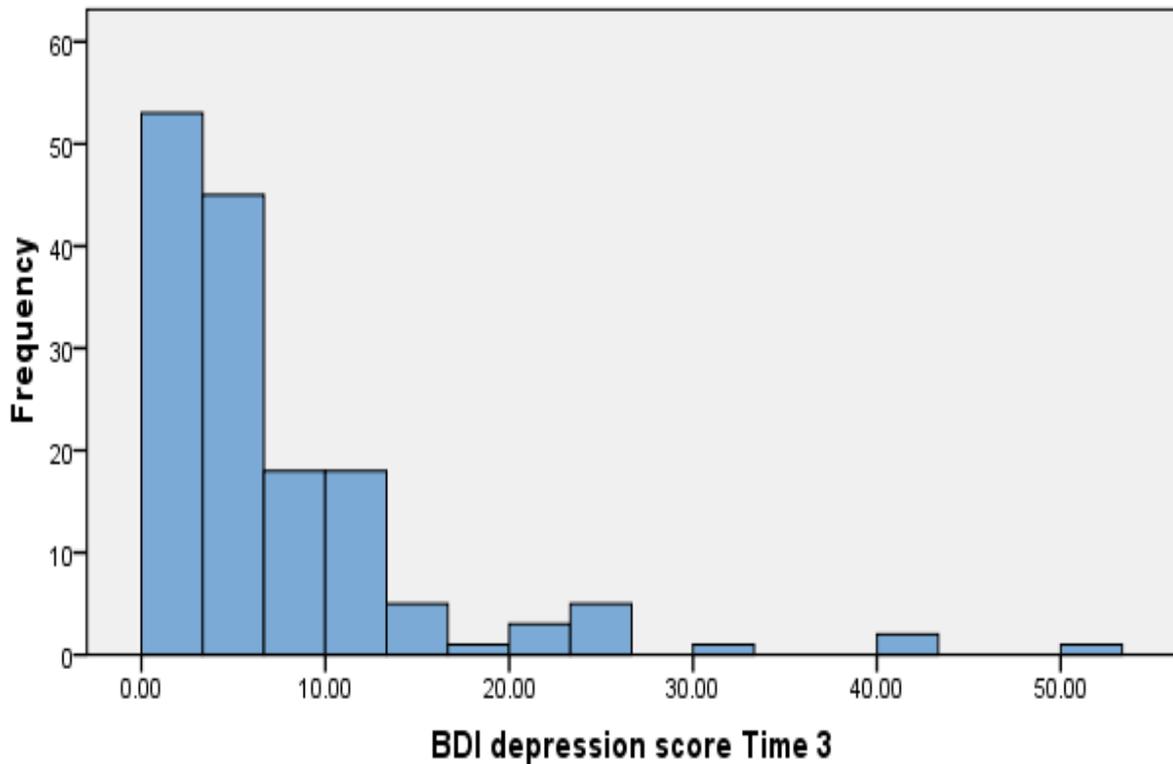
	<b>BDI</b>	<b>HADS</b>
<b>Mean (SD)</b>	7.23 (8.12)	4.28 (4.31)
<b>Range</b>	0 – 51	0 – 20
<b>N</b>	152	155

The score frequency and distribution of depression and anxiety scores at Time 3 are presented in Figures 6.3 and 6.4. The depression and anxiety scores were highly positively correlated suggesting significant comorbidity with 20 patients exceeding the threshold criteria for significant anxiety and depression. Analogous to Time 2, both score sets were positively skewed and both included a number of outliers including 4 extreme outliers for the depression scores. The 5% trimmed mean (6.13) is different from the mean (7.23) depression score indicating an influence of outliers on the mean score. The 5% trimmed mean (3.82) of the anxiety scores is not substantially different from the mean (4.28) which indicates no undue influence from outliers.

**Figure 6.3 Score frequency for HADS-Anxiety scores Time 3**



**Figure 6.4 Score frequency for BDI depression scores Time 3**



### **6.1.3.1 The influence of demographic and clinical variables on psychological distress at Time 3**

In order to determine the influence of Time 1 demographic and clinical variables on Time 3 depression and anxiety scores, BDI depression and HADS anxiety scores were analysed as both continuous and categorical variables as described previously. A series of one way between group's analyses of covariance were conducted with continuous anxiety and depression scores as the dependent variables, age, gender were entered as covariates and ethnicity (white/non-white), marital status (married/unmarried), employment status (employed/not employed), educational status (basic/secondary/degree), Time 2 depression score, deprivation index (low/moderate/high), history of depression (yes/no), the presence of

diabetes (yes/no), prior heart disease (yes/no) and GRACE score (low/moderate/high) as independent variables. The results showed that depression at Time 3 did not significantly vary according to ethnicity, marital status, educational status, deprivation index, depression history, GRACE score or whether the patient reported diabetes or a previous heart condition. Patients who had higher depression scores at Time 2 were significantly more likely to have higher depression scores at Time 3 ( $F(32, 101)=7.57, p<0.05, \text{partial } \eta^2 =0.71$ ). Unemployed patients were significantly also more depressed than employed patients ( $F(1, 147) =4.75, p<0.05, \text{partial } \eta^2 =0.03$ ). Gender also showed near significant effects with female patients having higher depressed scores than male patients ( $F(1, 149) =3.78, p<0.054, \text{partial } \eta^2 =0.025$ ).

A multiple regression analysis was conducted using Time 3 depression scores as the dependent variable and age, gender, employment status and depression score at Time 2 as the independent variables. The model explained a significant proportion of variance in depression scores ( $R^2=0.58, F(4, 130) = 44.54, p<0.05$ ) with patient depression score at Time 2 being the only independent predictor (Table 6.8). The model was rerun excluding Time 2 depression score to determine whether this variable may be obscuring other relationships. The model remained significant and both age ( $\beta=-0.28, p<0.05$ ) and employment status ( $\beta=-0.22, p<0.05$ ) were found to be significant independent predictors.

**Table 6.8 Demographic and clinical predictors of depression at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	T	Sig.
Constant	-3.11	-11.41 – 5.19		-0.74	0.460
Age	0.44	-0.07 – 0.16	0.06	0.79	0.428
Gender	1.14	-1.47– 2.24	0.05	0.87	0.389
Employment status	-0.07	-3.88 - -0.13	-0.00	-0.06	0.954
Time 2 depression score*	1.02	0.85 – 1.19	0.76	12.37	0.001

\*Significant independent predictor

Anxiety at Time 3 did not significantly vary according to age, gender, ethnicity, educational level, employment status, GRACE score or whether the patient reported diabetes or a previous heart condition. Unmarried patients reported more anxiety than married patients ( $F(1, 151) = 3.97, p < 0.05, \text{partial } \eta^2 = 0.03$ ). Deprivation level showed a near significant effect with more deprived patients experiencing greater anxiety than less deprived patients ( $F(2, 147) = 2.99, p = 0.053, \text{partial } \eta^2 = 0.04$ ). Patients with higher anxiety scores at Time 2 were also found to have higher anxiety at Time 3 ( $F(20, 116) = 6.70, p < 0.05, \text{partial } \eta^2 = 0.536$ ).

Based on the findings of these ANCOVA analyses, a multiple regression analysis was conducted using Time 3 anxiety scores as the dependent variable and age, gender, marital status, deprivation and Time 2 anxiety score as the independent variables. The model explained a significant proportion of variance in anxiety scores ( $R^2 = 0.54, F(5, 130) = 12.21, p < 0.05$ ) with Time 2 anxiety score being the strongest independent predictor (Table 6.9). Marital status was also found to independently predict Time 3 anxiety scores. The model was repeated excluding Time 2 anxiety score. The model remained significant and age was identified as a significant independent predictor ( $\beta = -0.18, p < 0.05$ ). None of the variables included in this any of the Time 3 models showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.9 Demographic and clinical predictors of anxiety at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	T	Sig.
Constant	0.31	-3.13 – 3.76		0.18	0.859
Age	-0.01	-0.06 - 0.04	-0.03	-0.50	0.619
Gender	0.93	-0.57 – 2.42	0.08	1.23	0.223
Deprivation	-0.73	-1.77 – 0.32	-0.10	-1.38	0.170
Marital status*	1.22	0.01 – 2.43	0.13	1.99	0.048
Time 2 anxiety score*	0.56	0.62 – 0.90	0.73	10.95	0.001

\* Significant independent predictor

Analogous to the Time 2 analyses, binary variables were also created for both the Time 3 depression and anxiety scores based on the cut off threshold ( $\geq 10$  BDI,  $\geq 8$  HADS) to create two status categories for each scale: non-depressed versus depressed, non-anxious versus anxious. Mean scores, sample sizes and % for non-depressed/depressed and non-anxious/anxious groups for depression and anxiety are described in Table 6.10.

**Table 6.10 Mean depression and anxiety scores by depression and anxiety status at Time 3**

	<i>Mean (SD)</i>	<i>N</i>	<i>%N</i>
<b><i>BDI Depression T3</i></b>			
Non-depressed	3.83 (2.45)	116	76
Depressed	18.20 (10.13)	36	24
Total	7.23 (8.1)	152	100
<b><i>HADS Anxiety T3</i></b>			
Non-anxious	2.80 (2.32)	131	85
Anxious	12.89 (3.72)	24	15
Total	4.28 (4.31)	155	100

Logistic regression was performed to assess the influence of demographic and clinical factors on depression above the cut off threshold at Time 3. The model contained ten categorical independent variables (gender, ethnicity, marital status, educational level, employment status, depression history, depression status at Time 2, deprivation level, previous CHD, and presence of diabetes) and two continuous independent variables (age and GRACE score). The full model containing all the predictors was statistically significant ( $X^2 (14, 132) = 43.31, p < 0.05$ ) indicating that the model was able to distinguish between patients who did and did not report depression and the model is depicted in Table 6.11. The full model explained 41% of the variance in depression status and correctly classified 86.4% of cases. The strongest independent predictor of depression status at Time 3 was depression status at Time 2 with an odds ratio of 14.56 suggesting that patients who scored over the threshold for depression at Time 2 were over 14 times more likely to score over the threshold at Time 3. The only other significant independent predictor was depression history

recording an odds ratio of 4.87 indicating that patients with a history of depression were nearly 5 times more likely to score over the depression threshold at Time 3.

**Table 6.11 Logistic regression predicting likelihood of depression at Time 3**

Variable	Categories	Adjusted odds ratio	95% C.I.	p
Age	<i>Annual increase</i>	0.95	0.87 to 1.04	0.27
Gender	<i>Male</i>	1		
	<i>Female</i>	1.11	0.21 to 3.94	0.90
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.27	0.35 to 4.60	0.72
	<i>High</i>	4.76	0.20 to 116.14	0.34
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.15	0.35 to 3.75	0.83
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	2.27	0.59 – 8.67	0.23
Education	<i>Basic</i>	1		
	<i>Secondary</i>	0.62	0.20 – 1.95	0.42
	<i>Degree</i>	0.45	0.10 – 2.15	0.32
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.62	0.43 – 6.05	0.48
Previous CHD	<i>No</i>	1		
	<i>Yes</i>	1.02	0.29 – 3.56	0.98
Diabetes	<i>No</i>	1		
	<i>Yes</i>	1.53	0.36 – 6.46	0.56
Depression history*	<i>No</i>	1		
	<i>Yes</i>	4.87	1.65 – 14.39	0.00
T2 Depression status*	<i>Not depressed</i>	1		
	<i>Depressed</i>	14.56	3.17 – 58.60	0.00
GRACE score	<i>Score increase</i>	1.04	1.00 – 1.08	0.27

\* Significant independent predictor

Logistic regression was also performed to assess the influence of demographic and clinical factors on anxiety above the cut off threshold at Time 3. The model contained ten categorical independent variables (gender, ethnicity, marital status, educational level, employment status, depression history, anxiety status at Time 2, deprivation level, previous CHD, and presence of diabetes) and two continuous independent variables (age and GRACE score). The full model containing all the predictors was statistically significant ( $X^2(14, 135) = 58.09$ ,

p<0.05) indicating that the model was able to distinguish between patients who did and did not report anxiety and the model is depicted in Table 6.12. The model explained 60.9% of the variance in anxiety status and correctly classified 91.1% of cases. Anxiety status at Time 2 was the largest significant independent predictor in the model with an odds ratio of 96.32 indicating that patients who scored over the threshold for anxiety at Time 2 were over 96 times more likely to score over the threshold at Time 3. Depression history was also a significant independent predictor noting an odds ratio of 4.96 suggesting that patients with a history of depression prior to their ACS were nearly 5 times more likely to report significant anxiety at Time 3. The only other significant independent predictor was age with an odds ratio of 0.84.

**Table 6.12 Logistic regression predicting likelihood of anxiety at Time 3**

Variable	Categories	Adjusted odds ratio	95% C.I.	p
Age*	<i>Annual increase</i>	0.84	0.73 to 0.98	0.02
Gender	<i>Male</i>	1		
	<i>Female</i>	2.87	0.32 to 25.42	0.34
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	0.64	0.08 to 5.04	0.67
	<i>High</i>	0.96	0.06 to 15.14	0.98
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.33	0.22 to 8.03	0.76
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	1.72	0.17 - 2.17	0.64
Education	<i>Basic</i>	1		
	<i>Secondary</i>	1.02	0.17 – 6.07	0.98
	<i>Degree</i>	8.28	0.75 – 91.47	0.09
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	4.76	0.68 - 33.33	0.12
Previous CHD	<i>No</i>	1		
	<i>Yes</i>	0.67	0.12 – 3.73	0.65
Diabetes	<i>No</i>	1		
	<i>Yes</i>	5.12	0.56 – 46.45	0.15
Depression history*	<i>No</i>	1		
	<i>Yes</i>	4.96	1.09 – 22.47	0.04
Anxiety status at Time 2*	<i>Not anxious</i>	1		
	<i>Anxious</i>	96.32	12.01 – 772.44	0.00
GRACE score	<i>Score increase</i>	1.05	1.00 – 1.10	0.18

\*Significant independent predictors

### 6.1.3.2 Summary: Psychological distress at Time 3

The findings from the Time 3 analyses indicate that mean depression and anxiety levels were similar to those reported at Time 2 indicating persistence in symptomatology over the follow up period. There was a significant increase in mean BDI score; however, the number of patients scoring above the threshold for depression at Time 3 was similar to the number at Time 2. There was no significant increase in mean HADS-A score between Time 2 and Time 3 with the number of patients scoring over the threshold for anxiety dropping between Time 2 and Time 3. With regard to the clinical and demographic influences on psychological

response at Time 3, the findings from the ANCOVA, linear regression and logistic regression analyses indicate that the Time 2 depression and anxiety scores were the strongest significant predictors of both depression and anxiety at Time 3. Thus, the experience of psychological distress shortly after ACS can be understood as strongly predictive of psychological distress 6 months following their hospital admission. Being younger, unemployed and with a history of depression also appeared to increase the risk of psychological disturbance at Time 3.

#### **6.1.4 Psychological distress after ACS at Time 4**

The psychological response findings at Time 4 were extremely similar to those reported at Time 2 and Time 3. Due to this likeness, full descriptive analysis is provided in Appendix V and a brief summary will be provided in this section.

##### **6.1.4.1 Summary of psychological distress after ACS at Time 4**

Mean depression score ( $M=7.62$ ,  $SD=8.12$ ) and mean anxiety score ( $M=4.43$ ,  $SD=4.28$ ) were not significantly different from those reported at Time 3 suggesting persistence of symptomatology, although a significant increase in mean depression score was noted between Time 2 and Time 4 ( $t(135) = -2.72$ ,  $p < 0.05$ ). Examining the score frequency revealed that 41 (26.6%) patients exceeded the clinical threshold ( $score \geq 10$ ) for significant depressive symptomatology on the BDI and 33 (21.3%) patients exceeded the cut-off  $F$  ( $score \geq 8$ ) for moderate anxiety on the HADS-A. There was also a significant increase in the number of patients scoring above the clinical threshold for depression from Time 2 – Time 3 - Time 4 ( $\chi^2(2, 108) = 9.75$ ,  $p < 0.05$ ). In order to determine the influence of demographic and clinical variables collected at Time 1 on depression and anxiety scores at Time 4, BDI depression and HADS-anxiety scores were analysed as both continuous and categorical outcomes. I found that history of depression increased risk of depression and anxiety at Time 4 (adjusted OR = 3.07 and 4.23 respectively, both  $p < 0.05$ ). Thus, the experience of psychological distress shortly after ACS has considerable predicative efficacy with regard to

distress at 12 months. There was also a tendency for female patients to experience more depression at Time 4 (adjusted OR=3.13,  $p<0.074$ ).

### **6.1.5 Summary: Psychological distress after ACS**

Depression and anxiety were common reactions to ACS with almost a quarter of patients reporting these symptoms shortly following their discharge from hospital. This distress continued throughout the follow up period with no significant reduction observed over time. Furthermore, depressive symptomatology and the occurrence of clinically significant depression actually increased over the follow up period. A number of demographic and clinical factors were found to have predictive efficacy with regard to psychological disturbance. In particular, age, gender, marital status, employment status, GRACE score and deprivation were shown to be of consistent importance. These factors may be useful for identification of patients who may be more vulnerable to experiencing psychological disturbance following an ACS and whom could be targeted for preventative intervention. These variables also represent important covariates in my analysis of the role of social support in the occurrence of post ACS distress which is described in detail in the following section.

## **6.2 Functional and structural social support as correlates and predictors of post ACS psychological distress**

### **6.2.1 Introduction**

The previous sections in this chapter have explored in detail both the psychological response of patients as well as the social support reported by patients at each follow up assessment. This section seeks to explore how these factors are related; how patient's levels of structural and functional social support may be associated or predictive of the patients experience of psychological distress. As discussed in Chapters 1 and 2, there is a large body of research highlighting the close relationship between social support and various psychological states with higher levels of social support generally revealed as protective

against negative psychological states including anxiety and depression. In the case of ACS patients, it is hypothesised that higher levels of functional and structural social support will be predictive of lower levels of psychological distress at each follow up assessment. This relationship between social support and psychological response will be explored cross sectionally each of the three follow up assessments individually and also longitudinally for Time 3 and Time 4 distress using Time 2 measures of social support.

### **6.2.2 Data analysis**

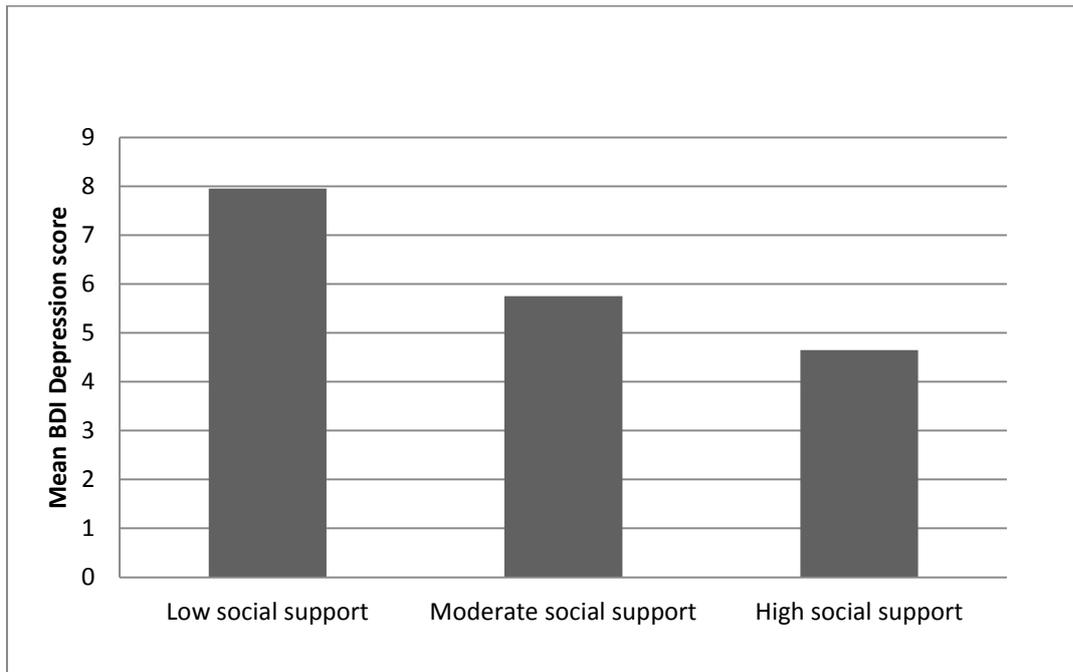
The association between social support and immediate psychological response (anxiety, depression) at Time 2 were examined using multivariate ANOVA and multiple regression analysis. The predictive efficacy of social support with regard to longer term psychological response (anxiety, depression) at Time 3 and Time 4 was explored using multiple regression and logistic regression analysis.

### **6.2.3 Functional social support and psychological distress at Time 2**

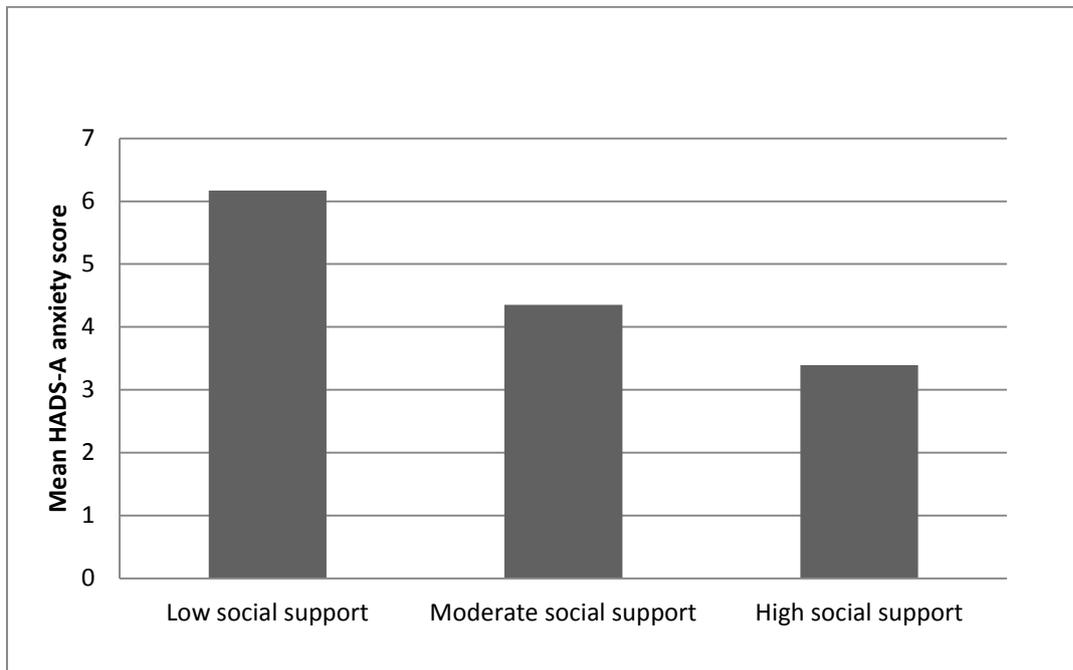
At Time 2, there was a significant negative correlation between BDI score and ESSi score ( $r(164) = -0.23, p < 0.001$ ), and also between HADS-A score and ESSi score ( $r(163) = -0.27, p < 0.001$ ) These findings suggest an inverse relationship between psychological distress and functional social support that merits further exploration.

Patients were divided according to their score on the ESSi to create three groups; Low social support ( $\leq 23$ ), moderate social support (24-31) and high social support ( $\geq 32$ ). The group aggregation by ESSi score was described in more detail in section 5.3.4. Mean depression and anxiety scores by functional social support group are depicted in Figures 6.5 and 6.6. Statistically significant decreases in both depression and anxiety score as levels of functional social support increased were observed (Depression:  $F(2, 161) = 4.18, p < 0.05$  Anxiety:  $F(2, 160) = 6.80, p < 0.05$ ).

**Figure 6.5 Mean depression score by level of functional social support at Time 2**



**Figure 6.6 Mean anxiety score by level of functional social support at Time 2**



Multiple regression analysis was conducted using either BDI depression score or HADS-A anxiety score as the dependent variable with functional social support (ESSI score), age, gender, employment status, marital status, GRACE score and deprivation as the

independent variables. Using depression as the dependent variable, the model explained a significant proportion of variance in depression scores ( $R^2=0.17$ ,  $F(7, 159) = 3.78$ ,  $p<0.05$ ) with age, employment status and deprivation being the only significant independent predictors (Table 6.13). There was a near significant effect of functional support on depression scores ( $p=0.06$ ).

**Table 6.13 Association between functional social support and depression at Time 2**

	<b>B</b>	<b>95% C.I for <math>\beta</math></b>	<b>Standardised <math>\beta</math></b>	<b>t</b>	<b>Sig.</b>
Constant	20.92	11.90 – 29.93		4.58	0.001
T2 ESSI score	-0.16	-0.34 – 0.08	-0.15	-1.89	0.061
Age*	-0.20	-0.35- 0.04	-0.38	-2.44	0.016
Gender	1.13	-1.50 – 3.76	0.07	-0.85	0.398
Marital status	0.32	-1.76 – 2.41	0.03	0.31	0.761
Ethnicity	1.03	-1.52 – 3.59	0.06	0.80	0.425
Employment status	-2.92	-5.19 - -0.65	-0.25	-2.54	0.012
GRACE score	0.01	-0.05 – 0.08	0.06	0.44	0.658
Deprivation*	1.71	0.06 – 3.36	0.17	2.05	0.042

\* Significant independent predictor

Using anxiety as the dependent variable, the model explained a significant proportion of variance in anxiety scores ( $R^2=0.20$ ,  $F(8, 158) = 4.64$ ,  $p<0.05$ ) with functional social support and deprivation being the only significant independent predictors (Table 6.14). There was a near significant effect of age on anxiety scores ( $p=0.06$ ). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.14 Association between functional social support and anxiety at Time 2**

	<b>B</b>	<b>95% C.I for <math>\beta</math></b>	<b>Standardised <math>\beta</math></b>	<b>t</b>	<b>Sig.</b>
Constant	13.59	7.71 – 19.48		4.56	0.001
T2 ESSI score*	-0.14	-0.25 - -0.02	-0.18	-2.36	0.020
Age	-0.10	-0.20 - 0.00	-0.29	-1.90	0.060
Gender	0.25	-1.47– 1.97	0.02	0.29	0.774
Marital status	-0.55	-1.92 –0.82	-0.06	-0.79	0.429
Ethnicity	0.90	-0.77 – 2.57	0.08	1.07	0.288
Employment status	-0.51	-1.99 – 0.98	-0.07	-0.67	0.501
GRACE score	0.00	-0.04 – 0.04	0.02	0.15	0.881
Deprivation*	1.71	0.63 – 2.79	0.25	3.13	0.002

\* Significant independent predictor

As discussed in Section 5.5, patients could be categorised into two groups according to their BDI depression or HADS-A anxiety score depending on whether their score exceeded a clinical threshold. Mean functional social support score was significantly lower in patients scoring above the clinical threshold for depression ( $F(1, 161) = 8.68, p < 0.05$ ) and anxiety ( $F(1, 161) = 8.92, p < 0.05$ ) than those scoring below (Table 6.15) indicating that the presence of marked depression and anxiety was associated with lower levels of functional social support.

**Table 6.15 Mean functional social support (ESSI) score by depression and anxiety status at Time 2**

	<b>Depressed</b>	<b>Non-depressed</b>	<b>Anxious</b>	<b>Non-anxious</b>
<b>Mean ESSI score (SD)</b>	24.73 (6.70)	28.18 (4.80)	25.33 (5.99)	28.30 (4.86)
<b>N (%)</b>	23 (14)	141 (86)	33 (20)	130 (80)

Logistic regression was performed to determine the relationship between functional social support and depression or anxiety above the cut off threshold at Time 2. The model contained five categorical independent variables (gender, marital status, ethnicity, employment status, deprivation level) and three continuous independent variables (functional social support, age and GRACE score with either anxiety status or depression

status entered as the dependent variable. For depression status, the full model (Table 6.16) was statistically significant ( $X^2(8, 160) = 24.19, p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 25.0% of the variance in depression status and correctly classified 88.1% of cases. Deprivation was the largest significant independent predictor in the model with an odds ratio of 10.02 indicating that deprived patients were ten times more likely to experience depression. Age was the only other significant independent predictor. A near significant ( $p = 0.07$ ) effect of functional social support was also observed with an odds ratio of 1.08.

**Table 6.16 Logistic regression determining the relationship between functional social support and depression at Time 2**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Functional social support	<i>Score increase</i>	1.08	0.99 to 1.19	0.07
Age*	<i>Annual increase</i>	0.89	0.81 to 0.98	0.016
Gender	<i>Male</i>	1		
	<i>Female</i>	5.49	0.76 to 40.00	0.09
Social deprivation*	<i>Low</i>	1		
	<i>Intermediate</i>	2.04	0.59 to 7.09	0.26
	<i>High</i>	10.02	1.47 to 59.43	0.011
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.30	0.39 to 4.27	0.67
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	2.32	0.65 to 8.28	0.19
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	3.14	0.79 to 12.50	0.10
GRACE score	<i>Score increase</i>	1.03	0.99 to 1.06	0.17

\*Significant independent predictors

For anxiety status, the full model (Table 6.17) was statistically significant ( $X^2(9, 159) = 23.84, p < 0.05$ ) and able to distinguish between patients who did and did not report anxiety above the threshold. The model explained 21.8% of the variance in anxiety status and correctly

classified 81.5% of cases. As we observed with depression, deprivation was the largest significant independent predictor in the model with an odds ratio of 7.96 suggesting that patients who were deprived were over seven times more likely to report anxiety above the threshold. Functional support was the only other significant independent predictor with an adjusted odds ratio of 1.09 indicating that patients with lower functional social support were more likely to experience anxiety than patients with higher functional social support.

**Table 6.17 Logistic regression determining the relationship between functional social support and anxiety at Time 2**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 functional social support*	<i>Score increase</i>	1.09	1.00 to 1.78	0.041
Age	<i>Annual increase</i>	0.97	0.89 to 1.05	0.43
Gender	<i>Male</i>	1	0.47 to 8.13	0.36
	<i>Female</i>	1.95		
Social deprivation*	<i>Low</i>	1	0.54 to 5.16	0.36
	<i>Intermediate</i>	1.66		
	<i>High</i>	7.96		
Marital status	<i>Married</i>	1	0.50 to 4.50	0.46
	<i>Not married</i>	1.51		
Ethnicity	<i>White</i>	1	0.66 to 5.60	0.67
	<i>Non-white</i>	1.93		
Employment	<i>Employed</i>	1	0.51 to 4.04	0.46
	<i>Not employed</i>	1.28		
GRACE score	<i>Score increase</i>	0.99	0.96 to 1.03	0.68

\*Significant independent predictors

### 6.2.3.1 Summary: Functional social support and psychological response at Time 2

Functional social support was identified as a significant independent predictor of anxiety at Time 2 suggesting that the amount of functional social support perceived by an ACS patient is an important contributory factor to the experience of anxiety shortly after discharge from hospital. Those patients who reported feeling socially supported were significantly less likely

to report anxiety symptomatology at Time 2. Conversely, those patients reporting a lack of social support were much more likely to experience anxiety, and also reported more severe and clinically relevant symptoms of anxiety. There was a near significant effect of functional social support on the experience of depression at Time 2 suggesting that functional support may also have a lesser influence on depression. Age and deprivation were also notable in their impact on psychological disturbance with younger patients and more deprived patients at significantly higher risk of depression and anxiety at Time 2. These effects were observed both in analyses of continuous anxiety and depression scores, and in categorical analyses of scores above threshold.

#### **6.2.4 Structural social support and psychological response at Time 2**

There was no significant correlation between psychological distress and SNI score. Patients were subdivided according to their level of structural social support to form two groups: Low structural social support (1 or fewer people in social network, Mean SNI = 0.92 (0.29), N=12) and adequate structural social support (2 or more people, Mean SNI = 4.25 (1.44), N=155). Patients who reported low structural social support reported significantly higher levels of depression ( $F(1, 163) = 4.78, p < 0.05$ ) compared with those who reported high structural social support (High,  $M=9.47, SD=4.07$  v Low,  $M=5.72, SD= 5.81$ ). There was no significant difference in anxiety scores by structural support level. These results indicate that the perception of higher structural social support shortly after hospital discharge may be associated with lower depression but does not affect anxiety levels.

Multiple regression analysis was conducted using either BDI depression score or HADS- A anxiety score as the dependent variable with structural social support (SNI score), age, gender, employment status, marital status, GRACE score, and deprivation as the independent variables. Using depression as the dependent variable, the model (Table 6.18) explained a significant proportion of variance in depression scores ( $R^2 = 0.15, F(7, 153) =$

3.87,  $p < 0.05$ ) with age, employment status and deprivation being the main significant independent predictors.

**Table 6.18 Association between structural social support and depression at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	17.82	9.92 – 25.72		4.45	0.001
T2 SNI score	-0.30	-0.86 – 0.26	-0.09	-1.07	0.288
Age*	-0.20	-0.36 - 0.04	-0.39	-2.48	0.014
Gender	1.44	-1.17 – 4.06	0.09	1.09	0.278
Marital status	0.56	-1.53 – 2.65	0.04	0.53	0.598
Employment status	-2.76	-5.05 - -0.47	-0.24	-2.38	0.019
GRACE score	0.01	-0.05 – 0.07	0.05	0.32	0.749
Deprivation*	1.90	0.26 – 3.54	0.19	2.30	0.023

\* Significant independent predictor

Using anxiety as the dependent variable, the model (Table 6.19) explained a significant proportion of variance in anxiety scores ( $R^2 = 0.16$ ,  $F(7, 152) = 4.23$ ,  $p < 0.05$ ) with deprivation being the only significant independent predictors. None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.19 Association between structural social support and anxiety at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	10.53	5.31 – 15.75		3.99	0.001
T2 SNI score	-0.07	-0.44 – 0.30	-0.03	-0.39	0.698
Age	-0.10	-0.21 - 0.00	-0.29	-1.89	0.060
Gender	0.54	-1.19 – 2.27	0.05	0.62	0.559
Marital status	-0.33	-1.72 – 1.06	-0.04	-0.47	0.642
Employment status	-0.54	-2.06 – 0.97	-0.07	-0.71	0.480
GRACE score	0.00	-0.05 – 0.04	-0.02	-0.15	0.883
Deprivation*	1.90	0.82 – 2.99	0.29	3.47	0.001

\* Significant independent predictor

Mean structural social support score was found to be significantly lower in patients scoring above the threshold for depression ( $F(1, 163) = 4.72$ ,  $p < 0.05$ ) compared with those scoring

below. No significant differences were noted for anxiety scores suggesting that depression was associated with lower levels of structural social support (Table 6.20).

**Table 6.20 Mean structural social support (SNI) score by depression and anxiety status at Time 2**

	<b>Depressed</b>	<b>Non-depressed</b>	<b>Anxious</b>	<b>Non-anxious</b>
<b>Mean SNI score (SD)</b>	3.33 (1.66)	4.13 (1.62)	3.88 (1.77)	4.05 (1.61)
<b>N (%)</b>	24 (15)	140 (85)	34 (21)	130 (79)

Logistic regression was also performed to determine the influence of structural social support on the likelihood that patients would report depression above the cut off threshold at Time 2. The model contained four categorical independent variables (gender, marital status, employment status and deprivation level) and three continuous independent variables (structural social support, age and GRACE score with either anxiety status or depression status entered as the dependent variable. For depression status, the full model (Table 6.21) was statistically significant ( $X^2 (8, 161) = 20.64, p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 20.6% of the variance in depression status and correctly classified 85.7% of cases. Deprivation was the largest significant independent predictor in the model with an odds ratio of 10.47 indicating that deprived patients were over ten times more likely to experience depression. Age was the only other significant independent predictor. Structural social support did not make an independent contribution to depression scores.

**Table 6.21 Logistic regression determining the relationship between structural social support and depression at Time 2**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Structural social support	<i>Score increase</i>	1.34	0.96 to 1.86	0.10
Age*	<i>Annual increase</i>	0.91	0.84 to 0.99	0.033
Gender	<i>Male</i>	1		
	<i>Female</i>	3.64	0.60 to 21.74	0.16
Social deprivation*	<i>Low</i>	1		
	<i>Intermediate</i>	2.35	0.73 to 7.54	0.15
	<i>High</i>	10.47	2.00 to 54.84	0.005
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.10	0.38 to 3.15	0.86
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	2.00	0.56 to 7.19	0.32
GRACE score	<i>Score increase</i>	1.02	0.99 to 1.06	0.25

\*Significant independent predictors

For anxiety status, the full model (Table 6.22) was statistically significant ( $X^2(8, 160) = 19.01$ ,  $p < 0.05$ ) and able to distinguish between patients who did and did not report anxiety above the threshold. The model explained 17.4% of the variance in anxiety status and correctly classified 80.6% of cases. Deprivation was the only significant independent predictor in the model with an odds ratio of 9.42 suggesting that patients who were deprived were over nine times more likely to report anxiety above the threshold. Structural social support was not significantly associated with anxiety status.

**Table 6.22 Logistic regression determining the relationship between structural social support and anxiety at Time 2**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Structural social support	<i>Score increase</i>	1.01	0.77 to 1.33	0.93
Age	<i>Annual increase</i>	0.98	0.91 to 1.05	0.54
Gender	<i>Male</i>	1	0.35 to 5.15	0.67
	<i>Female</i>	1.34		
Social deprivation*	<i>Low</i>	1	0.63 to 5.52 1.90 to 46.65	0.26 0.006
	<i>Intermediate</i>	1.87		
	<i>High</i>	9.42		
Marital status	<i>Married</i>	1	0.39 to 2.94	0.89
	<i>Not married</i>	1.08		
Employment	<i>Employed</i>	1	0.40 to 3.74	0.73
	<i>Not employed</i>	1.22		
GRACE score	<i>Score increase</i>	0.99	0.96 to 1.02	0.38

\*Significant independent predictors

#### **6.2.4.1 Summary: Structural social support and psychological response at Time 2**

Structural social support was found to be significantly associated with depression level and status in univariate analysis. However, this relationship was not significant in the multivariate or logistic models. Structural social support was not significantly associated with anxiety level in any analysis. These findings suggest that the level of structural support perceived by a patient was not associated with their risk of psychological disturbance in the early weeks following ACS. Younger and more deprived patients were identified as most at risk for depression and anxiety at Time 2.

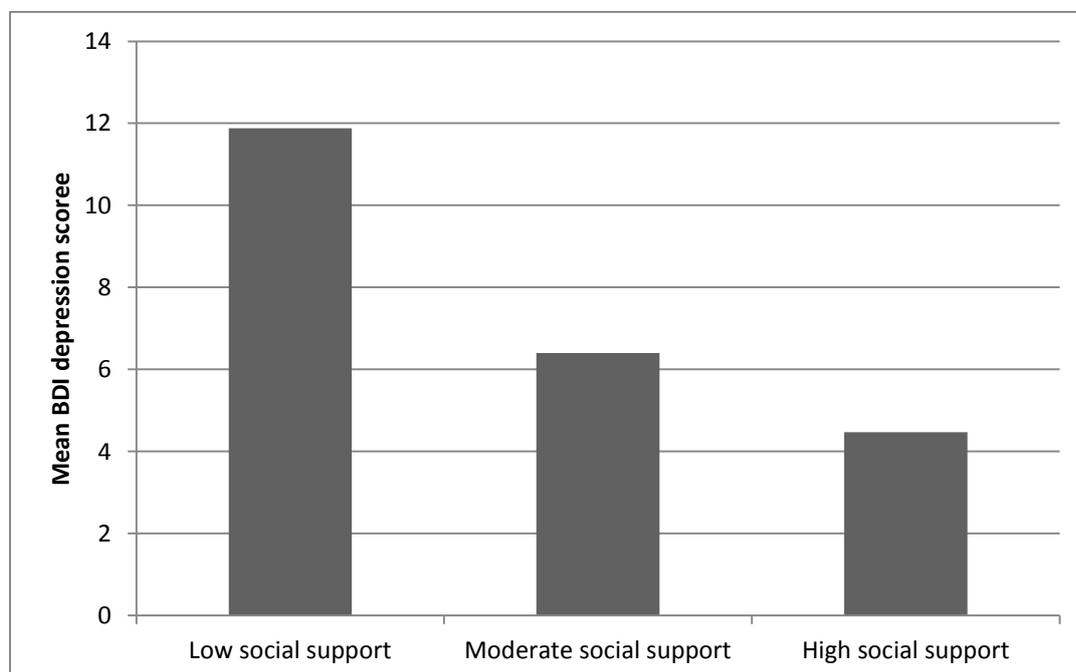
#### **6.2.5 Social support and psychological response at Time 3**

##### **6.2.5.1 Cross sectional analysis: Exploring the association between Time 3 functional social support and Time 3 psychological response**

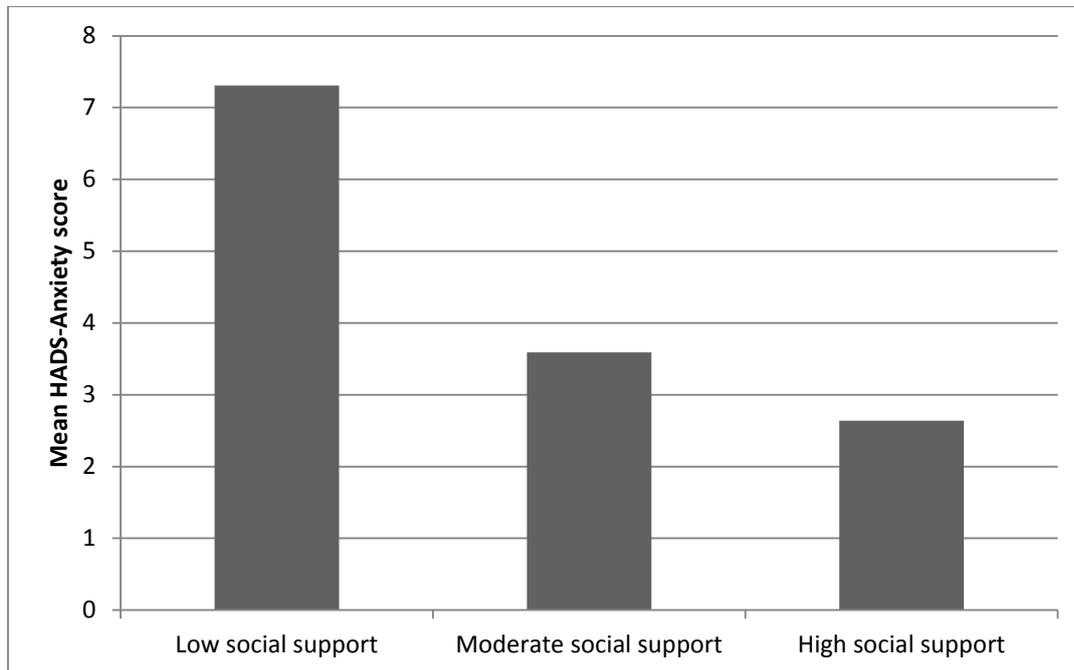
At Time 3, there was a significant negative correlation between BDI score and ESSI score ( $r(151) = -0.31, p < 0.001$ ), and also between HADS-A score and ESSI score ( $r(154) = -0.42, p < 0.001$ ) suggesting an inverse relationship between psychological distress and functional social support at Time 3 that requires exploration.

Patients were divided according to their score on the Time 3 ESSI assessment to create three groups; Low social support ( $\leq 23$ ), moderate social support (24-33) and high social support ( $\geq 34$ ). The group aggregation by ESSI score was described in more detail in section 5.3.4. Mean depression and anxiety scores by functional social support group are depicted in Figures 6.7 and 6.8. A clear decrease in both depression and anxiety score as levels of functional social support increase was observed which was statistically significant (Depression:  $F(2, 148) = 10.19, p < 0.05$  Anxiety:  $F(2, 150) = 16.45, p < 0.05$ ).

**Figure 6.7 Mean depression score at Time 3 by level of functional support at Time 3**



**Figure 6.8 Mean anxiety score at Time 3 by level of functional social support at Time 3**



Multiple regression analysis was conducted using either BDI depression score or HADS-A anxiety score as the dependent variable with functional social support (ESSI score), age, gender, employment status, marital status, GRACE score and deprivation and Time 2 BDI or HADS-A score as the independent variables. Using depression as the dependent variable, the model explained a significant proportion of variance in depression scores ( $R^2=0.60$ ,  $F(8, 122) = 3.33$ ,  $p<0.05$ ) with functional social support and Time 2 depression score being the main significant independent predictors of depression (Table 6.23). The regression was also re-run omitting Time 2 depression score to explore whether this variable may be obscuring other findings. The results were similar with functional social support again identified as the main significant predictor, however, employment status was also found to make an independent contribution to the model ( $\beta=-3.70$ ,  $p=0.037$ ).

**Table 6.23 Association between functional social support at Time 3 and depression at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	2.50	-7.48 – 12.47		0.50	0.621
T3 ESSI score*	-0.21	-0.41 – -0.02	-0.17	-2.17	0.032
Age	-0.03	-0.14 - 0.20	0.04	0.35	0.731
Gender	0.83	-1.92 – 3.57	0.04	0.60	0.553
Marital status	-1.46	-4.11 – 1.19	-0.08	-1.09	0.277
Employment status	-0.27	-2.09 – 2.63	0.02	0.23	0.821
GRACE score	0.02	-0.04 – 0.09	0.07	0.72	0.475
Deprivation	-0.81	-2.71 – 1.09	-0.05	-0.85	0.399
Time 2 depression score*	1.02	0.84 – 1.19	0.76	11.63	001

\* Significant independent predictor

Using anxiety as the dependent variable, the model explained a significant proportion of variance in anxiety scores ( $R^2=0.59$ ,  $F(8, 125) = 5.40$ ,  $p<0.05$ ) with functional social support and Time 2 anxiety score being the only significant independent predictors (Table 6.24). Repeating the regression model with the omission of Time 2 anxiety score did not reveal any additional findings. None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.24 Association between functional social support at Time 3 and anxiety at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	5.70	0.17 – 11.23		2.04	0.043
T3 ESSI score*	-0.19	-0.30 - -0.08	-0.29	-3.54	0.001
Age	0.01	-0.09 – 0.09	-0.01	0.01	0.949
Gender	0.56	-0.91 – 2.02	0.05	0.05	0.452
Marital status	-0.33	-1.80 – 1.13	-0.04	-0.04	0.651
Employment status	0.07	-1.9 – 1.32	-0.01	0.01	0.919
GRACE score	-0.00	-0.03 – 0.04	0.01	0.01	0.903
Deprivation	-0.86	-1.88 – 0.15	-0.12	-0.12	0.095
Time 2 anxiety score*	0.71	0.57 – 0.85	0.68	0.68	0.001

\* Significant independent predictor

Mean functional social support score was found to be significantly lower in patients scoring above the threshold for depression ( $F(1, 150) = 12.99$ ,  $p<0.05$ ) compared to those scoring

below, and was also significantly lower in patients with above threshold anxiety scores ( $F(1,153) = 14.04, p < 0.05$ ). Means scores are displayed in Table 6.25.

**Table 6.25 Mean functional social support (ESSI) score by depression and anxiety status at Time 3**

	Depressed	Non-depressed	Anxious	Non-anxious
<b>Mean ESSI score (SD)</b>	24.50 (7.51)	28.93 (6.09)	23.42 (6.96)	28.67 (6.19)
<b>N (%)</b>	36 (24)	116 (76)	24 (15)	131 (85)

Logistic regression was also performed to establish the impact of functional social support on the likelihood that patients would report above threshold distress at Time 3. The model contained five categorical independent variables (gender, marital status, employment status, deprivation level and Time 2 depression or anxiety status) and three continuous independent variables (functional social support, age and GRACE score) with either anxiety status or depression status entered as the dependent variable. For depression status, the full model (Table 6.26) was statistically significant ( $X^2(9, 132) = 39.53, p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 38.3% of the variance in depression status and correctly classified 84.8% of cases. Time 2 depression status was the largest significant independent predictor in the model with an odds ratio of 18.48. Functional social support was the only other significant independent predictor with an adjusted odds ratio of 1.12. The omission of Time 2 depression status from the model did not reveal any new significant findings.

**Table 6.26 Logistic regression determining the relationship between functional social support at Time 3 and depression at Time 3**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T3 Functional social support*	<i>Score increase</i>	1.12	1.02 to 1.24	0.018
Age	<i>Annual increase</i>	0.97	0.89 to 1.07	0.56
Gender	<i>Male</i>	1		
	<i>Female</i>	1.07	0.24 to 4.75	0.93
Social deprivation*	<i>Low</i>	1		
	<i>Intermediate</i>	1.47	0.43 to 5.06	0.54
	<i>High</i>	2.28	0.13 to 38.84	0.57
Marital status	<i>Married</i>	1		
	<i>Not married</i>	2.70	0.67 to 11.11	0.16
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.43	0.40 to 5.15	0.59
GRACE score	<i>Score increase</i>	1.03	0.99 to 1.07	0.13
Time 2 Depression status*	<i>Not depressed</i>	1		
	<i>Depressed</i>	18.48	4.44 to 76.87	0.001

\*Significant independent predictor

For anxiety status, the full model (Table 6.27) was statistically significant ( $X^2(9, 135) = 53.09, p < 0.05$ ) and able to distinguish between patients who did and did not report anxiety above the threshold. The model explained 56.2% of the variance in anxiety status and correctly classified 88.9% of cases. Time 2 anxiety was the main significant independent predictor with an odds ratio of 35.58. Both age and functional social support were near significant independent predictors (age:  $p = 0.055$ , functional social support:  $p = 0.053$ ). The inclusion of Time 2 anxiety status may have potentially obscured relevant findings. The logistic regression was re-run with the omission of Time 2 anxiety status. In this model, functional social support became a significant independent predictor ( $p = 0.003$ ) with an adjusted odds ratio of 1.14. The effect of age also became significant ( $p = 0.045$ ) with an odds ratio of 0.92.

**Table 6.27 Logistic regression determining the relationship between functional social support at Time 3 and anxiety at Time 3**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T3 Functional social support	<i>Score increase</i>	1.12	1.00 to 1.27	0.053
Age	<i>Annual increase</i>	0.88	0.78 to 1.00	0.055
Gender	<i>Male</i>	1		
	<i>Female</i>	1.35	0.18 to 10.48	0.77
Social deprivation*	<i>Low</i>	1		
	<i>Intermediate</i>	1.77	0.26 to 11.90	0.56
	<i>High</i>	1.21	0.11 to 13.33	0.88
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.78	0.30 to 10.75	0.53
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	3.61	0.59 to 22.22	0.16
GRACE score	<i>Score increase</i>	1.03	0.99 to 1.08	0.17
Time 2 anxiety status*	<i>Not anxious</i>	1		
	<i>Anxious</i>	35.58	7.49 to 168.99	0.001

\*Significant independent predictor

In summary, at Time 3, the cross sectional results suggest that functional social support was a significant independent predictor of both depression and anxiety. Patients who reported higher levels of functional social support at Time 3 were significantly less likely to report psychological distress 6 months following their admission for ACS. Age, employment status and Time 2 psychological distress were also found to be important to Time 3 psychological distress.

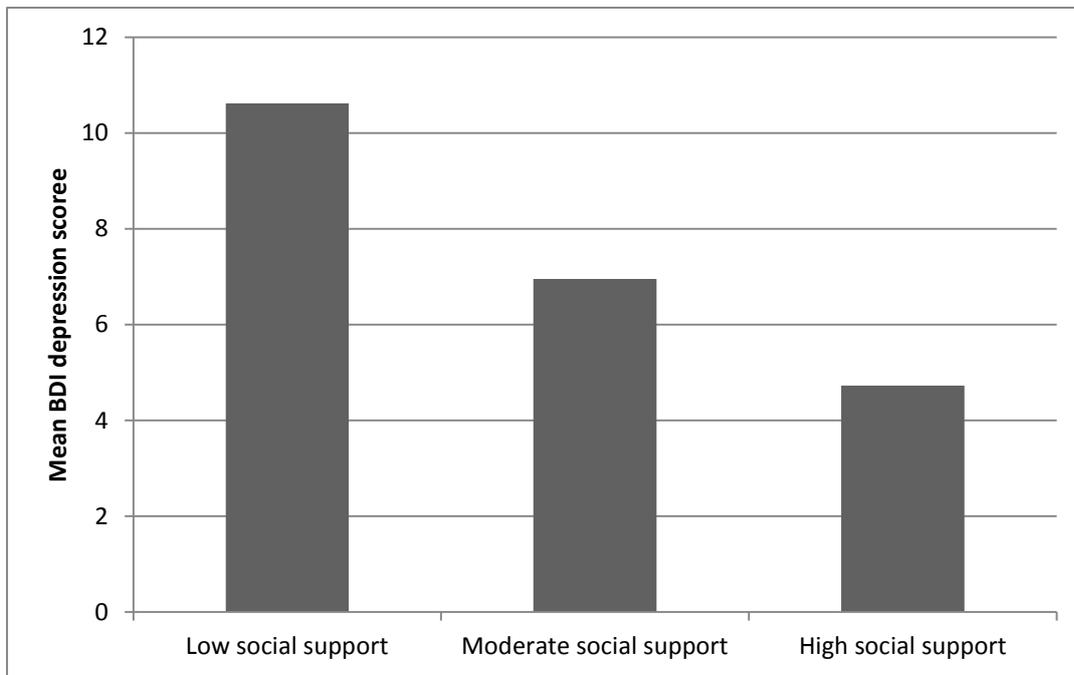
#### **6.2.5.2 Longitudinal analysis: Exploring the predictive efficacy of Time 2 social support for Time 3 psychological distress**

A key hypothesis within my thesis concerns the predictive power of social support measured at Time 2 with regard to the occurrence of psychological distress at Time 3 which is the area explored in the following section. There was a significant negative correlation between Time 3 BDI score and Time 2 ESSI score ( $r(126) = -0.24, p < 0.01$ ), and also between Time 3 HADS-A score and Time 2 ESSI score ( $r(129) = -0.43, p < 0.01$ ) suggesting an inverse

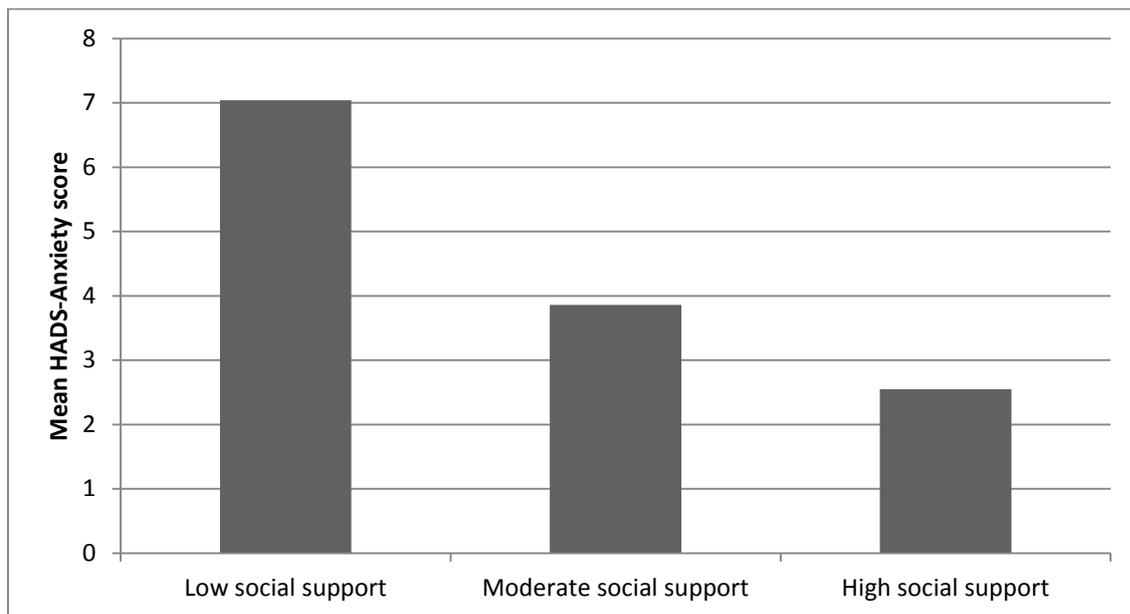
association between psychological distress at Time 3 and functional social support at Time 2.

Patients were divided according to their score on the Time 2 ESSI assessment to create three groups as previously described. Mean Time 3 depression and anxiety scores by Time 2 functional social support group are depicted in Figures 6.9 and 6.10. A clear decrease in both depression and anxiety score as levels of functional social support increase was observed which was statistically significant (Depression:  $F(2, 123) = 5.54, p < 0.05$ , Anxiety:  $F(2, 126) = 13.40, p < 0.05$ ).

**Figure 6.9 Mean depression score at Time 3 by level of functional support at Time 2**



**Figure 6.10 Mean Time 3 anxiety score by level of functional social support at Time 2**



Multiple regression analysis was conducted using either Time 3 BDI depression score or HADS-A anxiety score as the dependent variable with Time 2 functional social support (ESSI score), age, gender, ethnicity, employment status, marital status, GRACE score and deprivation and Time 2 BDI or HADS-A score as the independent variables. Using depression as the dependent variable, the model explained a significant proportion of variance in depression scores ( $R^2=0.60$ ,  $F(9, 112) = 18.71$ ,  $p<0.05$ ) with Time 2 depression score being the only significant independent predictors of depression (Table 6.28). The regression was also re-run omitting Time 2 depression score to explore whether this variable may be obscuring other findings. The model remained significant and age was also found to make an independent contribution to the model ( $\beta=-0.36$ ,  $p=0.044$ ).

**Table 6.28 Functional social support at Time 2 as a predictor of depression at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-2.08	-13.28 – 9.13		-0.37	0.714
T2 ESSI score	-0.05	-0.25 – 0.15	-0.03	-0.51	0.609
Age	0.03	-0.16 - 0.21	0.03	0.26	0.789
Gender	1.54	-1.53 – 4.61	0.06	0.99	0.323
Marital status	-0.18	-2.46 – 2.11	-0.01	-0.15	0.879
Employment status	0.16	-2.41 – 2.73	0.01	0.13	0.900
Ethnicity	1.05	-2.10 -4.16	0.04	0.65	0.515
GRACE score	0.01	-0.06 – 0.08	0.04	0.34	0.739
Deprivation	-0.79	-2.92 – 1.35	-0.05	-0.73	0.468
Time 2 depression score*	1.04	0.85 – 1.22	0.77	11.08	0.001

\* Significant independent predictor

Using anxiety as the dependent variable, the model explained a significant proportion of variance in anxiety scores ( $R^2 = 0.55$ ,  $F(9, 115) = 15.69$ ,  $p < 0.05$ ) with functional social support and Time 2 anxiety score being the only significant independent predictors (Table 6.29). The omission of Time 2 anxiety score produced a significant model with functional social support identified as the only significant predictor ( $\beta = -0.36$ ,  $p = 0.001$ ). None of the variables included in any of the Time 3 regression models showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.29 Functional social support at Time 2 as a predictor of anxiety at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	5.12	-1.14 – 11.38		1.62	0.108
T2 ESSI score*	-0.15	-0.27 - -0.04	-0.20	-2.76	0.007
Age	0.01	-0.11 – 0.09	-0.02	-0.14	0.889
Gender	0.64	-1.04 – 2.32	0.05	0.76	0.451
Marital status	1.00	-0.28 – 2.28	0.11	1.55	0.124
Employment status	-0.06	-1.45 – 1.33	-0.01	-0.09	0.929
Ethnicity	-0.42	-2.14 – 1.30	-0.03	-0.48	0.630
GRACE score	-0.00	-0.03 – 0.04	0.01	-0.01	0.956
Deprivation	-0.88	-2.03 – 0.28	-0.11	-1.51	0.135
Time 2 anxiety score*	0.72	0.56 – 0.87	0.66	9.01	0.001

\* Significant independent predictor

Mean Time 2 functional social support score was found to be significantly lower in patients scoring above the threshold for depression at Time 3 ( $F(1, 125) = 8.88, p < 0.05$ ) and was also significantly lower in patients with above threshold anxiety scores ( $F(1, 128) = 18.36, p < 0.05$ ) at Time 3. Mean scores are displayed in Table 6.30.

**Table 6.30 Mean functional social support (ESSI) score at Time 2 by depression and anxiety status at Time 3**

	Depressed	Non-depressed	Anxious	Non-anxious
<b>Mean ESSI score (SD)</b>	25.66 (5.46)	28.73 (4.82)	23.40 (5.92)	28.61 (4.83)
<b>N (%)</b>	31 (24)	96 (76)	20 (15)	110 (85)

Logistic regression was also performed to determine the relationship between Time 2 functional social support and depression or anxiety above the cut off threshold at Time 3. The model contained six categorical independent variables (gender, marital status, ethnicity, employment status, deprivation level and Time 2 depression or anxiety status) and three continuous independent variables (Time 2 functional social support, age and GRACE score) with either Time 3 anxiety status or depression status entered as the dependent variable. For depression status, the full model (Table 6.31) was statistically significant ( $X^2(10, 123) = 31.62, p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 33.5% of the variance in depression status and correctly classified 82.9% of cases. Time 2 depression status was the only significant independent predictor in the model with an odds ratio of 12.71. The omission of Time 2 depression status from the model did not reveal any new significant findings, however, Time 2 functional social support did reach near significance ( $\beta = 0.921, p = 0.064$ ).

**Table 6.31 Logistic regression predicting likelihood of depression at Time 3 using Time 2 functional social support**

Variable	Categories	Adjusted odds ratio	95% C.I.	p
T2 Functional social support	<i>Score increase</i>	1.08	0.98 to 1.19	0.14
Age	<i>Annual increase</i>	0.95	0.87 to 1.05	0.33
Gender	<i>Male</i>	1		
	<i>Female</i>	1.37	0.30 to 6.29	0.69
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.56	0.44 to 5.55	0.49
	<i>High</i>	1.71	0.09 to 34.08	0.73
Marital status	<i>Married</i>	1		
	<i>Not married</i>	2.70	0.42 to 4.63	0.58
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.30	0.37 to 4.66	0.68
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	2.22	0.50 to 9.90	0.29
GRACE score	<i>Score increase</i>	1.03	0.99 to 1.07	0.14
Time 2 Depression status*	<i>Not depressed</i>	1		
	<i>Depressed</i>	12.71	3.11 to 62.01	0.001

\*Significant independent predictor

For anxiety status, the full model (Table 6.32) was statistically significant ( $X^2(10, 126) = 49.96, p < 0.05$ ) and able to distinguish between patients who did and did not report anxiety above the threshold. The model explained 56.1% of the variance in anxiety status and correctly classified 88.9% of cases. Time 2 anxiety was the main significant independent predictor with an odds ratio of 36.39. Functional social support was the only other significant predictor with an odds ratio of 1.14. The omission of Time 2 anxiety status from the model did not reveal any new significant findings, however, age became a near significant predictor ( $\beta = 0.90, p = 0.056$ ).

**Table 6.32 Logistic regression predicting likelihood of anxiety at Time 3 using Time 2 social support**

Variable	Categories	Adjusted odds ratio	95% C.I.	p
T2 Functional social support*	<i>Score increase</i>	1.14	1.01 to 1.29	0.041
Age	<i>Annual increase</i>	0.90	0.79 to 1.02	0.11
Gender	<i>Male</i>	1		
	<i>Female</i>	2.23	0.27 to 18.33	0.46
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.80	0.25 to 12.99	0.56
	<i>High</i>	1.27	0.09 to 17.86	0.86
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.02	0.19 to 5.52	0.98
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	2.50	0.37 to 16.67	0.35
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	1.37	0.18 to 10.02	0.76
GRACE score	<i>Score increase</i>	1.03	0.98 to 1.08	0.27
Time 2 anxiety status*	<i>Not anxious</i>	1		
	<i>Anxious</i>	36.39	7.34 to 180.32	0.001

\*Significant independent predictor

Overall, the results suggest that functional social support assessed at Time 2 was a significant independent predictor of both the experience of anxiety symptomatology and above threshold anxiety. Age and Time 2 anxiety were also found to make a contribution to the experience of anxiety at Time 3 with younger patients and patients with higher anxiety at Time 2 more vulnerable to anxiety at Time 3. There is less evidence for a predictive relationship between Time 2 functional social support and depression at Time 3 with age and Time 2 depression being the main predictor of Time 3 depression. However, the results from the logistic regression did show a near significant contribution of functional social support to the likelihood of above threshold depression.

### **6.2.5.3 Longitudinal analysis: Exploring the predictive efficacy of Time 2 structural social support for Time 3 psychological distress**

This section addresses a further hypothesis within my thesis which involves the ability of structural social support measured at Time 2 to predict psychological distress at Time 3. There was a significant negative correlation between Time 3 BDI score and Time 2 structural social support ( $r(127) = -0.18, p < 0.05$ ) and between Time 3 HADS-A score and Time 2 structural social support ( $r(130) = -0.18, p < 0.05$ ) indicative of an association. Patients completing the Time 3 assessment were subdivided according to their level of structural social support to form two groups: Low structural social support (1 or fewer people in social network, Mean SNI = 0.88 (0.35), N=8) and adequate structural social support (2 or more people, Mean SNI = 4.23 (1.47), N=123). There was no significant difference in depression ( $F(1, 125) = 1.20, p = 0.28$ ) or anxiety ( $F(1, 128) = 0.65, p = 0.47$ ) level between patients reporting low or adequate structural social support.

Multiple regression analysis was conducted using either BDI depression score or HADS- A anxiety score as the dependent variable with structural social support (SNI score), age, gender, employment status, marital status, GRACE score, deprivation and Time 2 BDI or HADS-A score as the independent variables. Using depression as the dependent variable, the model (Table 6.33) explained a significant proportion of variance in depression scores ( $R^2 = 0.60, F(9, 113) = 19.18, p < 0.05$ ) with Time 2 BDI score being the only significant predictor. Running the regression without Time 2 BDI score produced a significant model with age ( $\beta = -0.35, P < 0.05$ ) and ethnicity ( $\beta = 0.18, p < 0.05$ ) identified as significant predictors. Gender also emerged as a borderline significant predictor ( $\beta = 0.18, p = 0.054$ ). Structural social support did not emerge as significant in either model.

**Table 6.33 Structural social support at Time 2 as a predictor of depression at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-4.59	-14.35 – 5.17		-0.93	0.353
T2 SNI score	-0.07	-0.69 – 0.56	-0.01	-0.21	0.832
Age	0.04	-0.14- 0.23	0.06	0.45	0.653
Gender	1.94	-1.07 – 4.96	0.08	1.28	0.204
Marital status	-0.12	-2.43 – 2.19	-0.01	-0.10	0.917
Employment status	0.46	-2.12 – 3.04	0.03	0.35	0.724
Ethnicity	1.13	-1.93 – 4.19	0.05	0.73	0.466
GRACE score	0.01	-0.06 – 0.08	0.02	0.21	0.836
Deprivation	-0.45	-2.53 – 1.64	-0.03	-0.42	0.673
Time 2 Depression score*	1.05	0.86 – 1.24	0.77	11.16	0.001

\* Significant independent predictor

Using anxiety as the dependent variable, the model (Table 6.34) explained a significant proportion of variance in anxiety scores ( $R^2 = 0.53$ ,  $F(9, 116) = 14.31$ ,  $p < 0.05$ ) with Time 2 anxiety score being the only significant independent predictors. Repeating the regression without Time 2 anxiety score produced a non-significant model with no significant predictors. None of the variables included in either depression or anxiety regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.34 Structural social support at Time 2 as a predictor of anxiety at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	1.27	-4.18 – 6.71		0.46	0.646
T2 SNI score	-0.16	-0.52 – 0.19	-0.06	-0.92	0.361
Age	-0.10	-0.11 - 0.09	-0.03	-0.25	0.799
Gender	0.87	-0.81 – 2.55	0.07	1.03	0.305
Marital status	1.11	-0.21 – 2.43	0.12	1.67	0.097
Employment status	-0.47	-1.47 – 1.38	-0.01	-0.07	0.948
Ethnicity	0.02	-1.72 – 1.75	0.00	0.02	0.986
GRACE score	-0.00	-0.04 – 0.04	-0.00	-0.05	0.959
Deprivation	-0.74	-1.89 – 0.41	-0.10	-1.27	0.205
Time 2 anxiety score*	0.77	0.61 – 0.92	0.71	9.66	0.001

\* Significant independent predictor

Mean structural social support score was found to be significantly lower in patients scoring above the threshold for anxiety ( $F(1, 129) = 4.94$ ,  $p < 0.05$ ) compared with those scoring

below. No significant differences in structural support score were noted between the depressed and non-depressed groups (Table 6.35).

**Table 6.35 Mean Time 2 structural social support (SNI) score by depression and anxiety status at Time 3**

	<b>Depressed</b>	<b>Non-depressed</b>	<b>Anxious</b>	<b>Non-anxious</b>
<b>Mean SNI score (SD)</b>	3.88 (1.74)	4.10 (1.63)	3.30 (1.53)	4.17 (1.83)
<b>N (%)</b>	32 (25)	96 (75)	20 (15)	111 (85)

Logistic regression was also performed to determine the relationship between structural social support and depression above the cut off threshold at Time 3. The model contained five categorical independent variables (gender, marital status, employment status, deprivation level and either depression or anxiety status at Time 2) and three continuous independent variables (Time 2 structural social support, age and GRACE score) with either anxiety status or depression status at Time 3 entered as the dependent variable. For depression status, the full model (Table 6.36) was statistically significant ( $X^2(9, 124) = 30.34$ ,  $p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 31.3% of the variance in depression status and correctly classified 82.9% of cases. The only significant predictor was Time 2 depression status recording an odds ratio of 17.57. Structural social support did not make an independent contribution to depression scores. The model was re-run excluding Time 2 depression status and was no longer significant with no new predictors identified.

**Table 6.36 Logistic regression predicting likelihood of depression at Time 3 using Time 2 structural social support**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Structural social support	<i>Score increase</i>	1.11	0.81 to 1.53	0.51
Age	<i>Annual increase</i>	0.96	0.88 to 1.05	0.39
Gender	<i>Male</i>	1		
	<i>Female</i>	1.20	0.40 to 7.57	0.46
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.67	0.50 to 5.55	0.41
	<i>High</i>	2.30	0.13 to 41.94	0.58
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.04	0.37 to 3.89	0.76
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.02	0.99 to 1.06	0.84
GRACE score	<i>Score increase</i>	1.02	0.99 to 1.06	0.24
Time 2 Depression score*	<i>Depressed</i>	1		
	<i>Not depressed</i>	17.57	4.24 to 72.83	0.001

\*Significant independent predictors

For anxiety status, the full model (Table 6.37) was statistically significant ( $X^2(9, 127) = 46.71$ ,  $p < 0.05$ ) and able to distinguish between patients who did and did not report anxiety above the threshold. The model explained 52.9% of the variance in anxiety status and correctly classified 90.6% of cases. Anxiety status at Time 2 was the only significant predictor with an odds ratio of 38.55. Structural social support did not make a significant independent prediction regarding anxiety status. The model remained significant with the omission of Time 2 anxiety status with age ( $\beta = 0.89$ ,  $p < 0.05$ ) and deprivation ( $\beta = 9.13$ ,  $p < 0.05$ ) identified as significant predictors

**Table 6.37 Logistic regression predicting likelihood of anxiety at Time 3**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Structural social support	<i>Score increase</i>	1.39	0.91 to 2.15	0.14
Age	<i>Annual increase</i>	0.89	0.79 to 1.01	0.06
Gender	<i>Male</i>	1		
	<i>Female</i>	1.69	0.20 to 14.11	0.63
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.42	0.23 to 8.70	0.71
	<i>High</i>	1.30	0.11 to 15.87	0.84
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.04	0.21 to 5.29	0.96
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	3.39	0.57 to 16.67	0.18
GRACE score	<i>Score increase</i>	1.03	0.98 to 1.08	0.46
Time 2 Anxiety score*	<i>Anxious</i>	1		
	<i>Not anxious</i>	38.55	7.99 to 186.13	0.001

\*Significant independent predictors

In summary, structural social support did not make an independent contribution to depression or anxiety scores at Time 3 suggesting that the level of structural support perceived by a patient at Time 2 was not associated with their risk of psychological disturbance 6 months following their admission for ACS.

## 6.2.6 Social support and psychological distress at Time 4

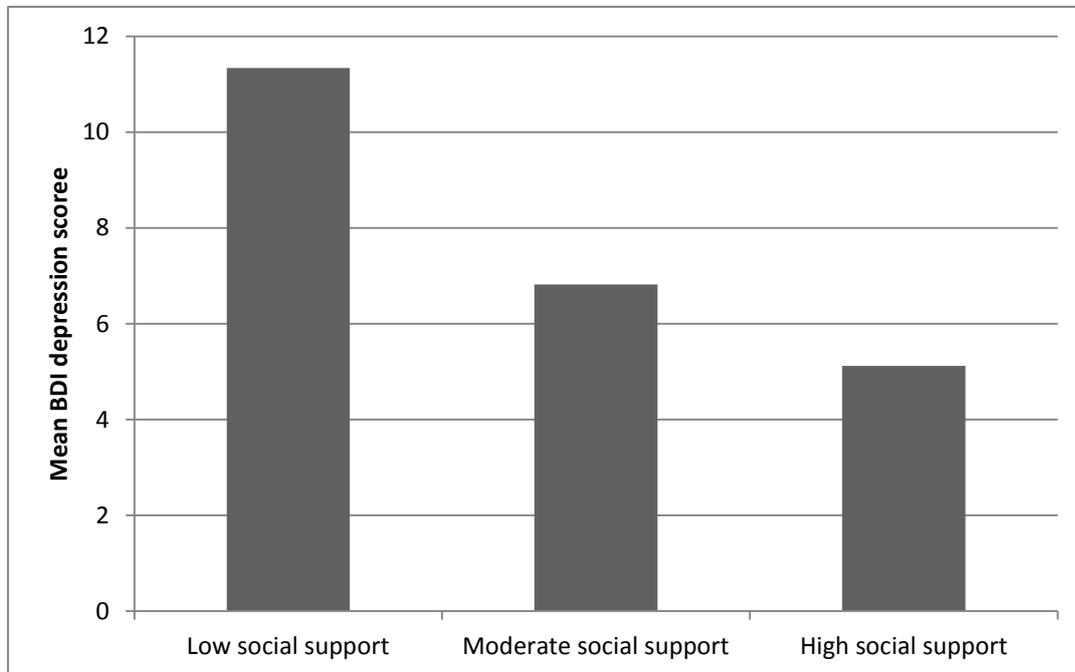
### 6.2.6.1 Cross sectional analysis: Exploring the association between Time 4 functional social support and Time 4 psychological distress

There was a significant negative correlation between Time 4 BDI score and Time 4 ESSI score ( $r(151) = -0.35, p < 0.001$ ), and also between Time 4 HADS-A score and Time 4 ESSI score ( $r(152) = -0.27, p < 0.001$ ) suggestive of an inverse relationship between psychological distress and functional social support at Time 4 meriting exploration.

Mean depression and anxiety scores by functional social support group (group aggregation described in Section 5.3.4) are depicted in Figures 6.11 and 6.12. A statistically significant

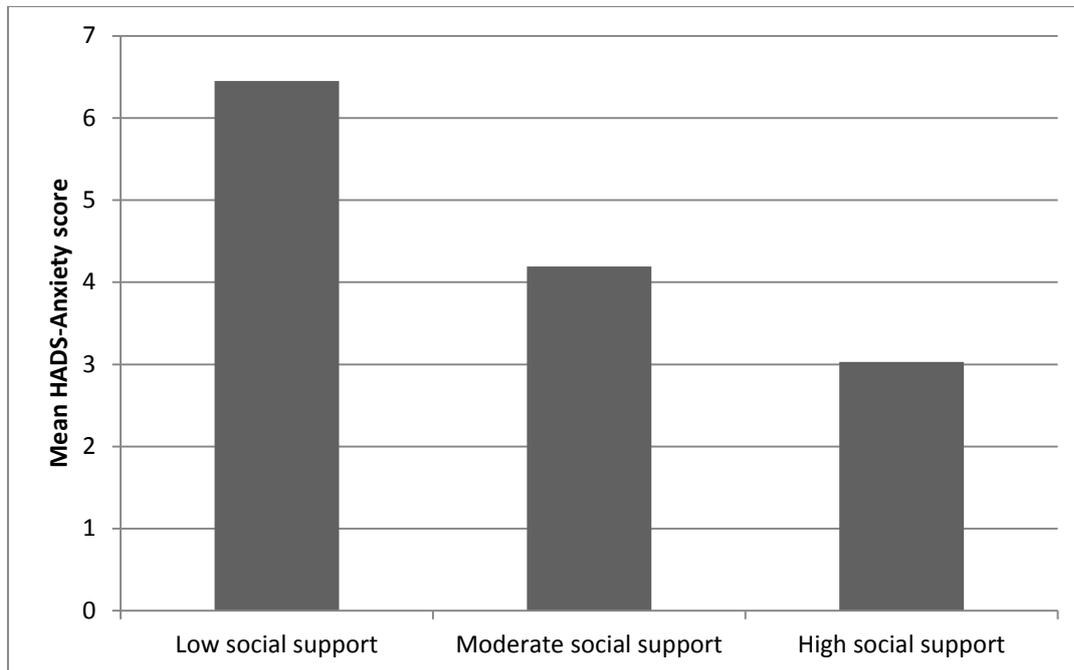
decrease in both depression and anxiety score as levels of functional social support increase was noted (Depression:  $F(2, 148) = 7.43, p < 0.05$ , Anxiety:  $F(2, 149) = 7.67, p < 0.05$ ).

**Figure 6.11 Mean depression score at Time 4 by level of functional support at Time 4**



**Figure 6.12 Mean anxiety score at Time 4 by level of functional social support at Time 4**

**4**



Multiple regression analysis was conducted using either Time 4 BDI depression score or HADS-A anxiety score as the dependent variable with Time 4 functional social support (ESSI score), age, gender, employment status, marital status, GRACE score, deprivation and Time 2 BDI or HADS-A score as the independent variables. Using depression as the dependent variable, the model explained a significant proportion of variance in depression scores ( $R^2 = 0.43$ ,  $F(8, 121) = 11.29$ ,  $p < 0.05$ ) with functional social support and Time 2 depression score being the main significant independent predictors (Table 6.38). The regression was also re-run omitting both Time 2 depression score to explore whether these variables may be obscuring other findings. The model remained significant with functional social support found to be the only significant independent predictor ( $\beta = -0.34$ ,  $p = 0.037$ ).

**Table 6.38 Association between Time 4 functional social support and depression at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	4.37	-6.06 – 14.80		0.83	0.409
T4 ESSi score*	-0.24	-0.43 – -0.05	-0.22	-2.49	0.014
Age	-0.04	-0.21 - 0.14	-0.06	-0.41	0.686
Gender	2.51	-0.38 – 5.40	0.13	1.72	0.088
Marital status	-1.70	-4.45 – 1.04	-0.11	-1.23	0.222
Employment status	1.05	-1.65 – 3.74	0.07	0.77	0.443
GRACE score	0.04	-0.03 – 0.12	0.15	1.20	0.234
Deprivation	-0.78	-2.52 – 0.96	-0.07	-0.89	0.376
Time 2 depression score*	0.81	0.60 – 1.02	0.57	7.53	0.001

\* Significant independent predictor

Using anxiety as the dependent variable, the model explained a significant proportion of variance in anxiety scores ( $R^2 = 0.65$ ,  $F(8, 121) = 11.16$ ,  $p < 0.05$ ) with functional social support, gender and Time 2 anxiety score revealed as significant independent predictors (Table 6.39). The omission of Time 2 anxiety, the model remained significant and functional social support emerged as the only significant predictor ( $\beta = -0.38$ ,  $p < 0.05$ ). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.39 Association between Time 4 functional social support and anxiety at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	T	Sig.
Constant	3.60	-1.85 – 9.05		1.31	0.194
T4 ESSi score*	-0.15	-0.26 – -0.05	-0.26	-2.94	0.004
Age	-0.01	-0.11 – 0.08	-0.04	-0.27	0.789
Gender*	1.65	0.09 – 3.20	0.15	2.09	0.038
Marital status	-1.14	-2.66 – 0.38	-0.13	-1.48	0.140
Employment status	0.38	-1.06 – 1.82	0.05	0.52	0.605
GRACE score	0.02	-0.02 – 0.06	0.11	0.90	0.372
Deprivation	-0.60	-1.56 – 0.36	-0.10	-1.24	0.219
Time 2 anxiety score*	0.56	0.40 – 0.73	0.52	6.76	0.001

\* Significant independent predictor

Mean functional social support score was found to be significantly lower in patients scoring above the threshold for depression ( $F(1, 149) = 6.11, p < 0.05$ ) compared to those scoring below, and was also significantly lower in patients with above threshold anxiety scores ( $F(1, 150) = 18.84, p < 0.05$ ). Means scores are displayed in Table 6.40.

**Table 6.40 Mean functional social support (ESSI) score at Time 4 by depression and anxiety status at Time 4**

	Depressed	Non-depressed	Anxious	Non-anxious
<b>Mean ESSI score (SD)</b>	24.70 (7.68)	27.97 (7.00)	22.44 (8.05)	28.39 (6.56)
<b>N (%)</b>	40 (26)	111 (74)	32 (21)	120 (79)

Logistic regression was also performed to ascertain the role of functional social support in above threshold depression or anxiety at Time 4. The model contained five categorical independent variables (gender, marital status, employment status, deprivation level, and Time 2 depression or anxiety status) and three continuous independent variables (Time 4 functional social support, age and GRACE score) with either Time 4 anxiety status or depression status entered as the dependent variable. For depression status, the full model (Table 6.41) was statistically significant ( $\chi^2(9, 130) = 26.88, p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 28% of the variance in depression status and correctly classified 80% of cases. Time 2 depression status was the largest significant independent predictor in the model with an odds ratio of 13.52 suggesting that those patients reporting above threshold depression at Time 2 were over 13 times more likely to report depression over the threshold at Time 4. Gender was found to be a significant predictor. Female patients were almost 4 times more likely to report above threshold depression than male patients at Time 4. GRACE score was also a significant predictor with an odds ratio of 1.04 suggesting that patients with higher GRACE score were slightly more at risk to have depression at Time 4. Functional social support was not a significant independent predictor. However, both functional social support

and marital status were nearing significance. The model was repeated with the omission of Time 2 depression status, and the new model was no longer significant. However, functional social support did emerge as the only significant predictor ( $\beta=0.93$ ,  $p=0.023$ ).

**Table 6.41 Logistic regression determining the relationship between functional social support at Time 4 and depression at Time 4**

Variable	Categories	Adjusted odds ratio	95% C.I.	p
T4 Functional social support	<i>Score increase</i>	1.08	0.99 to 1.17	0.07
Age	<i>Annual increase</i>	0.94	0.87 to 1.02	0.15
Gender*	<i>Male</i>	1		
	<i>Female</i>	3.73	1.10 to 12.59	0.034
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.45	0.35 to 6.25	0.61
	<i>High</i>	1.37	0.27 to 6.96	0.71
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.06	0.96 to 15.15	0.06
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.19	0.36 to 3.93	0.78
GRACE score*	<i>Score increase</i>	1.04	1.00 to 1.07	0.041
Time 2 Depression status*	<i>Not depressed</i>	1		
	<i>Depressed</i>	7.95	2.27 to 27.85	0.001

\*Significant independent predictors

For anxiety status, the full model (Table 6.42) was statistically significant ( $X^2$  (9, 130) = 37.27,  $p<0.05$ ) and able to distinguish between patients who did and did not report anxiety above the threshold. The model explained 39.9% of the variance in anxiety status and correctly classified 83.8% of cases. Time 2 anxiety status was the largest significant independent predictor with an odds ratio of 9.81 indicating that above threshold anxiety at Time 2 was predictive of above threshold anxiety at Time 4. Functional social support was also a significant predictor with an odds ratio of 1.16. The logistic regression was repeated with the omission of Time 2 anxiety status. The model remained significant with functional

social support identified as a significant predictor of anxiety with an odds ratio of 1.13. No new predictors were identified.

**Table 6.42 Logistic regression determining the relationship between functional social support at Time 4 and depression at Time 4**

Variable	Categories	Adjusted odds ratio	95% C.I.	p
T4 Functional social support*	<i>Score increase</i>	1.16	1.05 to 1.29	0.003
Age	<i>Annual increase</i>	1.00	0.92 to 1.10	0.94
Gender	<i>Male</i>	1		
	<i>Female</i>	3.24	0.78 to 13.54	0.11
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.91	0.39 to 9.43	0.43
	<i>High</i>	9.52	0.77 to 111.11	0.08
Marital status	<i>Married</i>	1		
	<i>Not married</i>	6.10	0.57 to 66.67	0.14
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	3.89	0.83 to 18.06	0.08
GRACE score	<i>Score increase</i>	1.03	0.92 to 1.10	0.19
Time 2 Anxiety status*	<i>Not anxious</i>	1		
	<i>Anxious</i>	9.81	2.73 to 35.27	0.001

\*Significant independent predictors

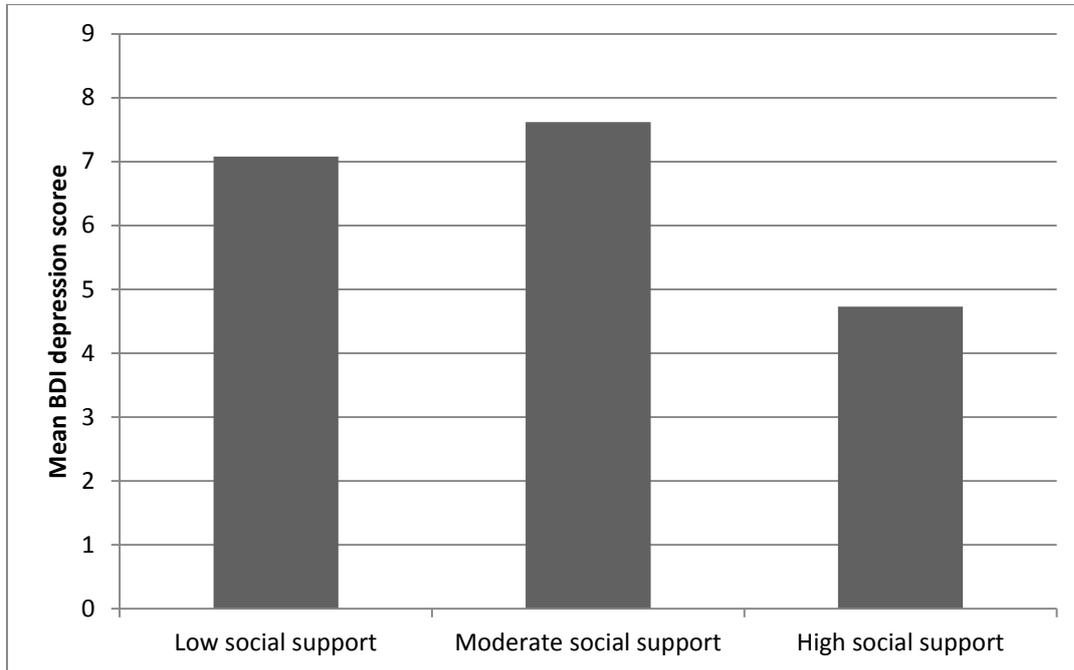
Overall, functional social support measured at Time 4 was found to be associated with the occurrence of distress at Time 4. In particular, functional social support was strongly associated with the occurrence of anxiety and anxiety over the threshold at Time 4. Functional social support was also found to be associated with depression level at Time 4 but did not emerge as a significant predictor of above threshold depression in the logistic regression. Gender, marital status and employment status were also found to be important to psychological distress at Time 4 with unmarried, unemployed and female patients most at risk of persistent distress. Time 2 levels of psychological distress were unsurprisingly highly predictive of distress at Time 4 indicating the continuance of such states over the long term.

### **6.2.6.2 Longitudinal analysis: Exploring the predictive efficacy of Time 2 functional social support for Time 4 psychological response**

A second key hypothesis addressed in my thesis relates to the predictive relationship between Time 2 functional social support and Time 4 psychological distress. There was a significant negative correlation between Time 4 HADS-A score and Time 2 ESSi score ( $r(123) = -0.21, p < 0.05$ ) suggesting an inverse association between anxiety at Time 4 and functional social support at Time 2. There was no significant correlations between Time 4 BDI score and Time 2 functional social support ( $r(122) = -0.02, p = 0.87$ ).

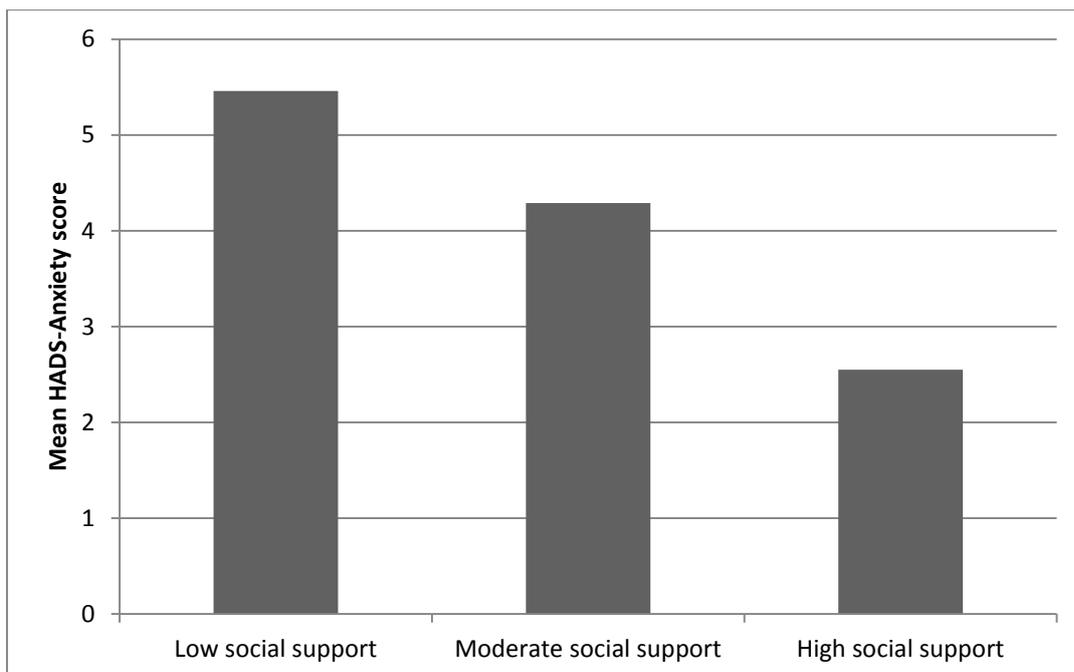
Patients were divided according to their score on the Time 2 ESSi assessment to create three groups as detailed previously. Mean Time 4 depression and anxiety scores by Time 2 functional social support group are depicted in Figures 6.13 and 6.14. A significant decrease in Time 4 anxiety score as levels of Time 2 functional social support increase was observed which was statistically significant ( $F(2, 120) = 4.24, p < 0.05$ ). There was no significant association between depression at Time 4 and functional social support at Time 2 ( $F(2, 119) = 0.84, p = 0.75$ ).

**Figure 6.13 Mean depression score at Time 4 by level of functional support at Time 2**



**Figure 6.14 Mean anxiety score at Time 4 by level of functional social support at Time**

**2**



Multiple regression analysis was conducted using either Time 4 BDI depression score or HADS-A anxiety score as the dependent variable with Time 2 functional social support (ESSI score), age, gender, ethnicity, employment status, marital status, GRACE score and deprivation and Time 2 BDI or HADS-A score as the independent variables. Using depression as the dependent variable, the model explained a significant proportion of variance in depression scores ( $R^2=0.50$ ,  $F(9, 108) = 11.81$ ,  $p<0.05$ ) with Time 2 depression score, gender and ethnicity revealed as significant independent predictors of depression (Table 6.43). Time 2 functional social support was also found to be a borderline significant predictor of depression ( $\beta=0.15$ ,  $p=0.05$ ). The regression was also re-run omitting Time 2 depression score and the model was no longer significant with no new predictors identified.

**Table 6.43 Functional social support at Time 2 as a predictor of depression at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-13.92	-25.57 – -2.57		-2.43	0.017
T2 ESSI score	0.21	0.00 – 0.41	0.15	1.99	0.050
Age	0.04	-0.14 - 0.22	0.07	0.49	0.626
Gender*	3.79	0.82 – 6.76	0.18	2.53	0.013
Marital status	-0.96	-3.40 – 1.47	-0.06	-0.78	0.435
Employment status	1.12	-1.57 – 3.81	0.08	0.83	0.410
Ethnicity*	4.38	1.07 – 7.68	0.19	2.62	0.010
GRACE score	0.02	-0.05 – 0.09	0.07	0.54	0.591
Deprivation	-1.38	-3.31 – 0.56	-0.11	-1.41	0.162
Time 2 depression score*	1.00	0.80 – 1.21	0.71	9.66	0.001

\* Significant independent predictor

Using anxiety as the dependent variable, the model explained a significant proportion of variance in anxiety scores ( $R^2=0.45$ ,  $F(9, 108) = 10.00$ ,  $p<0.05$ ) with gender and Time 2 anxiety score being the only significant independent predictors (Table 6.44). There was a borderline significant effect of deprivation ( $\beta= -0.16$ ,  $p= 0.051$ ). The regression was repeated with the omission of Time 2 anxiety score and the model was no longer significant with no new predictors identified. None of the variables included in any of the Time 4 regression models showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.44 Functional social support at Time 2 as a predictor of anxiety at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-0.61	-6.79 – 5.58		-0.20	0.846
T2 ESSI score	-0.09	-0.21 – 0.02	-0.12	-1.57	0.118
Age	0.01	-0.09 – 0.11	0.04	0.26	0.796
Gender*	2.35	0.66 – 4.05	0.21	2.76	0.007
Marital status	-0.37	-1.77 – 1.03	-0.04	-0.53	0.600
Employment status	0.60	-0.90 – 2.09	0.08	0.79	0.431
Ethnicity	0.30	-1.59 – 2.18	0.02	0.31	0.755
GRACE score	-0.01	-0.03 – 0.05	0.06	0.48	0.631
Deprivation	-1.14	-2.28 – 0.01	-0.16	-1.97	0.051
Time 2 anxiety score*	0.69	0.53 – 0.86	0.63	8.13	0.001

\* Significant independent predictor

Mean Time 2 functional social support score was found to be significantly lower in patients scoring above the threshold for anxiety ( $F(1,121) = 9.90, p < 0.05$ ) at Time 4. No significant association was found between Time 4 depression status and Time 2 functional social support ( $F(1, 120) = 0.04, p = 0.85$ ). Means scores are displayed in Table 6.45.

**Table 6.45 Mean functional social support (ESSI) score by depression and anxiety status at Time 4**

	Depressed	Non-depressed	Anxious	Non-anxious
<b>Mean ESSI score (SD)</b>	27.91 (4.35)	28.12 (5.42)	24.98 (4.54)	28.69 (5.11)
<b>N (%)</b>	27 (22)	95 (78)	22 (18)	101 (82)

Logistic regression was also performed to assess the impact of Time 2 functional social support on the likelihood that patients would report above threshold depression or anxiety at Time 4. The model contained six categorical independent variables (gender, marital status, ethnicity, employment status, deprivation level and Time 2 depression or anxiety status) and three continuous independent variables (Time 2 functional social support, age and GRACE score) with either Time 4 anxiety status or depression status entered as the dependent

variable. For depression status, the full model (Table 6.46) was statistically significant ( $X^2(10, 118) = 27.42, p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 31.5% of the variance in depression status and correctly classified 81.4% of cases. Time 2 depression status was the largest significant independent predictor in the model with an odds ratio of 16.14. Gender and marital status were also found to be important with female patients over 6 times more likely, and unmarried patients over 5 times more likely to experience above threshold depression at Time 4. The model was no longer significant with the omission of Time 2 depression status and no new predictors were identified.

**Table 6.46 Logistic regression predicting likelihood of depression at Time 4 using Time 2 functional social support**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
Time 2 Functional social support	<i>Score increase</i>	1.04	0.94 to 1.16	0.44
Age	<i>Annual increase</i>	0.96	0.88 to 1.05	0.83
Gender*	<i>Male</i>	1		
	<i>Female</i>	6.22	1.48 to 26.06	0.012
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.37	0.30 to 6.32	0.68
	<i>High</i>	1.50	0.19 to 11.76	0.70
Marital status*	<i>Married</i>	1		
	<i>Not married</i>	5.59	1.24 to 25.00	0.025
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.02	0.29 to 3.63	0.97
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	3.43	0.75 to 15.72	0.11
GRACE score	<i>Score increase</i>	1.04	1.0 to 1.07	0.08
Time 2 Depression status*	<i>Not depressed</i>	1		
	<i>Depressed</i>	16.14	3.57 to 73.06	0.001

\*Significant independent predictor

For anxiety status, the full model (Table 6.47) was statistically significant ( $X^2(10, 118) = 36.40, p < 0.05$ ) and able to distinguish between patients who did and did not report anxiety

above the threshold. The model explained 43.0% of the variance in anxiety status and correctly classified 84.7% of cases. Time 2 anxiety was the largest significant independent predictor with an odds ratio of 16.51. Functional social support was identified as a significant predictor with an odds ratio of 1.15. Gender was also found to be a significant predictor with an odds ratio of 2.23. The model remained significant with the omission of Time 2 anxiety status from the model and functional social support was revealed as the only significant predictor of anxiety status ( $\beta=0.88$ ,  $p<0.05$ ).

**Table 6.47 Logistic regression predicting likelihood of anxiety at Time 4 using Time 2 social support**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Functional social support*	<i>Score increase</i>	1.15	1.04 to 1.28	0.009
Age	<i>Annual increase</i>	0.99	0.90 to 1.11	0.14
Gender	<i>Male</i>	1		
	<i>Female</i>	2.23	0.27 to 18.33	0.46
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.49	0.23 to 9.52	0.67
	<i>High</i>	12.05	0.57 to 250.00	0.11
Marital status	<i>Married</i>	1		
	<i>Not married</i>	1.15	0.23 to 5.65	0.98
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	3.15	0.63 to 15.76	0.16
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	2.09	0.43 to 10.12	0.36
GRACE score	<i>Score increase</i>	1.03	0.98 to 1.07	0.26
Time 2 anxiety status*	<i>Not anxious</i>	1		
	<i>Anxious</i>	16.15	3.92 to 66.41	0.001

\*Significant independent predictor

The overall findings suggest that functional social support assessed at Time 2 was a significant independent predictor of above threshold anxiety at Time 4. Anxiety at Time 2, being female and more deprived was also found to increase the risk of anxiety at Time 4. Functional social support was not found to predict the occurrence of depression at Time 4.

Time 2 depression, gender, ethnicity and marital status were identified as important predictors of Time 4 depression.

### **6.2.6.3 Longitudinal analysis: Exploring the predictive efficacy of Time 2 structural social support for Time 4 psychological distress**

It was predicted that lower levels of structural social support measured at Time 2 would be predictive of higher levels of psychological distress at Time 4. There was no significant correlation between Time 4 BDI score and Time 2 structural social support ( $r(123)=-0.04$ ,  $p=0.65$ ), nor between Time 4 HADS-A score and Time 2 structural social support ( $r(124)=-0.05$ ,  $p=0.61$ ) suggesting no association. Patients completing the Time 4 assessment were categorised according to their level of structural social support to form two groups: Low structural social support (1 or fewer people in social network, Mean SNI = 0.86 (0.38),  $N=7$ ) and adequate structural social support (2 or more people, Mean SNI = 4.24 (1.45),  $N=132$ ). There was no significant difference in depression ( $F(1, 121) = 0.002$ ,  $p=0.97$ ) or anxiety ( $F(1, 122) = 0.37$ ,  $p=0.54$ ) level between patients reporting low or adequate structural social support.

Multiple regression analysis was conducted using either Time 4 BDI depression score or HADS- A anxiety score as the dependent variable with structural social support (SNI score), age, gender, employment status, marital status, GRACE score, deprivation and Time 2 BDI or HADS-A score as the independent variables. Using depression as the dependent variable, the model (Table 6.48) explained a significant proportion of variance in depression scores ( $R^2=0.47$ ,  $F(8, 110) = 12.00$ ,  $p<0.05$ ) with Time 2 BDI score being the only and gender being the only significant predictors. Running the regression without Time 2 BDI score produced a non-significant model with no new predictors identified. Structural social support did not emerge as significant in either model.

**Table 6.48 Structural social support at Time 2 as a predictor of depression at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-8.53	-18.71 – 1.66		-1.66	0.100
T2 SNI score	-0.08	-0.63 – 0.80	0.02	0.23	0.822
Age	0.05	-0.14- 0.24	0.07	0.51	0.611
Gender*	3.89	0.84 – 6.93	0.19	2.53	0.013
Marital status	-0.72	-3.30 – 1.86	-0.05	-0.55	0.582
Employment status	1.41	-1.39 – 4.21	0.10	1.00	0.321
GRACE score	0.02	-0.06 – 0.09	0.07	0.52	0.603
Deprivation	-1.33	-3.30 – 0.65	-0.10	-1.33	0.186
Time 2 Depression score*	1.00	0.79 – 1.21	0.69	9.33	0.001

\* Significant independent predictor

Using anxiety as the dependent variable, the model (Table 6.49) explained a significant proportion of variance in anxiety scores ( $R^2=0.45$ ,  $F(8, 110) = 11.30$ ,  $p<0.05$ ) with Time 2 anxiety score, gender and deprivation identified as the only significant independent predictors. Repeating the regression without Time 2 anxiety score produced a non-significant model with no new predictors. None of the variables included in either depression or anxiety regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.49 Structural social support at Time 2 as a predictor of anxiety at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-2.05	-7.39 – 3.29		-0.76	0.449
T2 SNI score	-0.24	-0.63 – 0.15	-0.10	-1.23	0.222
Age	0.00	-0.10 - 0.11	0.01	0.08	0.934
Gender*	2.53	0.87 – 4.19	0.23	3.02	0.003
Marital status	-0.41	-1.82 – 1.00	-0.05	-0.57	0.569
Employment status	0.69	-0.81 – 2.18	0.09	0.91	0.363
GRACE score	0.01	-0.03 – 0.05	0.07	0.53	0.599
Deprivation*	-1.13	-2.24 – 0.02	-0.17	-2.01	0.047
Time 2 anxiety score*	0.72	0.55 – 0.88	0.65	8.51	0.001

\* Significant independent predictor

Mean structural social support score was found to be significantly lower in patients scoring above the threshold for anxiety compared with those scoring below. No significant differences in structural support score were noted between the depressed and non-depressed groups ( $F(1, 121) = 0.48, p=0.48$ ), nor between the anxious and non-anxious groups ( $F(1, 122) = 0.04, p=0.84$ ) (Table 6.50).

**Table 6.50 Mean structural social support (SNI) at Time 2 score SNI score by depression and anxiety status at Time 4**

	Depressed	Non-depressed	Anxious	Non-anxious
<b>Mean SNI score (SD)</b>	4.39 (1.62)	4.15 (1.60)	4.13 (1.69)	4.21 (1.59)
<b>N (%)</b>	28 (23)	95 (77)	23 (19)	101 (81)

Logistic regression was also performed to determine the influence of structural social support on the likelihood that patients would report depression above the cut off threshold at Time 4. The model contained five categorical independent variables (gender, marital status, employment status, deprivation level and either depression or anxiety status at Time 2) and three continuous independent variables (Time 2 structural social support, age and GRACE score) with either anxiety status or depression status at Time 3 entered as the dependent variable. For depression status, the full model (Table 6.51) was statistically significant ( $\chi^2(9, 119) = 28.81, p < 0.05$ ) and able to distinguish between patients who did and did not report depression above the threshold. The model explained 32.4% of the variance in depression status and correctly classified 82.9% of cases. The largest significant predictor was Time 2 depression status recording an odds ratio of 20.36. Gender was also found to be a significant predictor with an odds ratio of 6.19. Marital status made a borderline significant contribution to the model with an odds ratio of 4.15 ( $p=0.059$ ). Structural social support did not make an independent contribution to depression scores. The model was re-run excluding Time 2 depression status and was no longer significant with no new predictors identified.

**Table 6.51 Logistic regression predicting likelihood of depression at Time 4**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Structural social support	<i>Score increase</i>	1.24	0.89 to 1.73	0.21
Age	<i>Annual increase</i>	0.97	0.88 to 1.05	0.43
Gender*	<i>Male</i>	1		
	<i>Female</i>	6.19	1.59 to 24.04	0.008
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	1.40	0.30 to 6.49	0.67
	<i>High</i>	1.48	0.22 to 10.21	0.69
Marital status	<i>Married</i>	1		
	<i>Not married</i>	4.16	0.37 to 18.18	0.059
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.02	0.29 to 3.57	0.98
GRACE score	<i>Score increase</i>	1.03	0.99 to 1.07	0.10
Time 2 Depression score*	<i>Depressed</i>	1		
	<i>Not depressed</i>	20.36	4.48 to 92.58	0.001

\*Significant independent predictors

For anxiety status, the full model (Table 6.52) was statistically significant ( $X^2(9, 119) = 30.07$ ,  $p < 0.05$ ) and able to distinguish between patients who did and did not report anxiety above the threshold. The model explained 35.7% of the variance in anxiety status and correctly classified 83.2% of cases. Anxiety status at Time 2 was the main significant predictor with an odds ratio of 16.13. Gender was also found to make a significant contribution to the model with an odds ratio of 5.77. Structural social support did not make a significant independent prediction regarding anxiety status. The model was no longer significant with the omission of Time 2 anxiety status no new predictors identified.

**Table 6.52 Logistic regression predicting likelihood of anxiety at Time 4**

Variable	Categories	Adjusted odds ratio	95% C.I.	P
T2 Structural social support	<i>Score decrease</i>	1.25	0.81 to 1.69	0.39
Age	<i>Annual increase</i>	0.99	0.90 to 1.09	0.90
Gender*	<i>Male</i>	1	0.39 to 24.03	0.016
	<i>Female</i>	5.77		
Social deprivation	<i>Low</i>	1	0.26 to 7.29	0.72
	<i>Intermediate</i>	1.36		
	<i>High</i>	4.11		
Marital status	<i>Married</i>	1	0.25 to 5.10	0.88
	<i>Not married</i>	1.13		
Employment	<i>Employed</i>	1	0.67 to 12.25	0.16
	<i>Not employed</i>	2.86		
GRACE score	<i>Score increase</i>	1.02	0.98 to 1.06	0.33
Time 2 Anxiety score*	<i>Anxious</i>	1	4.48 to 58.03	0.001
	<i>Not anxious</i>	16.13		

\*Significant independent predictors

In summary, structural social support was not independently predictive of depression or anxiety scores at Time 4. These findings suggest that the level of structural support reported by patients at Time 2 was not associated with their risk of psychological disturbance 12 months following their admission for ACS. Patients who reported distress at Time 2, and female patients were most likely to experience elevated distress at Time 4.

### 6.2.7 Overall summary: Social support and psychological distress after ACS

The findings discussed in this section reveal that social support contributes to the experience of psychological distress and the key findings from my analyses are summarised in Table 6.53.

**Table 6.53 Summary of key findings: Social support and psychological distress**

	Time 2		Time 3		Time 4	
	Functional	Structural	Functional	Structural	Functional	Structural
<b>T2 Distress</b>						
Anxiety	<b>SIG</b>	NON SIG	-	-	-	-
Depression	<b>Near SIG</b>	NON SIG	-	-	-	-
<b>T3 Distress</b>						
Anxiety	<b>SIG</b>	NON SIG	<b>SIG</b>	NON SIG	-	-
Depression	NON SIG	NONSIG	<b>SIG</b>	NON SIG	-	-
<b>T4 Distress</b>						
Anxiety	<b>SIG</b>	NON SIG	-	-	<b>SIG</b>	NON SIG
Depression	NON SIG	NON SIG	-	-	<b>SIG</b>	NON SIG

Key: SIG = significant association (p<0.05), Near SIG = near significant association (p=0.051-0.07), NON SIG= no significant association (p>0.071)

At Time 2, there was a clear association between functional social support and the occurrence of anxiety. There was also a near significant association between functional social support and depressive symptomatology. Patients who reported low functional social support at Time 2 were more likely to report higher distress at Time 2. The association between Time 2 functional social support and anxiety was also found to persist at Time 3 with patients who reported lower Time 2 functional social support more likely to report anxiety symptomatology and to report anxiety above the threshold than patients reporting higher functional support at Time 2. No significant relationship was found between Time 2 functional social support and Time 3 depression scores. The relationship between Time 2 functional social support and anxiety also persisted at Time 4. Patients reporting low functional social support at Time 2 were significantly more likely to report anxiety above the threshold at Time 2. No significant relationship was found between Time 2 functional social support and Time 4 depression. These findings provide support for my hypothesis that low social support reported shortly after ACS would be predictive of both short and long term

anxiety. The findings also support the notion that low social support at Time 2 would be associated with higher risk of both depression and anxiety at Time 2. However, the data did not support a persistent longitudinal relationship between Time 2 functional social support and depression at Time 3 and Time 4.

The results of the cross sectional analyses revealed significant associations between functional social support and distress at Time 3, and also between functional social support and distress at Time 4. The significant cross sectional associations between functional social support and depression examined within the context of the lack of predictive efficacy demonstrated by Time 2 functional social support with regard to depression does suggest that depressed patients may be more liable to make negative appraisals about their levels of social support, and subsequently current emotional state may be the influencing factor motivating these associations. However, this does not appear to be the case for the occurrence of anxiety as the longitudinal associations between Time 2 functional social support and Time 3/Time 4 anxiety were significant illustrating a more persistent relationship and a causal role for functional social support in the occurrence of anxiety. Structural social support at Time 2 was not independently associated with or predictive of distress at Time 2, Time 3 or Time 4 which does not provide support my hypothesis that lower structural social support would be predictive of both short and long term distress.

## **6.3 Chapter discussion**

### **6.3.1 Psychological distress following ACS**

The experience of psychological distress following ACS was common with approximately a quarter of patients reporting significant distress at each assessment. Mean BDI and HADS-A scores were not particularly elevated, however, the negative prognostic impact of even mildly elevated depression as measured on the BDI has been demonstrated in previous research (Lesperance, Frasure-Smith, Talajic, & Bourassa, 2002). A moderate proportion of patients reported elevated levels of depression and anxiety in the immediate weeks following

their ACS which persisted over the 12 month follow up. Anxiety levels remained fairly stable over time suggesting persistent anxiety which has been shown to have a particularly deleterious impact on post ACS prognosis (Moser et al., 2011). Depression levels actually increased between Time 2 and Time 4 suggesting that the negative affective impact of an ACS may increase during the first year. Previous research has also identified increasing depression over the first year post ACS (Kaptein, de, van den Brink, & Korf, 2006; Lane, Carroll, Ring, Beevers, & Lip, 2002). This increase in depression over time may be partially explained by the dual nature of ACS; that it is both an acute and chronic illness. The initial shock of the potentially life threatening nature of the acute MI is followed by a period of understanding and coming to terms with the lifestyle changes and chronic nature of their heart disease which may have substantial impact on the patient's everyday life. For example, the patient may not be able to engage in activities they enjoy because they do not fit in with their new lifestyle i.e. activities where they used to smoke or drink. Qualitative research suggests that post MI patients do report significant difficulty in integrating lifestyle recommendations into their normal life. Engaging in these lifestyle changes has been described as a continual reminder of their MI which provokes persistent uncertainty (Gregory, Bostock, & Backett-Milburn, 2006). The depressogenic facets of ACS and the theoretical perspectives that may underlie this association are discussed in detail by Davidson, Rieckmann, & Lesperance, (2004).

The Time 2 prevalence of depression in the TRACE sample (19.3%) was slightly lower than the prevalence rates reported by other studies using the BDI to classify depression in MI patients. In a review, Thombs et al, (2006) identified that approximately 31% (range 20 – 37%) of patients exceeded the cut-off of  $\geq 10$  for significant depressive symptoms on the BDI during hospitalisation for MI. Our prevalence rates refer to depression assessed at an average of 21 days post admission for MI rather than during hospitalisation which may account for the samples lower prevalence of depression; however, Lauzon et al, (2003) also reported a prevalence of 39% of patients exceeding the BDI cut-off at 30 days post

admission for MI which suggests that our sample did have slightly lower prevalence of depressive symptomatology. Recent research using other depression measures has also begun to identify a lower prevalence of post ACS depression than has been previously found which has been attributed to the continually improving prognosis and medical advances following ACS (Hanssen, Nordrehaug, Eide, Bjelland, & Rokne, 2009). The prevalence of anxiety within the sample (23.8%) was similar to that found in other studies of ACS patients using HADS which estimate prevalence of significant anxiety between 19 – 31% (Hanssen et al., 2009; Lane et al., 2002). Depression and anxiety were highly comorbid at this Time 2 assessment with 34 patients exceeding the cut-off for significant symptomatology on both the BDI and the HADS-A and this comorbidity persisted at Time 3 and 4. Comorbidity of depression and anxiety is common in post ACS patients (Lane et al., 2002). The persistence of psychological distress at 12 months following ACS observed in the TRACE sample has been demonstrated in other studies of ACS patients revealing the chronic nature of anxiety and depression following ACS (Thombs et al., 2006; Huffman, Celano, & Januzzi, 2010). Persistent comorbid depression and anxiety has been found to be particularly detrimental to post ACS recovery (Doering et al., 2010). A number of demographic factors were identified that increased risk of depression and anxiety in the TRACE sample. In particular, younger, female, unemployed patients, patients with a prior history of depression, and more deprived patients were the most at risk for distress at some point during the following year. The occurrence of distress at Time 2 was a strong predictor of distress at Time 3 and Time 4 indicating the significance of early psychological reaction to their ACS to long term psychological adjustment. Psychological response may also be influenced by other factors that were not explored in this study, particularly the clinical environment, the nature of the acute treatment and the level of information and communication with staff. For example, Oterhals, Hanestad, Eide, & Hanssen, (2006) identified that the sufficiency of information received during in hospital treatment for ACS influences patient experience and satisfaction with their healthcare.

### **6.3.2 Social support and psychological distress after ACS**

A central pathway through which social support may influence recovery following ACS is via the experience of psychological distress. Patients who experience significant depression and anxiety following ACS are much more likely to suffer greater morbidity and have a higher risk of mortality than patients who do not report significant distress (Roest, Martens, de Jonge, & Denollet, 2010; Ziegelstein, 2001; Kaptein et al., 2006). Previous research suggests that low social support increases the risk of anxiety and depression in a variety of different populations (Cohen & Wills, 1985; Kawachi & Berkman, 2001). Social support has also been found to have a direct etiologic role in CHD as well as influencing morbidity and mortality post ACS (Lett et al., 2005). This collective evidence suggests the presence of a psychological pathway through which social support may influence post ACS recovery and I hypothesised that low social support in the TRACE patients would be associated with more significant and persistent depression and anxiety after ACS. Consistent with this hypothesis, functional social support at Time 2 was found to be significantly associated with anxiety at Time 2 (OR, 1.09; 95% CI, 1.00 – 1.78), and significantly predictive of anxiety at Time 3 (OR, 1.14; 95% CI, 1.01 – 1.29) and Time 4 (OR, 1.15; 1.04 – 1.28). Those patients who reported low functional social support shortly after their admission to hospital for ACS were 14 – 15% more likely to experience significant anxiety symptomatology at 6 months post admission and at 12 months post admission, controlling for gender, age, marital status, ethnicity, employment status, GRACE score, deprivation and Time 2 anxiety status. Functional social support at Time 2 was also found to be significantly associated with continuous measures of anxiety at Time 2 ( $p=0.020$ ) and Time 3 ( $p=0.007$ ), independent of gender, age, marital status, ethnicity, employment status, GRACE score, deprivation and, for Time 3 anxiety, Time 2 anxiety score. Additionally, significant cross sectional relationships were noted between anxiety and functional social support at Time 3 and Time 4. These findings suggest a robust association between functional social support and anxiety (assessed categorically and continuously) following ACS with low functional social support conferring increased risk for anxiety that persisted over the long term.

I also hypothesised that there would be a strong predictive relationship between functional social support and depression; however, the data presented here do not support such a causal relationship. There were significant cross sectional relationships between functional social and depression at Time 3 and Time 4, and a near significant relationship at Time 2 which are comparable to other studies in CHD populations (for example, Frasure-Smith et al., 2000; Holahan, Moos, Holohan, & Brennan, 1995; Brummett et al., 1998) and also to clinical and community populations (Clara, Cox, Enns, Murray, & Torgrudc, 2003). These findings illustrate that low social support and depression are closely allied and reveal that individuals reporting higher depression are more likely to report lower levels of support. However, these findings do not explicate causal direction. There were no significant relationships between Time 2 functional social support and depression at either Time 3 or Time 4. The lack of a longitudinal relationship between social support and depression suggests that, in the TRACE sample, low social support was most likely a corollary of depression whereby depressed patients were more likely to evaluate a lower level of social support than non-depressed individuals. The lack of a longitudinal relationship between social support and depression is contrary to the current research base which has found significant prospective relationships between low social support and depression (Brummett et al., 1998; Lett et al., 2005; Lett et al., 2009; Fontana, Kerns, Rosenberg, & Colonese, 1989).

Lack of structural social support was also hypothesised to confer higher risk of anxiety and depression; however, structural social support did not demonstrate any predictive efficacy with regard to the occurrence of psychological distress, and was not cross sectionally associated with either measure of distress in the TRACE study. This lack of association has been reported by other studies (Lett et al., 2005; Hamalainen et al., 2000). However, other studies have reported cross sectional and prospective relationships between structural social support and distress (Horsten, Mittleman, Wamala, Schenck-Gustafsson, & Orth-Gomer, 2000; Barefoot et al., 2000; Lett et al., 2009). These mixed findings are further compounded

by the considerable heterogeneity in measures used to assess structural social support in cardiac populations (Lett et al., 2005). In the TRACE study, structural social support was moderate and there were very few patients who reported very low levels of structural social support (only one patient (0.6%) reported having no social ties at Time 2) and it may be that the psychopathological hazard of low structural social support only arises at a very low level (i.e. total social isolation). Thus, our conceptualisation of structural social support as a continuum of risk (and as a categorical risk of low versus adequate) may not have adequately captured the threshold effect on depression of social isolation versus adequate social support.

Overall, the inverse relationship between functional social support and anxiety over the short and long term is consistent with my hypothesis and reveals the presence of a psychological pathway through which lack of functional social support may negatively impact upon post ACS recovery. Low functional social support reported shortly after admission for ACS was significantly associated with concurrent anxiety level and predictive of anxiety at both 6 and 12 months following admission. This is an important finding as anxiety is highly prevalent in post ACS patients and there is robust and growing evidence that post ACS anxiety is associated with increased morbidity and mortality. Functional social support was also cross sectionally associated with depression at Time 2, 3 and 4 which is consistent with current research. However, functional social support was not prospectively associated with Time 3 or Time 4 depression which is contrary to the general research consensus. Structural social support was not found to be associated or predictive of anxiety or depression which was contrary to my hypothesis. There have been mixed research findings regarding the relationship between structural social support and distress, and this lack of effect has been reported previously in other studies and suggests that measures of structural social support may be less useful in predicting distress in cardiac patients. However, before the role of globally assessed social support in depression can be discounted, it is important to explore the potential reasons why social support was not useful in predicting depression in the

TRACE study and these will be discussed within the thesis discussion section in Chapter 9. It is clear that the relationship between social support and distress is complex and multifaceted. Our findings provide support for a potential psychological pathway between functional social support, anxiety and post ACS recovery but not for a pathway between social support, depression and recovery.

### **6.11 Chapter summary**

Depression and anxiety were prevalent amongst the TRACE patients with 19.3% of patients reporting above threshold depression and 23.8% patients reporting above threshold anxiety in the early weeks following ACS. Anxiety rates remained elevated and fairly stable over the follow up period; however, depression rates increased over the follow up reaching 26.6% at Time 4. Functional social support was found to play an important cross sectional and longitudinal role in the occurrence and severity of anxiety symptomatology analysed in both continuous and categorical form. This is an important finding within the social support and cardiac health literature illustrating the close alliance between low social support and the experience of anxiety following ACS. Functional social support was also found to have limited longitudinal impact upon depressive symptoms. Similarly, structural social support was found to have no cross sectional or longitudinal effect on measures of anxiety and depression at each assessment point suggesting that functional social support may be more important in the psychological recovery following ACS. The longitudinal assessment of patient psychological recovery over a year was a particular strength of this study as it provides a broader picture of the process of psychological rehabilitation beginning with the initial response to the ACS and following the gradual long term adjustment to their cardiac condition. Furthermore, the identification of a longitudinal significant association between social support and anxiety highlights the robustness of the role of social support in post ACS anxiety which is pertinent in the context of the research identifying a particularly adverse prognostic role for anxiety in post ACS recovery.

## CHAPTER 7 TRACE STUDY RESULTS PART 3 & 4

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### **Part 3: Quality of life following ACS and the relationship between social support and post ACS quality of life.**

The quality of life reported by patients at Time 2, 3 and 4 is described. This is followed by an appraisal of the relationship between patient social support and their quality of life at Time 2, Time 3 and Time 4. In addition, Part 4 discusses the association between patient social support and HRV at Time 2. The chapter closes with a discussion of the results presented.

#### **7.1 Quality of Life at Time 2, 3 and 4**

##### **7.1.1 Analytic dataset**

Of the 226 patients completing the Time 2 assessment, 203 had valid data for the measure of quality of life at Time 2. At Time 3, of the 200 patients completing the telephone interview, 146 had valid data for the quality of life assessment. At Time 4, of the 176 who completed the telephone interview, 147 returned data for the quality of life measure. The difference in numbers is because the quality of life measures were part of a postal questionnaire, and not all the participants completing the telephone follow up interviews returned this set of measures despite repeated follow up attempts. The quality of life and subsequent adjustment of patients following ACS was assessed using the MOS SF-12. These measures were assessed at Time 2, 3 and 4 and have been described in detail in Chapter 4.

##### **7.1.2 Quality of life at Time 2**

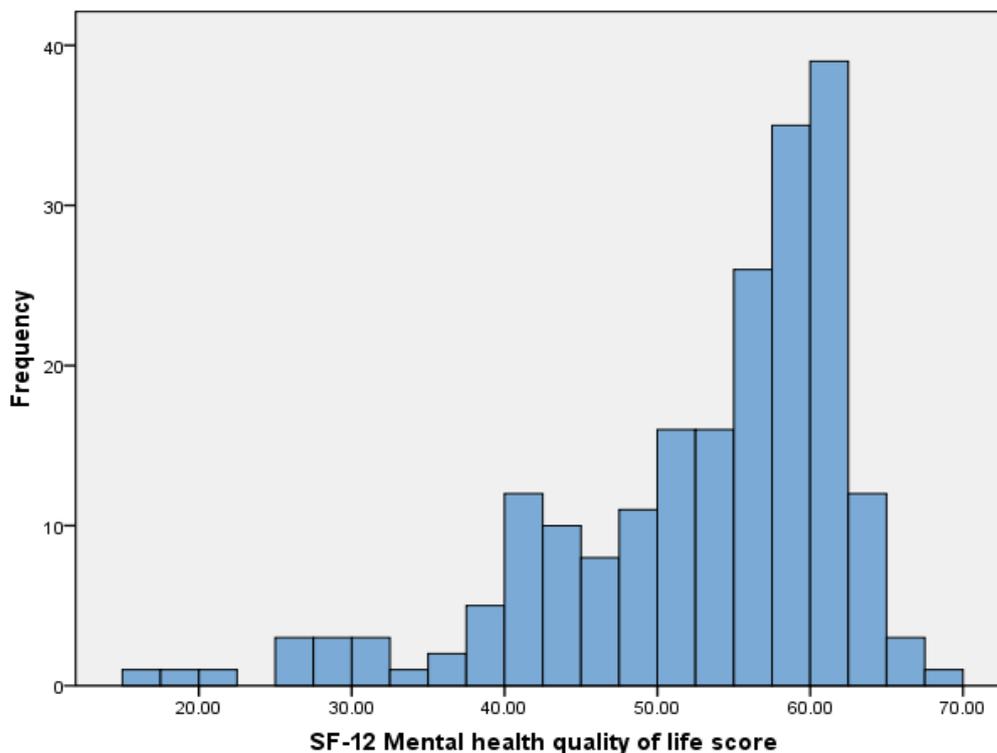
The mean scores for the SF-12 PCS and MCS at Time 2 are depicted in Table 7.1. The scores indicate slightly below average physical health and average mental health quality of life. The low physical health quality of life is unsurprising in the context of the recent health crisis.

**Table 7.1 Mean SF-12 quality of life scores at Time 2**

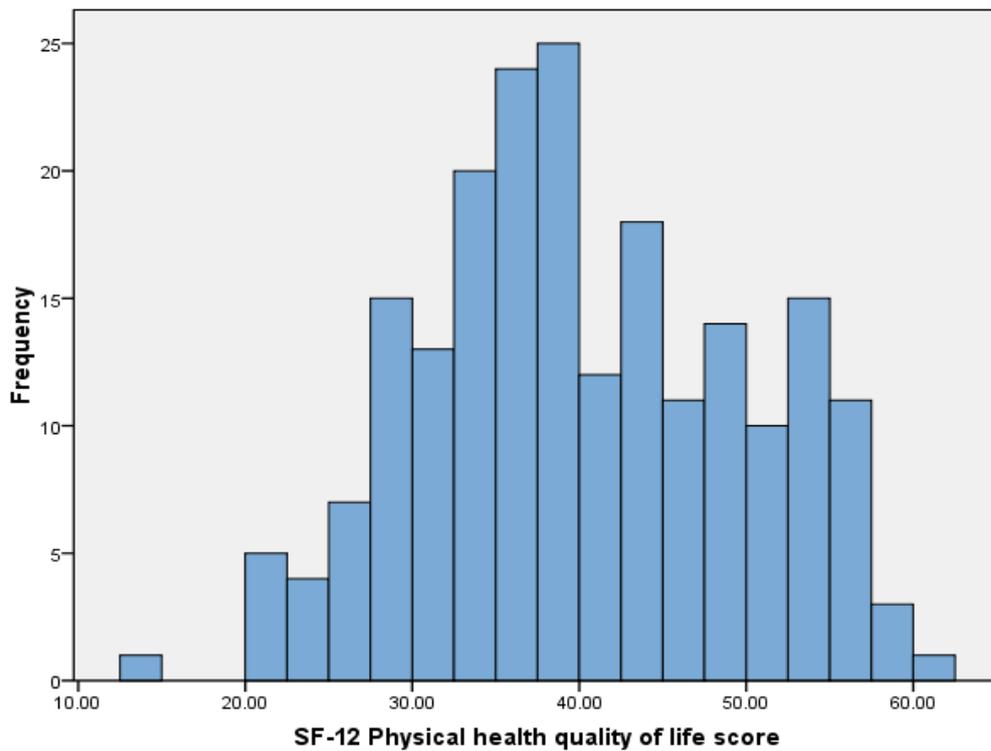
	SF-12 PCS	SF-12 MCS
<b>Mean (SD)</b>	40.20 (9.56)	53.07 (9.89)
<b>Range</b>	13.68 – 61.19	15.17 – 67.55
<b>N</b>	209	209

The score frequency and distribution of SF-12 PCS and MCS at Time 2 are presented in Figures 7.1 and 7.2. The MCS were highly positively skewed as is typical of this measure, indicating that most scores fell in the upper range suggestive of generally good mental health related quality of life. There were a number of outliers; however, the 5% trimmed mean (53.95) was not different from the mean suggesting no undue influence from these outliers. The PCS were normally distributed with no outliers suggesting that the majority of scores fell in the intermediate range.

**Figure 7.1 Score distribution for SF-12 MCS at Time 2**



**Figure 7.2 Score distribution for SF-12 PCS at Time 2**



In order to determine the influence of demographic and clinical variables collected at Time 1 on SF-12 scores at Time 2, a series of one way between group's analyses of covariance were conducted. Continuous Time 2 SF-12 PCS and MCS were the dependent variables, age and gender were entered as covariates and the independent variables were ethnicity (white/non-white), marital status (married/unmarried), employment status (employed/not employed), educational status (basic/secondary/degree), deprivation index (low/moderate/high), the presence of diabetes (yes/no), prior heart disease (yes/no) and GRACE score (low/moderate/high). Poorer physical quality of life (lower PCS) was significantly associated with higher deprivation levels ( $F(1, 207) = 3.42, p < 0.05, \text{partial } \eta^2 = 0.03$ ) and poorer mental health quality of life ( $F(1, 201) = 7.00, p < 0.05, \text{partial } \eta^2 = 0.07$ ). Unmarried patients also reported significantly worse mental health quality of life at time 2 ( $F(1, 205) = 6.08, p < 0.05, \text{partial } \eta^2 = 0.03$ ).

Based on the findings of these ANCOVA analyses, a multiple regression analysis was conducted using Time 2 SF-12 PCS as the dependent variable and age, gender and

deprivation as the independent variables. The model explained a significant proportion of variance in anxiety scores ( $R^2=0.05$ ,  $F(3, 205) = 3.70.61$ ,  $p<0.05$ ) with age and deprivation being significant independent predictors (Table 7.2).

**Table 7.2 Demographic and clinical predictors of SF-12 PCS at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	49.50	42.11 – 56.89		13.21	0.001
Age*	-0.12	-0.23 - -0.01	-0.15	-2.06	0.041
Gender	-0.98	-4.89 – 2.94	-0.04	-0.49	0.623
Deprivation*	-2.51	-4.50 – 0.53	-0.18	-2.50	0.013

\* Significant independent predictor

A similar multiple regression analysis was completed using S-12 MCS as the dependent variable and age, gender, marital status and deprivation the independent variables. The model explained a significant proportion of variance in MCS ( $R^2=0.09$ ,  $F(4, 205) = 5.14$ ,  $p<0.05$ ) with deprivation being the only significant independent predictor (Table 7.3). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.3 Demographic and clinical predictors of SF-12 MCS at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	48.91	41.37 – 56.46		12.78	0.001
Age	0.10	-0.02 – 0.22	0.12	1.73	0.085
Gender	-0.06	-4.09 – 3.98	-0.00	-0.03	0.979
Marital status	-2.69	-5.83 – 0.46	-0.12	-1.68	0.094
Deprivation*	-3.08	-5.18 – -0.99	-0.21	-2.90	0.004

\* Significant independent predictor

Correlational analysis revealed that PCS and MCS were significantly negatively correlated with BDI depression and HADS anxiety scores (Table 7.4) suggesting that at Time 2 the experience of psychological distress and poor health quality of life are closely allied.

**Table 7.4 Correlations between psychological distress and quality of life measures at Time 2**

	T2 SF-12 PCS	T2 SF-12 MCS
T2 BDI depression	-.255*	-.712*
T2 HADS anxiety	-.251*	-.674*

\*correlation is significant  $p < 0.001$

The findings from the Time 2 analyses indicate slightly below average PCS scores and average MCS scores that are comparable with other studies of ACS patients. Deprivation level offered the greatest predictive efficacy with regard to both physical and mental health quality of life at Time 2. This finding illustrates the pervasive impact of SES on health as the negative impact of deprivation on quality of life was independent of the clinical features of ACS. Thus, regardless of the severity of their ACS, patients living in high deprivation were more likely to report poor health related quality of life. Age was also shown to make an independent contribution to physical quality of life with younger patients reporting better physical quality of life. These preliminary analyses suggest that deprivation and age will be important covariates in the analysis of the impact of social support on quality of life at Time 2.

### 7.1.3 Quality of life at Time 3

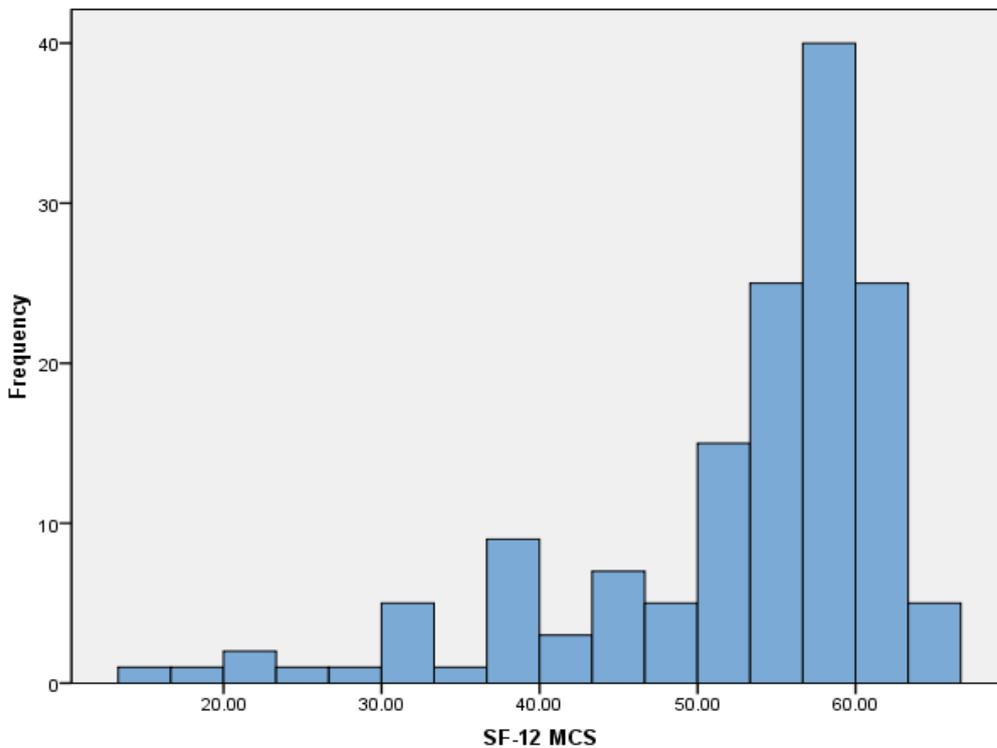
The mean scores for the SF-12 PCS and MCS at Time 3 (and Time 2 for comparison) are depicted in Table 7.5. The Time 3 scores indicate slightly below average physical health and average mental health quality of life. There was a significant increase in SF-12 PCS scores ( $t(123) = -4.53, p < 0.05$ ) between Time 2 and Time 3 indicating a general improvement in physical health quality of life within the 6 months following ACS. There was no significant change in SF-12 MCS score between Time 2 and Time 3. There was no significant correlation between SF-12 MCS and PCS scores at Time 3.

**Table 7.5 Mean SF-12 scores at Time 2 and Time 3**

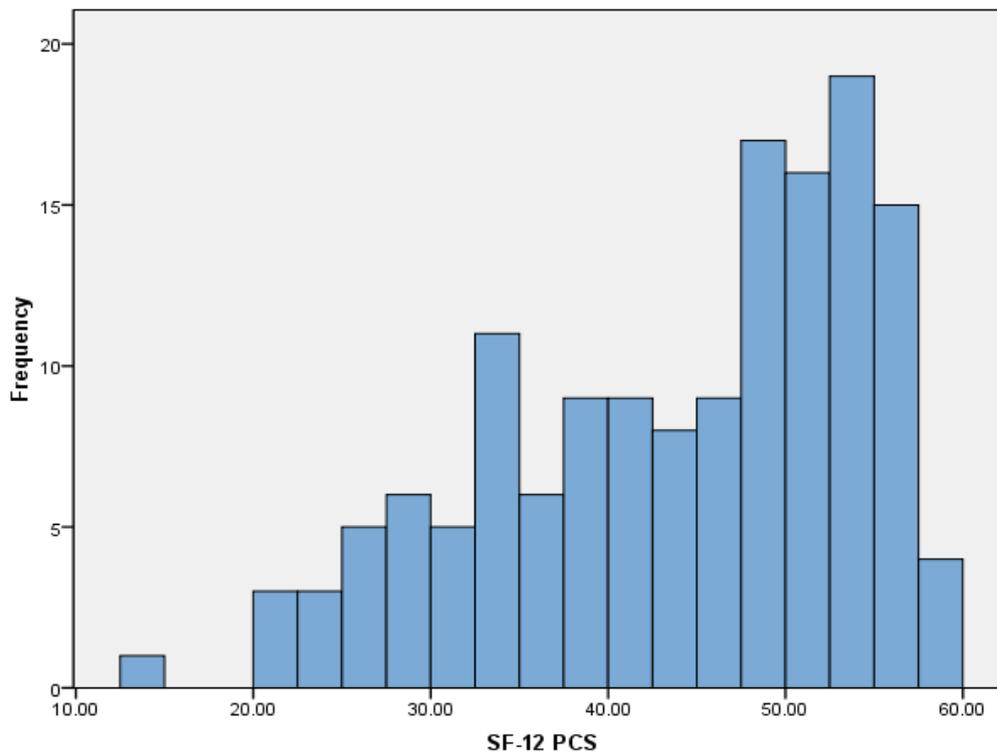
	Time 2		Time 3	
	SF-12 PCS	SF-12 MCS	SF-12 PCS	SF-12 MCS
<b>Mean (SD)</b>	40.20 (9.56)	53.07 (9.89)	44.06 (10.23)	52.72 (10.20)
<b>Range</b>	13.68 – 61.19	15.17 – 67.55	14.21 – 58.96	15.39 – 65.48
<b>N</b>	209	209	146	146

The score frequency and distribution of SF-12 PCS and MCS at Time 3 are presented in Figures 7.3 and 7.4. The MCS score distribution was similar to that observed at Time 2 with the MCS scores being highly positively skewed with a number of outliers. The 5% trimmed mean (53.72) was not substantially different from the mean indicative of no undue influence from these outliers. The PCS scores were also slightly positively skewed; however, no outliers were identified.

**Figure 7.3 Score distribution for SF-12 MCS at Time 3**



**Figure 7.4 Score distribution for SF-12 PCS at Time 3**



The same ANCOVA analysis utilised for the Time 2 SF-12 data was run using SF-12 PCS and MCS as the dependent variables. ANCOVA analysis revealed that patients with a history of CHD had significantly lower physical quality of life than patients with no CHD history ( $F(1, 142) = 9.01, p < 0.05, \text{partial } \eta^2 = 0.06$ ). Patients with higher GRACE scores at Time 1 (indicative of more severe ACS) were also significantly more likely to report poorer physical quality of life than patients with lower GRACE scores ( $F(2, 141) = 3.31, p < 0.05, \text{partial } \eta^2 = 0.04$ ). White patients reported better mental health quality of life than non-white patients ( $F(1, 142) = 4.13, p < 0.05, \text{partial } \eta^2 = 0.03$ ). Patients who were in employment at Time 1 also reported better mental health quality of life than non-employed patients ( $F(1, 141) = 5.44, p < 0.05, \text{partial } \eta^2 = 0.04$ ). Deprivation was also found to be important with more deprived patients indicating worse physical quality of life ( $F(2, 138) = 3.91, p < 0.05, \text{partial } \eta^2 = 0.05$ ) and mental quality of life ( $F(2, 138) = 3.67, p < 0.05, \text{partial } \eta^2 = 0.05$ ) than less deprived patients.

Based on the findings of these ANCOVA analyses, a multiple regression analysis was conducted using Time 3 SF-12 PCS as the dependent variable and age, gender and deprivation, previous CHD, GRACE score and Time 2 PCS score as the independent variables. The model explained a significant proportion of variance in PCS scores ( $R^2=0.24$ ,  $F(6, 114) = 5.93$ ,  $p<0.05$ ) with Time 2 PCS score being the only significant independent predictors (Table 7.6). The model remained significant with the omission of Time 2 PCS and patient history of previous CHD was a significant independent predictor ( $\beta=-4.77$ ,  $p<0.05$ ). None of the variables included in this model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.6 Demographic and clinical predictors of SF-12 PCS at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	42.45	29.71 – 55.18		6.60	0.001
Age	-0.02	-0.28 - 0.24	-0.02	-0.17	0.865
Gender	1.33	-4.38 – 7.04	0.04	0.46	0.645
Previous CHD	-2.98	-7.18 – 1.23	-0.12	-1.40	0.164
GRACE score	-0.10	-0.22 – 0.02	-0.25	-1.70	0.092
Deprivation	-2.08	-5.44 – 1.23	-0.11	-1.23	0.222
T2 PCS*	0.31	0.13 – 0.49	0.30	3.46	0.001

\* Significant independent predictor

Multiple regression analysis was also conducted using Time 3 SF-12 MCS as the dependent variable and age, gender, ethnicity, employment, deprivation and Time 2 MCS score as the independent variables. The model explained a significant proportion of variance in MCS scores ( $R^2=0.48$ ,  $F(6, 113) = 17.70$ ,  $p<0.05$ ) with age and Time 2 MCS being significant independent predictors. The model remained significant with the removal of Time 2 MCS score with age ( $\beta=0.32$ ,  $p<0.05$ ) and employment status ( $\beta=4.54$ ,  $p<0.05$ ) identified as significant independent predictors (Table 7.7). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.7 Demographic and clinical predictors of SF-12 MCS at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	2.48	-12.50 – 17.45		0.33	0.744
Age*	0.22	0.05 – 0.39	0.23	2.51	0.014
Gender	-0.78	-5.46 – 3.91	-0.02	-0.33	0.744
Ethnicity	-1.41	-5.62 – 2.80	-0.05	-0.67	0.507
Employment	2.94	-0.74 – 6.61	0.15	1.59	0.116
Deprivation	0.63	-2.16 – 3.41	0.03	0.45	0.657
T2 MCS*	0.68	0.53 – 0.83	0.66	9.06	0.001

\* Significant independent predictor

As identified with the Time 2 scores, correlational analysis revealed that PCS and MCS were significantly negatively correlated with BDI depression and HADS anxiety scores at Time 3 (Table 7.8) indicating the close association between the experience of psychological distress and poor health quality of life.

**Table 7.8 Correlations between psychological distress and quality of life measures at Time 3**

	SF-12 PCS	SF-12 MCS
<b>BDI depression</b>	-.346*	-.806*
<b>HAS anxiety</b>	-.249*	-.758*

\*correlation is significant  $p < 0.001$

Overall, mean physical health quality of life increased between Time 2 and Time 3 which is likely to reflect the improving physical condition and recovery of the patients over the 6 months from their ACS. There was no change in mental health quality of life between Time 2 and Time 3. The findings from the ANCOVA and regression analyses suggest that Time 2 physical health quality of life and previous CHD are the most important predictors of physical quality of life at Time 3. Time 2 mental health quality of life and age were the most significant predictors of mental quality of life at Time 3 with younger patients and patients with better mental health quality of life at Time 2 reporting better mental health quality of life at Time 3.

These preliminary analyses suggest that previous CHD, Time 2 PCS/MCS scores and age will be important covariates in the analysis of the impact of social support on quality of life at Time 3.

#### **7.1.4 Quality of life at Time 4**

The quality of life findings at Time 4 were extremely similar to those reported at Time 2 and Time 3. Subsequently, full descriptive analysis is provided in Appendix VI and a brief summary will be provided in this section.

The Time 4 scores indicated slightly below average physical health and average mental health quality of life. There was a significant increase in SF-12 PCS scores ( $t(126) = -4.72$ ,  $p < 0.05$ ) between Time 2 and Time 4, but not between Time 3 and Time 4 indicating an overall improvement in physical health quality of life within the 12 months following ACS with the majority of this improvement occurring in the first 6 months. There was no significant change in SF-12 MCS score between Time 2 and Time 4, nor between Time 3 and Time 4 suggesting stability over time. The findings from the ANCOVA and regression analyses suggest that Time 2 physical health quality of life ( $\beta = 0.25$ ,  $p < 0.05$ ) and previous CHD ( $\beta = -4.80$ ,  $p < 0.05$ ) are the most important predictors of physical quality of life at Time 4. I also found that Time 2 mental health quality of life was the only significant predictor of mental quality of life at Time 4 ( $\beta = 0.66$ ,  $p < 0.05$ ). Marital status ( $\beta = -0.20$ ,  $p < 0.05$ ) and age ( $\beta = 0.15$ ,  $p = 0.059$ ) also emerged as significant or near significant predictors of mental health quality of life at Time 4 when Time 2 mental health quality of life was not controlled. These factors will be included as covariates in the analysis of social support and quality of life at Time 4.

#### **7.1.5 Summary: Quality of Life after ACS**

Impaired physical quality of life was common in the immediate weeks following ACS and was below the US population normative score of 50. There was an improvement in physical health quality of life over the 12 month follow up period, with the greatest significant

improvement noted in the first six months following ACS. The mental health quality of life scores were within US population normative range and remained stable throughout the follow up period indicative of no significant change. The ANCOVA and regression analyses revealed that a number of demographic and clinical factors influenced quality of life. In particular, age, deprivation and previous CHD were important to physical health quality of life, and age, deprivation, employment status and marital status were important to mental health quality of life. Thus, younger, more deprived, unemployment and unmarried patients were identified as most at risk of experiencing poor mental health quality of life following ACS. These variables also represent important covariates in my analysis of the relationship between social support and quality of life following ACS which is described in detail in the following section.

## **7.2 Functional and structural social support as correlates and predictors of post ACS quality of life**

### **7.2.1 Introduction**

This section aims to examine how both functional and structural social support may also be related to patient quality of life in the 12 months following ACS. The research concerning the relationship between quality of life and social support has been discussed in Chapters 1 and 2 and suggests that higher levels of social support are generally associated with better quality of life in various population groups; although the research is scant in ACS patients. It was hypothesised that higher levels of functional and structural social support would be predictive of higher levels of quality of life at each follow up assessment. This relationship between social support and quality of life will be explored cross sectionally for each of the three follow up assessments individually and also longitudinally for Time 3 and Time 4.

### **7.2.2 Data analysis**

The relationship between social support and quality of life shortly following ACS at Time 2 were examined using multivariate ANOVA and multiple regression analysis. The predictive

efficacy of social support with regard to later term quality of life at Time 3 and Time 4 was explored using multiple regression and logistic regression analysis.

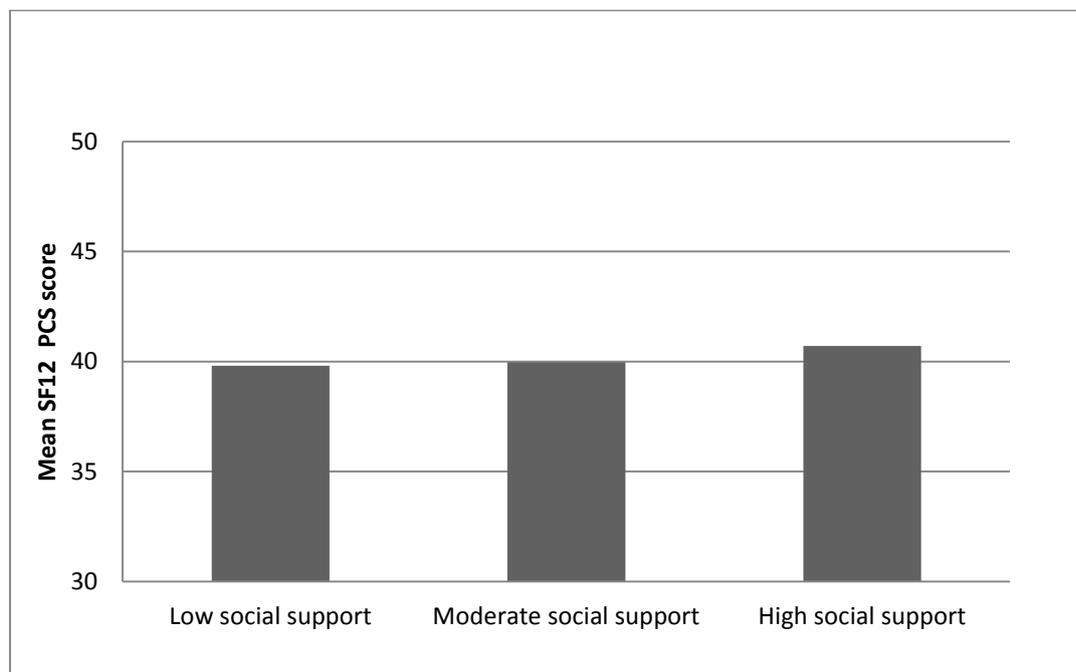
### 7.2.3 Social support and quality of life at Time 2

#### 7.2.3.1 Functional social support and quality of life at Time 2

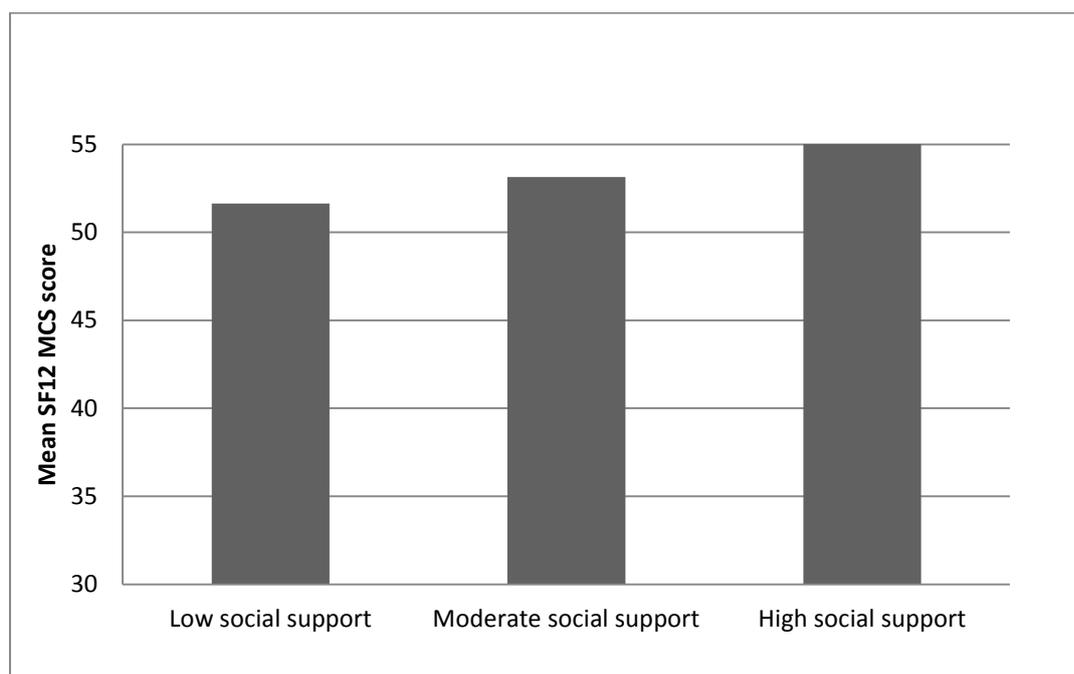
At Time 2, there was a significant positive correlation between MCS score and ESSi score ( $r(155) = 0.19, p < 0.001$ ) suggestive of a modest linear relationship between mental health quality of life and functional social support.

Patients were divided in three social support groups according to their score on the Time 2 ESSi assessment as described before. Mean PCS and MCS scores by functional social support group are depicted in Figures 7.5 and 7.6. There was no significant difference in PCS scores according to level of functional support ( $F(2, 162) = 0.13, p = 0.88$ ). There was a near significant effect of social support on MCS scores ( $F(2, 152) = 2.80, p = 0.064$ ).

**Figure 7.5 Mean PCS score by level of functional social support at Time 2**



**Figure 7.6 Mean MCS by level of functional social support at Time 2**



Multiple regression analysis was conducted using Time 2 PCS or MCS score as the dependent variable with Time 2 functional social support (ESSI score), age, gender, ethnicity, marital status, previous CHD, employment and deprivation as the independent variables. Using Time 2 PCS score as the dependent variable, the model explained only a small proportion of the variance in PCS scores and was not significant ( $R^2=0.09$ ,  $F(8, 142) = 1.64$ ,  $p=0.12$ ) with deprivation emerging as a single significant independent predictor (Table 7.9).

**Table 7.9 Functional social support as a correlate of PCS score at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	35.02	20.02 – 50.01		4.62	0.001
T2 ESSI score	0.07	-0.23 – 0.36	0.04	0.45	0.654
Age	0.03	-0.15- 0.21	0.04	0.34	0.732
Gender	0.22	-4.72 – 5.16	0.01	0.09	0.929
Marital status	1.44	-2.25 – 5.13	0.07	0.77	0.441
Previous CHD	-2.15	-5.92 – 1.62	-0.09	-1.13	0.262
Employment status	3.44	-0.58 – 7.47	0.18	1.69	0.093
Ethnicity	0.69	-3.78 – 5.16	0.03	0.31	0.759
Deprivation*	-3.77	-6.74 – -0.80	-0.22	-2.51	0.013

\* Significant independent predictor

Using Time 2 MCS score as the dependent variable, the model was not significant and only explained a small proportion of variance in MCS scores ( $R^2=0.20$ ,  $F(8, 142) = 1.77$ ,  $p=0.088$ ) with no significant predictors identified (Table 7.10). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.10 Functional social support as a correlate of MCS score at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	41.70	26.94 – 56.45		5.59	0.001
T2 ESSI score	0.21	-0.08 - 0.50	0.12	1.45	0.150
Age	0.12	-0.06 - 0.29	0.14	1.30	0.197
Gender	-0.34	-5.20– 4.53	-0.01	-0.14	0.892
Marital status	-2.58	-6.21 –1.05	-0.12	-1.41	0.162
Ethnicity	-2.84	-7.23 – 1.56	-0.11	-1.28	0.204
Employment status	0.99	-2.97 – 4.95	0.05	0.50	0.622
Previous CHD	1.10	-2.62 – 4.81	0.05	0.58	0.561
Deprivation	-1.27	-4.19 – 1.65	-0.07	-0.86	0.392

\* Significant independent predictor

Functional social support did not make an independent contribution to quality of life scores at Time 2 which suggests that quality of life during the early weeks following ACS was not associated with the functional support perceived by the patient.

### 7.2.3.2 Structural social support and quality of life at Time 2

At Time 2, there was no significant correlation between structural social support (SNI score) and either MCS or PCS scores suggesting no cross sectional association between structural social support measured at Time 2 and quality of life at Time 2. Patients completing the Time 2 assessment were subdivided according to their level of structural social support to form two groups: Low structural social support (1 or fewer people in social network, Mean SNI = 0.92 (0.29), N=12) and adequate structural social support (2 or more people, Mean SNI = 4.24 (1.45), N=153). Patients reporting low structural social support had significantly lower

PCS scores (M=34.49, SD=8.32) indicative of poorer physical health quality of life than those patients reporting adequate (M= 40.56, SD=9.16) structural social support ( $F(1, 154) = 4.55$ ,  $p < 0.05$ ). There was no significant difference in MCS score ( $F(1, 154) = 0.46$ ,  $p = 0.50$ ) between patients reporting low or adequate structural social support.

Multiple regression analysis was conducted using Time 2 PCS or MCS score as the dependent variable with Time 2 structural social support (SNI score), age, gender, marital status, previous CHD, employment and deprivation as the independent variables. Using Time 2 PCS score as the dependent variable, the model was not significant ( $R^2 = 0.09$ ,  $F(7, 144) = 1.92$ ,  $p = 0.07$ ) with deprivation emerging as the only significant independent predictor within the model (Table 7.11).

**Table 7.11 Structural social support as a correlate of PCS score at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	37.13	24.16 – 50.09		5.66	0.001
T2 SNI score	-0.05	-1.00 – 0.90	-0.01	-0.10	0.923
Age	0.03	-0.14– 0.21	0.04	0.37	0.710
Gender	0.10	-4.75 – 4.95	0.00	0.04	0.967
Marital status	1.34	-2.29 – 4.98	0.06	0.73	0.467
Previous CHD	-2.04	-5.74 – 1.67	-0.09	-1.09	0.279
Employment status	3.54	-0.50 – 7.58	0.19	1.73	0.085
Deprivation*	-3.86	-6.76 – -0.96	-0.23	-2.63	0.009

\* Significant independent predictor

Using Time 2 MCS score as the dependent variable, the model was not significant and identified no significant predictors ( $R^2 = 0.09$ ,  $F(7, 144) = 1.94$ ,  $p = 0.07$ ) (Table 7.12). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values. These findings suggest that structural social support was not an independent predictor of quality of life shortly after ACS.

**Table 7.12 Structural social support as a correlate of MCS score at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	46.28	33.17 – 59.38		6.98	0.001
T2 ESSI score	0.67	-0.29 – 1.63	0.12	1.38	0.170
Age	0.12	-0.06 - 0.29	0.14	1.28	0.201
Gender	-1.67	-6.07– 3.73	-0.04	-0.47	0.639
Marital status	-3.07	-6.75 –0.61	-0.15	-1.65	0.101
Employment status	0.01	-4.07 – 4.09	0.00	0.01	0.996
Previous CHD	0.45	-3.30 – 4.91	0.02	0.24	0.814
Deprivation	-1.88	-4.82 – 1.05	-0.11	-1.27	0.207

\* Significant independent predictor

Structural social support was not significantly associated with quality of life scores at Time 2 suggesting that the level of structural support perceived by a patient was not associated with their quality of life early weeks following ACS.

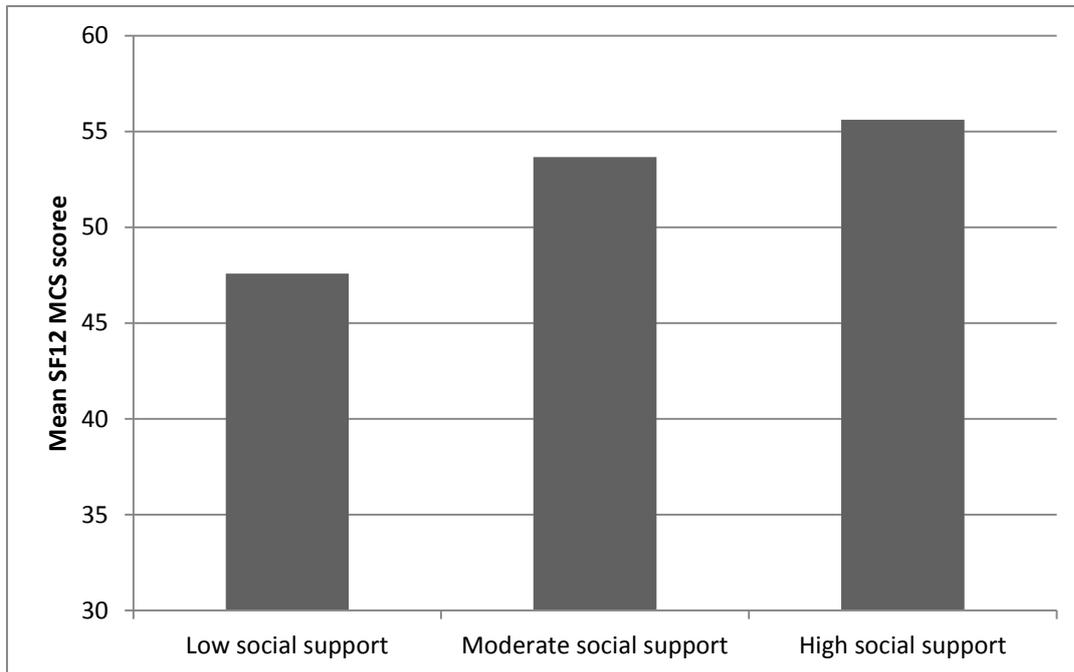
## 7.2.4 Social support and quality of life at Time 3

### 7.2.4.1 Cross sectional analysis: Exploring the association between Time 3 functional social support and Time 3 quality of life

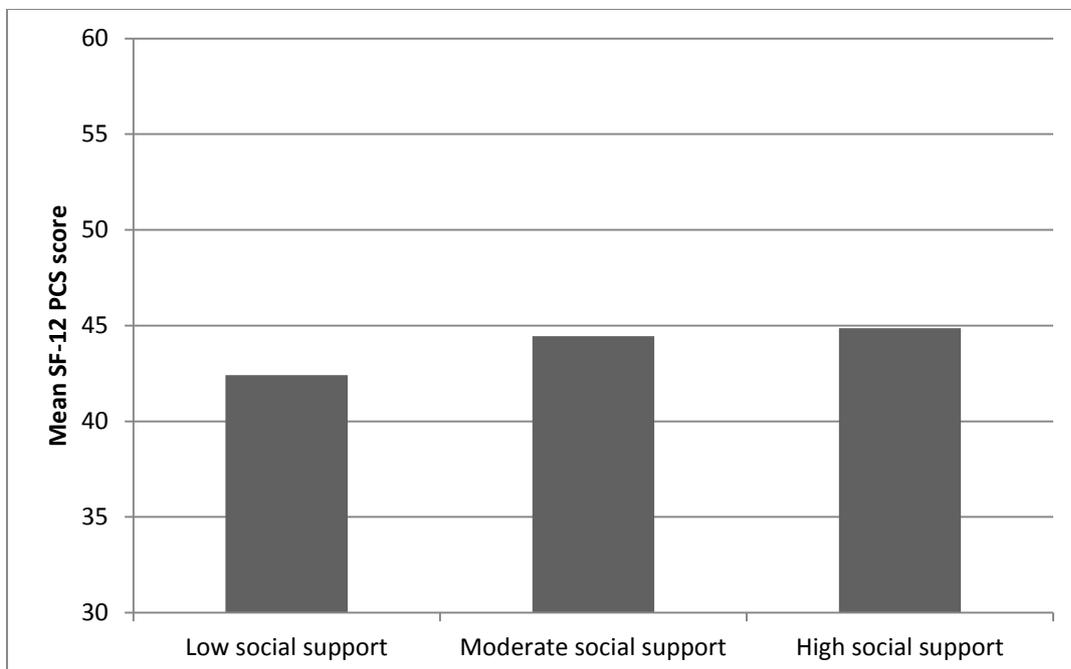
At Time 3, there was a significant positive correlation between SF-12 MCS score and Time 3 ESSI score ( $r(145) = 0.31, p < 0.001$ ) suggesting an association between mental health quality of life and functional social support at Time 3. There was no significant correlation between SF-12 PCS score and ESSI score at Time 3 ( $r(145) = 0.07, p = 0.38$ ).

Patients were divided in three social support groups according to their score on the Time 3 ESSI assessment as described previously. Mean PCS and MCS scores by functional social support group are detailed in Figures 7.7 and 7.8. A gradual increase in MCS scores as levels of functional social support increase was observed which was statistically significant ( $F(2, 141) = 6.84, p < 0.05$ ). No significant differences in PCS scores according to level of functional social support were noted ( $F(2, 141) = 0.62, p = 0.54$ ).

**Figure 7.7 Mean MCS score at Time 3 by level of functional support at Time 3**



**Figure 7.8 Mean PCS score at Time 3 by level of functional social support at Time 3**



Multiple regression analysis was conducted using Time 3 PCS or MCS score as the dependent variable with Time 3 functional social support (ESSI score), age, gender,

ethnicity, marital status, previous CHD, employment, deprivation and Time 2 PCS or MCS score as the independent variables. Using Time 3 PCS score as the dependent variable, the model explained a reasonable proportion of the variance in PCS scores ( $R^2=0.23$ ,  $F(9, 109) = 3.33$ ,  $p<0.05$ ) with age and Time 2 PCS being the only significant independent predictors of depression (Table 7.13). The regression was also re-run omitting Time 2 PCS score to explore whether this variable may be obscuring other findings. The model remained significant and previous CHD emerged as a significant predictor ( $\beta=-0.22$ ,  $p<0.05$ ).

**Table 7.13 Functional social support at Time 3 as a correlate of PCS score at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	38.39	-19.56 – 57.23		4.04	0.000
T3 ESSI score	-0.22	-0.16 – 0.59	0.13	1.12	0.263
Age*	-0.24	-0.46 - 0.03	-0.27	-2.24	0.027
Gender	2.16	-3.80 – 8.12	0.06	0.72	0.474
Marital status	0.77	-4.53 – 6.07	0.03	0.29	0.775
Previous CHD	-4.03	-8.43 – 0.38	-0.16	-1.81	0.073
Employment status	-0.54	-5.24 – 4.16	-0.03	-0.23	0.821
Ethnicity	-1.44	-6.80 – 3.92	-0.05	-0.53	0.595
Deprivation	-1.26	-5.01 – 2.50	-0.06	-0.66	0.508
Time 2 PCS score*	0.35	0.16 – 0.53	0.33	3.66	0.001

\* Significant independent predictor

Using Time 3 MCS score as the dependent variable, the model explained a significant proportion of variance in MCS scores ( $R^2=0.53$ ,  $F(9, 109) = 13.71$ ,  $p<0.05$ ) with functional social support, previous CHD and Time 2 MCS score being the only significant independent predictors (Table 7.14). Age was also a near significant predictor ( $p=0.054$ .) Repeating the regression model with the omission of Time 2 MCS score produced a significant model with functional social support and age emerging as significant predictors, and employment status emerging as a near significant factor ( $\beta=0.94$ ,  $p=0.054$ ). Previous CHD did not emerge as a significant predictor within this model. None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.14 Functional social support at Time 3 as a correlate of MCS score at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-6.61	-22.06 – 9.74		-0.77	0.444
T3 ESSI score*	0.43	0.13 – 0.73	0.26	2.84	0.005
Age	0.17	-0.00 – 0.34	0.18	1.95	0.054
Gender	-0.25	-4.89 – 4.39	-0.01	-0.11	0.916
Marital status	3.00	-1.13 – 7.14	0.13	1.44	0.153
Employment status	2.32	-1.31 – 5.94	0.12	1.27	0.208
Ethnicity	-0.21	-4.40 – 3.99	-0.01	-0.10	0.921
Deprivation	1.55	-1.39 – 4.49	0.08	1.05	0.297
Time 2 MCS score*	0.66	0.51 – 0.81	0.63	8.86	0.001

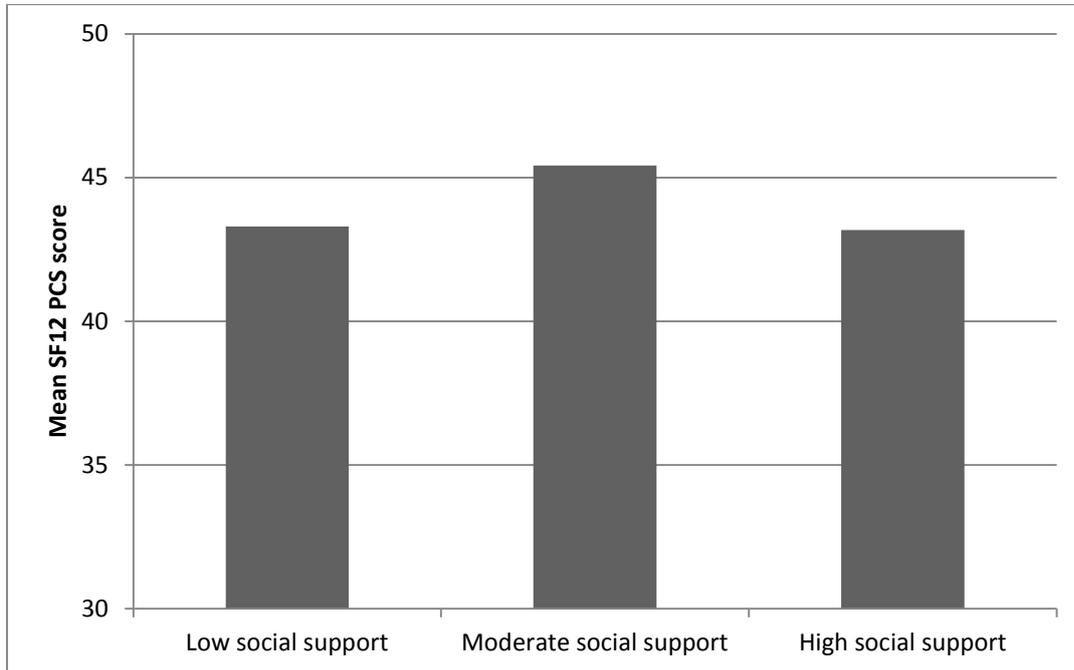
\* Significant independent predictor

#### 7.2.4.2 Longitudinal analysis: Exploring the predictive efficacy of Time 2 social support for Time 3 quality of life

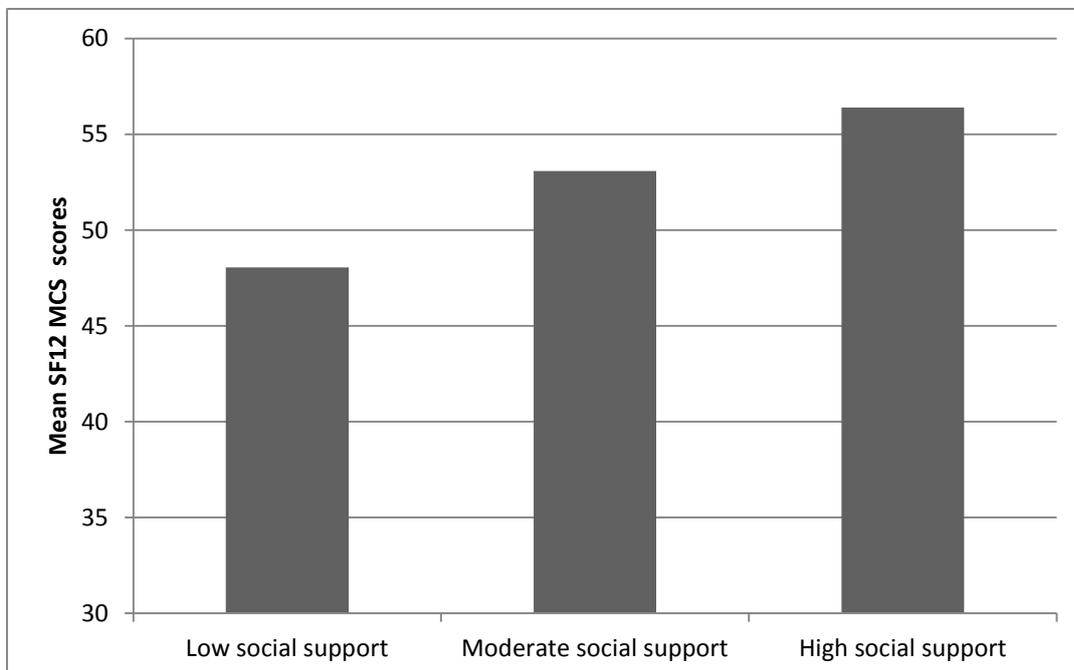
A key hypothesis within my thesis concerned my prediction that social support at Time 2 would be predictive of quality of life at Time 3 with greater social support associated with greater quality of life. There was a significant positive correlation between Time 3 MCS scores and Time 2 ESSI scores ( $r(120) = 0.30, p < 0.001$ ) suggesting an association between mental health quality of life at Time 3 and functional social support measured at Time 2. There was no significant correlation between Time 3 PCS scores and Time 2 ESSI scores ( $r(120) = -0.04, p = 0.68$ ).

Patients were divided in three social support groups according to their score on the Time 2 ESSI assessment as previously discussed. Mean Time 3 PCS and MCS scores by Time 2 functional social support group are depicted in Figures 7.9 and 7.10. A significant increase in MCS score as levels of functional social support increase was observed ( $F(2, 117) = 6.34, p < 0.05$ ). No significant relationship was found between PCS score and level of functional social support ( $F(2, 117) = 0.69, p = 0.51$ ).

**Figure 7.9 Mean PCS score at Time 3 by level of functional support at Time 2**



**Figure 7.10 Mean Time 3 MCS score by level of functional social support at Time 2**



Multiple regression analysis was conducted using either Time 3 PCS or MCS score as the dependent variable with Time 2 functional social support (ESSI score), age, gender, ethnicity, marital status, previous CHD, employment status, deprivation and Time 2 PCS or MCS score as the independent variables.

Using Time 3 PCS as the dependent variable, the model explained a moderate proportion of variance in PCS scores ( $R^2=0.22$ ,  $F(9, 100) = 3.04$ ,  $p<0.05$ ) with Time 2 PCS score being the only significant independent predictors of Time 3 PCS (Table 7.15). The regression was also re-run omitting Time 2 PCS to explore whether this variable may be obscuring other findings. The model remained significant with employment status emerging as a near significant predictor ( $\beta=0.23$ ,  $p= 0.067$ ).

**Table 7.15 Functional social support at Time 2 as a predictor of PCS score at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	40.89	20.99 – 60.79		4.08	0.001
T2 ESSI score	-0.15	-0.51 – 0.21	-0.08	-0.84	0.402
Age	-0.14	-0.37 - 0.10	-0.15	-1.16	0.249
Gender	2.42	-4.86 – 9.71	0.06	0.66	0.511
Marital status	-1.17	-5.79 – 3.45	-0.05	-0.50	0.617
Previous CHD	-2.95	-7.42 – 1.52	-0.12	-1.31	0.194
Employment status	1.43	-3.75 – 6.60	0.07	0.55	0.586
Ethnicity	-2.33	-8.57 – 3.90	-0.07	-0.74	0.460
Deprivation	-0.83	-5.21 – 3.55	-0.04	-0.37	0.709
Time 2 PCS score*	0.35	0.15 – 0.55	0.33	3.39	0.001

\* Significant independent predictor

Using Time 3 MCS as the dependent variable, the model explained a significant proportion of variance in MCS scores ( $R^2=0.51$ ,  $F(9, 100) = 11.61$ ,  $p<0.05$ ) with Time 2 MCS score being the only significant independent predictor and Time 2 functional social support score being a near significant predictor ( $p=0.068$ ) (Table 7.16). The regression was repeated with the omission of Time 2 MCS score and the model remained significant with functional social support ( $\beta= 0.22$ ,  $p<0.05$ ) and age ( $\beta=0.26$ ,  $p<0.05$ ) identified as significant independent

predictors. None of the variables included in any of the Time 3 regression models showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.16 Functional social support at Time 2 as a predictor of MCS at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-2.89	-19.79 – 14.01		-0.34	0.735
T2 ESSI score	0.26	-0.03 – 0.55	0.14	1.84	0.068
Age	0.17	-0.02 – 0.35	0.18	1.79	0.077
Gender	0.67	-4.96 – 6.29	0.02	0.23	0.815
Marital status	0.07	-3.59 – 3.72	0.00	0.04	0.971
Previous CHD	-2.52	-6.02 – 0.98	-0.10	-1.43	0.157
Employment status	2.13	-1.84 – 6.11	0.11	1.06	0.290
Ethnicity	-1.09	-6.03 – 3.85	-0.03	-0.44	0.664
Deprivation	1.24	-2.14 – 4.61	0.06	0.73	0.468
Time 2 MCS score*	0.68	0.53 – 0.84	0.64	8.54	0.001

\* Significant independent predictor

Functional social support at Time 2 was significantly predictive of mental health quality of life at Time 3 in the univariate analysis but this relationship only reached near significance in the multivariate model. Age was identified as important with younger patients more vulnerable to reduced mental health quality of life. Functional social support at Time 2 was not found to influence physical health quality of life at Time 3. Employment status was found to make a contribution with unemployed patients more likely to experience poorer physical health quality of life at Time 3.

#### **7.2.4.3 Longitudinal analysis: Exploring the predictive efficacy of Time 2 structural social support for Time 3 quality of life**

I hypothesised that structural social support measured at Time 2 would be predictive of quality of life at Time 3 with lower levels of structural social support predicting poorer quality of life, and this hypothesis is addressed in the following section. There was a significant correlation between structural social support (SNI score) and Time 3 MCS score ( $r(121) =$

0.18,  $p < 0.05$ ). There was no significant correlation between structural social support and Time 3 PCS score ( $r(121) = 0.04$ ,  $p = 0.70$ ).

Patients completing the Time 2 assessment were subdivided according to their level of structural social support to form two groups: Low structural social support (1 or fewer people in social network, Mean SNI = 0.88 (0.35),  $N = 8$ ) and adequate structural social support (2 or more people, Mean SNI = 4.23 (1.47),  $N = 123$ ). There were no significant difference between patients reporting low or adequate structural social support in PCS score ( $F(1, 119) = 1.19$ ,  $p = 0.28$ ) or MCS score ( $F(1, 119) = 0.004$ ,  $p = 0.95$ ). Multiple regression analysis was conducted using Time 3 PCS or MCS score as the dependent variable with Time 2 structural social support (SNI score), age, gender, marital status, previous CHD, employment, deprivation and Time 2 PCS or MCS score as the independent variables. Using Time 3 PCS score as the dependent variable, the model was significant ( $R^2 = 0.22$ ,  $F(8, 102) = 3.53$ ,  $p < 0.05$ ) with Time 2 PCS score emerging as the only significant independent predictor within the model (Table 7.17). The regression was repeated with the omission of Time 2 MCS score to ensure that this variable was not occluding significant predictors. The model remained significant and previous CHD was identified as a near significant predictor ( $\beta = -0.18$ ,  $p = 0.064$ ).

**Table 7.17 Structural social support at Time 2 as a predictor of PCS score at Time 3**

	<b>B</b>	<b>95% C.I for <math>\beta</math></b>	<b>Standardised <math>\beta</math></b>	<b>t</b>	<b>Sig.</b>
Constant	40.55	22.58 – 58.51		4.48	0.001
T2 SNI score	-0.22	-1.41 – 0.97	-0.04	-0.37	0.712
Age	-0.17	-0.40 - 0.06	-0.18	-1.44	0.153
Gender	1.61	-5.23 – 8.46	0.04	0.47	0.642
Marital status	-1.31	-5.92 – 3.31	-0.06	-0.56	0.576
Previous CHD	-3.30	-7.76 – 1.15	-0.14	-1.47	0.145
Employment status	0.85	-4.32 – 6.02	0.04	0.33	0.745
Deprivation	-1.41	-5.63 – 2.81	-0.07	-0.66	0.508
Time 2 PCS score*	0.35	0.14 – 0.55	0.33	3.38	0.001

\* Significant independent predictor

Using Time 3 MCS score as the dependent variable, the model was significant and identified ( $R^2=0.51$ ,  $F(8, 102) = 13.54$ ,  $p<0.05$ ) and both age and Time 2 MCS score were found to be significant independent predictors (Table 7.18). The model was repeated without Time 2 MCS score. The model remained significant with age identified as the only significant predictor ( $\beta=0.30$ ,  $p<0.05$ ). None of the variables included in any of these regression models showed multicollinearity according to variance inflation factor and tolerance values. These findings suggest that structural social support was not an independent predictor of quality of life 6 months following ACS.

**Table 7.18 Structural social support at Time 2 as a predictor of MCS score at Time 3**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	0.00	-15.73 – 15.73		0.00	1.000
T2 ESSI score	0.51	-0.44 – 1.46	0.08	1.07	0.286
Age*	0.19	0.01 - 0.37	0.20	2.06	0.042
Gender	0.37	-4.96– 5.71	0.01	0.14	0.890
Marital status	-0.05	-3.71 – 3.62	-0.00	-0.03	0.980
Previous CHD	-2.51	-6.01 – 0.99	-0.10	-1.43	0.157
Employment status	2.13	-1.85 – 6.12	0.11	1.06	0.290
Deprivation	0.92	-2.34 – 4.18	0.04	0.56	0.576
Time 2 MCS score*	0.71	0.56 – 0.86	0.67	9.12	0.001

\* Significant independent predictor

Structural social support did not make an independent contribution to quality of life scores at Time 3 indicating that the level of structural support reported by patients shortly after their ACS did not influence their quality of life 6 months after their ACS. The main factor contributing to reduced quality of life at Time 4 was reduced quality of life at Time 2. Age was again highlighted as important with younger patients identified as more at risk of poor mental health quality of life at Time 3.

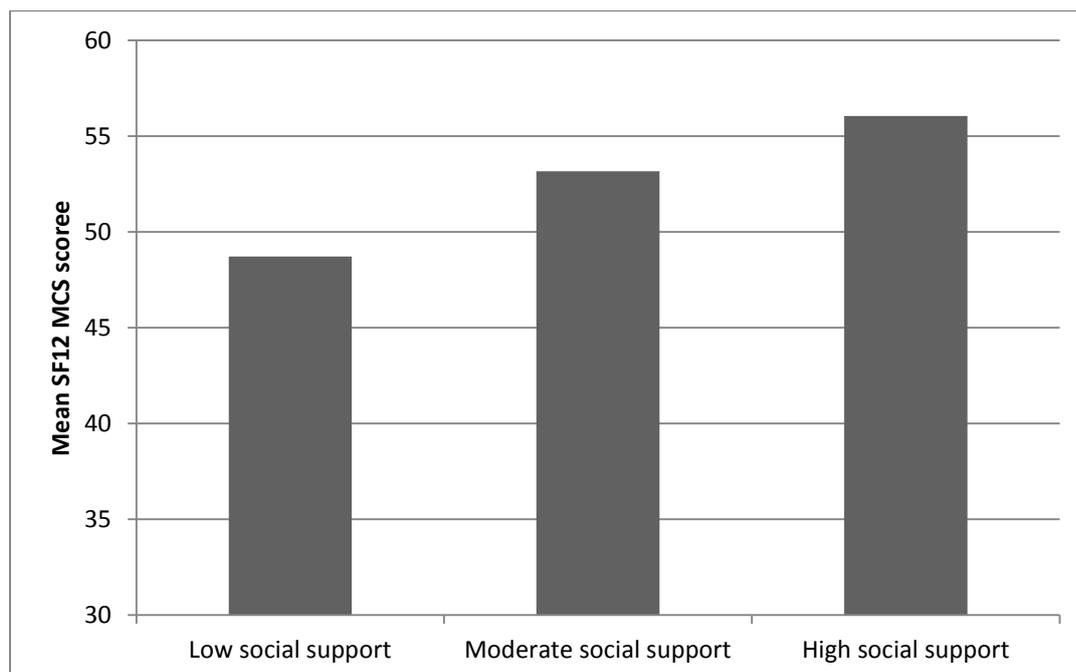
## 7.2.5 Social support and quality of life at Time 4

### 7.2.5.1 Cross sectional analysis: Exploring the association between Time 4 functional social support and Time 4 quality of life

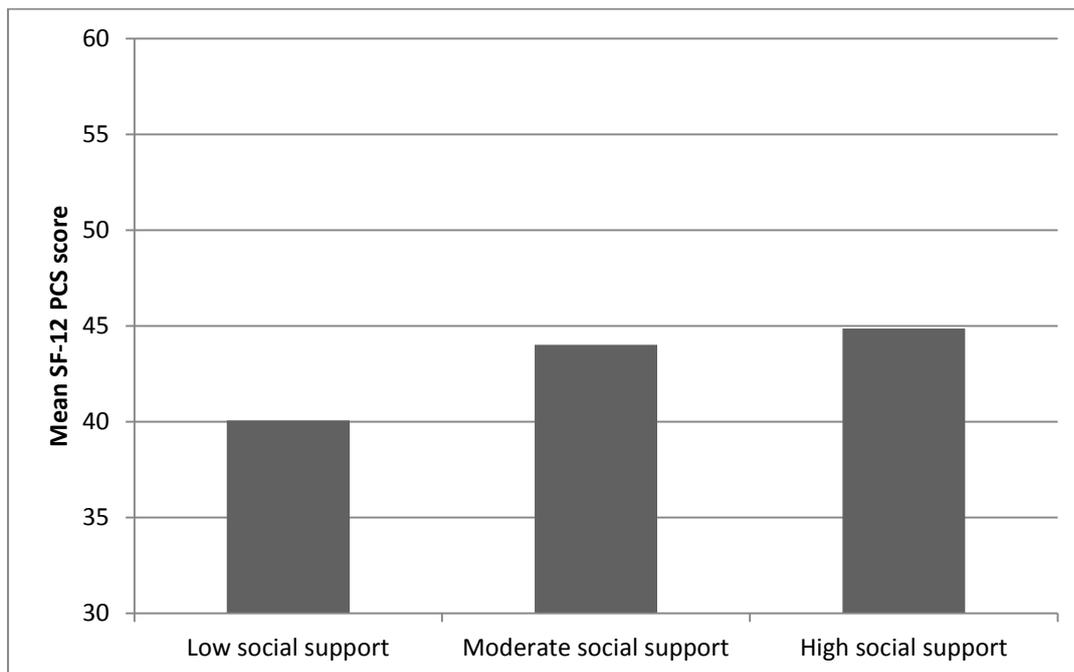
At Time 4, there was a significant positive correlation between Time 4 SF-12 PCS score and Time 4 ESSI score ( $r(145) = 0.17, p < 0.05$ ) and also between Time 4 SF-12 MCS score and Time 4 ESSI score ( $r(145) = 0.32, p = 0.001$ ).

Patients were divided in three social support groups according to their score on the Time 4 ESSI assessment as before. Mean PCS and MCS scores by functional social support group are depicted in Figures 7.11 and 7.12. A gradual increase in both PCS and MCS scores as levels of functional social support increase was observed which were statistically significant (PCS:  $F(2, 142) = 4.94, p < 0.05$ , MCS:  $F(2, 142) = 5.91, p < 0.05$ ) suggesting a positive association between functional social support and quality of life at Time 4.

**Figure 7.11 Mean MCS score at Time 4 by level of functional support at Time 4**



**Figure 7.12 Mean PCS score at Time 4 by level of functional social support at Time 4**



Multiple regression analysis was conducted using Time 4 PCS or MCS score as the dependent variable with Time 4 functional social support (ESSI score), age, gender, ethnicity, marital status, previous CHD, employment, deprivation and Time 2 PCS or MCS score as the independent variables. Using Time 4 PCS score as the dependent variable, the model explained a reasonable proportion of the variance in PCS scores ( $R^2=0.31$ ,  $F(9, 112) = 5.52$ ,  $p<0.05$ ) with social support, age, previous CHD and Time 2 PCS identified as significant predictors of Time 4 PCS score (Table 7.19). The regression was also re-run omitting Time 2 PCS to explore whether this variable may be obscuring other findings. The model remained significant and functional social support ( $\beta=0.24$ ,  $p<0.05$ ), age ( $\beta=-0.26$ ,  $p<0.05$ ) and previous CHD ( $\beta=-0.30$ ,  $p<0.05$ ) emerged as significant predictors.

**Table 7.19 Functional social support at Time 4 as a correlate of PCS score at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	41.97	25.02 – 58.92		4.91	0.001
T4 ESSI score*	0.32	0.03 – 0.60	0.21	2.18	0.032
Age*	-0.28	-0.47 - -0.10	-0.33	-3.04	0.003
Gender	1.56	-3.25 – 6.37	0.05	0.64	0.521
Marital status	1.69	-2.63 – 6.02	0.08	0.78	0.439
Previous CHD*	-6.12	-10.70 – -1.53	-0.23	-2.64	0.009
Employment status	-0.32	-4.52 – 3.89	-0.02	-0.15	0.881
Ethnicity	-2.27	-7.22 – 2.69	-0.07	-0.91	0.367
Deprivation	-0.87	-3.59 – 1.84	-0.06	-0.64	0.524
Time 2 PCS score*	0.26	0.08 – 0.44	0.25	2.88	0.005

\* Significant independent predictor

Using Time 4 MCS score as the dependent variable, the model explained a significant proportion of variance in MCS scores ( $R^2=0.52$ ,  $F(9, 112) = 13.35$ ,  $p<0.05$ ) with previous CHD and Time 2 MCS score being the only significant independent predictors (Table 7.20). Repeating the regression model with the omission of Time 2 MCS score produced a significant model with functional social support emerging as the only significant predictor ( $\beta=0.24$ ,  $p<0.05$ ). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.20 Functional social support at Time 4 as a correlate of MCS score at Time 4**

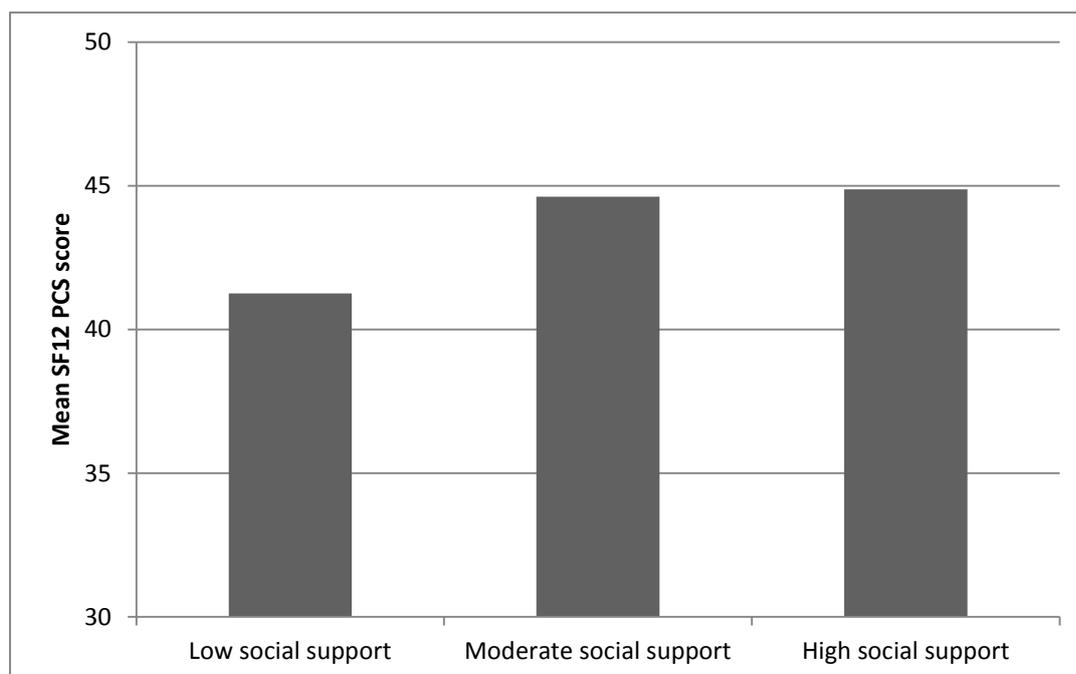
	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	9.93	-3.68 – 23.55		1.45	0.151
T4 ESSI score	0.13	-0.11 – 0.38	0.09	1.09	0.280
Age	0.12	-0.03 – 0.26	0.15	1.62	0.108
Gender	-2.74	-6.56 – 1.08	-0.10	-1.42	0.158
Marital status	0.07	-3.37 – 3.51	0.00	0.04	0.967
Previous CHD	-4.21	-7.65 - -0.78	-0.16	-2.43	0.017
Employment status	1.07	-2.27 – 4.40	0.06	0.64	0.527
Ethnicity	2.92	-1.01 – 6.84	0.10	1.47	0.143
Deprivation	-0.14	-2.32 – 2.04	-0.01	-0.13	0.900
Time 2 MCS score*	0.65	0.50 – 0.80	0.63	8.50	0.001

\* Significant independent predictor

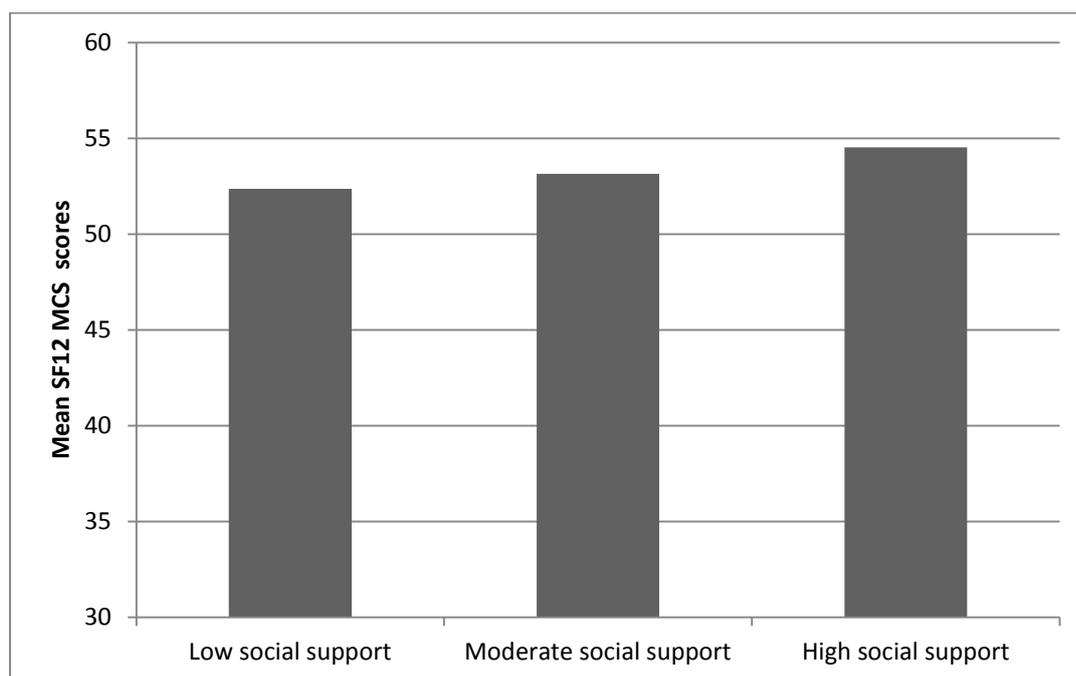
### 7.2.5.2 Longitudinal analysis: Exploring the predictive efficacy of Time 2 functional social support for Time 4 quality of life

A central hypothesis within my thesis concerns the ability of functional social support measured at Time 2 to predict patient quality of life at each follow up point. I hypothesised that greater social support at Time 2 would be predictive of greater quality of life at Time 4 and this is addressed in the following section. There were no significant correlations between Time 4 PCS scores and Time 2 ESSI scores ( $r(117) = 0.06, p=0.52$ ), nor between Time 4 MCS scores and Time 2 ESSI scores ( $r(120) = 0.09, p=0.31$ ) Patients were divided in three social support groups according to their score on the Time 2 ESSI assessment as before. Mean Time 4 PCS and MCS scores by Time 2 functional social support group are depicted in Figures 7.13 and 7.14. No significant differences in either PCS score ( $F(2, 114) = 1.25, p=0.29$ ) or MCS score (PCS  $F(2, 114) = 0.49, p=0.61$ ) according to level of social support were found.

**Figure 7.13 Mean PCS score at Time 4 by level of functional support at Time 2**



**Figure 7.14 Mean MCS score at Time 4 by level of functional social support at Time 2**



Multiple regression analysis was conducted using either Time 4 PCS or MCS score as the dependent variable with Time 2 functional social support (ESSI score), age, gender, ethnicity, marital status, previous CHD, employment status, deprivation and Time 2 PCS or MCS score as the independent variables.

Using Time 4 PCS as the dependent variable, the model explained a moderate proportion of variance in PCS scores ( $R^2 = 0.31$ ,  $F(9, 100) = 4.87$ ,  $p < 0.05$ ) with age, previous CHD and Time 2 PCS score identified as significant independent predictors of Time 4 PCS (Table 7.21). The regression was also re-run omitting Time 2 PCS to explore whether this variable may be obscuring other findings. The model remained significant with age ( $\beta = -0.30$ ,  $p < 0.05$ ) and previous CHD ( $\beta = 0.29$ ,  $p < 0.05$ ) identified as significant predictors.

**Table 7.21 Functional social support at Time 2 as a predictor of PCS score at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	44.69	25.56 – 63.82		4.63	0.001
T2 ESSI score	0.16	-0.19 – 0.51	0.08	0.89	0.378
Age*	-0.27	-0.48 - -0.07	-0.31	-2.67	0.009
Gender	0.22	-5.44 – 5.89	0.01	0.08	0.938
Marital status	0.28	-3.93 – 4.49	0.01	0.13	0.895
Previous CHD*	-5.20	-10.17 – -0.23	-0.19	-2.08	0.040
Employment status	0.26	-4.36 – 4.87	0.01	0.11	0.913
Ethnicity	-4.39	-10.25 – 1.46	-0.13	-1.49	0.140
Deprivation	0.91	-2.40 – 4.23	0.05	0.55	0.586
Time 2 PCS score*	0.32	0.13 – 0.52	0.31	3.30	0.001

\* Significant independent predictor

Using Time 4 MCS as the dependent variable, the model explained a significant proportion of variance in MCS scores ( $R^2 = 0.53$ ,  $F(9, 100) = 12.70$ ,  $p < 0.05$ ) with previous CHD and Time 2 MCS score being the only significant independent predictors (Table 7.22). The regression was repeated with the omission of Time 2 MCS score and the model was no longer significant with no predictors highlighted. None of the variables included in any of the Time 4 regression models showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.22 Functional social support at Time 2 as a predictor of MCS at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	10.71	-4.80 – 26.21		1.37	0.174
T2 ESSI score	-0.05	-0.32 – 0.22	-0.03	-0.35	0.726
Age	0.13	-0.03 – 0.29	0.15	1.63	0.107
Gender	-4.06	-8.45 – 0.34	-0.13	-1.83	0.070
Marital status	-0.24	-3.49 – 3.02	-0.01	-0.15	0.884
Previous CHD*	-4.06	-7.67 – -0.45	-0.16	-2.23	0.028
Employment status	0.85	-2.71 – 4.42	0.05	0.48	0.636
Ethnicity	2.60	-61.94 – 7.15	0.08	1.14	0.259
Deprivation	-0.09	-2.68 – 4.49	-0.01	-0.07	0.942
Time 2 MCS score*	0.75	0.59 – 0.90	0.70	9.68	0.001

\* Significant independent predictor

Overall, the data indicate that functional social support at Time 2 was not significantly associated with quality of life at Time 4. Previous CHD and quality of life score at Time 2 were predictive of both physical and mental health quality of life. Individuals reporting previous CHD prior to their ACS admission were more likely to have reduced physical and mental health quality of life at Time 4. Patients reporting reduced quality of life at Time 2 were more likely to report reduced quality of life at Time 4. Age was also identified as important with older patients more vulnerable to reduced physical health quality of life at Time 4.

#### **7.2.5.3 Longitudinal analysis: Exploring the predictive efficacy of Time 2 structural social support for Time 4 quality of life**

The following section addresses the hypothesis that higher levels of structural social support assessed Time 2 predict greater quality of life at Time 4. There were no significant correlations between structural social support (SNI score) and MCS score ( $r(118) = 0.12$ ,  $p = 0.21$ ) or PCS ( $r(118) = 0.01$ ,  $p = 0.92$ ).

Patients completing the Time 2 assessment were grouped according to their level of structural social support to form two groups: Low structural social support (1 or fewer people in social network, Mean SNI = 0.86 (0.38), N=7) and adequate structural social support (2 or more people, Mean SNI = 4.24 (1.45), N=132). There were no significant difference between patients reporting low or adequate structural social support in PCS score ( $F(1, 116) = 1.74$ ,  $p = 0.19$ ) or MCS score ( $F(1, 119) = 0.31$ ,  $p = 0.58$ ) suggesting that quality of life at Time 4 did not vary according to the level of structural social support reported by the patient at Time 2.

Multiple regression analysis was conducted using Time 4 PCS or MCS score as the dependent variable with Time 2 structural social support (SNI score), age, gender, marital status, previous CHD, employment, deprivation and Time 2 PCS or MCS score as the

independent variables. Using Time 4 PCS score as the dependent variable, the model was significant ( $R^2 = 0.29$ ,  $F(8, 102) = 5.24$ ,  $p < 0.05$ ) with age, previous CHD and Time 2 PCS score emerging as the only significant independent predictors within the model (Table 7.23). The regression was repeated with the omission of Time 2 PCS score to ensure that this variable was not occluding significant predictors. The model remained significant and previous CHD ( $\beta = -0.28$ ,  $p < 0.05$ ) and age ( $\beta = -0.28$ ,  $p < 0.05$ ) remained the only significant predictors.

**Table 7.23 Structural social support as a predictor of PCS score at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	T	Sig.
Constant	51.19	33.69 – 68.69		5.80	0.001
T2 SNI score	-0.45	-1.59 – 0.69	-0.07	-0.78	0.437
Age*	-0.26	-0.46 - -0.06	-0.29	-2.54	0.013
Gender	-0.73	-6.28 – 4.82	0.02	-0.26	0.795
Marital status	-0.90	-5.14 – 3.35	-0.04	-0.42	0.676
Previous CHD*	-4.98	-9.79 – -0.17	-0.19	-2.05	0.043
Employment status	0.39	-4.31 – 5.09	0.02	0.17	0.869
Deprivation	0.14	-3.13 – 3.41	0.01	0.08	0.933
Time 2 PCS score*	0.32	0.13 – 0.52	0.30	3.26	0.001

\* Significant independent predictor

Using Time 4 MCS score as the dependent variable, the model was significant and identified ( $R^2 = 0.54$ ,  $F(8, 102) = 15.10$ ,  $p < 0.05$ ) and both previous CHD and Time 2 MCS score were found to be significant independent predictors (Table 7.24). The model was repeated without Time 2 MCS score. The model was no longer significant with no new predictors identified. None of the variables included in any of these Time 4 regression models showed multicollinearity according to variance inflation factor and tolerance values. These findings suggest that structural social support was not an independent predictor of quality of life 12 months following ACS.

**Table 7.24 Structural social support as a predictor of MCS score at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	10.07	-4.06 – 24.20		1.41	0.161
T2 ESSI score	0.21	-0.67 – 1.08	0.03	0.47	0.640
Age	0.13	-0.03 - 0.28	0.15	1.59	0.114
Gender	-3.76	-8.03– 0.51	-0.13	-1.75	0.084
Marital status	0.32	-2.92 – 3.57	0.02	0.20	0.844
Previous CHD*	-4.07	-7.53 – -0.60	-0.16	-2.33	0.022
Employment status	0.84	-2.74 – 4.41	0.04	0.46	0.644
Deprivation	0.13	-2.41 – 2.67	0.01	0.10	0.918
Time 2 MCS score*	0.72	0.57 – 0.86	0.68	9.63	0.001

\* Significant independent predictor

Structural social support did not make an independent contribution to quality of life scores at Time 4 indicating that the level of structural support reported by patients shortly after their ACS did not influence their quality of life 12 months after their ACS. Previous CHD and Time 2 quality of life scores were shown to be important to both physical and mental health quality of life. Older age was also found to be predictive of reduced physical health quality of life at Time 4.

### **7.2.6 Overall summary: Social support and quality of life after ACS**

The findings discussed in this section reveal that social support makes some contribution to the quality of life reported by patients following ACS. For clarity, the key findings from my analyses are summarised in Table 7.25.

**Table 7.25 Summary of key findings: Social support and quality of life**

	Time 2		Time 3		Time 4	
	Functional	Structural	Functional	Structural	Functional	Structural
<b>T2 QoL</b>						
Physical	NON SIG	NON SIG	-	-	-	-
Mental	NON SIG	NON SIG	-	-	-	-
<b>T3 QoL</b>						
Physical	NON SIG	NON SIG	NON SIG	NON SIG	-	-
Mental	<b>Near SIG</b>	NON SIG	<b>SIG</b>	NON SIG	-	-
<b>T4 QoL</b>						
Physical	NON SIG	NON SIG	-	-	<b>SIG</b>	NON SIG
Mental	NON SIG	NON SIG	-	-	<b>SIG</b>	NON SIG

Key: SIG = significant association ( $p < 0.05$ ), Near SIG = near significant association ( $p = 0.051 - 0.07$ ), NON SIG = no significant association ( $p > 0.071$ )

The relationship between social support and quality of life was less robust and clear than the relationship with distress. Functional or structural social support were not independently associated with physical or mental health quality of life at Time 2 suggesting that social support does not play a role in determining quality of life shortly after ACS. However, the relationship between Time 2 functional social support and Time 3 mental health quality of life was significant in the univariate analysis and there was a near significant association between Time 2 functional social support and mental health quality of life at Time 3 in the multivariate analysis suggesting that functional social support may be important to mental health quality of life 6 months following ACS. However, this relationship was not significant and did not persist at Time 4. There were no significant associations between Time 2 structural social support and quality of life at any follow up point. The data did not provide support for my prediction that both structural and functional social support assessed at Time 2 would be associated with quality of life at all follow up time points.

There were significant cross sectional relationships between Time 3 functional social support and Time 3 mental health quality of life, and between Time 4 functional social support and Time 4 physical and mental health quality of life. These findings suggest that social support and quality of life are associated; however causal direction cannot be established due to the cross sectional nature of this analysis. In the context of a lack of significant longitudinal relationships between social support and quality of life, these findings suggest that patients experiencing poor quality of life may tend to make more negative evaluations regarding the support available to them. However, the near significant longitudinal relationship between Time 2 functional social support and Time 3 mental health related quality of life does indicate a potential predictive relationship and thus the data does not entirely reject a predictive role for social support in quality of life.

Overall, the results do not support the hypothesised robust longitudinal relationship between Time 2 functional social support and quality of life. Functional social support and quality of life were significantly cross sectional associated at Time 3 and Time 4 indicative of an associative relationship between them. Structural social support did not to demonstrate any predictive efficacy or association with quality of life.

## 7.3 Part 4: The role of social support in heart rate variability after ACS

### 7.3.1 Analytic dataset

The Time 2 assessment was completed by 226 patients and 151 of these patients provided valid heart rate variability data during this assessment. The difference in numbers is due to patient request for postal involvement instead of a home visit, patient refusal for monitor attachment and technical problems with the monitor during recording. The details of how HRV data were gathered are detailed in Chapter 4.

### 7.3.2 HRV at Time 2

Heart rate variability (HRV) was analysed in terms of frequency domain measures (LF-HRV, VLF-HRV, and HF-HRV) and time domain measures (heart rate, pNN50 and RMSSD). The specifics of these measures are described in more detail in Chapter 4. All of the HRV measures (except heart rate) were logged transformed prior to analysis. The mean values for each of these HRV measures are depicted in Table 7.26.

**Table 7.26 Mean HRV measures at Time 2**

	<b>N</b>	<b>Mean</b>	<b>SD</b>
<b>Heart rate</b>	151	66.66	12.20
<b>LF</b>	151	4.89	1.28
<b>VLF</b>	151	4.58	1.10
<b>HF</b>	151	4.61	1.50
<b>RMSSD</b>	151	3.63	0.78
<b>pNN50</b>	151	15.32	16.41

### 7.3.3 Social support and HRV at Time 2

The relationship between HRV and social support was explored using continuous (ESSI/SNI score) and categorical (low/med/high functional social support on ESSI; low/adequate structural social support on SNI) measures of both functional and structural social support. All multivariate analyses included control for age, gender, beta blocker usage at Time 2,

depression status at Time 2 and anxiety status at Time 2 as these have been shown to influence HRV.

### 7.3.3.1 Functional social support and HRV

There were no significant correlations between any HRV measure and the ESSI (functional social support). Multiple regression analysis was completed separately using each measure of HRV as the dependent variable and age, gender, beta blocker usage at Time 2, depression status at Time 2 and anxiety status at Time 2 as covariates with ESSI score as the independent variable. Using RMSSD as the dependent variable, the model was non-significant ( $R^2 = 0.03$ ,  $F(6, 103) = 0.59$ ,  $p = 0.74$ ) with no significant predictors emerging suggesting that functional social support did not contribute to RMSSD measures of HRV (Table 7.27).

**Table 7.27 Functional social support at Time 2 as a correlate of RMSSD at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	4.69	3.21 – 6.17		6.27	0.001
T2 ESSI score	-0.02	-0.05 - 0.01	-0.13	-1.25	0.214
Age	0.00	-0.01 – 0.02	0.01	0.05	0.959
Gender	-0.27	-0.88 – 0.34	-0.09	-0.88	0.382
Depression status	-0.16	-0.70 – 0.38	-0.07	-0.60	0.552
Anxiety status	-0.09	-0.54 – 0.35	-0.05	-0.42	0.679
Beta blocker use	-0.22	-0.65 – 0.22	-0.10	-0.99	0.326

\* Significant independent predictor

Using HF-HRV as the dependent variable, the model was non-significant ( $R^2 = 0.02$ ,  $F(6, 103) = 0.43$ ,  $p = 0.86$ ) with no significant predictors emerging suggesting that functional social support did not contribute to HF measures of HRV (Table 7.28).

**Table 7.28 Functional social support at Time 2 as a correlate of HF-HRV at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	6.22	3.38 – 9.06		4.34	0.001
T2 ESSI score	-0.03	-0.10 - 0.03	-0.11	-1.07	0.288
Age	0.00	-0.03 – 0.03	0.01	-0.11	0.914
Gender	-0.25	-1.42 – 0.92	-0.04	-0.43	0.672
Depression status	-0.26	-1.30 – 0.77	-0.06	-0.50	0.616
Anxiety status	-0.27	-1.12 – 0.58	-0.07	-0.63	0.531
Beta blocker use	-0.27	-1.10 – 0.55	-0.07	-0.65	0.516

\* Significant independent predictor

The same multiple regression analysis was also repeated for the remaining HRV variables (VLF, LF, Heart rate, pNN50) with functional social support as the independent variable and, similar to the findings with RMSSD and HF-HRV, none of the models were significant. These non-significant results are not reported here for brevity. Patients were also grouped according to their score on the ESSI at Time 2 into three groups; low, moderate and high social support as previously described. Values for each HRV measure according to social support group are listed in Table 7.33.

**Table 7.29 Mean HRV values by functional social support group at Time 2**

	Low social support	Moderate social support	High social support
<b>N</b>	31	45	34
<b>Heart rate</b>	69.39 (13.47)	64.38 (9.84)	67.23 (13.52)
<b>RMSSD</b>	3.64 (0.74)	3.69 (0.78)	3.56 (0.89)
<b>pNN50</b>	14.72 (15.10)	15.65 (15.79)	13.65 (15.31)
<b>HF</b>	4.66 (1.35)	4.68 (1.54)	4.47 (1.68)
<b>LF</b>	4.90 (1.26)	4.97 (1.29)	4.86 (1.31)
<b>VLF</b>	4.53 (1.25)	4.63 (1.00)	4.46 (1.16)

A series of ANCOVA analyses were conducted with each HRV measure as the dependent variable, functional social support group as the independent variable and age, gender,

depression status at Time 2, anxiety status at Time 2 and beta blocker use at Time 2 as covariates. The only significant factor was lower heart rate among those taking beta blockers compared to those patients not taking beta blocker. There were no significant differences by functional social support group for any measure of HRV.

### 7.3.3.2 Structural social support and HRV

There were no significant correlations between SNI (structural social support) scores and any measure of HRV. However, there was a significant negative correlation between marital status and LF power ( $r(151) = -0.17, p < 0.05$ ) and a near significant correlation between marital status and HF power ( $r(151) = -0.15, p = 0.06$ ) indicative of a potential relationship between marital status and HRV that is further explored in Chapter 8.

The relationship between structural social support and HRV measures was also explored in using multiple regression analysis with each measure of HRV as the dependent variable and age, gender, beta blocker usage at Time 2, depression status at Time 2 and anxiety status at Time 2 as covariates with SNI score as the independent variable. Using RMSSD as the dependent variable, the model was non-significant ( $R^2 = 0.02, F(6, 104) = 0.40, p = 0.88$ ) with no significant predictors emerging suggesting that level of structural social support did not contribute to RMSSD measures of HRV (Table 7.30).

**Table 7.30 Structural social support at Time 2 as a correlate of RMSSD at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	4.36	2.91 – 5.80		5.97	0.001
T2 ESSI score	-0.03	-0.13 - 0.07	-0.06	-0.56	0.575
Age	-0.00	-0.02 – 0.01	-0.02	-0.22	0.823
Gender	-0.24	-0.84 – 0.36	-0.08	-0.79	0.433
Depression status	-0.18	-0.71 – 0.36	-0.08	-0.66	0.514
Anxiety status	-0.06	-0.50 – 0.39	-0.03	-0.25	0.802
Beta blocker use	-0.25	-0.70 – 0.19	-0.12	-1.13	0.260

\* Significant independent predictor

Using HF-HRV as the dependent variable, the model was non-significant ( $R^2 = 0.02$ ,  $F(6, 104) = 0.30$ ,  $p=0.94$ ) with no significant predictors emerging indicating that structural social support did not make a contribution of HF measures of HRV (Table 7.31).

**Table 7.31 Structural social support as a correlate of HF-HRV at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	5.74	2.97 – 8.51		4.11	0.001
T2 ESSI score	-0.06	-0.25 – 0.14	-0.06	-0.58	0.562
Age	0.01	-0.03 – 0.02	0.04	-0.36	0.719
Gender	-0.20	-1.35 – 0.94	-0.04	-0.35	0.724
Depression status	-0.29	-1.31 – 0.74	-0.07	-0.56	0.578
Anxiety status	-0.21	-1.05 – 0.64	-0.06	-0.49	0.626
Beta blocker use	-0.35	-1.20 – 0.51	-0.08	-0.80	0.423

\* Significant independent predictor

Multiple regression analysis was also completed for the remaining HRV measures. None of the regression models were significant and structural social support did not emerge as a predictor in any model.

Patients were also grouped according to their score on the SNI at Time 2 into two groups; low structural social support (N=9, M=1.00 (SD=0.00) or adequate structural social support (N=102, M=4.25 (SD=1.47)). HRV values for each by structural social support group are listed in Table 7.32.

**Table 7.32 Mean HRV values by structural social support group at Time 2**

	Low social support	Adequate social support
<b>N</b>	9	102
<b>Heart rate</b>	66.03 (11.56)	66.79 (12.26)
<b>RMSSD</b>	3.39 (0.54)	3.65 (0.81)
<b>pNN50</b>	1.69 (0.86)	2.16 (1.19)
<b>HF</b>	4.27 (1.18)	4.64 (1.54)
<b>LF</b>	4.53 (1.22)	4.88 (1.31)
<b>VLF</b>	4.58 (0.90)	4.55 (1.14)

A number of ANCOVA analyses were conducted using each HRV measure as the dependent variable, structural social support group as the independent variable and age, gender, depression status at Time 2, anxiety status at Time 2 and beta blocker use at Time 2 as covariates. There were no significant differences by structural social support group for any measure of HRV (results not shown).

### **7.3.4 Summary: Social support and HRV after ACS**

The findings suggest that the functional and structural social support reported by patients shortly after ACS were not associated with any measure of HRV measured concurrently.

## **7.4 Chapter Discussion**

### **7.4.1 Quality of life after ACS**

The quality of life scores indicate that patients had mean physical health related quality of life scores below US population norms for this age group which is likely reflective of their recent physical health crisis. Mean mental health quality of life was consistent with the US population norm. The scores for both measures of quality of life were similar to the US population norms for post MI populations (PCS= 40 (SD=12), MCS = 50 (SD=12)) (Ware, Kosinski, & Keller, 1998). These scores were also comparable with the scores reported by other more recent studies using this measure in post ACS patients. For example, Muller-

Nordhorn, Roll, & Willich, (2004) reported mean PCS and MCS scores of 40 and 47 respectively in post ACS, PCTA and CABG patients during the first three weeks following admission. Failde, Medina, Ramirez, & Arana, (2009) identified mean PCS and MCS scores of 43.5 and 48.5 in post MI patients during hospitalisation. McBurney et al, (2002) measured quality of life 7 months post MI using the SF-12 and found mean PCS score of 40.6 and a mean MCS score of 52.1. Among the TRACE patients, mental health quality of life remained stable over the follow up period; however, there was a significant increase in physical health quality of life reflective of physical recovery over the 12 month follow up period. A number of factors were found to influence quality of life over time. Age, deprivation and prior CHD history were associated with physical health quality of life with older, more deprived patients, and patients reporting prior CHD more likely to report impaired physical health quality of life. Similarly, age and deprivation were associated with mental health quality of life with more deprived and younger patients reporting worse quality of life. Marital status and employment status were also identified as potentially important although these findings were based on the univariate rather than the multivariate analyses (the role of marital status and quality of life will be explored in detail in Chapter 8). Quality of life reported at Time 2 was highly predictive of quality of life at Time 3 and Time 4 indicating the importance of early post ACS reactions for long term adjustment. Few studies have explored the demographic predictors of quality of life in post ACS patients with most studies tending to have greater focus on clinical and cardiovascular antecedents. However, as seen in the TRACE sample findings, age and previous cardiac history have previously been associated with poorer quality of life following ACS (Brown et al., 1999; McBurney et al., 2002; Schweikert et al., 2009; Perers et al., 2006).

#### **7.4.2 Social support and quality of life after ACS**

Functional social support was cross-sectionally associated with Time 3 mental health related quality of life, and also with Time 4 physical and mental health related quality of life. Functional social support was not significantly associated with quality of life at Time 2.

Structural social support did not demonstrate any significant cross sectional association with quality of life at any assessment point. These findings suggest that level of social support perceived by the patient in period shortly after post ACS hospital discharge does not contribute to the level of quality of life experienced by the patient during this period. However, functional social support was correlated with mental health quality of life at 6 months and 12 months post ACS, and also with physical health quality of life at 12 months suggesting that functional social support may be important at these later stages. However, due to the cross sectional nature of these significant associations, causal direction cannot be determined and it could be that experiencing poor quality of life may lead to less favourable evaluations regarding social support.

The findings of the longitudinal analyses provide tentative suggestion that functional social support may contribute to mental health quality of life at Time 3 as Time 2 functional social support was significantly predictive of Time 3 mental health quality of life in the univariate analysis, and was near significant in the multivariate analysis. However, this relationship did not persist at Time 4. No longitudinal associations between Time 2 functional social support and physical health quality of life at Time 3 or Time 4 were identified. Similarly, structural social support did not demonstrate any predictive efficacy with regard to Time 3 or Time 4 quality of life. Overall, these results do not provide robust support for my hypotheses and suggest that social support was not a strong predictor of quality of life. However, the potential longitudinal link between Time 2 functional social support and Time 3 mental health quality of life does merit further inquiry.

#### **7.4.3 Social support and HRV after ACS**

Contrary to my hypothesis that lower functional and structural social support would be associated with lower HRV, the results suggest that neither functional nor structural social support had an influence on the HRV of post ACS patients. These results differ from the association found between social support and HRV in a non-clinical population in the only

other study investigating this relationship (Shin et al., 2012). In the light of research reported in this thesis (Chapter 3) and elsewhere (Horsten et al., 1999) suggesting the presence of marital status differentials in HRV, an exploration of the role of marital status on patient HRV was conducted on the TRACE data and the findings are presented in Chapter 8 which suggest a more prominent role of marital status in HRV. Overall, the findings do not provide evidence for a relationship between low social support and HRV among post ACS patients although it is clear that more research is required.

#### **7.4.4 Chapter summary**

Quality of life was slightly below population norms in the physical domain shortly after ACS but this improved over the follow up period. Mental health quality of life was not significantly lower than population norms and remained stable throughout follow up suggesting limited quality of life impact in terms of mental health functioning. Both functional and structural social were found to exert a limited cross sectional or longitudinal impact on quality of life, although functional social support did exhibit some cross sectional associations with quality of life at Time 3 and 4, and a longitudinal association with Time 3 mental health quality of life providing some evidence of a relationship between social support and quality of life at these later stages that merits further research.

The HRV values derived shortly after discharge from hospital were not associated with either measure of social support suggesting that level of social support were not associated with HRV at Time 2. A notable strength of the TRACE study was the inclusion of longitudinal assessment of quality of life as it provides insight into both the immediate and longer term impact of ACS. In the case of the TRACE patients, for the majority of patients there was limited long term impact upon their physical or mental wellbeing (although as identified in Chapter 6 there was increased prevalence of depression and anxiety amongst the patients). A further asset of the study was the inclusion of HRV assessment as this affords a more comprehensive psychobiological picture of the relationship between social support and post

ACS recovery, and the possible pathways through which social support may operate. The assessment of HRV and identification of psychosocial correlates is extremely salient given the important prognostic role of post ACS HRV.

## CHAPTER 8 TRACE STUDY RESULTS PART 1

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### **Part 5: Marital status and marital satisfaction after ACS, and their relationship with psychological distress, quality of life and HRV**

#### **8.1 Introduction**

This chapter focuses on the same patient sample from the TRACE study as Chapter 5, 6 and 7. However, specific attention is given to the role of marital status and, amongst married patients, the impact of marital satisfaction on psychological adjustment and HRV. Baseline characteristics and attrition analyses for married and unmarried patients at each assessment point are summarised. The pattern of psychological response and quality of life experienced by married and unmarried patients over time is compared. The association between marital status and HRV at Time 2 is also described. This is followed by exploration of the marital satisfaction reported by married patients at Time 2, 3 and 4, and the relationship between marital satisfaction and psychological health, as well as marital satisfaction and quality of life at Time 2, 3 and 4. Finally, the association between marital satisfaction and HRV at Time 2 is examined. The chapter finishes with a discussion and summary of the results presented.

#### **8.2 Data analysis**

Descriptive data regarding the marital status, marital satisfaction, psychological distress and quality of life reported by married and unmarried patients over the 12 month follow up period was explored. Associations between marital status and immediate psychological adjustment (anxiety, depression and quality of life) at each time point had already been examined in Chapter 6 and Chapter 7. In this chapter, these findings were extended with a series of repeated measures ANOVA which examined marital status patterns in change over time in distress and quality of life. The relationship between marital satisfaction and psychological adjustment (anxiety, depression and quality of life) was also further explored through a

series of ANCOVA analyses. The association between marital status, marital satisfaction and HRV at Time 2 was also investigated using multivariate ANOVA analyses.

### 8.3 Married and unmarried patient characteristics Time 1 – Time 4

Demographic and clinical characteristics of married versus unmarried patients at Time 1 and also at Time 2 – 4 are depicted in Table 8.1 and 8.2 respectively. Significant differences at a single assessment are indicated (\*) and differences that were significant or borderline significant ( $p < 0.055$ ) at all follow up periods are bolded.

**Table 8.1 Characteristics and comparison of married and unmarried patients at Time 1**

	Time 1			
	Married		Unmarried	
	Mean (SD) / N (%) Range	n	Mean (SD) / N (%) Range	n
Age	60.34 (11.11) 36 - 86	203	59.75 (11.57) 32 - 88	95
Gender*		203		95
Female	25 (12)		22 (23)	
Male	178 (88)		58 (61)	
Marital status		203		95
Single	-		31 (33)	
Married	185 (91)			
Living as married	18 (9)			
Divorced	-		32 (34)	
Separated	-		9 (10)	
Widowed	-		22 (23)	
Marital duration	30.81 (14.33) 1.5 - 60		-	
Lives alone*	4 (2)	203	65 (68)	95
Ethnicity		203		95
White	168 (83)		79 (83)	
Asian	26 (13)		9 (10)	
Black	5 (2)		5 (5)	
Other	4 (2)		2 (2)	
Education		203		95
Basic	109 (54)		49 (52)	
Secondary	58 (28)		35 (37)	
Degree	36 (18)		10 (11)	
Employment		202		94
Employed	118 (58)		51 (54)	

Unemployed			43 (46)	
Deprivation*		200		94
Low	148 (74)		40 (43)	
Moderate	37 (19)		33 (35)	
High	15 (7)		21 (22)	
GRACE score	92.98 (27.77) 33 - 179	203	92.57 (27.77) 35 - 160	95
ACS type		203		95
STEMI	176 (87)		84 (88)	
NSTEMI	27 (13)		11 (12)	
Cardiac arrest	15 (7)	203	7 (7)	95
Previous MI	31 (15)	203	8 (8)	95
Previous CHD	44 (22)	203	22 (23)	95
Family history CHD	131 (65)	203	58 (61)	95
URTI 2 mths	68 (36)	187	23 (29)	80
Diabetic	32 (16)	203	15 (16)	95
Current smoker at T1	69 (34)	203	48 (51)	95
BMI	27.36 (4.36) 19 - 48	188	27.94 (5.23) 35 - 160	89
Drink alcohol	138 (68)	202	64 (69)	93

**Table 8.2 Characteristics and comparison of married and unmarried patients at Time 2, Time 3 and Time 4**

	Time 2				Time 3				Time 4			
	Married		Unmarried		Married		Unmarried		Married		Unmarried	
	Mean (SD)/ N (%) Range	n	Mean (SD)/ N (%) Range	n	Mean (SD) / N (%) Range	n	Mean (SD) / N (%) Range	n	Mean (SD) / N (%) Range	n	Mean (SD) / N (%) Range	n
Age	59.27 (11.03) 36 – 88	162	60.95 (13.41) 32 - 88	64	60.63 (10.76) 37 - 86	141	61.36 (11.27) 38 - 86	59	61.12 (10.33) 38 - 86	120	61.29 (12.73) 32 - 88	56
<b>Gender*</b>		<b>162</b>		<b>64</b>		<b>141</b>		<b>59</b>		<b>120</b>		<b>56</b>
<i>Female</i>	<b>18 (11)</b>		<b>18 (28)</b>		<b>15 (11)</b>		<b>13 (22)</b>		<b>11 (9)</b>		<b>14 (25)</b>	
<i>Male</i>	<b>144 (89)</b>		<b>46 (72)</b>		<b>126 (89)</b>		<b>46 (78)</b>		<b>109 (91)</b>		<b>42 (75)</b>	
Marital status		162		64		141		59		120		56
<i>Single</i>	-		21 (33)		-		20 (34)		-		21 (37)	
<i>Married</i>	145 (90)				130 (92)		-		112 (93)			
<i>Living as married</i>	17 (10)				11 (8)		-		8 (7)			
<i>Divorced</i>	-		19 (30)		-		19 (32)		-		14 (27)	
<i>Separated</i>	-		17 (26)		-		5 (9)		-		6 (11)	
<i>Widowed</i>	-		7 (11)		-		5 (9)		-		14 (25)	
Marital duration	30.81 (14.33) 1.5 – 60	162	-	-	30.59 (13.89) 15 - 60	138	-	-	31.77 (13.33) 37 - 166	120	-	
<b>Lives alone*</b>	<b>2 (1.2)</b>	<b>162</b>	<b>42 (66)</b>	<b>64</b>	<b>1 (0.7)</b>	<b>141</b>	<b>44 (75)</b>	<b>59</b>	<b>1 (0.8)</b>	<b>120</b>	<b>39 (70)</b>	<b>56</b>
Ethnicity		162		64		141		59		120		56
<i>White</i>	136 (84)		52 (81)		120 (85)		50 (85)		107 (89)		49 (88)	
<i>Asian</i>	18 (11)		7 (11)		15 (11)		6 (10)		10 (8)		4 (7)	
<i>Black</i>	5 (3)		4 (6)		4 (3)		3 (5)		1 (1)		2 (4)	
<i>Other</i>	3 (2)		1 (2)		2 (1)		0		2 (2)		1 (2)	
Education		162		63		141		58				
<i>Basic</i>	88 (54)		36 (57)		77 (55)		31 (53)		69 (57)	120	32 (58)	55
<i>Secondary</i>	48 (30)		20 (32)		41 (29)		21 (36)		31 (26)		19 (35)	

<i>Degree</i>	26 (16)		7 (11)		23 (16)		6 (10)		20 (17)		4 (7)	
Employment		161		63		140		59		119		56
<i>Employed</i>	99 (61)*		28 (44)		81 (58)		29 (49)		68 (57)		28 (50)	
<i>Unemployed</i>	62 (39)		35 (56)		59 (42)		30 (51)		51 (43)		28 (50)	
<b>Deprivation*</b>		<b>160</b>		<b>63</b>		<b>139</b>		<b>58</b>		<b>118</b>		<b>55</b>
<i>Low</i>	<b>123 (77)</b>		<b>29 (46)</b>		<b>110 (79)</b>		<b>29 (50)</b>		<b>94 (80)</b>		<b>27 (49)</b>	
<i>Moderate</i>	<b>26 (16)</b>		<b>19 (30)</b>		<b>21 (15)</b>		<b>19 (33)</b>		<b>19 (16)</b>		<b>17 (31)</b>	
<i>High</i>	<b>11 (7)</b>		<b>15 (24)</b>		<b>8 (6)</b>		<b>10 (17)</b>		<b>5 (4)</b>		<b>11 (20)</b>	
GRACE score	89.94 (25.79) 33 - 166	162	96.52 (27.68) 45 - 160	64	93.86 (26.00) 37 - 166	141	94.93 (25.76) 51 - 158	59	94.15 (25.03) 37 - 166	120	97.09 (25.41) 45 - 158	56
ACS type		162		64		141		59		120		56
<i>STEMI</i>	144 (89)		55 (86)		124 (88)		51 (86)		106 (88)		49 (88)	
<i>NSTEMI</i>	18 (11)		9 (14)		17 (12)		8 (14)		14 (12)		7 (12)	
Cardiac arrest	10 (6)	162	6 (9)	64	10 (7)	141	3 (5)	59	8 (7)	120	6 (11)	56
Previous MI	23 (14)	162	5 (8)	64	25 (18)	141	6 (10)		20 (17)	120	4 (7)	56
Previous CHD	28 (17)	162	14 (22)	64	31 (22)	141	18 (31)	59	23 (20)	120	14 (25)	56
Family history CHD	107 (66)	62	34 (53)	64	92 (65)	141	37 (63)	59	79 (66)	120	33 (59)	56
URTI 2 mths	55 (37)*	147	11 (20)	54	48 (37)	130	14 (29)	49	45 (40)	112	12 (25)	48
Diabetic	24 (15)	162	11 (17)	64	16 (11)	141	9 (15)	59	14 (12)	120	6 (11)	56
Current smoker at T1	55 (34)	162	29 (45)	64	46 (33)	141	30 (51)	59	39 (33)	120	29 (52)	56
Smoker at T3 / T4	-		-		9 (9)*	104	9 (22)	41	8 (8)*	103	9 (19)	48
BMI	27.44 (4.52) 19.20 – 48.39	150	27.77 (5.17) 17.53 – 44.79	60	27.32 (4.53) 19 - 48	131	28.02 (4.81) 20 - 44	57	27.34 (4.34) 19 - 44	115	28.05 (4.88) 20 – 45	54
Drink alcohol	112 (69)	162	42 (68)	62	103 (73)	141	40 (70)	59	89 (74)	120	38 (70)	54

Overall, there were some consistent significant demographic differences between married and unmarried patients that persisted throughout the study period. Unmarried patients were significantly more likely to live alone, to be living in high or moderate deprivation and to have been a current smoker at the time of their ACS. Unmarried patients were also more likely to be current smokers at 6 and 12 months following their ACS compared to married patients. There were also a higher proportion of female patients in the unmarried group compared to in the married group. However, on most demographic and clinical measures, the two groups are comparable.

## **8.4 Attrition analysis**

### **8.4.1 Time 2 attrition analysis**

Of the 298 patients who completed the Time 1 in-hospital assessments, 203 were married or living as married. A total of 226 patients (76%) also completed the Time 2 home assessment (including 11 patients who completed a postal version of the assessment). Of the 203 married patients, 162 completed Time 2 (80% of the married patient sample). Married completers and non-completers were similar in clinical, demographic and psychosocial baseline variables. Married patients who did not complete Time 2 were more likely to have had a higher GRACE score ( $F(1, 201) = 9.98, p < 0.05$ ), to have a longer marital duration ( $F(1, 194) = 4.07, p < 0.05$ ) and were more likely to have had a previous heart condition ( $X^2 = 9.11, p < 0.05$ ) than married patients who completed Time 2. Of the 95 unmarried patients, 64 completed the Time 2 home assessment (67% of unmarried patient sample). Unmarried non-completers were more likely to have had a lower GRACE score ( $F(1, 93) = 4.09, p < 0.05$ ), were more likely to have had an URTI in the two weeks preceding their MI ( $X^2 = 5.70, p < 0.05$ ) and were more likely to be employed ( $X^2 = 7.41, p < 0.05$ ).

The Time 2 home assessment comprised of an in-home interview and a postal questionnaire. Of the 162 married patients who completed the Time 2 home assessment interview, 121 (59.6%) also completed and returned the Time 2 postal questionnaire.

Comparing married patients who completed both the Time 2 interview and questionnaire (N=121) and those who completed only the Time 2 interview (N=41) revealed that Time 2 interview and questionnaire completers were more likely to be classified as living in low deprivation ( $X^2 = 21.27$ ,  $p < 0.05$ ), more likely to drink alcohol ( $X^2 = 5.13$ ,  $p < 0.05$ ), less likely to be a current smoker ( $X^2 = 9.62$ ,  $p < 0.05$ ) and more likely to have had an ST elevation MI ( $X^2 = 4.60$ ,  $p < 0.05$ ).

Analysis comparing unmarried patients who completed both the Time 2 interview and questionnaire (N=46) and those who completed only the Time 2 interview (N=18) revealed that Time 2 interview and questionnaire completers were older ( $F(1, 92) = 10.41$ ,  $p < 0.05$ ), had higher GRACE scores ( $F(1, 92) = 8.29$ ,  $p < 0.05$ ), were more likely to be employed ( $X^2 = 6.73$ ,  $p < 0.05$ ) and more likely to be a current smoker ( $X^2 = 8.43$ ,  $p < 0.05$ ) than the questionnaire non completers.

In summary, married patients completing the full assessment (interview and questionnaire) were less deprived, had a shorter marital duration and lower initial GRACE score. They were more likely to drink alcohol, to have had a previous heart condition and to have had an ST elevation MI, and were also less likely to smoke than married patients who did not complete or only partially completed the Time 2 assessment. Unmarried patients completing the full assessment (interview and questionnaire) were older, had higher GRACE, were more likely to be employed and a current smoker, and were also more likely to have had an URTI in the two weeks preceding their hospital admission than those who did not complete or only partially completed Time 2.

#### **8.4.2 Time 3 attrition analysis**

A total of 200 patients (67%) completed the Time 3 telephone assessment, of whom 160 patients also returned their completed postal questionnaire assessment. Of the 141 married patients who completed the Time 3 telephone assessment, 114 also returned the postal questionnaire. Of the 59 unmarried patients who completed Time 3 telephone assessment,

46 also returned the postal questionnaire. Since the Time 2 assessment, 14 (10%) of married patients and 7 (12%) of unmarried patients reported another major cardiac event, with 9 (7%) of the married patients and 9 (16%) of the unmarried patients describing experiencing recurrent cardiac symptoms during the 6 months post ACS. Married non-completers were more likely to have diabetes ( $X^2 = 5.37$ ,  $p < 0.05$ ), less likely to have had a previous MI ( $X^2 = 4.70$ ,  $p < 0.05$ ), more likely to be non-white ( $X^2 = 4.48$ ,  $p < 0.05$ ) and more likely to be moderately or highly deprived ( $X^2 = 13.14$ ,  $p < 0.05$ ). There were no significant differences noted between unmarried completers and non-completers.

#### **8.4.3 Time 4 attrition analysis**

A total of 176 patients (59%) completed the Time 4 assessment, of whom 94 also returned their postal questionnaire. Of the 120 married patients who completed the Time 4 telephone assessment, 68 also returned their questionnaire. Of the 56 unmarried patients who completed the Time 4 telephone assessment, 26 also returned their questionnaire. Since the Time 3 assessment, 11 (20%) married patients and 16 (29%) of unmarried patients reported a further major cardiac event, with 28 (24%) of married patients and 16 (29%) of unmarried patients reporting on-going cardiac symptoms. Married completers at Time 4 were more likely to be white ( $X^2 = 8.45$ ,  $p < 0.05$ ), more likely to drink alcohol ( $X^2 = 4.67$ ,  $p < 0.05$ ) and more likely to report low levels of deprivation ( $X^2 = 6.23$ ,  $p < 0.05$ ) than non-completers. There were no significant differences between unmarried completers and non-completers.

### **8.5 Psychological distress by marital status**

#### **8.5.1 Analytic data set**

Of the 226 patients completing the Time 2 assessment, 162 (71%) were married or living as married, 21 (9%) were single, 19 (8%) were divorced, 8 (3.5%) were separated and 17 (7.5%) were widowed. For the purposes of analysis, patients were categorised as either; **Married** (married and cohabiting, N=162) or **unmarried** (single, divorced, separated and

widowed, N=64). At Time 2, 160 married patients and 63 unmarried patients had valid data for the measure of depression at Time 2. 161 married patients and 62 unmarried patients had valid data for the measure of anxiety. At Time 3, 149 married and 59 unmarried patients completed the assessment. 109 married patients and 43 unmarried patients had valid data for the measure of depression at Time 2. 111 married patients and 44 unmarried patients had valid data for the measure of anxiety. At Time 4, of the 176 patients who completed the telephone interview, 120 were married and 56 were unmarried. 104 married patients and 50 unmarried patients had valid data for the measure of depression at Time 2. 105 married patients and 50 unmarried patients had valid data for the measure of anxiety.

### 8.5.2 Psychological distress at Time 2, 3 and 4 by marital status

The mean scores for the BDI and HADS-anxiety scale for married and unmarried patients at each assessment are depicted in Table 8.3 and 8.4 respectively. For both married and unmarried patients, Time 2, 3 and 4 anxiety scores and Time 2, 3 and 4 depression scores were significantly positively correlated.

**Table 8.3 Mean depression score by marital status at Time 2, 3 and 4**

	Time 2		Time 3		Time 4	
	M (SD) Range	N	M (SD) Range	N	M (SD) Range	N
Married	6.04 (5.37) 0 - 38	160	6.50 (7.00) 0 - 43	109	6.77 (6.61) 0 - 37	104
Unmarried	8.33 (9.12) 0 - 38	63	9.09 (10.28) 0 - 51	43	9.37 (10.46) 0 - 51	50

**Table 8.4 Mean anxiety score by marital status at Time 2, 3 and 4**

	Time 2		Time 3*		Time 4	
	M (SD) Range	N	M (SD) Range	N	M (SD) Range	N
Married	4.61 (3.92) 0 - 17	161	3.30 (3.90) 0 - 20	111	4.23 (3.99) 0 - 19	105
Unmarried	5.55 (5.14) 0 - 20	62	5.48 (5.07) 0 - 18	44	4.93 (4.84) 0 - 19	50

\*Significant difference between married and unmarried patients

The results indicated that unmarried patients had higher anxiety and depression scores at every assessment; however, this difference only reached significance for anxiety scores at Time 3. Multivariate analysis of marital status differentials in distress levels has already been examined within the multiple regression models investigating clinical and demographic influences on distress reported in Chapter 5 of this thesis. There were no significant differences between married and unmarried patient distress at any time point with the exception of anxiety at Time 3 (see section 6.2.1 of Chapter 6) where marital status was found to independently predict level of anxiety at Time 3 ( $\beta=1.02$ ,  $p<0.05$ ) with unmarried patients experiencing significantly higher levels of anxiety. In order to further explore any marital status differences in distress, a series of repeated measures ANOVA were conducted to identify whether there were any differences over time in the patterns of depression and anxiety experienced by married compared with unmarried patients. The first set examined marital status differences in depression or anxiety between Time 2 and Time 3 with marital status as the within person factor, time as the between person factor with age and gender entered as covariates. For depression scores, there was no interaction effect between marital status and time ( $F(1, 131) = 0.02$ ,  $p=0.88$ ). Similarly, for anxiety scores, no interaction between marital status and time was observed ( $F(1, 135) = 0.84$ ,  $p=0.36$ ). These findings indicate the pattern of depression between Time 2 and Time 3 was similar for both married and unmarried patients. However, unmarried patients experienced significantly higher anxiety at Time 3 compared to married patients.

The second set of ANOVA's examined marital status differences in depression or anxiety between Time 2 and Time 4 with age and gender entered as covariates. For depression scores, there was no interaction effect between marital status and time ( $F(1, 132) = 0.53$ ,  $p=0.47$ ). For anxiety scores, no interaction between marital status and time was observed ( $F(1, 132) = 1.04$ ,  $p=0.31$ ). These findings indicate that the pattern of depression and anxiety between Time 2 and Time 4 was also similar for married and unmarried patients suggesting that marital status does not influence the experience of post ACS distress.

The number of married and unmarried patients exceeding the clinical threshold for significant depressive symptomatology on the BDI (score $\geq$ 10) and moderate anxiety on the HADS-A (score $\geq$ 8) is depicted in Table 8.5. At any one assessment point, 13-26% of married patients and 21-30% of unmarried patients reported notable psychological disturbance.

**Table 8.5 Number of patients (%) exceeding depression and anxiety thresholds by marital status at Time 2, 3 and 4**

	Married	Unmarried
<b>Time 2</b>	<b>N= 160 (d), 161 (a)</b>	<b>N= 63 (d), 62 (a)</b>
Depression $\geq$ 10	27 (17%)	16 (25%)
Anxiety $\geq$ 8	36 (22%)	17 (27%)
<b>Time 3</b>	<b>N = 110 (d), 112 (a)</b>	<b>N = 43 (d), 44 (a)</b>
Depression $\geq$ 10	23 (21%)	13 (30%)
Anxiety $\geq$ 8	15 (13%)	9 (21%)
<b>Time 4</b>	<b>N = 104 (d), 105 (a)</b>	<b>N = 50</b>
Depression $\geq$ 10	27 (26%)	14 (28%)
Anxiety $\geq$ 8	20 (19%)	13 (26%)

The percentage of unmarried patients exceeding the cut-off for depression and anxiety was greater than for married patients at every assessment; however, none of these differences reached significance. The proportion of married and unmarried patients reporting above threshold anxiety remained stable over time suggesting no increase in the number of married or unmarried patients reporting high anxiety. There was a significant increase in the proportion of married patients reporting above threshold depression ( $X^2(2) = 5.39, p < 0.05$ ) over time. This increase was not found in the unmarried patients with the proportion reporting above threshold depression remaining stable over time.

In summary, the findings described here suggest that there is limited association between marital status and the experience of distress following ACS. Unmarried patients reported

higher levels of anxiety and depression than married patients at all follow up points but these differences did not reach significance except for a single significant association between anxiety and marital status at Time 3 with unmarried patients more likely to report greater anxiety than married patients. However, this relationship did not persist at Time 4. No interaction effects were identified between marital status and time for either anxiety or depression suggesting similar patterns of distress over the follow up period for both married and unmarried patients. Thus, these results suggest that being married or living with a partner does not significantly reduce risk of developing post ACS distress, nor does being unmarried increase risk of post ACS distress. It is important to consider here that assessment of marital status does not capture the dynamics of marriage and that some marriages may be health enhancing and others may be health impairing. In the TRACE sample, the results suggest that simply being married or cohabiting does not confer protection against post ACS distress; however, the role of marital satisfaction in post ACS distress will be further explored in Section 8.8 in this chapter.

## **8.6 Quality of life by marital status**

### **8.6.1 Analytic data set**

Of the 226 patients who completed Time 2, 162 were married and 64 were unmarried. At Time 2, 152 married and 57 unmarried patients had valid quality of life data. At Time 3, 105 married and 41 unmarried patients had valid data. At Time 4, 101 married and 46 unmarried patients provided complete data.

### **8.6.2 Quality of life at Time 2, 3 and 4 by marital status**

The mean scores for the PCS and MCS quality of life scales scale for married and unmarried patients at each assessment are depicted in Table 8.6 and 8.7 respectively.

**Table 8.6 Mean PCS scores by marital status at Time 2, 3 and 4**

	Time 2		Time 3		Time 4	
	M (SD) Range	N	M (SD) Range	N	M (SD) Range	N
Married	40.31 (10.00) 14 - 59	152	44.56 (9.97) 14 - 59	105	44.75 (10.05) 17 - 64	101
Unmarried	39.90 (8.34) 22 - 58	57	42.78 (10.91) 22 - 59	41	41.83 (10.03) 16 - 57	46

**Table 8.7 Mean MCS scores by marital status at Time 2, 3 and 4**

	Time 2		Time 3		Time 4	
	M (SD) Range	N	M (SD) Range	N	M (SD) Range	N
Married	54.10 (8.78) 26 - 67	152	53.75 (9.07) 22 - 65	105	54.12 (9.32) 19 - 68	101
Unmarried	50.32 (12.04)	57	50.11 (12.39) 15 - 63	41	49.33 (11.09)* 22 - 64	46

The mean scores indicated that unmarried patients had lower quality of life scores than married patients at every follow point. Multivariate analysis of marital status differentials in quality of life has already been examined within the multiple regression models investigating clinical and demographic influences on quality of life reported in Chapter 7 of this thesis. These findings revealed that there were no significant differences between married and unmarried patient physical health related quality of life at any time point. There were, however, some marital status differences in mental health related quality of life. At Time 2, in the univariate analyses, unmarried patients reported significantly worse mental health quality of life compared to married patients. However, this effect did not remain in the multivariate model (see section 7.1 of Chapter 7). Marital status was significantly predictive of mental health quality of life at Time 4, independent of age and gender ( $\beta=-0.20$ ,  $p<0.05$ ) (see section 7.3 of Chapter 7).

To further explore potential marital status variations in quality of life over time, a series of repeated measures ANOVA were conducted to identify variations over time in the patterns of quality of life experienced by married compared with unmarried patients. The first set

examined marital status differences in physical or mental quality of life between Time 2 and Time 3 with marital status as the within person factor, time as the between person factor with age and gender entered as covariates. For PCS scores, there was no interaction effect between marital status and time ( $F(1, 120) = 0.83, p=0.36$ ) although there was a significant main effect of time ( $F(1, 120) = 6.24, p<0.05$ ). For MCS scores, no interaction between marital status and time was observed ( $F(1, 120) = 0.49, p=0.49$ ) but the main effect for marital status was significant ( $F(1, 120) = 7.44, p<0.05$ ). These results suggest that both married and unmarried patients experienced a similar pattern of physical quality of life between Time 2 and Time 3. However, unmarried patients experienced significantly poorer mental health related quality of life at Time 2 and Time 3 compared to married patients.

The second set of ANOVA's explored marital status differences in physical or mental quality of life between Time 2 and Time 4 with age and gender again entered as covariates. There was no interaction effect between marital status and time for PCS ( $F(1, 123) = 0.02, p=0.88$ ) or MCS ( $F(1, 123) = 0.04, p=0.84$ ). There was however a significant effect of marital status in the MCS analysis ( $F(1, 123) = 4.56, p<0.05$ ). These findings indicate that the pattern of PCS between Time 2 and Time 4 was similar for married and unmarried patients while mental health related quality of life was poorer in unmarried patients.

In summary, physical health related quality of life did not significantly vary according to marital status but was more impacted by factors such as age. However, mental health quality of life was significantly better in married compared with unmarried patients at all follow up points. There were no interaction effects between time and marital status which suggests that there were no differences between married and unmarried patients in improvement or deterioration of quality of life over time.

## 8.7 Heart rate variability and marital status

### 8.7.1 Analytic dataset

Heart rate variability (HRV) was collected during the Time 2 home assessment. This assessment was completed by 226 patients and 151 of these patients provided valid heart rate variability data during this assessment. The difference in numbers is due to patient request for postal involvement instead of a home visit, patient refusal for monitor attachment and technical problems with the monitor during recording. The details of how HRV data were gathered are detailed in Chapter 4.

### 8.7.2 Heart rate variability at Time 2 by marital status

HRV was analysed in terms of frequency domain measures (LF-HRV, VLF-HRV, and HF-HRV) and time domain measures (heart rate, pNN50 and RMSSD). The specifics of these measures are described in more detail in Chapter 4. All of the HRV measures (except heart rate) were logged transformed prior to analysis. Mean HRV values by marital status (adjusted for age, gender, ethnicity, deprivation, GRACE score, history of depression, T2 depression status, T2 anxiety status, smoking status and beta-blocker use) are displayed in Table 8.8.

**Table 8.8 Adjusted mean HRV values by marital status at Time 2**

	Married	Unmarried
<i>N</i>	34	113
Heart rate	65.47 (1.12)	69.79 (2.14)
RMSSD	3.70 (0.08)	3.49 (0.15)
pNN50	2.24 (0.11)	1.96 (0.22)
HF*	4.81 (0.14)	4.14 (0.28)
LF*	5.06 (0.12)	4.49 (0.23)
VLF*	4.71 (0.10)	4.22 (0.20)

\*significant difference

Means adjusted for age, gender, ethnicity, deprivation, GRACE score, history of depression, T2 depression status, T2 anxiety status, T1 smoking status and beta-blocker use

Examination of the mean scores revealed that heart rate was higher in unmarried compared with married patients, and all other HRV measures were lower in unmarried compared to married patients suggesting a pattern of impaired heart rate variability in unmarried patients. To determine the significance of these differences, a series of ANCOVA analyses were completed using each measure of HRV as the dependent variable, marital status as the independent variable and age, gender, ethnicity, deprivation, GRACE score, history of depression, Time 2 depression and anxiety status, smoking status (current, previous, never) and beta blocker use at Time 2 as covariates. A number of significant differences were observed. Unmarried patients had significantly impaired HF power ( $F(1, 146) = 4.47, p < 0.05$ ), LF power ( $F(1, 146) = 4.64, p < 0.05$ ) and VLF power ( $F(1, 146) = 4.48, p < 0.05$ ) compared to married patients. Heart rate was elevated in patients with a higher GRACE score at Time 1 ( $F(1, 146) = 4.89, p < 0.05$ ) and older patients ( $F(1, 146) = 7.26, p < 0.05$ ). These findings suggest the presence of a significant marital status effect on frequency domain measures of HRV with unmarried patients having reduced LF, VLF and HF power compared to married patients.

Overall, marital status was found to have a significant association with HRV with reduced HF, VLF and LF power noted in unmarried patients. These effects were independent of age, gender, ethnicity, deprivation, GRACE score, T2 depression and anxiety status, history of depression, smoking status and beta blocker use suggesting a unique role of marriage in biological response following ACS. I noted a similar impact of marital status on VLF and LF power in a sample of suspected CAD patients as reported in Chapter 3, although significant marital status effects were also found for time domain measures in that sample. In the present sample, the RMSSD findings were in the right direction (greater RMSSD in married patients), but the difference was small. These findings are particularly interesting in the light of the lack of association between HRV and the global measures of functional and structural social support (reported in Chapter 7). This suggests that there is something distinctive about being married that is associated with greater HRV which is unrelated to increased

functional social support and also to simply having another person in the social network. Further research is required to further explicate the origins of the marital status differentials in HRV identified here and in Chapter 3.

## 8.8 Marital satisfaction at Time 2, 3 and 4

### 8.8.1 Analytic dataset and measures

Of the 161 married or cohabiting patients, 117 (73%) patients had valid data for the measure of marital satisfaction. Marital satisfaction was assessed at Time 2, 3 and 4 using a 7 item measure self-report measure with scores ranging between 0 – 21 whereby higher scores indicate greater marital satisfaction (Troxel et al., 2005).

Descriptive details of the marital satisfaction scores at Time 2, 3 and 4 are provided in Table 8.9. Marital satisfaction remained stable throughout the 12 month follow up with no significant differences noted between the mean scores at Time 2 and Time 3 ( $F(1, 83) = 1.48, p = 0.23$ ), nor between mean scores at Time 2 and Time 4 ( $F(1, 79) = 3.62, p = 0.06$ ). All the scores were highly positively correlated.

**Table 8.9 Marital satisfaction at Time 2, 3 and 4**

	Time 2	Time 3	Time 4
<b>Marital satisfaction</b>			
<b>Mean (SD)</b>	15.80 (3.88)	16.37 (4.26)	16.55 (3.74)
<b>Range</b>	0 - 21	1 - 21	7 - 21
<b>N</b>	117	97	98

The marital satisfaction score distribution was highly positively skewed at all assessments with the 25<sup>th</sup> percentile represented by a score of <14 and the mean score close to the maximum score of 21. Subsequently, scores were aggregated into three categories according to three equal tertiles: low marital satisfaction (<14), moderate marital satisfaction (15 - 18 percentile score) and high marital satisfaction (>19). The frequency of scores within each marital satisfaction category is displayed in Table 8.10.

**Table 8.10 Aggregated marital satisfaction score frequency at Time 2, 3 and 4**

	<b>Time 2</b>	<b>Time 3</b>	<b>Time 4</b>
	<i>N (%)</i>	<i>N (%)</i>	<i>N (%)</i>
<b>Total N</b>	117	84	80
<b>Low</b>	38 (33)	25 (30)	22 (28)
<b>Moderate</b>	46 (39)	32 (38)	32 (40)
<b>High</b>	33 (28)	27 (32)	26 (32)

### 8.8.2 The association between demographic factors and marital satisfaction

Marital satisfaction may vary according to certain demographic and clinical features. A series of Pearson and point-by-serial correlations were run between Time 2 marital satisfaction scores and a number of important demographic and clinical factors including age, gender, deprivation, education, ethnicity, GRACE score, previous MI and history of depression to identify any significant associations. Age ( $r(115) = 0.26, p < 0.05$ ), gender ( $r(115) = -0.25, p < 0.05$ ), GRACE score ( $r(115) = 0.22, p < 0.05$ ) and history of depression ( $r(115) = -0.23, p < 0.05$ ) were all found to be significantly related to marital satisfaction. Based on these correlations, multiple regression analysis was conducted using Time 2 marital satisfaction as the dependent variable with age, gender, GRACE score and history of depression as independent variables. The model explained a significant proportion of variance in marital satisfaction ( $R^2 = 0.15, F(4, 112) = 5.03, p < 0.05$ ) with only gender identified as a significant independent predictor (Table 8.11) with female patients reported lower marital satisfaction compared to men.

**Table 8.11 Demographic influences on marital satisfaction at Time 2**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	14.54	9.62 – 19.46		5.83	0.001
Age	0.07	-0.04 – 0.19	0.20	1.30	0.196
Gender*	-3.05	-5.46 – - 0.63	-0.22	-2.50	0.014
GRACE score	0.00	-0.04 – 0.05	0.03	0.18	0.858
Depression history	-1.43	-2.97 – 0.12	-0.16	-1.82	0.070

\*Significant independent predictor

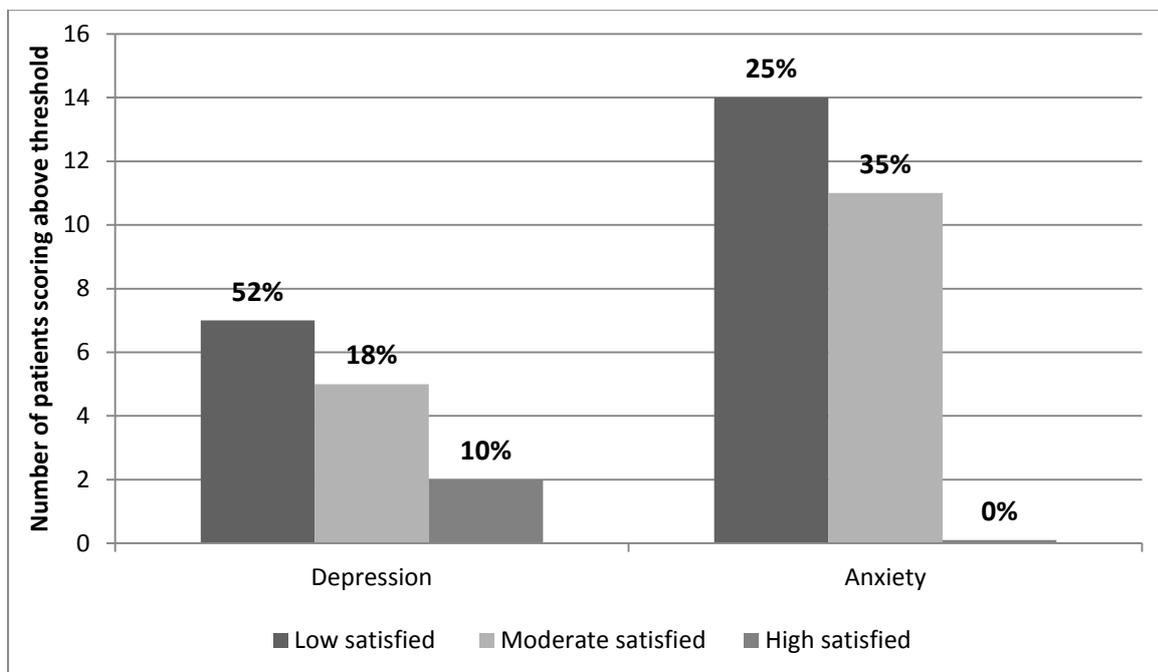
### 8.8.3 Summary: Marital satisfaction after ACS

The majority of the sample were married and reported moderate to high marital satisfaction, with a third or less reporting low marital satisfaction at each follow up point. Marital satisfaction remained stable over time with no significant change noted in mean marital satisfaction score between Time 2 and Time 3, nor between Time 2 and Time 4. Marital satisfaction was found to be significantly lower among female patients compared to male patients.

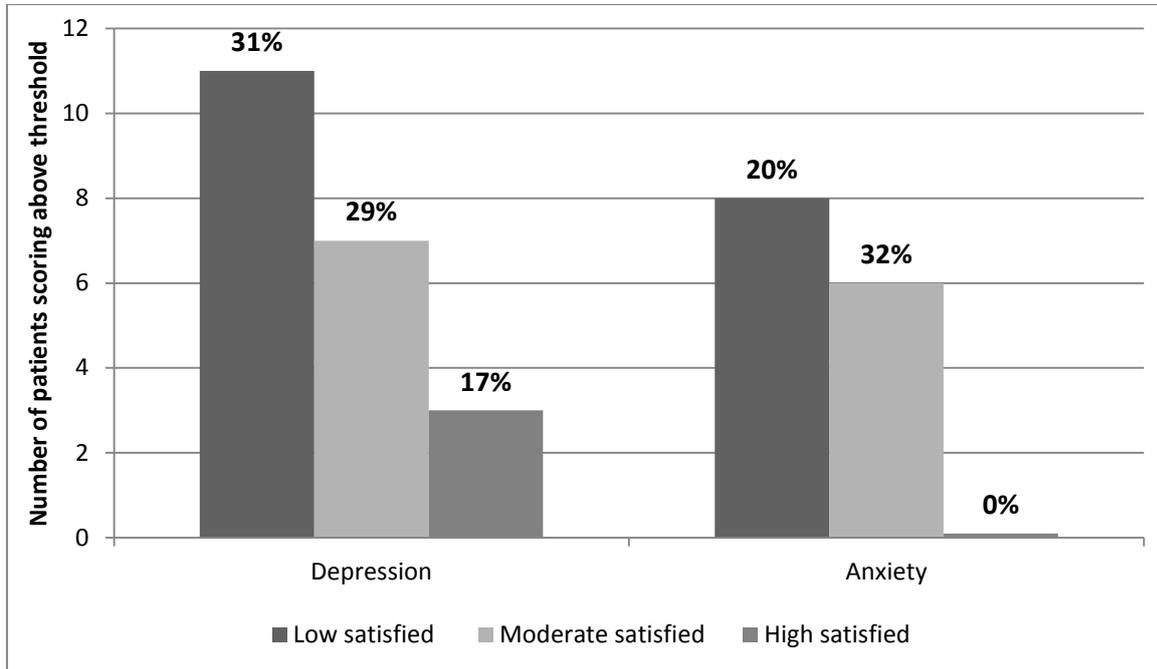
## 8.9 Comparing psychological distress at Time 2, 3 and 4 by level of marital satisfaction

The number of patients exceeding the clinical threshold for significant depressive symptomatology on the BDI (score $\geq$ 10) and moderate anxiety on the HADS-A (score $\geq$ 8) by marital satisfaction was explored and is depicted for each assessment point in Figures 8.1, 8.2 and 8.3.

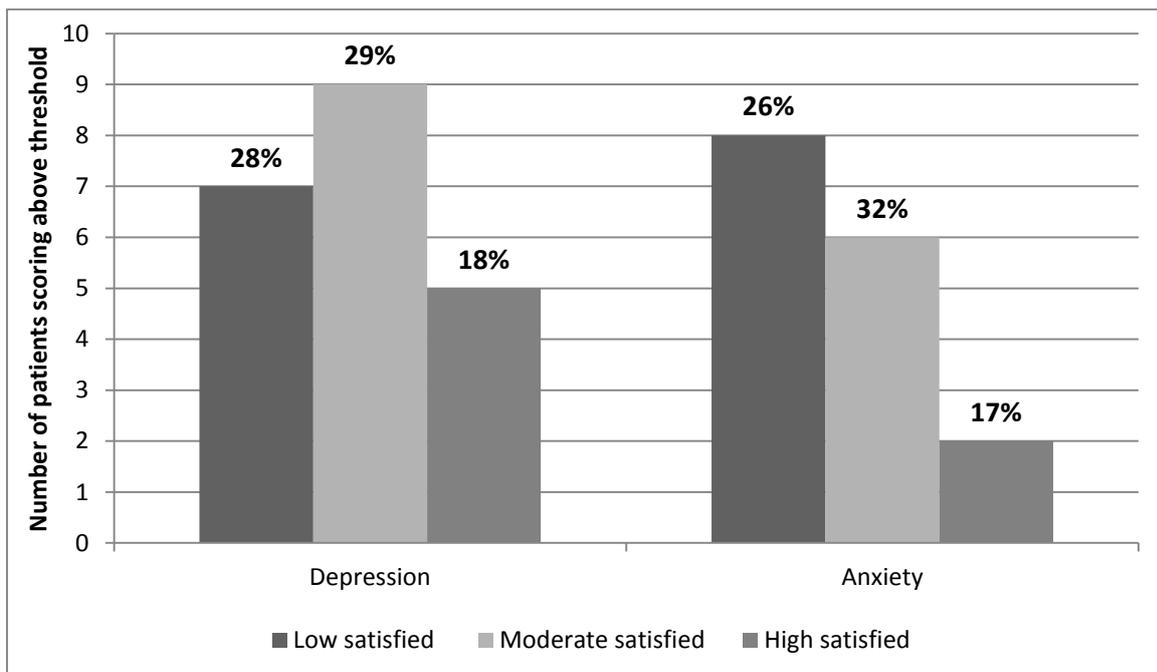
**Figure 8.1 Number of patients (% of each marital group) exceeding depression and anxiety thresholds by marital satisfaction at Time 2**



**Figure 8.2 Number of patients (% of each marital group) exceeding depression and anxiety thresholds by marital satisfaction at Time 3**



**Figure 8.3 Number of patients (% of each marital group) exceeding depression and anxiety thresholds by marital satisfaction at Time 4**



A gradient effect was observed for married patients with the percentage of low satisfied married patients exceeding the distress thresholds being higher than moderate and high satisfied patients, and the percentage of high satisfied married patients exceeding the distress threshold being lower than moderate or low satisfied patients at every assessment, except Time 4 depression. These differences reached significant for depression at Time 3 ( $X^2(2) = 7.85, p < 0.05$ ), and for anxiety at Time 2 ( $X^2(2) = 13.15, p < 0.05$ ) and at Time 3 ( $X^2(2) = 6.31, p < 0.05$ ). The proportion of patients from all marital groups reporting above threshold anxiety remained stable over time. The proportion of low satisfied patients reporting above threshold depression also remained stable over time. There was a significant increase in the number of moderately satisfied patients reporting above threshold depression ( $X^2(2) = 6.33, p < 0.05$ ), and a borderline significant increase in the number of high satisfied patients reporting above threshold depression ( $X^2(2) = 6.00, p = 0.05$ ).

In order to determine whether level of marital satisfaction was associated with distress at any follow up point, a series of ANOVA analyses were conducted with marital satisfaction group (low, moderate, high) as the independent variable, anxiety or depression score (Time 2, Time 3 or Time 4) as the dependent variable with age, gender, deprivation level, ethnicity, GRACE score and depression history included as covariates. The adjusted mean depression and anxiety scores by level of marital satisfaction at each assessment point are displayed in Table 8.12 and Table 8.13. At Time 2, level of marital satisfaction was significantly associated with depression score ( $F(2, 112) = 3.56, p < 0.05$ ) and anxiety score ( $F(2, 105) = 2.83, p < 0.05$ ) suggesting an independent association between marital satisfaction and distress at Time 2.

At Time 3, an additional covariate, anxiety or depression score at Time 2, was included in the model. The results indicated that only depression score at Time 2 was significantly associated with depression score at Time 3 ( $F(1, 78) = 20.44, p < 0.05$ ). However, repeating the analysis without Time 2 depression score as a covariate, revealed that marital

satisfaction level was significantly associated with Time 3 depression level ( $F(2, 79) = 2.20, p < 0.05$ ). Deprivation was also found to be associated with Time 3 depression score ( $F(1, 79) = 7.47, p < 0.05$ ). Similarly, only anxiety score at Time 2 was independently associated ( $F(1, 79) = 63.82, p < 0.05$ ) with anxiety score at Time 3. However, when Time 2 anxiety was not included, level of marital satisfaction emerged as significant ( $F(1, 80) = 2.64, p < 0.05$ ). These findings indicate an association between level of marital satisfaction and distress at Time 3, although this was not independent of distress at Time 2. Patients reporting low marital satisfaction at Time 2 were more likely to experience elevated anxiety and depression at Time 3.

At Time 4, only depression score at Time 2 was significantly related to depression score at Time 4 ( $F(1, 70) = 56.51, p < 0.05$ ). No additional findings were identified with exclusion of Time 2 depression score. Anxiety score at Time 2 was the only independent associate of anxiety at Time 4 ( $F(1, 79) = 63.82, p < 0.05$ ); however, exclusion of Time 2 anxiety score as a covariate revealed that level of marital satisfaction was significantly associated with Time 4 anxiety ( $F(2, 72) = 2.07, p < 0.05$ ). These results indicate that marital satisfaction did not exhibit any relationship with depression at Time 4. Level of marital satisfaction was found to be significantly associated with anxiety at Time 4, when anxiety at Time 2 was not controlled.

#### 8. 12 Mean depression score by marital satisfaction at Time 2, 3 and 4

	Time 2	Time 3	Time 4
<b>Low marital satisfaction</b>	6.37 (0.66)	7.39 (0.96)	6.96 (1.15)
<b>Moderate marital satisfaction</b>	5.54 (0.60)	6.09 (0.82)	7.57 (0.99)
<b>High marital satisfaction</b>	3.70 (0.74)	4.39 (1.00)	4.39 (1.15)

*Means adjusted for age, gender, deprivation level, GRACE score, ethnicity and history of depression*

### 8. 13 Mean anxiety score by marital satisfaction at Time 2, 3 and 4

	Time 2	Time 3	Time 4
<b>Low marital satisfaction</b>	4.95 (0.54)	4.66 (0.67)	5.05 (0.74)
<b>Moderate marital satisfaction</b>	4.57 (0.50)	3.80 (0.57)	4.27 (0.67)
<b>High marital satisfaction</b>	3.04 (0.62)	2.41 (0.68)	2.87 (0.76)

*Means adjusted for age, gender, deprivation level, GRACE score, ethnicity and history of depression*

To summarise, a clear gradient of distress by level of marital satisfaction emerged when a categorical approach to distress was utilised. This reached significance at Time 3 for depression scores and at Time 2 and Time 3 for anxiety scores. These findings suggest that low levels of marital satisfaction are associated with above threshold distress particularly within the first six months following ACS. Level of marital satisfaction was associated with mean anxiety score at every follow up and was also associated with mean depression score at Time 2 and Time 3. These associations were independent of age, gender, deprivation, GRACE score and history of depression. These findings suggest that post ACS patients in marriages or cohabiting relationships demarcated by low marital satisfaction are more vulnerable to anxiety and depression during recovery and beyond whereas those patients in relationships with high marital satisfaction are less at risk of post ACS distress.

### 8.10 Comparing quality of life at Time 2, 3 and 4 by level of marital satisfaction

The influence of marital satisfaction on level of quality of life experienced by patients was explored in a series of ANOVA analyses using marital satisfaction group (low, moderate, high) as the independent variable, PCS or MCS score (Time 2, Time 3 or Time 4) as the dependent variable with age, gender, deprivation level, GRACE score, previous CHD, employment status and depression history included as covariates. The adjusted mean PCS and MCS scores by level of marital satisfaction at each assessment point are displayed in Table 8.14 and Table 8.15.

#### 8.14 Mean PCS scores by marital satisfaction level at Time 2, 3 and 4

	Time 2	Time 3	Time 4
<b>Low marital satisfaction</b>	40.13 (1.63)	44.31 (1.93)	43.31 (1.94)
<b>Moderate marital satisfaction</b>	40.78 (1.52)	45.29 (1.65)	46.49 (1.69)
<b>High marital satisfaction</b>	39.02 (1.91)	44.00 (2.05)	43.30 (2.11)

*Means adjusted for age, gender, deprivation level, previous CHD, employment status, ethnicity, GRACE score and history of depression. Time 3 and Time 4 means also adjusted for Time 2 mean score.*

#### 8.15 Mean MCS scores by marital satisfaction level at Time 2, 3 and 4

	Time 2*	Time 3	Time 4
<b>Low marital satisfaction</b>	52.54 (1.37)	54.05 (1.62)	54.23 (1.57)
<b>Moderate marital satisfaction</b>	55.04 (1.28)	53.83 (1.37)	53.35 (1.35)
<b>High marital satisfaction</b>	57.15 (1.61)	55.56 (1.70)	54.96 (1.69)

*Means adjusted for age, gender, deprivation level, previous CHD, employment status, ethnicity, GRACE score and history of depression. Time 3 and Time 4 means also adjusted for Time 2 mean score.*

At Time 2, level of marital satisfaction was significantly associated with MCS score ( $F(2, 107) = 3.26, p < 0.05$ ) indicative of an independent association between marital satisfaction and mental health related quality of life at Time 2. No significant association between marital satisfaction and PCS score was observed. The analysis also showed that level of deprivation was also associated with PCS score ( $F(1, 107) = 4.46, p < 0.05$ ).

At Time 3, an additional covariate, PCS or MCS score at Time 2, was also included in the model. The results indicated that at Time 3 neither PCS nor MCS score were significantly associated with marital satisfaction with Time 2 scores being the only independent predictors of Time 3 scores (PCS:  $F(1, 78) = 9.48, p < 0.05$ ), MCS: ( $F(1, 78) = 15.21, p < 0.05$ ). The exclusion of Time 2 PCS or MCS score did not reveal any different findings. These findings suggest no relationship between quality of life at Time 3 and marital satisfaction.

The same analyses utilised for Time 3 were run using Time 4 PCS and MCS score. Analogous to the Time 3 findings, no significant relationship was observed between Time 4

PCS or MCS score and marital satisfaction. Previous CHD was significantly related to PCS score ( $F(1, 74) = 4.84, p < 0.05$ ). Time 2 scores were strongly predictive of Time 4 scores (PCS:  $F(1, 74) = 10.15, p < 0.05$ ), MCS: ( $F(1, 74) = 35.15, p < 0.05$ ). No additional findings were identified with the exclusion of Time 2 scores. Level of marital satisfaction was not associated with quality of life at Time 4.

Overall the findings indicate that marital satisfaction was cross sectionally associated with mental health quality of life at Time 2 independent of age, gender, deprivation, previous CHD, employment status, ethnicity, GRACE score and history of depression. This finding, in the context of the elevated levels of anxiety and depression in low satisfied married patients compared to high satisfied married patients at Time 2, reflects the burden of distress upon patients in poor quality relationships. The association between mental health quality of life and marital satisfaction did not persist at Time 3 or Time 4 suggesting a short term quality of life impact. Causal direction cannot be established due to the cross sectional nature of this association. Nonetheless, my previous identification of higher levels of distress and lower levels of quality of life in low satisfied married patients highlight the increased negative emotional impact of ACS on this group of patients which in turn poses concomitant negative prognostic implications. No association was found between level of marital satisfaction and physical health quality of life at any follow up point.

## **8.11 HRV and marital satisfaction**

### **8.11.1 Analytic dataset**

Heart rate variability (HRV) was collected during the Time 2 home assessment and has been previously described in section 8.6.1 of this chapter. Of the patients providing valid HRV data, 115 were married and 87 of these patients provided valid marital satisfaction data. The details of how HRV data were gathered are detailed in Chapter 4.

### 8.11.2 Heart rate variability and marital satisfaction at Time 2

HRV was analysed in terms of frequency domain measures (LF-HRV, VLF-HRV, and HF-HRV) and time domain measures (heart rate, pNN50 and RMSSD). The specifics of these measures were described in more detail in Chapter 4. All of the HRV measures (except heart rate) were logged transformed prior to analysis. Adjusted mean HRV values by level of marital satisfaction (low, moderate and high; group aggregation is described in section 8.8) are displayed in Table 8.16.

**Table 8.16 Adjusted mean HRV values by level of marital satisfaction at Time 2**

Marital satisfaction	LOW	MODERATE	HIGH
<i>N</i>	29	38	21
<b>Heart rate</b>	65.88 (2.32)	65.69 (1.96)	69.38 (2.64)
<b>RMSSD</b>	3.78 (0.16)	3.77 (0.14)	3.37 (0.19)
<b>pNN50</b>	2.34 (0.24)	2.32 (0.20)	1.67 (0.27)
<b>HF</b>	4.92 (0.31)	4.84 (0.27)	4.25 (0.36)
<b>LF</b>	4.99 (0.26)	5.23 (0.22)	4.46 (0.29)
<b>VLF</b>	4.45 (0.22)	4.92 (0.19)	4.29 (0.25)

Adjusted for age, gender, deprivation, ethnicity, history of depression, GRACE score, beta blocker use, depression score at T2 and T1 smoking status

Examination of the adjusted mean scores revealed no consistent pattern or variation in heart rate variability according to level of marital satisfaction. A series of ANCOVA analyses were completed using each measure of HRV as the dependent variable, marital satisfaction group (low, moderate, high) as the independent variable and age, gender, ethnicity, history of depression, depression score at Time 2, smoking status at T1 (current, previous, never) and beta blocker use as covariates. No significant differences were observed according to level of marital satisfaction. Gender, ethnicity, depression score at Time 2 and history of depression were found to have a significant effect on various indices of HRV.

In summary, the results indicate that HRV in married patients was not significantly influenced by the level of marital satisfaction reported at Time 2. These are particularly intriguing results in the context of my previous findings that identified the presence of marital status differentials in HRV whereby unmarried patients had significantly reduced HRV compared to married patients, and also identified no association between functional and structural social support and HRV. These combined results suggest that, in the TRACE sample, simply being married or cohabiting had a protective effect on HRV regardless of the satisfaction experienced in that relationship or the social support derived from that relationship. There is indeed something unique about being married when it comes to HRV.

## **8.12 Chapter discussion**

### **8.12.1 Marital status and distress after ACS**

A key pathway through which being married may facilitate better recovery and prognosis following ACS may be due to the differential experience of distress in married compared to unmarried patients. I explored whether marital status influenced post ACS short and long term distress as the identification of this relationship would provide good evidence for the presence of a psychological pathway between marital status and ACS outcomes. However, the results provided limited support for my hypothesis that being married or cohabiting offers protection against distress in ACS patients. Married patients reported lower anxiety and depression at every follow up assessment; however, these differences were only significant at Time 3 where unmarried patients had significantly elevated anxiety compared with married patients ( $\beta=1.02$ ,  $p<0.05$ ). The repeated measures analysis also revealed no significant relationship between marital status and change in anxiety or depression between follow up points. High deprivation, unemployment, younger age and being female were associated with greater risk of distress at various points during the 12 month follow up.

A number of recent studies have begun to identify similar findings regarding marital status differentials in distress in coronary patients. In a sample of 288 MI patients followed up for 18

months, Hanssen, Nordrehaug, Eide, Bjelland, & Rokne, (2009) reported no marital status differences in anxiety or depression. Chung et al, (2009) found no difference between married and unmarried patients regarding depressive symptoms assessed using the BDI in 166 heart failure patients followed up for 4 years, although married patients had longer event free survival compared to unmarried patients. In a study of nearly 500 ACS patients, marital status was not related to the presence of a clinical anxiety disorder as defined by the DSM-IV (Parker, Owen, Brotchie, & Hyett, 2010). Similarly, Akhtar, Malik, & Ahmed, (2004) found no marital status difference in depression or anxiety at one week post event in a sample of 100 MI patients. The limited marital status effects in distress observed within the TRACE sample fits within these recent research findings. My findings do, however, contrast with the increased risk of psychological disorder amongst never married compared with married healthy populations (Scott et al., 2010).

The relationship between marriage and distress in post ACS patients does not appear to be as simple as hypothesised. It has been discussed throughout this thesis that there is clear and consistent research linking marital status and post ACS survival. Within a psychobiological framework marital status is hypothesised to influence ACS outcomes through a variety of mechanisms including lower levels of psychological distress among the married. It has been suggested that being married buffers an individual against the risk of becoming depressed or anxious or against the negative corollaries of these states if they do occur following an ACS. My results reveal a non-significant trend towards lower depression and anxiety in married compared with unmarried patients. However, these differences did not reach significance except for anxiety at Time 3 providing limited overall support for my hypotheses and indicating a lesser role for psychological distress in trajectories between marital status and post ACS outcome than hypothesised.

### **8.12.2 Marital status and quality of life after ACS**

Quality of life may represent an important part of the pathway between marital status and ACS prognosis as better quality of life has been associated with better recovery, and quality of life has been found to be influenced by marital status. My results revealed that unmarried patients reported lower mean quality of life scores at every follow up point suggesting worse quality of life among unmarried compared with married patients. But, the results from the multivariate analyses reported in Chapter 7 (sections 7.1, 7.2 and 7.3.) and the repeated measures analyses completed in this chapter revealed that these differences were not significant at any follow up point for physical health related quality of life suggesting that unmarried and married patients experienced similar levels of quality of life with regard to their physical functioning. However, there were some significant differences in mental health quality of life. In the multivariate analyses reported previously in Chapter 7, marital status was predictive of quality of life at Time 4 ( $\beta=-0.20$ ,  $p<0.05$ ) independent of age and gender. Furthermore, the results of the repeated measures ANOVA indicated a significant marital status difference in MCS score across Time 2 and Time 3, and Time 2 and Time 4. These findings do suggest a trend towards worse mental health related quality of life over time in unmarried patients. The lack of an associative relationship at Time 2 and Time 3 and the presence of such a relationship at Time 4 suggest that the importance of marital status in influencing mental health related quality of life may emerge later in post ACS recovery.

The research base investigating marital status differences in post ACS quality of life is currently limited and heterogeneous. In a study of predictors of health related quality of life in women with coronary artery disease who had been hospitalised for an acute event and were attending a secondary prevention programme, Christian, Cheema, Smith, & Mosca, (2007) found that being married was associated with significantly better quality of life than being unmarried at 6 months. Lane, Carroll, Ring, Beevers, & Lip, (2001) found that living alone and not having a partner were significantly predictive of quality of life at 12 months in a sample of 288 post MI patients. Interestingly, Lie, Arnesen, Sandvik, Hamilton, & Bunch,

(2010) identified marital status differentials in physical health related quality of life but not mental health quality of life in 185 patients followed up at 6 months following CABG surgery utilising the SF-36, which contrasts with our results indicating no marital status influence on physical health related quality of life. In a randomised controlled trial, Oldridge et al., (1998) explored the sociodemographic and clinical predictors of health related quality of life following a cardiac rehabilitation intervention in 201 post MI patients. Health related quality of life was assessed using a number of different instruments (Quality of Life After Myocardial Infarction Questionnaire, The Quality of Wellbeing Scale and the Time Trade Off scale) during hospitalisation, at 8 weeks and 12 months post MI. They identified a very limited role of marital status. Marital status explained a small amount of variance (4.5%) in baseline Time Trade Off (TTO) scale scores but did not explain change over time in TTO score at 8 weeks or 12 month follow. Marital status was not associated with any other quality of life measure at any time point. The findings of these different studies are hard to integrate as they have used diverse quality of life measures within varied cardiac populations and were conducted in different eras of cardiac care. My findings using a standardised measure of quality of life in a large sample of post ACS patients indicate no role for marital status in predicting physical health related quality of life but do suggest a tendency for unmarried patients to have poorer mental health related quality of life which may increase risk of poor ACS outcome.

### **8.12.3 Marital status and HRV**

A potential trajectory through which marital status may directly impact upon ACS prognosis and outcome is via HRV with reduced HRV identified as a clear indicator of higher post ACS mortality (Bigger et al., 1992; La Rovere, Bigger, Jr., Marcus, Mortara, & Schwartz, 1998). The TRACE results indicated that HF, VLF and LF power were significantly reduced in unmarried compared with married patients independent of age, gender, ethnicity, deprivation, GRACE score, history of depression, T2 depression and anxiety status, smoking status and beta-blocker use. The particularly prominent impact of marital status on frequency

domain power is an important finding as increased risk of post ACS mortality has been most strongly associated with reduced frequency domain power (Bigger et al., 1992). Thus, these findings suggest the presence of a biological trajectory from unmarried state to poorer survival via reduced HRV indicating a biological impact of the most common social tie, the marital relationship. These findings expand the current extremely limited research base which has highlighted associations between social isolation (Horsten et al., 1999), lower social support (Shin et al., 2012) and reduced HRV to suggest that marital status may also influence HRV. These results are also consistent with my previous results reported in Chapter 3 indicating significant marital status differentials in HRV in a sample of suspected CAD patients. Overall, reduced HRV appears to be an important explanatory mechanism for the greater risk of poorer prognosis noted amongst unmarried patients.

There are a number of potential factors that may explain these marital status differentials in HRV which have been previously discussed in Chapter 3. One possibility is that being married buffers the negative HRV impact of psychopathological states. Negative emotional states, in particular depression, have been found to negatively impact upon HRV in physically healthy, CHD and MI populations (Thayer & Lane, 2007; Rottenberg, 2007) and as previously discussed throughout this thesis are common following ACS. However, the TRACE analyses controlled for both depression at Time 2 and history of depression, and the findings were independent of these covariates. Furthermore, this chapter has revealed limited marital status differences in the experience of post ACS distress at Time 2. A further possible explanation is that these differences in HRV may be attributable to differences in health behaviour between unmarried and married patients. Health damaging behaviours (in particular, smoking and low physical activity) have been associated with lowered HRV (Thayer & Lane, 2007) and are more prominent among unmarried individuals. The greater level of social control exerted by marital partners may contribute to health behaviour differences between married and unmarried populations (Umberson, Crosnoe, & Reczek, 2010). Furthermore, unhealthy behaviours have also been found to play a central role in

explaining marital status differentials in CHD mortality (Molloy, Stamatakis, Randall, & Hamer, 2009). Recent research also suggests that HRV (and in particular HF-HRV) may be a physiological indicator of self-regulatory effort (Segerstrom & Nes, 2007; Reynard, Gevirtz, Berlow, Brown, & Boutelle, 2011). Successful post ACS recovery and CAD management involves significant lifestyle change and adjustment of behaviour requiring considerable self-regulation. Although behaviour is not explicitly addressed within this thesis, a brief analysis revealed that in the TRACE sample there were no marital status differences in physical activity<sup>†</sup> but unmarried patients were more likely to be a current smoker at the time of their ACS compared to married patients. In the HRV analysis, smoking status was included as a covariate and was not significant in influencing HRV. These findings suggest the marital status differentials in HRV observed in the TRACE are unlikely to be due to differences in physical activity or smoking behaviour at Time 2. It is possible that other behaviours may influence HRV and these merit investigation. There is also a significant body of research indicating an association between negative marital interaction and conflict with increased cardiovascular reactivity and reduced HRV (Smith et al., 2011; Kiecolt-Glaser & Newton, 2001). This raises the possibility of a gradient of HRV amongst married patients with happily married patients exhibiting greatest HRV and unhappily married patients having the lowest HRV. My investigation into marital satisfaction and HRV in the TRACE patients, discussed later in this discussion, suggests that the presence of such a gradient is unlikely. Thus, there are many potential mechanisms through which marital status may influence HRV, and my identification of marital status differences in HRV does provide evidence of a biological trajectory between marital status and post ACS prognosis.

*† Physical activity was assessed as the amount of walking in minutes per day reported by the patient at the Time 2 assessment. No difference was found between married ( $M=32.92$ ,  $SD=36.25$ ) and unmarried patients ( $M=44.00$ ,  $SD=69.09$ ) at Time 2.*

#### **8.12.4 Marital satisfaction and distress after ACS**

Lower levels of marital satisfaction have been associated with higher levels of depression and anxiety and various other psychological disorders in community and clinical samples (for example, Whisman, 2007; Whisman, 1999). Various measures of poor marital functioning including poor marital satisfaction have also been associated with worse prognosis amongst coronary patients (Orth-Gomer et al., 2000; Rohrbaugh, Shoham, & Coyne, 2006; Coyne & Anderson, 1999; King & Reis, 2012). Based on this research, I hypothesised that marital satisfaction would be predictive of post ACS distress. The results illustrated a significant gradient association between mean level of marital satisfaction assessed at Time 2 and anxiety at all follow points, as well as between level of marital satisfaction and depression at Time 2 and 3. This association was independent of age, gender, deprivation, education, ethnicity, GRACE score, previous MI and history of depression. Patients reporting lower levels of marital satisfaction were more likely to experience elevated anxiety and depression compared to those patients reporting higher levels of marital satisfaction. Similarly, using a categorical approach, a gradient of distress was observed by level of marital satisfaction which reached significance at Time 3 for depression and at Time 2 and Time 3 for anxiety. Patients indicating lower levels of marital satisfaction were more likely to experience above threshold depression at the 6 month follow up, and above threshold anxiety at both ten days post ACS and the 6 month follow up. In the context of the limited association between marital status and distress, these findings illustrate that simply being married does not offer protection against distress; but being in a highly satisfying marriage does reduce risk of distress compared to those in a low satisfaction marriage.

The relationship between marital satisfaction and psychological distress following ACS has not been well explored. One study identified a similar association between psychological adjustment and marital quality in 198 male MI or cardiac surgery patients at 3 months post cardiac event (Brecht, Dracup, Moser, & Riegel, 1994). However, this study was conducted in a very different time with regard to cardiac care, utilised only male CABG patients and

was based only upon post CABG patients followed up for a short time suggesting a clear need for updating and extending. The TRACE study provides a more robust investigation in a larger sample of mixed gender ACS patients who were followed up for one year. The findings reveal that patients in low satisfaction marriages were at increased risk of both anxiety and depression that persists beyond hospital discharge. This suggests that the marital satisfaction-distress link may be an important mediating pathway between marital satisfaction and coronary outcome, in the context of the prognostic dangers of psychological distress in ACS. Identification of factors that can help to stratify patients needing more intensive support may help to improve outcome and subsequently married patients who report low marital satisfaction during admission may benefit from more support during cardiac rehabilitation. It is also plausible that there may be aspects of marital satisfaction that are particularly protective. For example, research suggests that sexual functioning is often particularly adversely impacted by ACS which may have concomitant effects on marital satisfaction. Research suggests that fears surrounding post ACS sexual functioning may negatively influence marital satisfaction and increase risk of distress (Kazemi-Saleh, Pishgou, Assari, & Tavallaii, 2007; Kazemi-Saleh, Pishgoo, Farrokhi, Fotros, & Assari, 2008a; Kazemi-Saleh et al., 2008b). Thus, it may be that problems relating to the sexual aspect of marital satisfaction may be particularly important in the development of distress. Dimensional analysis may provide greater insight in the specific marital issues that post ACS patients face and how these specific issues are differentially related to distress. Overall, the robust association between marital satisfaction and distress is a valuable finding, particularly in the context of the limited marital status differences in distress, as it highlights the vital importance of considering the quality of marital relationships.

#### **8.12.5 Marital satisfaction and quality of life after ACS**

There has been limited research investigating differences in quality of life following ACS due to variations in levels of marital satisfaction. Research findings have identified marital satisfaction differentials in quality of life in post CABG and recently diagnosed CHD patients

(Elizur & Hirsh, 1999; Brecht et al., 1994). Furthermore, research into other chronic illnesses and conditions (including diabetes, poor vision, physical disability) has found that marital satisfaction does influence quality of life (for example, Trief, Himes, Orendorff, & Weinstock, 2001; Bookwala, 2011; Bookwala & Franks, 2005). In the context of these findings and the relationship between quality of life and prognosis following ACS, I hypothesised that lower levels of marital satisfaction would predict worse quality of life over time. My results indicated that low levels of marital satisfaction at Time 2 were significantly associated with lower levels of mental health quality of life at Time 2 independent of age, gender, deprivation, previous CHD, employment status, ethnicity, GRACE score and history of depression. The association between mental health quality of life and marital satisfaction did not persist at Time 3 or Time 4 suggesting a short term quality of life impact. As previously discussed, elevated levels of depression and anxiety were also noted in low satisfied married patients at Time 2. Thus, married patients in poor quality marriages were more likely to experience a greater burden of distress and poor quality of life in the early weeks following ACS compared to married patients in better quality marriages. No differences were noted in physical health related quality of life. These findings are the first to illustrate an independent association between marital satisfaction and early post ACS quality of life. However, as these analyses were cross sectional in nature and no longitudinal association between marital satisfaction at Time 2 and quality of life at Time 3 or 4 was noted, it is also possible that poor quality of life may have negatively impact upon marital satisfaction whereby a patient who was not coping well and experiencing poor quality of life may increase strain and conflict within their marital relationship resulting in concomitant reduced marital satisfaction. It is not possible to distinguish which of these causal patterns is correct and subsequently it cannot be assumed that marital satisfaction is the driving force.

These findings are important as they provide the first well-controlled, longitudinal test of the relationship between marital satisfaction and quality of life during post ACS recovery, and significantly extend the current literature in coronary patients. My findings draw attention to

the relationship between marital satisfaction and quality of life in the early weeks following discharge which is particularly noteworthy as the TRACE patients had a generally high level of marital satisfaction and quality of life. Thus, the associations may be even greater where individuals experience very poor marital satisfaction and/or quality of life.

#### **8.12.6 Marital satisfaction and HRV**

The analyses revealed no significant association between level of marital satisfaction and various indices of HRV. This is interesting in the context of the significant association between marital status and various indices of HRV. No other studies were identified that investigated the influence of marital satisfaction on HRV in post ACS patients. A few general population studies have found associations between low marital satisfaction and reduced HRV. For example, Smith et al, (2011) identified a significant correlation between HF-HRV and self-reported marital quality in 114 married females. Similarly, Carrere et al., (2005) found a significant main effect of marital satisfaction on HF-HRV and IBI in a sample of 54 married couples. Lower marital satisfaction was associated with reduced HF-HRV power and shorter IBI. However, most studies investigating links between HRV and marital quality have focused more upon the immediate impact of marital interaction on cardiovascular reactivity which has often been conducted within laboratory settings (Nealey-Moore, Smith, Uchino, Hawkins, & Olson-Cerny, 2007; Smith et al., 2009; Carels, Szczepanski, Blumenthal, & Sherwood, 1998). These studies have identified patterns of reduced HRV during and following negative marital interactions that have been identified as particularly pronounced among women. In a more naturalistic setting, Holt-Lunstad, Birmingham, & Jones, (2008) identified that marital adjustment and satisfaction were significantly predictive of ambulatory blood pressure. However, the study utilised a sample of 303 healthy and young (mean age=31) adult participants which differs substantially from the characteristics of the TRACE sample.

Thus, the lack of marital satisfaction effect on HRV is surprising in the context of this albeit somewhat limited research. Gender may be an important factor as marital conflict has been found to exert a greater cardiovascular impact on women compared to men (Robles & Kiecolt-Glaser, 2003) and marital satisfaction has been observed as lower in wives compared to husbands (Schumm, Webb, & Bollman, 1998). The TRACE sample was predominantly male and it is possible that HRV-marital satisfaction effects may have been more prevalent within a female sample. Gender was controlled for in all the analyses and was not found to be significant; however, there were very few females within the sample (T2 N= 36, 16%) and lack of power to detect these gender differences may have been an issue. Furthermore, mean levels of marital satisfaction observed in the sample were generally high and there may not have been enough variation in marital satisfaction to detect HRV differences. It is possible that HRV effects may only occur at extremely low levels of marital satisfaction and the majority of married TRACE patients reported marital satisfaction above this threshold. Marital satisfaction was assessed approximately 10 days following hospital discharge and it is possible that the scores may be inflated due to the immediate effects of the ACS on the couple. The experience of such a crisis combined with the need for the couple to pull together to aid early recovery may lead to more elevated appraisals of the marital situation which may have ameliorated any HRV effects. Further exploration of these possibilities is warranted to fully explore the influence of marital satisfaction on HRV. Overall, the results reported here do not support a relationship between marital satisfaction and HRV in married post ACS patients indicating that it is being married that matters to HRV rather than the quality of the marriage.

### **8.12.7 Chapter summary**

It was hypothesised that marital status would have a significant impact upon both the experience of distress and quality of life following ACS, with unmarried patients expected to report elevated distress and poorer quality of life compared to their married counterparts. However, the findings illustrated limited significant associations with distress although

unmarried patients reporting greater anxiety at Time 3 compared to married patients. There was also a tendency towards worse mental health quality of life amongst unmarried patients, although no differences in physical health quality of life were noted. It was also hypothesised that greater marital satisfaction would be associated with lower levels of distress and better quality of life. The findings provided substantial support for this prediction. Elevated levels of anxiety were reported by low satisfied married patients compared with high satisfied married patients at all follow up points, and higher depression levels were noted in low satisfied patients at Time 2 and 3. Furthermore, low satisfied married patients also reported significantly poorer mental health related quality of life at Time 2 compared with high satisfied patients.

Significant marital status differentials in HRV were identified which were congruent with my hypothesis. Unmarried patients were found to have significantly reduced LF-HRV, HF-HRV and VLF HRV power compared to married patients which was independent of age, gender, ethnicity, deprivation, GRACE score, history of depression, Time 2 depression score, smoking status (current, previous, never) and beta blocker use. But, contrary to my hypothesis, level of marital satisfaction was not associated with any measure of HRV. Overall, some interesting patterns have emerged. The findings provide support for a biological trajectory between marital status and post ACS outcome, although suggest a more limited role for a psychological pathway. However, the results also demonstrate a clear and robust gradient of distress associated with marital satisfaction which persists over the long term and particularly impacts quality of life in the early post ACS weeks. It can be concluded that both marital status and marital satisfaction do contribute to recovery after ACS and should be considered as important prognostic factors. Marital status appears to have a primarily biological influence whereas marital satisfaction contributes more to psychological adjustment.

## CHAPTER 9 OVERALL DISCUSSION

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### 9.1 Introduction

Functional, structural and marital social support has been associated with prognosis in post ACS patients and CHD patients. Numerous pathways have been proposed to explain these differences. The research reported in this thesis investigated potential psychobiological pathways that may account for these support and marital differentials in post ACS and CHD outcomes. The pathways investigated were depression, anxiety, quality of life and heart rate variability (HRV). The TRACE study enabled analysis of social and marital patterns in depression, anxiety and quality of life at 2 weeks, 6 months and 12 months following an ACS, as well as assessment of HRV shortly after discharge from hospital (2 weeks). The results of this study have been presented in Chapter 6, 7 and 8. The suspected CAD patients study investigated marital status differentials in HRV in a sample of patients with suspected coronary heart disease, a relationship which is currently unexplored in the literature. The results of this study have been described in Chapter 3. In this chapter, I will begin by presenting a summary of the hypotheses and key findings of the TRACE study regarding the role of social support in adjustment and HRV in the context of the current research as well as summarising the key messages within my findings. Following this, I will also detail the hypotheses, central findings and key messages from the TRACE and suspected CAD studies regarding the associations between marital status and satisfaction, adjustment and HRV. I will also consider the limitations of these studies as well as the clinical implications and will present my ideas for future research direction. Finally, I will summarise the contribution these studies make to our comprehension of the role of social and marital support in CHD and ACS will be discussed

## **9.2 Key hypotheses and findings of the TRACE study: Social support, psychological distress, quality of life and HRV**

The TRACE study aimed to investigate a wide range of psychobiological indicators of outcome and recovery in a large consecutive sample of post ACS patients. I made a number of hypotheses regarding the relationship between social support (functional and structural) and psychological distress, quality of life and HRV.

### ***9.2.1 Lower levels of functional and structural social support, assessed soon after hospital discharge for ACS (T2), will be associated with depression at Time 2, and predictive of depression at six months (T3) and 12 months (T4).***

The results provided some limited support for this hypothesis. There was a near significant cross sectional association between functional social support and depression at Time 2 when depression was assessed continuously ( $p < 0.06$ ) and categorically (OR 1.08, 95% CI: 0.99-1.19,  $p < 0.07$ ). This suggests a tendency towards patients with lower levels of social support to report higher levels of depression symptoms and to be more likely to report clinically significant depressive symptoms at Time 2. However, causal direction cannot be assumed as the association was cross sectional. No significant or near significant associations were noted between functional support at Time 2 and depression at Time 3 or Time 4 indicating no longitudinal relationship. Significant cross sectional relationships were observed for social support and depression at Time 3, as well as social support and depression at Time 4. Finally, no significant or near significant associations were found between depression and structural social support at any assessment point.

These findings suggest a fairly limited role for social support in the development of post ACS depression. The near significant association observed at Time 2, and the significant cross sectional associations observed at Time 3 and Time 4 most likely reflect the impact of low mood on appraisals of social support considering the lack of a longitudinal trajectory. Cross sectional associations between functional social support and depression are widely reported

in the ACS and general population literature (Grav, Hellzèn, Romild, & Stordal, 2012; Barth, Schneider, & von Kanel, 2010). The lack of longitudinal association between functional social support and depression in the TRACE study contrasts with current research which suggests a consistent prospective relationship between low functional social support and increased risk of depression in post ACS patients in the short and long term (Brummett et al., 1998; Lett et al., 2005; Lett et al., 2009; Fontana, Kerns, Rosenberg, & Colonese, 1989; Leifheit-Limson et al., 2010; Hamalainen et al., 2000).

There are a number of potential explanations for the lack of relationship between depression and social support. The TRACE sample was characterised by high levels of functional social support with the mean score being close to the maximum score at each assessment and only 13.9% of patients classified as having low perceived social support (based on the ENRICH criteria) at Time 2. The patients in our low social support group had low social support relative to the TRACE sample; however, social support in the “low” group was not particularly low within a wider context. It is possible that the depressogenic effects of low functional social support may only be observed where functional social support is extremely low, and may be better measured in a binary fashion (no social support versus any level of social support) rather than on a continuum from low to high. With such a small number of patients reporting no or very low functional social support, this would not be possible to operationalize in this sample. It may also be that the relationship between depression and low social support was present for certain subsections of the sample that were not fully explored in the analysis, as although I controlled for a number of demographic and clinical factors, moderation analysis that would identify significant interactions was not conducted. A number of moderators have been found to be important to the relationship between social support and depression in cardiac patients including depression severity, age and SES whereby social support was found to have a more pronounced effect for patients with severe depression, younger patients and patients reporting low income (Barefoot et al., 2000). I also did not examine patient depression trajectories by tracking rates of improvement and

deterioration in depressive symptoms over time. It is possible that social support may exert an influence on depression over time whereby patients with higher social support would be more likely to have improvements in depression levels over time compared to those patients with lower social support. This dynamic impact has been noted in a number of studies (Barefoot et al., 2000; Frasure-Smith et al., 2000).

It may also be that rather than acting to reduce the likelihood or severity of depression post ACS, social support may instead buffer the effects of depression on cardiac outcomes (Frasure-Smith et al., 2000). Thus, high social support may ameliorate the negative prognostic implications of post ACS depression rather than prevent or reduce the severity of depression which would not have been detected in my analyses. Research has also found that the synergistic combination of low social support and depression may be particularly deleterious to cardiac morbidity and mortality in ACS (Horsten, Mittleman, Wamala, Schenck-Gustafsson, & Orth-Gomer, 2000; Wang, Mittleman, Leineweber, & Orth-Gomer, 2006). Thus, the lack of an association between depression and social support found within the TRACE study does not preclude social support as an important contributory factor in post ACS depression as there are other ways in which social support may exert an impact on depression that merit exploration.

I also hypothesised that low levels of structural social support would confer a higher risk of post ACS depression. However, the results demonstrated no cross sectional or longitudinal association between social support and depression. These findings that globally assessed structural support may not be an efficacious means of predicting post ACS depression adding to the current research where the findings have been mixed and considerable heterogeneity of measure noted (Hamalainen et al., 2000; Horsten et al., 2000; Barefoot et al., 2000; Lett et al., 2009; Lett et al., 2005). As previously noted in Chapter 6 discussion, very low levels of structural social support were extremely rare in the TRACE study and it is possible that the depressogenic impact of low structural social support only occurs at the

level of total social isolation. This notion is supported by the general research consensus that social isolation has the most deleterious effects of health and mortality in general (House, Landis, & Umberson, 1988). However, Brummett et al, (2001) found that low structural social support defined as *3 or fewer social ties* exerted a negative impact on morbidity and mortality after ACS suggesting a higher threshold than total isolation. It is possible that structural social support exerts an influence through many different mechanisms (i.e. direct, biological, behavioural) and the threshold levels for causing harm are also different for each mechanism. Thus, a higher threshold of social isolation (no social ties) may trigger distress effects whereas a less stringent threshold may trigger direct or behavioural effects.

It may also be important to consider the key demographic characteristics of the TRACE population which consisted mainly of married, middle aged or older men. Research suggests that within this age group the presence of a stable relationship is the facet of structural social support most closely allied with health protective effects (Robles & Kiecolt-Glaser, 2003). Subsequently, continuum measures of structural social support may be less relevant than to younger cohorts where wider social networks may be more important to wellbeing. This supposition would propose that a binary measure of marital status would provide better predictive efficacy for distress in the TRACE sample which has been addressed within this thesis. The findings are discussed in more detail later in this Chapter; however, marital status was not found to be predictive of distress. My findings suggest that neither a global assessment of structural social support nor a more specific marital approach were directly predictive of post ACS depression.

The impact of structural social support on the prognosis of CHD has also been questioned in a recent review. Barth et al, (2010) examined the role of social support (functional and structural) on the prognosis of CHD (cardiac mortality and all-cause mortality existing CHD patients). They reviewed 26 prognostic studies (where 15 of those studies included

measures of structural social support). They found mixed outcomes with regard to the prognostic influence of structural social support on CHD, with a significant relationship observed for all-cause mortality and no significant relationship found with cardiac mortality. Conversely, the results of the studies including measures of functional social support provided much more robust and significant evidence of a prognostic role in CHD. These findings suggest that the “power” of social support within cardiac health may not lie in the size of the network itself but in the functional support derived from this network. The lack of predictive efficacy with regard to psychological distress found in the TRACE study does fit within this paradigm. Finally, as discussed regarding functional social support, it is possible that structural social support exerts a prognostic impact at a different stage in the pathway, by reducing the negative effects of distress rather than preventing distress itself.

***9.2.2 Lower levels of functional and structural social support, assessed soon after hospital discharge for ACS (T2) will be associated with anxiety at Time 2, and will be predictive of anxiety at six months (T3) and 12 months (T4).***

The results provide considerable support for my hypothesis. Functional social support at Time 2 was found to be significantly associated with anxiety assessed categorically at Time 2 (OR, 1.09; 95% CI, 1.00 – 1.78), and significantly predictive of anxiety at Time 3 (OR, 1.14; 95% CI, 1.01 – 1.29) and Time 4 (OR, 1.15; 1.04 – 1.28). Similarly functional social support at Time 2 was also found to be significantly associated with continuous measures of anxiety at Time 2 ( $p=0.020$ ) and Time 3 ( $p=0.007$ ). Significant cross sectional relationships were also noted for functional social support and anxiety assessed at Time 3 and Time 4. All these significant relationships were independent of gender, age, marital status, ethnicity, employment status, GRACE score, deprivation and (where applicable) Time 2 anxiety.

Our findings extend the current research base documenting significant cross sectional associations between low functional social support and the experience of anxiety in cardiac patients (for example, Koivula, Paunonen-Ilmonen, Tarkka, Tarkka, & Laippala, 2002;

Hughes et al., 2004; Leon, Nouwen, Sheffield, Jaumdally, & Lip, 2010; Pedersen, Middel, & Larsen, 2002; Okkonen & Vanhanen, 2006; Connell & Bennett, 1997; Pignalberi, Patti, Chimenti, Pasceri, & Maseri, 1998). There have been few longitudinal studies examining the predictive power of social support with regard to post ACS anxiety, and no recent (post 2000) studies were identified in my literature review that utilised standardised assessments of anxiety and social support. Hamalainen et al, (2000) found no significant association between low functional social support assessed using a study devised measure at hospitalisation and increased anxiety (assessed using the Symptom Checklist – 90) at one year post ACS. Drory, Kravetz, & Hirschberger, (2002) found that long term (5 year post MI) psychological wellbeing (assessed using the Mental Health Inventory) was predicted by high social support assessed by the Multidimensional scale of Perceived Social Support. This lack of longitudinal research is particularly noticeable in the light of the large research base dedicated to depression and social support suggesting a substantial gap in the literature. My findings address this gap and are the first to identify a longitudinal prospective association between low functional social support assessed shortly after ACS and elevated anxiety assessed cross sectionally and at 6 and 12 months following ACS. My findings are particularly robust due to the analysis of anxiety both categorically and continuously, and the control of numerous sociodemographic and clinical confounders.

This association between functional social support and anxiety is particularly salient in the light of the increasing evidence of the considerable impact of anxiety on ACS prognosis and mortality. In a recent meta-analysis, Roest, Martens, Denollet, & de Jonge, (2010) concluded that post MI anxiety was associated with a 36% increased risk of adverse medical outcomes (cardiac events, cardiac mortality, all-cause mortality). Recently, Moser et al, (2011) found an independent relationship between anxiety (in particular, persistent anxiety) and outcome (all-cause mortality, hospitalisation for ACS, hospitalisation for other cardiac event) in a sample of 3048 CHD patients. In their meta-analysis, Roest et al acknowledge that the anxiety effect is smaller than has been found with regard to the prognostic impact of

depression; however, in the knowledge of the greater prevalence of anxiety compared to depression in post ACS populations (and identified in the TRACE sample), the implications of the negative prognostic capacity of anxiety may be greater. Beyond clinical prognostic issues, post ACS anxiety has also been found to have far reaching negative consequences including slower return to work, poorer adaptation to lifestyle changes and worse quality of life (Moser, 2007). It is clear that anxiety poses a substantial threat to recovery and prognosis after ACS. My identification of a longitudinal association between anxiety and functional social support provides evidence of the presence of a psychological pathway between functional social support and outcome after ACS that operates via the experience of anxiety.

***9.2.3 Lower levels of functional and structural social support, assessed soon after hospital discharge for ACS (T2) will be associated with quality of life at Time 2, and will be predictive of poorer quality of life at six months (T3) and 12 months (T4).***

There was no significant association between functional social support and quality of life at Time 2. Functional social support was significantly cross sectionally associated with mental health related quality of life at Time 3, and with both physical and mental health related quality of life at Time 4 independent of age, gender, marital status, previous CHD, employment status, ethnicity, deprivation and Time 2 physical or mental health related quality of life score.

These findings suggest that functional support may contribute to quality of life during later stages of recovery. However, causal direction cannot be established from these analyses and it is also possible that poorer quality of life may negatively influence perceptions of social support. The results of the longitudinal analysis do shed light on this relationship as Time 2 functional social support was significantly predictive of Time 3 mental health quality of life in the univariate analysis, and was near significant ( $p=0.068$ ) in the multivariate analysis suggesting a potential relationship. However, this relationship was not confirmed at

Time 4. No longitudinal associations between Time 2 functional social support and physical health quality of life at Time 3 or Time 4 were identified. Similarly, structural social support did not demonstrate any cross sectional or longitudinal association with quality of life at any follow up. Overall, these results are contrary to my hypothesis of a relationship between social support and quality of life suggesting that level of social support was not a strong predictor of quality of life. However, the significant cross sectional relationships between functional social support and quality of life at Time 3 and Time 4, as well as the near significant multivariate relationship Time 2 functional social support and Time 3 mental health quality of life provides tentative evidence of a role for social support in post ACS quality of life.

The research base examining links between social support and quality of life in cardiac patients is highly heterogeneous in terms of both measures and sample cardiac characteristics, and the current conclusions are mixed. Some research has reported significant cross sectional and longitudinal associations (for example, Barry, Kasl, Lichtman, Vaccarino, & Krumholz, 2006; Thomson, Molloy, & Chung, 2012; Leifheit-Limson et al., 2010). However, other studies have identified no or limited relationships between social support and quality of life (for example, Rantanen et al., 2009; Bucholz et al., 2011; Panagopoulou, Montgomery, & Benos, 2006).

There may be a number of reasons for the lack of robust longitudinal social support effects in the TRACE sample. Similar to the discussion regarding the relationship between social support and post ACS distress, it may be that more dimensional and specific aspects of both quality of life and social support are relevant which are not captured adequately by generic global measures. There may be elements of social support that are particularly important to certain aspects of quality of life. For example, instrumental support has been shown to be repeatedly associated with mental health related quality of life in a number of studies (Thomson et al., 2012; Barry et al., 2006). Similarly, certain types of social ties may be

differentially important for quality of life. For example, Bisschop et al, (2003) found that number of daughters and non-kin ties was positively associated with less physical functioning decline in older adults with chronic disease. Furthermore, improvement and deterioration within individual patient trajectories of quality of life were not examined and it is possible that social support may be related to quality of life in a more dynamic fashion than explored in my thesis. I could not find any research pertaining to the influence of social support on quality of life trajectories after ACS. However, research in other chronic illness populations has identified a relationship between social support and quality of life trajectories which, consistent with my findings, co-occur with no significant longitudinal association between baseline social support and longitudinal quality of life (Song et al., 2011). Furthermore, quality of life scores were not particularly low and it may be that the protective impact of social support is only initialised at very low levels of quality of life buffering against longer term impaired quality of life. Thus, the lack of variation in social support and quality of life scores may have reduced the power to detect relationships between them. In the light of the near significant longitudinal association between functional social support and mental health quality of life, this may be particularly relevant.

Overall, my findings did not support my hypothesis that greater social support and social network resources would be longitudinally associated with better quality of life. The identification of significant cross sectional relationships between functional social support and quality of life underscores the close affiliation between these constructs. However, the lack of longitudinal associations does not endorse the presence of a psychological pathway between functional social support, quality of life and ACS outcome. These results add to the current research body by providing a rigorous and well controlled longitudinal assessment of quality of life, functional and structural social support utilising standardised measures in an well-defined ACS population.

***9.2.4 Lower levels of functional and structural social support, assessed at Time 2, will be predictive of reduced HRV at Time 2.***

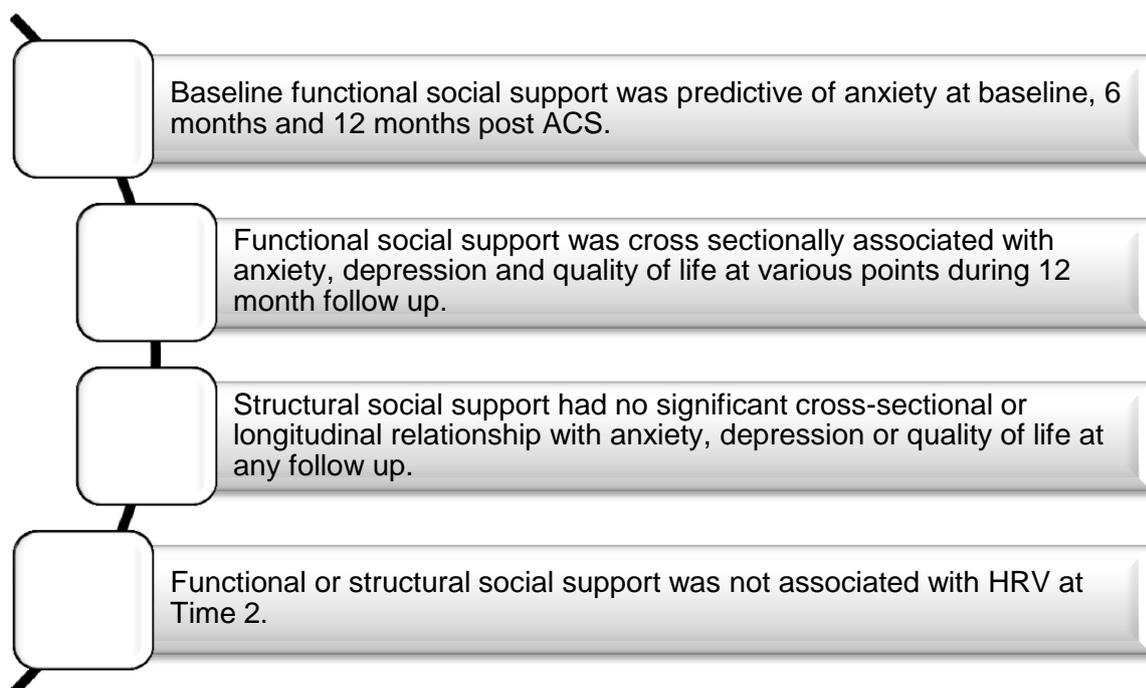
The analysis of the relationship between functional social support, structural social support, and HRV assessed at Time 2 revealed no significant cross sectional relationships indicating that neither lower functional nor structural social support influenced HRV shortly after hospital discharge for ACS as hypothesised. This finding adds to the current research base as it is the first investigation to explore social support differentials in HRV in a clinical population. There is limited current research examining the association between social support and HRV. In a sample of 1727 individuals living in an agricultural district in Korea, Shin et al, (2012) found lower HRV in individuals with lower social support compared with those reporting higher social support on the Medical Outcomes Study– Social Support Survey. However, their sample was a non-clinical population recruited from a single district in Korea reducing generalizability to my clinical sample of ACS. Horsten et al. (1999) also identified an association between decreased HRV and lower social support. They found that smaller household size, lower appraisal, tangible and belonging social support exhibited associations with lower SDNN index, LF, VLF and HF power. The Horsten findings also raise the possibility that there may be specific elements of functional social support that influence HRV which are not captured by the global measures utilised here. Echoing the previous discussions of both the psychological distress and quality of life associations with social support, a more dimensional approach to the assessment of functional social support may provide greater clarity of the presence of social support differentials in HRV. Furthermore, the high level of functional social support reported by the TRACE sample may also preclude the identification of HRV effects as the relationship between social support and HRV may only operate where social support levels are extremely low. Social support was also high in the Horsten sample but this sample consisted of healthy women whereas the TRACE study was constituted by primarily male ACS patients. Furthermore, the Horsten sample monitored HRV over a 24 hour period of everyday life which contrasts with the TRACE study's shorter

interview based assessment and these differences reduce comparability. Overall, my findings do not provide evidence for a relationship between low social support and HRV among post ACS patients suggesting that pathways between social support and ACS outcome may not mediated by HRV.

### 9.2.5 Summary of social support hypotheses and results: Key messages for social support and psychobiological pathway research in ACS

The key findings identified with regard to the relationship between social support, adjustment and HRV in the TRACE sample are listed in Figure 9.1.

Figure 9.1 Key findings: Social support, adjustment and HRV in ACS patients



My results provide support for a buffering effect of functional social support against the adverse psychological impact of ACS and suggest the presence of a psychology pathway between functional social support and ACS prognosis operating through the negative prognostic impact of elevated anxiety. However, clear evidence for a pathway role of depression and quality of life was not identified. This differential impact of social support on anxiety compared with depression and quality of life is an interesting finding. The focus of

prior research examining distress after ACS has predominantly focused on the depressogenic impact of ACS and the subsequent negative prognostic impact of depression on ACS outcomes. This has been attributed to the tendency for anxiety to be viewed as an appropriate and adaptive response to a crisis such as ACS (Moser, 2007). Consequently, this view has been echoed in social support research which has maintained depression as the chief psychological conduit between social support and ACS outcome. However, there is an emergent theme whereby an awareness of the maladaptive nature of severe or persistent anxiety in ACS and the subsequent prognostic impact of this anxiety has become more prominent (Moser, 2007). This is supplemented by research suggesting that the strength of the prognostic relationship between depression and ACS outcome has declined in recent years (Spijkerman et al., 2006). Furthermore, studies investigating the concurrent impact of depression and anxiety on ACS and CHD prognosis have identified comparable and sometimes greater prognostic impact of anxiety compared with depression (Rothenbacher, Hahmann, Wusten, Koenig, & Brenner, 2007; Strik, Denollet, Lousberg, & Honig, 2003). There is also evidence suggesting that anxiety effects on prognosis are often independent of depression effects indicative of greater prognostic risk (Kubzansky, Cole, Kawachi, Vokonas, & Sparrow, 2006; Huffman, Smith, Blais, Januzzi, & Fricchione, 2008; Shen et al., 2008), although this finding has not been consistent with other studies identifying greater prognostic impact of depression or comorbid depression and anxiety (Frasure-Smith & Lesperance, 2008; Doering et al., 2010). There has also been a recent shift towards including anxiety within physiological pathway models between distress and outcome in ACS with preliminary findings suggesting a significant role for anxiety (Zafar et al., 2010).

It is also important to bear in mind that intervention research aimed at improving social support and reducing depression has had limited efficacy in improving ACS outcomes (Berkman et al., 2003), although more research is required. There are many reasons for this lack of efficacy that have been detailed in Chapter 1, but the lack of impact does highlight the complexity of the social support-depression-outcome trajectory in ACS which presents a

significant clinical and research challenge. These findings combined with the established high prevalence of elevated anxiety and the negative prognostic impact of anxiety on ACS outcome has resulted in an emerging shift towards anxiety as an endpoint for treatment. The findings suggest that anxiety may be more amenable to change than depression. Kronish, Chaplin, Rieckmann, Burg, & Davidson, (2012) explored the impact of usual care versus problem solving therapy and/or anti-depressant treatment on anxiety in a sample of consecutively recruited ACS patients. They found that HADS-A score significantly decreased between 3 and 9 month following up which was not observed in the usual care group. It is also interesting that the treatment effect remained significant when change in depression score was controlled for. In a second intervention study, O'Neil et al, (2012) investigated the effects of a telephone delivered health coaching programme on 6 month anxiety and depression in 430 MI patients assessed using the HADS. They found that the intervention group had a significant reduction in anxiety score over time compared to the usual care group. They observed similar patterns in depression score but the effect did not reach significance. An intervention study currently underway (completion 2016) is investigating the impact of anti-depressant treatment on anxiety in ACS patients and the subsequent impact on treatment use (hospital stays, cardiac surgery, emergency care) and cardiac symptoms over a 6 month follow up (Henry Ford Health System, 2012) which will provide more insight into the prognostic implications of treating post ACS anxiety and reflects the growing move towards anxiety as an important contributor to ACS outcome. I did not identify any studies examining interventions aimed at both low social support and anxiety and this is an area requiring investigation, particularly in the light of the relationship between anxiety and social support highlighted in my thesis. My findings endorse the shift in focus towards incorporating anxiety as a critical component in the social support–distress–outcome ACS pathway, and offer substantial research foundation for the development of interventions aimed at concurrently treating low social support and elevated anxiety in ACS patients. Depression remains an important risk factor to prognosis but the impact of anxiety needs also to be

recognised. This is particularly important in the light of the greater prevalence of anxiety compared with depression in ACS populations.

The consistent lack of association between structural social support and any of my adjustment outcomes (anxiety, depression and quality of life) also has significant consequences for the conceptualisation of pathways between social support and ACS prognosis. There are a number of potential reasons why structural social support effects were not observed and these have been discussed previously with regard to depression within this Chapter. However, the consistency of my finding across outcomes and at different follow up points does imply a fundamental lack of structural support effects. This resides well within current theoretical models of social support in health whereby functional support is proposed to primarily function by buffering against the negative ramifications of health stressors whereas structural social support provides more direct effects that operate regardless of health stressors. This delineation has been identified in aetiological and prognostic research in ACS patients (for example, Barth et al., 2010). My findings provide support for a buffering effect of functional support and also indicate no buffering effect of structural social support on distress. It is possible that structural social support exerts a direct effect on ACS outcome, rather than operating through the experience of increased distress. Thus, structural social support may be important to outcomes but not to adjustment or protecting against the negative prognostic impact of maladjustment.

The final key missive from my findings is the lack of evidence for HRV as an important biological correlate of social support as neither structural or functional social support were associated with various measures of HRV. The relationship between HRV and social support is a relatively unexplored area; the current research findings tentatively suggest that more diminutive and specific aspects of structural social support (for example, marital status, living alone) may have greater influence on HRV than more global measures although this is far

from conclusive. The lack of association identified within my data does fit into this paradigm, particularly in the light of my identification of marital status differentials in HRV in the TRACE and HRV study described in this thesis. The importance of marital status and the implications this has for a HRV mediated pathway will be discussed in more detail later in this Chapter. My findings with regard to social support effects do imply that HRV may not be an important contributor to social support differentials in ACS outcome.

Overall, my results provide some important steps forward for clarifying the psychobiological pathways between social support and ACS outcome. My findings also raise further questions that highlight the need for considerable further research and my suggestions for future research will be described in Section 9.6.3 of this chapter.

### **9.3 Key hypotheses and findings of the TRACE study: Marital status, marital satisfaction, psychological distress, quality of life and HRV**

I also made a number of hypotheses regarding the relationship between marital status, marital satisfaction and psychological distress, quality of life and HRV. Some of the individual hypotheses have been combined for ease of interpretation.

#### ***9.3.1 Married patients are predicted to experience lower levels of anxiety and depression at home assessment (T2), six months (T3) and 12 months (T4).***

There is considerable evidence documenting substantial marital status differentials in ACS morbidity and mortality outcomes with unmarried patients at significantly greater risk of poorer outcome (King & Reis, 2012a; Nielsen, Faergeman, Larsen, & Foldspang, 2006; Eaker, Sullivan, Kelly-Hayes, D'Agostino, Sr., & Benjamin, 2007; Gerward, Tyden, Engstrom, & Hedblad, 2010). A key pathway through which being married may facilitate better recovery and prognosis following ACS may be due to the differential experience of distress in married compared to unmarried patients, and the subsequent negative impact of

this distress on prognosis. I explored whether marital status influenced post ACS short and long term distress as the identification of this relationship would provide good evidence for the presence of a psychological pathway between marital status and ACS outcomes. However, the results provided limited support for my hypothesis that being married or cohabiting offers protection against distress in ACS patients. No marital status differentials were observed in depression at any follow up point or in anxiety at Time 2 and Time 4. At Time 3, unmarried patients did report significantly greater anxiety compared with married patients. However, this did not persist at Time 4. The results of the repeated measures analysis revealed no relationship between marital status and change in anxiety or depression between follow up points. As previously described in Chapter 8, recent studies have documented comparable findings (Hanssen, Nordrehaug, Eide, Bjelland, & Rokne, 2009; Chung et al., 2009; Parker, Owen, Brotchie, & Hyett, 2010; Akhtar, Malik, & Ahmed, 2004).

Increasing research seems to support a more direct effect of marital status on survival rather than a buffering effect with a more limited role for psychological distress variables in explaining coronary mortality differential. Panagiotakos et al, (2008) found no relationship between marital status and depression in a longitudinal study of over 2000 post ACS patients, but found clear evidence of mortality and morbidity marital differentials. In a prospective study of 13, 889 Scottish men and women without a history of cardiovascular disease, Molloy et al (2009) found that psychological distress variables explained the least amount of variance when compared to behavioural and metabolic dysregulation factors in marital status differentials in coronary mortality. Behaviour may also be particularly important as marital status differences in health behaviour, medication adherence and CR attendance in ACS populations have been observed (Molloy, Hamer, Randall, & Chida, 2008; Trivedi, Ayotte, Edelman, & Bosworth, 2008; Bovbjerg et al., 1995). The limited marital status effects in distress observed in the TRACE study and other recent studies suggests that a

psychological pathway between marriage and ACS outcomes is not a predominant explanatory mechanism.

There were also factors within the TRACE study that may have reduced sensitivity for detecting marital status differences in post ACS distress. The role of marital history over the lifespan rather than current marital state has become an area of interest with particular patterns of marital history identified as particularly health enhancing or impairing. For example, individuals in a first marriage who remain married have lowest risk of psychological disorder than individuals with any other marital state pattern (LaPierre, 2009; Scott et al, (2010). In the TRACE study we did not consider the marital history of patients but instead examined only their current marital situation. It is possible that analysis of marital history would have identified more associations with distress and further research is required to elucidate the role of marital history in post ACS distress. There is also the possibility of selection effects with the most psychologically distressed patients less likely to be included within the study due to worse physical health, death or attrition from the study. This could limit the variation in distress that could be accounted for by marital status. Finally, assessment of marital status provides only a small piece within a complex puzzle linking an individual's social situation to their health outcomes. Fundamentally, not all marriages are of the same quality. Some may confer increased psychological risk and others may offer protection against post ACS distress. The importance of considering aspects of marital satisfaction in influencing post ACS has been identified within this thesis and illustrates a more robust association among the TRACE patients which is discussed in detail later in this Chapter.

**9.3.2 Married patients will be predicted to experience higher levels of quality of life at home assessment (T2), six months (T3) and 12 months (T4).**

A further trajectory through which marital status may influence prognosis following ACS is by influencing quality of life. Although the findings are currently mixed, research suggests that quality of life after ACS tends to be better among married compared with unmarried patients (for example, Christian, Cheema, Smith, & Mosca, 2007; Lie, Arnesen, Sandvik, Hamilton, & Bunch, 2010). As better quality of life is associated with better clinical outcome following ACS, this may represent an important pathway between marital status and prognosis. The results showed that unmarried patients reported lower mean physical and mental health related quality of life scores at every follow up point, but these differences did not reach significance at any point for physical quality of life. However, marital status was found to be significantly predictive of mental health quality of life at Time 4 independent of age and gender. Repeated measures results also highlighted significant worsening of mental health related quality of life over time in unmarried compared married patients. Overall, the results do not support the presence of marital status differences in physical health related quality of life; however, there was some support for my hypothesis suggesting poorer mental health related quality of life in unmarried compared with married patients that merits further investigation. Current research is limited by considerable methodological heterogeneity and mixed findings regarding the association between marital status and quality of life. My longitudinal and robustly controlled analysis using standardised measures of quality of life provides some clarification of the relationships indicating a greater influence on mental health rather than physical health related quality of life that emerges later in recovery.

This trend towards worse mental health related quality of life over time in unmarried patients and the significant marital status differential identified at Time 4 but not at earlier follow up points is an interesting finding. It suggests that marital status differences in mental health related quality of life may only begin to emerge at a later stage in recovery and adaptation. It

is possible that longitudinal assessments beyond 12 months may yield even greater differences. Currently the research literature has identified marital status differences in quality of life at early (< 2 months) (Oldridge et al., 1998), mid (6 month) (Christian et al., 2007; Lie et al., 2010) and long term (>12 month) stages of recovery (Lane, Carroll, Ring, Beevers, & Lip, 2001). These studies were not specifically assessing mental health quality of life but focus on a variety of different aspects of quality of life using different measures. The one study incorporating a specific assessment of mental health quality of life (SF-12) found no marital status differentials at 6 month assessment which concurs with my findings (Oldridge et al., 1998). The appearance of marital differences in mental related quality of life at later stages of recovery and adaption rather than earlier stages does fit into current understanding of the reciprocal relationship between illness and social support resources whereby the burden of illness leads to a concomitant attrition of social support (Uchino, 2006). It may be that the burden of living with CHD after ACS may levy greater functional impairment on unmarried compared to married patients in the long term because there is no partner to provide the assistance needed to facilitate and encourage day to day activities and maintain emotional support in the long term milieu of chronic illness. Immediately following ACS unmarried patients may be more likely to be able to gain this type of support from friends, family and health care professionals due to the crisis nature of their situation. However, as the crisis recedes, recovery is assumed and these initial support providers are likely to return to their lives making it harder for unmarried patients to garner such support over the long term. For many married patients, this type of daily support is often an integral part of the marital relationship. This type of low level daily (often termed invisible) social support has been found to be related to successful adjustment to stress (Bolger, Zuckerman, & Kessler, 2000). It may be that this type of social support becomes more important during later stages of recovery and this is the type of support that is more easily accessible from a spouse compared to other members of the social network.

The lack of association between physical quality of life and marital status is noteworthy because one would expect that the care of a partner would help to overcome or minimise any physical limitations imposed by an ACS and thus improve quality of life. Research has previously demonstrated an association between being married and better physical health related quality of life at 6 months in a sample of myocardial infarction patients attending CR (Oldridge et al., 1998). There may be a number of reasons for this lack of significant effect. Physical health related quality of life within the whole TRACE sample was only moderately impaired in the early weeks following ACS and returned to within population norms within 6 months. These findings suggest that the majority of patients experienced minimal disruption to their functioning and quality of life following their ACS. Thus, it is plausible that the low levels of impairment experienced by patients provided little variation to be explained by marital status. It is possible that the importance of marital status on quality of life may only emerge when quality of life is more significantly threatened. Furthermore, assessment of marital status does not provide insight into the support or the strain engendered by that marital relationship as all marital relationships are different and vary in terms of quality. The role of marital satisfaction in quality of life after ACS has been explored within this thesis and suggests a cross sectional link between marital satisfaction and mental health quality of life at Time 2 indicative that the nature of the marital relationship may be the more important to quality of life.

In summary, my findings provide some support for the hypothesised relationship between marital status and post ACS quality of life with preliminary evidence that mental health quality of life may be poorer in unmarried compared with married patients particularly during later stages of recovery. However, marital status made no significant contribution to physical health quality of life suggesting a differential marital status influence on mental health rather than physical health related quality of life. The evidence presented here provides some tentative support for a trajectory between marital status and outcome that operates via mental health related aspects of quality of life.

**9.3.3 Unmarried patients will have lower HRV compared to married patients (TRACE and suspected CAD study), and low satisfied married patients will have lower HRV compared to high satisfied married patients at Time 2 (TRACE).**

I predicted that there would be a direct biological association between marital status and HRV which may contribute to the established marital status differentials in mortality and morbidity in CHD because HRV (particularly frequency domain power) has been found to have a strong predictive relationship with post ACS mortality (Bigger et al., 1992). I hypothesised that unmarried patients would have lower HRV compared with married patients because of emerging findings indicating a relationship between social isolation and reduced HRV (Horsten et al., 1999). My findings offered substantial support for this hypothesised relationship with significantly reduced HF, VLF and LF power and borderline significant elevations of heart rate observed in unmarried compared with married patients adjusted for age, gender, ethnicity, deprivation, GRACE score, history of depression, T2 depression score, smoking status and beta-blocker use identified in the TRACE study. This was further complemented by the identification of significantly reduced LF, VLF, RMSSD, and pNN50 in unmarried compared to married patients independent of age, gender, beta-blocker use, and definite CAD diagnosis in my study of suspected coronary artery disease patients. There are similarities and differences between the two sets of findings but the particularly prominent impact of marital status on frequency domain power (particularly VLF power) in both studies is a notable finding as increased risk of post ACS mortality has been most strongly associated with reduced VLF power (Bigger et al., 1992). Both studies identified marital status differences in LF and VLF which suggests a particular marital influence on sympathetic activity. The influence on parasympathetic activity was more ambiguous with reduced HF power noted among unmarried in the TRACE sample but not in the suspected CAD sample whereas reduced RMSSD and pNN50 were identified in unmarried suspected CAD patients but not among the TRACE patients sample. Overall my findings indicate the presence of a biological trajectory from unmarried state to poorer survival via reduced HRV.

Reduced HRV is conceptualised as reflecting reduced physiological flexibility which increases vulnerability to the biological impact of stress which, in turn, can lead to concomitant health effects. These findings suggest that lack of physiological flexibility among unmarried patients may make them more vulnerable to the negative impact of the physical and psychological stress endured during post ACS recovery which adversely impact upon their prognosis.

There are a number of potential factors that may explain these marital status differentials in HRV which have been previously discussed in Chapter 3 and Chapter 8. Marriage may buffer against the negative impact of depression and other emotional states on HRV as there is clear evidence suggesting that psychopathological states reduce HRV (Thayer & Lane, 2007; Rottenberg, 2007). However, my analyses of the TRACE data found that the relationship between HRV and marital status was independent of both depression and anxiety at Time 2, anxiety as well as history of depression. As previously discussed, I also identified limited marital status differences in post ACS distress. Similarly, in the study of suspected CAD patients, there was no association between BDI depression score and HRV, and there were also no marital status differences in BDI score. It should be noted that relationships were observed when depressed mood over the sampling period were measured using the Day Reconstruction Method (Bhattacharyya, Whitehead, Rakhit, & Steptoe, 2008) suggesting that depression assessed in this manner may contribute. This suggests that there may be some aspects of depression captured using this method that may influence HRV that were not captured using the BDI which warrants future investigation.

Another potential mechanism refers to marital status differentials in health behaviour because health impairing behaviours are more common amongst unmarried populations, which negatively influence HRV (Thayer & Lane, 2007) and it has been proposed that HRV (and in particular HF-HRV) may be a physiological indicator of self-regulatory effort (Seegerstrom & Nes, 2007; Reynard, Gevirtz, Berlow, Brown, & Boutelle, 2011). However, my analyses suggested that marital status differentials in HRV observed in the TRACE study

may not be due to differences in health behaviour at Time 2 (in terms of physical activity and smoking). Although these results support a more direct impact of marital status on HRV, the presence of a behavioural pathway cannot yet be discounted. There is the possibility that the relationship between HRV and health behaviour is more dynamic than our cross sectional assessment captures. It may be that HRV differences do reflect marital status differences in early decisions to change behaviour not yet reflected in actual behavioural change, or it may be that more married compared to unmarried patients had given up smoking at Time 2. I also only examined the role of smoking and physical activity as these have a well documented relationship with HRV. However, other behaviours may also contribute as all behavioural change requires self-regulation. In the suspected CAD patients study, I did not examine or control for behavioural factors. Potential behavioural mechanisms may be operating between marital status and HRV that merit future investigation as they offer the opportunity for modification which may help to influence prognosis.

Recent research has also begun to call attention to the links between marital interaction and HRV with negative marital interactions and conflict associated with reduced HRV and increased cardiovascular reactivity (Smith et al., 2011a; Kiecolt-Glaser & Newton, 2001). Extending these findings, I explored the idea that individuals in poor quality marriages may have reduced HRV compared to individuals in high quality marriages and hypothesised that individuals reporting low marital satisfaction at Time 2 may be more likely to have reduced HRV compared to individuals reporting high marital satisfaction. My analyses revealed no significant association between level of marital satisfaction and various indices of HRV at Time 2 which is particularly noteworthy in the context of the significant association between marital status and various indices of HRV. The TRACE study was the first to address the influence of marital satisfaction on HRV in post ACS patients although a small number of population studies have identified associations between low marital satisfaction and reduced HRV, particularly HF-HRV power (Smith et al., 2011a; Carrere et al., 2005). As previously

mentioned, research has found that HRV, in particular HF-HRV, is a physiological marker of self-regulatory effort (Reynard et al., 2011; Segerstrom & Nes, 2007) which coalesces with research indicating that greater self-regulation of behaviour and emotion is related to better marital satisfaction (Wilson, Charker, Lizzio, Halford, & Kimlin, 2005). In both the TRACE and suspected CAD samples, the increased HF-HRV power noted amongst married patients may reflect the greater self-regulatory effort of married patients compared with unmarried patients required during ACS recovery. This self-regulation may apply to behavioural aspects of recovery but may also emulate the greater need for emotional regulation amongst married patients as they cope and adjust to their ACS in the context of their marital relationship. In the light of the previous research illustrating marital satisfaction differences in HRV in general population samples, the lack of association between marital satisfaction and HRV in the TRACE sample is surprising, and numerous reasons for this have been described in Chapter 8 including the predominance of male patients in the sample, the lack of variation in marital satisfaction scores and the possibility of inflated view of marital satisfaction due to the health crisis.

In the context of a particularly strong association between reduced frequency domain (especially VLF power) measures of HRV and post MI mortality (Bigger et al., 1992), my observation of a marital status HRV differential in the TRACE sample and suspected CAD patients suggests that unmarried patients may be at particular risk. This is salient following ACS and indicates that unmarried patients may benefit from closer monitoring and greater support for lifestyle change. Further research is required to determine the role of health behaviour, health behaviour change and intra marital mechanisms in this relationship. My findings provide further endorsement of a biological link between marital status and cardiac outcome and reveal the distinctive and valuable nature of marital status as a potential prognostic indicator in clinical cardiac care. The lack of association with marital satisfaction indicates that this particular aspect of the marital relationship does not impact upon HRV

although this does not rule out the possibility that other qualitative aspects of the marital relationship (for example, conflict) may be important.

***9.3.4 Lower levels of marital satisfaction, assessed soon after hospital discharge for ACS (T2) will be associated with higher levels of anxiety and depression at Time 2, and will be predictive of higher levels of anxiety and depression (T3) and 12 months (T4).***

There is a significant literature indicating that lower marital satisfaction is associated with greater psychological distress and higher prevalence of psychological disorders in community and clinical samples (for example, Whisman, 2007; Whisman, 1999). Various qualitative facets of the marital relationship including satisfaction and discord have also been implicated in different aspects of coronary heart disease including the development of CAD (Smith, Uchino, Berg, & Florsheim, 2012; Smith et al., 2011b; Gallo et al., 2003), the incidence of CAD (de Vogli, Chandola, & Marmot, 2007) and worse prognosis amongst coronary patients (Orth-Gomer et al., 2000; Rohrbaugh, Shoham, & Coyne, 2006; Coyne & Anderson, 1999; King & Reis, 2012b; Rosland, Heisler, & Piette, 2012). In the light of this research, I hypothesised that patients reporting greater marital satisfaction at Time 2 would be less likely to experience elevated anxiety and depression in the short term (Time 2) and long term (6 months and 12 months). My findings provided support for this hypothesis and revealed a significant association between Time 2 marital satisfaction and mean anxiety score at Time 2, 3 and 4 whereby patients reporting lower marital satisfaction were more likely to report higher anxiety scores at every follow up point. I also found a significant association between lower marital satisfaction at Time 2 and higher depression at Time 2 and 3. These associations were independent of age, gender, deprivation, education, ethnicity, GRACE score, previous MI and history of depression. Similar findings were also noted utilising a categorical assessment of distress with patients reporting lower levels of

marital satisfaction more likely to experience above threshold depression at the 6 month follow up, and above threshold anxiety at both ten days post ACS and the 6 month follow up. Again these findings were independent of age, gender, deprivation, education, ethnicity, GRACE score, previous MI and history of depression. It should be noted that associations with Time 3 and Time 4 distress were not independent of Time 2 distress suggesting that initial distress remains the greatest predictor of later distress. My findings add significantly to the current limited research exploring potential psychological pathways between marital satisfaction and post ACS outcome. Only one other study that investigated the role of marital quality in post-surgical psychological adjustment was identified and noted that better marital quality was associated with better psychological adjustment at 3 months post-surgery in 198 male MI or cardiac surgery patients at 3 months post cardiac event (Brecht, Dracup, Moser, & Riegel, 1994). The results from the TRACE study extend and update these findings; asserting a clear relationship between marital satisfaction and psychological distress in ACS patients. Patients in low satisfaction marriages are at increased risk of both anxiety and depression that persists beyond hospital discharge indicating that marital satisfaction is an important predictor of adjustment among married ACS patients.

The marital satisfaction findings are particularly interesting because, as reported previously, limited associations were identified between marital status and distress in the TRACE sample which contrasts with my finding that marital satisfaction is robustly associated with distress. This suggests that simply being married does not confer reduced risk of post ACS distress but being in a highly satisfying marriage does reduce the risk of distress compared to being in a low satisfaction marriage. It would have been interesting to further extend the analysis to include comparison of the varying marital satisfaction groups with the unmarried group as research has found that low satisfied married individuals have worse health outcomes than unmarried individuals (Holt-Lunstad, Birmingham, & Jones, 2008). However, sample size and thesis scope constraints precluded this analysis. These results are consistent with a substantial body of research illustrating that marital satisfaction is a better

predictor of various health outcomes than marital status including ambulatory blood pressure, negative affect, stress, metabolic syndrome, cardiovascular risk factors and atherosclerosis (Gallo, Troxel, Matthews, & Kuller, 2003; Grewen, Girdler, & Light, 2005; Gallo et al., 2003; Troxel, Matthews, Gallo, & Kuller, 2005; Gove, Hughes, & Style, 1983). Based on this research, being in a highly satisfying marriage confers significant health benefits compared to being unmarried or in a less satisfying marriage. However, being in an unsatisfactory marriage is associated with worse health than being unmarried. Our findings add further evidence of the greater importance of marital satisfaction compared with marital status in predicting health outcomes and suggests that post ACS distress can be added to the list of health outcomes differentially impact by marital satisfaction versus marital status.

Marital dissatisfaction and distress are closely allied and there has been debate in the literature regarding the typical causal direction of this relationship (Fincham & Beach, 2010; Rehman, Gollan, & Mortimer, 2008). The TRACE study findings do suggest a causal pattern whereby marital dissatisfaction reported in the early days following ACS was predictive of greater distress 6 and 12 months later. A criticism frequently levied at this type of research is the possibility of reverse causality due to lack of control for prior history of distress which has been noted as an important predictor of marital dissatisfaction (Rehman et al., 2008). In the case of the TRACE sample, history of depression was controlled for in all analyses and the relationship between marital satisfaction and distress were found to be independent of prior depression history indicating that marital dissatisfaction increases vulnerability to post ACS distress regardless of prior distress. However, in the light of significant research illustrating a bidirectional relationship between distress and dissatisfaction (Kouros, Papp, & Cummings, 2008; Davila, Karney, Hall, & Bradbury, 2003), this increased vulnerability to distress caused by marital dissatisfaction is also likely to have reciprocal impact on marital satisfaction resulting in further distress. Thus, the predictive relationship between marital satisfaction and distress, combined with the knowledge of the reciprocal nature of this relationship has significant consequences for ACS patients in low satisfaction marriages as the negative

prognostic implications of increased post ACS distress are well established and reported throughout this thesis.

Overall, the association between marital satisfaction and distress in a sample of ACS patients is a valuable finding highlighting the importance of marital satisfaction in post ACS recovery and providing evidence of a psychological pathway through which marital satisfaction may exert an impact of ACS prognosis. These findings also draw attention to the importance of considering qualitative aspects of relationship. Based on my analysis of the relationship between marital status and distress, it could be assumed that marriage confers little protection against post ACS distress. However, the identification of marital satisfaction differentials in distress suggests that high satisfaction marriages may indeed confer such protection.

***9.3.5 Lower levels of marital satisfaction, assessed soon after hospital discharge for ACS will be associated with poorer quality of life at home assessment (T2) and predictive of poorer quality of life at six months (T3) and 12 months (T4).***

A significant association between marital satisfaction and mental health related quality of life was observed at Time 2 with low satisfied married patients reporting worse mental health related quality of life than highly satisfied patients independent of age, gender, deprivation, previous CHD, employment status, ethnicity, GRACE score and history of depression. However, this relationship did not persist at Time 3 or 4. This finding suggests that poor marital satisfaction and reduced quality of life are associated although it is not possible to distinguish whether low marital satisfaction reduces quality of life or whether reduced quality of life increases marital dissatisfaction. These findings do, however, reveal that marital satisfaction and quality of life are related during the early stages of post ACS recovery. It is also important to consider these results in the context of the significant relationship between marital satisfaction and distress identified at Time 2. As discussed in Chapter 8, the

combination of both elevated distress and reduced quality of life indicate a substantial burden experienced by patients in low satisfying marriages and highlight a particular at risk group of ACS patients. I proposed that the relationship between marital satisfaction and ACS related factors may be explained by its influence on quality of life which has been found to be significantly associated with ACS prognosis. My findings provide some support for this pathway through the identification of a triad (marital dissatisfaction, increased distress, reduced quality of life) of comorbid states which represent a significant risk to post ACS outcome as each of these factors is associated with increased morbidity and/or mortality after ACS.

There has been limited research exploring marital satisfaction influences on post illness quality of life, although cross sectional and prospective marital satisfaction differentials in quality of life have been identified in patients with diabetes, poor vision, physical disability and cancer (Bookwala, 2011; Trief, Himes, Orendorff, & Weinstock, 2001; Trief, Wade, Britton, & Weinstock, 2002; Hannum, Giese-Davis, Harding, & Hatfield, 1991; Bookwala, 2011). It should be noted that these studies utilised a battery of quality of life assessments and also include measures of disease specific quality of life which differ from my single assessment of general health related quality of life. With regard to coronary populations, marital satisfaction differences in quality of life have been observed in post CABG and recently diagnosed CHD populations (Elizur & Hirsh, 1999; Brecht et al., 1994). However, the TRACE study was the first to investigate cross sectional and prospective relationships between marital satisfaction and quality of life in a post ACS population.

I found no relationship between marital satisfaction and physical health quality of life which was surprising. However, there are a number of potential reasons for this lack of relationship. As discussed throughout this thesis with regard to numerous psychological parameters, levels of quality of life and marital satisfaction were generally high in the TRACE

study reducing the variation to be explained by marital satisfaction differences. Gender may be an important factor here as although gender was controlled for in all analyses, gender was found to be an important predictor of quality of life with female patients reporting poorer quality of life compared to male patients. In general, marital satisfaction tends to be lower among women compared to men, and the impact of marital dissatisfaction appears to be greater among women (Fowers, 1991; Schumm, Webb, & Bollman, 1998). The recovery behaviour of post ACS patients has also been found to be strongly influenced by gender (Kristofferzon, Lofmark, & Carlsson, 2003). Following ACS female patients report receiving less assistance with household duties, were less likely to involve their spouses in their recovery and tended to minimise the impact of their ACS. They also showed that female patients tend to resume responsibility for domestic tasks and tend to engage in too much activity post ACS (Kerr & Fothergill-Bourbonnais, 2002; Lemos et al., 2003). Thus, these differences in early recovery behaviour may influence quality of life, marital satisfaction and their interaction. It could be postulated that because female patients are more likely to resume activity earlier than men, physical and mental health limitations may be more obvious to them and have a greater impact. It would be interesting to conduct a more gender stratified analysis of quality of life and marital satisfaction. However, the TRACE sample was mainly composed of male patients and the small sample size of married women providing marital satisfaction data prevented such analysis. The gender differential impact of marital satisfaction on quality of life merits investigation. It is also important to consider the complex and dynamic nature of the marital relationship and that marital satisfaction is only one aspect of this relationship. Thus, although marital satisfaction exhibited mixed associations with aspects of quality of life, other marital factors (for example conflict or intimacy) may be important to quality of life.

My findings are consistent with the current quality of life literature which has been described as inconsistent and paradoxical (Rapkin & Schwartz, 2004; Dempster, Carney, &

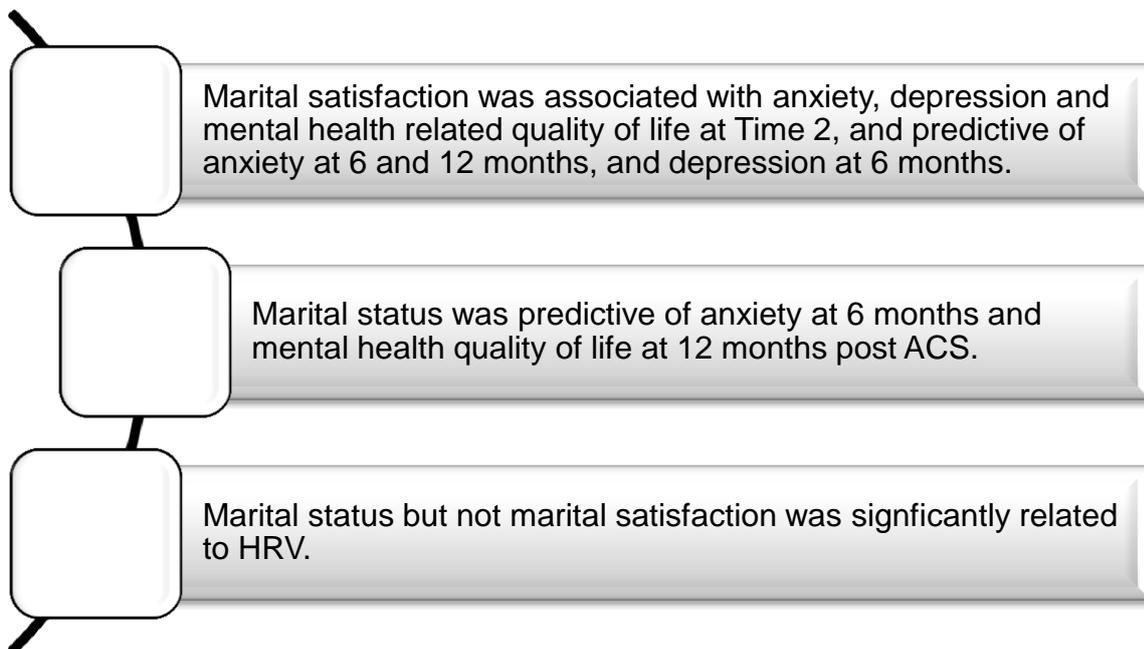
McClements, 2010). It has been suggested that these inconsistencies are less a reflection of methodological issues and bias but more a manifestation of a lack of sensitivity within quality of life measures to detect the significant variation in how quality of life is appraised at different times, as the result of varying events and by different people. This is particularly important among ACS patients who have experienced both an acute health event followed by adaptation to life with a chronic illness. These contrasting experiences are likely to influence how quality of life is appraised. As a way of increasing sensitivity, recent models of quality of life have begun to incorporate the concept of response shift. Response shift refers to the change in an individual's conceptualisation of quality of life, as well as the personal values and benchmarks that underlie this conceptualisation, as a result of a change in health state (Dempster et al., 2010; Sprangers & Schwartz, 1999). A recent longitudinal study by Dempster et al., (2010) of 57 ACS patients participating in a cardiac rehabilitation programme observed that patients retrospectively rated their functioning as significantly lower than their original baseline rating. The authors highlight that this discrepancy indicates that the patients had engaged in response shift and the changing nature of patient's perceptions of their own functioning. ACS is both an acute event and part of a chronic illness resulting in both sudden and gradual changes in health state and functioning. The acute event is typically followed by a period of convalescence and reduced health and functioning with gradual improvements occurring over time which may even exceed pre ACS levels as treatment for underlying CHD takes effect. As a result, significant changes over time in the way the patient appraises and compares their own functioning are likely. It may be that the limited association between marital satisfaction (as well as marital status and social support) and quality of life observed in my thesis may in part be due to a lack of sensitivity to variation in quality of life appraisal over time. Incorporation of measures of response shift may be important to determine quality of life changes in ACS patients. In summary, the cross sectional association between marital satisfaction and mental health quality of life at Time 2 indicates that these two factors are related and supports my hypothesis. However, the lack of longitudinal associations is contrary to my hypotheses and prevents the establishment of

causal direction. Similarly, the lack of relationship between marital satisfaction and physical health quality of life at any assessment is not consistent with my hypothesis.

### 9.3.6 Summary of marital status and satisfaction hypotheses and results: Key messages for marital and psychobiological pathway research in ACS

The key findings identified with regard to the relationship between marital status, marital satisfaction, adjustment and HRV in the TRACE sample are listed in Figure 9.2.

**Figure 9.2 Key findings: Marital status, marital satisfaction, adjustment and HRV in ACS and suspected CAD patients**



My results provide some support for a psychological pathway between marital satisfaction and ACS outcomes. Reduced marital satisfaction was cross sectionally and prospectively associated with anxiety and depression, and was also cross sectionally associated with mental health quality of life. These combined findings suggest a substantial burden of distress experienced by ACS patients in low satisfaction marriages and indicate a group of patients at elevated risk of poor prognosis due to the increased risk of comorbid distress and

poor quality of life. My study is the first to demonstrate prospective marital satisfaction differentials in distress. According to marital role theory (Tharp & Otis, 1966), poor marital satisfaction is proposed to create a marital environment whereby change and the need for adaptation results in stress and conflict. The experience of an ACS can be understood as both an acute and chronic condition requiring considerable short and long term lifestyle adaptations by both the patient and their spouse. For example, adherence to medication and dietary regimes, attendance at CR and smoking cessation. Marital role theory states that where these changes occur within a supportive and satisfying marital relationship, the couple will work together and provide mutual support for successful adaptation. For a couple in a less satisfying and poor quality marriage, the need to adapt to these lifestyle changes may result in significant stress and conflict which may subsequently reduce adjustment and quality of life. Furthermore, marital satisfaction has also been found to influence the types of coping methods used by couples when faced with the challenge of chronic illness. Higher marital satisfaction has been associated with greater adaptive dyadic coping and reduced maladaptive coping, and may also buffer against the negative impact of maladaptive coping in diverse chronic diseases (Bodenmann, Pihet, & Kayser, 2006; Coyne & Smith, 1991; Hagedoorn et al., 2000; Schokker et al., 2010; Hinnen, Hagedoorn, Ranchor, & Sanderman, 2008). Thus, the experience of elevated distress and poorer quality of life among ACS patients in low satisfaction marriages may arise from the conflict and strain induced by the need for adaptation and change instigated by the ACS and the utilisation of less adaptive dyadic coping behaviours which represents an interesting area for further exploration.

Marital status offered less predictive or associative efficacy with non-significant trends towards elevated distress and poorer mental health quality of life among unmarried compared to married patients. However, these relationships only reached significance at Time 3 (distress) and Time 4 (quality of life) suggesting that marital differences only emerge during later stages of recovery. Complementing these findings, marital status was also found to predict functional support at Time 3 and Time 4 but not at Time 2 suggesting that support

was similar for married and unmarried patients during the early stages of recovery but in the later stages of recovery married patients reported greater functional social support. These combined findings suggest that the psychological and support benefits derived from being married may emerge later during post ACS recovery. There is some evidence for this in the TRACE data where marital status predicted later (T3 and T4) but not earlier (T2) levels of functional social support. Temporal patterns of support within the context of chronic illness are currently not well understood (Revenson, 2003; Berg & Upchurch, 2007) and no studies investigating marital differences in changes in social support over the long term course of a chronic illness were identified illustrating a significant research gap.

Finally, my identification of marital status differentials in HRV in both the TRACE study and in the study of suspected coronary artery disease patients provides significant support for a biological pathway between marital status and post ACS outcome. These are important findings as they are the first studies to demonstrate marital status differentials in HRV in two different cardiac populations and highlight the biological vulnerability of unmarried following ACS. The lack of influence of marital satisfaction on HRV in the TRACE study also provides further insight into the characteristics of this biological pathway. It may be that other qualitative aspects, for example marital conflict, may have a greater influence than global evaluation of marital satisfaction and this warrants investigation. Overall, my results suggest a more direct biological impact of marital status and a greater psychological impact of marital satisfaction.

## **9.4 Limitations, clinical implications and future research direction**

### **9.4.1 Limitations**

#### **9.4.1.1 Scope and approach**

The TRACE study gathered a huge array of data from ACS patients and their partners, and subsequently the data gathered was greater than my thesis scope allowed for. I had hoped to include data from patient's partners and more varied assessment of marital factors; however, limitations had to be made in order to develop this thesis into a concise piece of research. My approach was to establish relationships between single constructs (for example, functional social support and anxiety) using standardised assessments to provide evidence to support particular pathways to post ACS adjustment. However, this method did not allow for a more dynamic and full assessment of the inter relationships between these constructs (for example, quality of life and depression and functional social support). This approach is artificially narrow as in real life these constructs all coexist and interact. However, this approach provides a structured way of identifying individual relationships.

#### **9.4.1.2 Assessment of social support, marital status and satisfaction**

Social support was assessed using standardised and well established measures of functional and structural social support. However, there were aspects of social support that were not assessed by these measures that may be important. My focus was on beneficial forms of social support; however there is research illustrating that some forms of social support (for example, unwanted or unhelpful social support) can have a negative impact resulting in increased stress and distress (Boutin-Foster, 2005; Linden & Vodermaier, 2012; Stafford, McMunn, Zaninotto & Nazroo, 2011). These negative aspects of social support have shown particular predictive utility with regard to quality of life (Helgeson, 2003). The relationship between social network resources and the functional support derived from specific sources of support was not explored although prior research has illustrated that the match between source, support and situation may be important in ACS patient adjustment (Friedman, 1993; Yates, 1995).

Furthermore, there has been recent debate regarding the ability of global measures to accurately capture the predictive relationship between social support and depression in CHD

patients (Lett et al., 2009). There may be specific dimensions of low social support that are particularly distress invoking that were not adequately captured by the measures we used and it may also be that the TRACE population did not have particularly high levels of these specific elements of low social support. Lett et al (2009) recommend the use of more dimensional measures of social support to improve the accuracy and sensitivity of predictive models in cardiac patients. There are currently few studies that prospectively examine the association between dimensional aspects of social support and emotional distress in cardiac patients. However, esteem, informational/tangible, and emotional/intimacy aspects of social support have been identified in cross sectional and prospective studies as offering the most promise with regard to preventing emotional distress (King, Reis, Porter, & Norsen, 1993; Yu, Lee, Woo, & Thompson, 2004; Lett et al., 2009; Hamalainen et al., 2000). This same issue of construct heterogeneity is also problematic in the evaluation of distress with corresponding current debate regarding the specific elements of depression that are most detrimental to CHD recovery (Doyle, McGee, Conroy, & Delaney, 2011) and different depression scales demonstrating differing predictive capacity with regard to CHD morbidity and mortality (Doyle, McGee, De La Harpe, Shelley, & Conroy, 2006). Certain dimensions of depression have been identified as particularly valuable in the prediction of post ACS mortality and morbidity including anhedonia (Doyle, 2010; Davidson et al., 2010) and somatic/affective factors (de Jonge et al., 2006; Poole, Dickens, & Steptoe, 2011; Bekke-Hansen, Trockel, Burg, & Taylor, 2012). Correspondingly, there may be specific elements of depression that are most influenced by social support that would not be captured by a generic depression measure such as the BDI. In the only study investigating dimensional associations between social support and depression, Lett et al., (2009) conducted confirmatory factor analysis of the most commonly used measures of social support (including the ESSI) and depression (including the BDI) in a sample of 705 cardiac patients enrolled in the ENRICHD study. They found that the most optimal measurement of the social support-depression relationship in CHD patients incorporated somatic, cognitive/affective and anxiety factors of depression with perceived functional support from intimate

relationships, perceived tangible support from peripheral contacts, as well as number of children, relatives and friends in social network. These findings suggest the important potential of dimensional measurement. This discussion also taps into the broader debate within social support literature regarding the importance of an optimal match between the type of social support and the situational stressor in activating and facilitating the buffering effects of social support (Thoits, 2011). Determining and capturing this optimal match between social support and depression is an important next step in the cardiac and social support research field.

There are also aspects of structural social support that were not evaluated in either study that may influence psychological outcome. In particular, centrality (whether an individual is at the centre or periphery of a social network) and community participation have been found to be particularly related to depression (Rosenquist, Fowler, & Christakis, 2011; Ahern & Hendryx, 2008). My social network measure did not assess the centrality of the patient within their social network and subsequently may have a relationship with distress in the TRACE population. However, aspects of community participation (volunteer work, social clubs, and religious groups) were assessed within my network questionnaire. It is possible that a more specific analysis of community participation and depression may identify a relationship. Factors such as aging and life events (e.g. retirement, widowhood, marital dissolution) associated with these populations have also been found to impact upon many aspects of the social network (Ajrouch, Blandon, & Antonucci, 2005; Aartsen, van Tilburg, Smits, & Knipscheer, 2004) and there is a need to garner greater understanding of the fundamental nature of middle aged/older adults social networks to inform the development of social network measures that accurately capture any potential psychopathological association in cardiac patients.

Social support was assessed at three different time points; however the temporal pattern of social support for each patient and how this pattern links to the evolution of distress was not

explored. This may be relevant as research suggests that illness can erode social support and it is possible that this erosion of social support may result in poorer adjustment (Uchino, 2004). Similarly, marital satisfaction represents only one facet of the marital relationship and was selected because it is the most utilised measure of the marital relationship in health. Changes in marital satisfaction were not explored and may be important as illness has been found to have a concomitant negative impact on marital satisfaction (Bradbury, Fincham, & Beach, 2000). Numerous other facets of the marital relationship may also be important including equity, strain, and attachment style which have all been found to contribute various health outcomes (Umberson & Williams, 2005; Umberson, Williams, Powers, Liu, & Needham, 2006; Hirschberger, Srivastava, Marsh, Cowan, & Cowan, 2009).

A limitation of both studies was the binary classification of patients as married or unmarried. Both states actually encompass a wide range of different marital statuses and histories. An individual may report being married which may reflect a single continuous marriage or may reflect an individual who has been divorced and married a number of times. Similarly, an unmarried individual may be single and never married or may be divorced, widowed or separated. The TRACE study and the suspected CAD study did not investigate the impact of these different statuses and histories which was largely due to small sample sizes. As previously discussed in detail in Chapter 3 and Chapter 8, research has identified that both marital history and specific marital status may impact upon outcomes and it is possible that these facets of marital life may differentially influence psychological or biological parameters.

#### **9.4.1.3 Design**

The TRACE study enabled ACS patients to be followed up regularly during the first year of recovery but it was not possible to obtain pre event measurement of any variables and thus baseline levels could not be controlled for in my analyses. However, the TRACE study did gather data regarding history of depression which was included in many of the analysis and

this provides some control for prior depression. Similarly, in the suspected CAD patient study, patients were recruited from a Chest Pain clinic and subsequently pre-referral assessment was not possible. All patients included in the TRACE study were hospitalised due to their ACS and the sample excludes patients who were not hospitalised following an ACS and those patients who died during or immediately after their ACS. Conducting research with cardiac patients inevitably involves some compromise with regard to design and sample rigour due to the unpredictable and nature of cardiac events and the utilisation of a hospitalised patient sample. Finally, both the TRACE and suspected CAD study did not incorporate any experimental manipulation or control groups which would have enabled more robust conclusions regarding the relationship between specific variables.

#### **9.4.1.4 Assessment**

A large proportion of questionnaire measures were used which were often completed in the presence of the researcher at the Time 2 assessment in order to support completion. This raises the possible of social desirability issues influencing responses which is an issue inherent in all questionnaire based research. All questionnaires were presented with information explaining that there were no right answers to help to minimise this problem. In the TRACE study, the Time 2 interviews were conducted within the patient's home. Researchers conducting these interviews attempted to ensure the patient was alone and comfortable to disclose personal information. However, this was not always possible due to the presence of their partner, relatives or others within the household which may have influenced patient responses. This may be a particularly salient issue with regard to sensitive affective, support and marital issues. Finally, the large amount of questionnaires and the repeated follow ups may have reduced response rate due to the questionnaire burden. This is particularly salient considering that the patient sample were recovering from a serious cardiac event.

#### **9.4.1.5 Sample size, sample characteristics and attrition**

The TRACE sample predominantly consisted of white, middle aged men. The sample included few ethnic minority patients (11-17%) and few female patients (14-16%) suggesting that these patients were under represented within the sample as ACS rates have been found to be elevated among certain ethnic minority groups (British Heart Foundation, 2010) and female patients report worse prognosis and increased risk of post ACS distress (Reina et al., 2007; El-Menyar & Al, 2009; Norris, Hegadoren, & Pilote, 2007). Response rate declined over the study period but remained satisfactory. At recruitment, 45% of eligible patients were recruited to the study. Attrition was mainly due to poor clinical condition or transfer/discharge from hospital. Only a small percentage (9%) of eligible patients refused to take part in the study. Working with clinical populations requires sensitivity to the patient's health needs and also to hospital protocol and procedures which inevitably leads to some sample loss.

A good response rate of 76% was noted at Time 2 with non-completers more likely to be moderate to highly deprived, unmarried, living alone and with prior heart disease. The greater loss of unmarried patients and patients living alone may reduce the representativeness of the sample of unmarried patients included in the TRACE study. It is possible that these patients were too distressed or physically unwell to complete the measures and thus the marital status relationships described may have been weakened. At Time 3, 67% of patients completed the follow up with non-completers more likely to be younger, non-white, diabetic and living in moderate to high social deprivation. At the final 12 month follow, 59% of patients responded with non-completers more likely to be diabetic, non-white and less likely to report prior heart disease. Thus, there may be a subsection of patients, particularly non-white, diabetic, highly deprived and unmarried patients, who were less well represented in later follow ups and who may be more vulnerable to social isolation and distress suggesting that the core patient sample consisted of patients who were more likely to adapt successfully to their ACS. Thus, the sample limitations may reduce the

generalisability of my findings and also imply that the most isolated and distressed patients may not have been reached.

#### **9.4.2 Clinical implications**

A key facet of research in clinical populations is the ability for research to inform practice and to improve clinical and psychosocial outcome. The identification of a prospective association between low functional social support and anxiety, as well as between low marital satisfaction and longitudinal distress highlights two potential routes for risk stratification amongst ACS patients whereby patients reporting low functional social support and/or low marital satisfaction may benefit from greater support and information from health care providers. Both are easy and quick to assess with standardised measures providing health care staff with a simple means of identifying those patients at increased risk for distress.

These associations also highlight potential entry points for intervention aimed at reducing post ACS distress and improving cardiac outcome. Interventions aimed at increasing functional social support and at improving marital satisfaction and targeted more specifically at anxiety occurring early in recovery may impact upon current and later distress. Current intervention research in ACS is limited with the few studies investigating the impact of treating anxiety on outcome and distress documenting mixed findings (Merswolken, Siebenhuener, Orth-Gomer, Zimmermann-Viehoff, & Deter, 2011; Bradt & Dileo, 2009; McLaughlin et al., 2005). Similarly, interventions aimed at ameliorating low social support and depression in ACS patients have demonstrated improvements in depression and social support but no impact upon outcomes (Berkman et al., 2003). Other studies aimed at improving social support and reducing distress have been found to be effective for chronically ill patients with comorbid high distress and low support (Hill, Schillo, & Weinert, 2004). A review and meta-analysis of couple oriented interventions for chronic illness by Martire, Schulz, Helgeson, Small, & Saghafi, (2010) concluded that greater intervention effects were found in studies focusing on couples with low partner support, poor marital

quality or more illness related conflict. Similarly, involvement of family members (primarily spouses) and focusing on relationship satisfaction in interventions aimed at improving outcome in chronic illness has also proven effective (Martire, Lustig, Schulz, Miller, & Helgeson, 2004; Hartmann, Bazner, Wild, Eisler, & Herzog, 2010). My findings suggest that interventions targeting low social support, poor marital satisfaction and anxiety may represent an important and currently unexplored means of improving both psychosocial and clinical outcome of ACS patients that warrant investigation.

Unmarried marital status was also found to be a significant indicator of biological risk in terms of reduced HRV in both ACS and suspected CAD patients, and represents another means of rapid risk assessment. Reduced HRV represents a substantial threat to prognosis and is a well-established indicator of mortality (La Rovere, Bigger, Jr., Marcus, Mortara, & Schwartz, 1998; Bigger et al., 1992). These results suggest that there may be value to selecting unmarried patients for more specialist intervention and attention with particular focus on modification of HRV. Behavioural change has been found to be effective in modifying HRV (Thayer & Lane, 2007) and thus focused and intensive lifestyle modification may help to reduce the biological risk associated with unmarried status in cardiovascular disease. A recent review also identified evidence supporting the use of biofeedback, relaxation and meditation techniques to increase HRV suggesting that these methods may represent a useful adjunct to cardiac rehabilitation for unmarried patients (Servant, Logier, Mouster, & Goudemand, 2009).

#### **9.4.3 Future research directions**

The findings from the studies reported within my thesis highlight numerous areas for future research. Most significantly, my identification of a prospective association between functional social support and anxiety amongst post ACS patients suggests that there is a need to shift focus towards anxiety as an important mediator of the trajectory between functional social support and outcome. Previous research has mainly focused on depression; however, there

is growing momentum within the research literature, further confirmed by my findings, suggesting that anxiety may play a pivotal role. There is also a need to explore the relationships between distress and social support on a more dimensional level to develop a more refined and sensitive understanding of the exact elements of social support that may be most depressogenic and/or anxiogenic, and the specific aspects of depression and anxiety that are most associated with these social support elements. Similarly, my identification of a relationship between marital satisfaction and long term distress in ACS provides a strong basis for further research to replicate these findings, as well as explore the differential role of gender and the specific aspects of marital dissatisfaction that impose the greatest risk to post ACS adjustment. The differential impact of varying levels of marital satisfaction compared to unmarried status also merits investigation. Research may need to examine the temporal relationship between marital factors and levels of specific marital support in the genesis of distress over time to identify the patterns and inter relationship between these two facets of the marital relationship. My research provided preliminary evidence of marital status effects on distress occurring later but not earlier in recovery, and there was evidence suggesting that changing levels of functional social support may contribute. In the light of the association between marital satisfaction and distress, there is a need to further clarify the prognostic role of marital satisfaction in ACS as the current research is limited.

The lack of significant quality of life findings in my research with regard to social support, marital status or satisfaction differences adds to the inconsistent research base associated with quality of life in chronic illness. Exploration of the role of aspects of response shift within quality of life assessment in ACS merits exploration before quality of life is discounted as a pathway mechanism between social and marital factors, and ACS outcomes. Measures that are more sensitive to the dynamic issues and challenges faced by patients during ACS recovery may identify relationships with quality of life.

Marital status was not found to exert a substantial influence on distress or quality of life after ACS until the 6 and 12 months follow up. Subsequently future research may need to explore the possibility of marital effects emerging later in recovery and it would be interesting to extend follow up times beyond 12 months to determine whether marital effects do become more pronounced later in recovery. Similarly these findings combined with the deficit in research suggest that the temporal course of social and marital support over the course of short and long term ACS recovery merits exploration. The limited support for a psychological pathway between marital status and ACS outcome identified here also suggests that other mechanisms may be important. Exploration of behavioural mechanisms was beyond the scope of my thesis; however, these merit examination and will form part of future research analyses of the TRACE data. The presence of a biological pathway was identified in the TRACE study as marital status was found to have a robust impact upon HRV in both the TRACE and suspected CAD patients; although the specific aspects of HRV effect did vary. Future research may need to focus upon clarifying the impact of marital status on specific aspects of parasympathetic and sympathetic modulation, and how these vary according to cardiac population and socio demographic factors.

Finally, my research focused on various social support and marital factors that may contribute to various measures of adjustment. I considered each of these factors and measures in relative isolation from the others to ensure clarity and distinguish specific relationships. However, these facets are highly interdependent and interactional. Future research may need to integrate these findings to provide a more coherent model of the pathways between social support, marital factors, adjustment and prognosis.

## **9.5 Overall conclusion**

My thesis presents research exploring the role of social support and marriage in psychobiological pathways to prognosis in ACS and CAD. Previous research has identified that varying aspects of social support and marriage are significantly related to morbidity and

mortality following ACS. Diverse pathways have been proposed to explain these outcome differentials including behavioural, psychological and biological mechanisms. The research described in this thesis illustrates that aspects of both social support and marriage contribute to psychological adjustment following ACS. In particular, functional social support and marital satisfaction offer considerable predictive utility with regard to the occurrence of distress which was most pronounced with regard to the experience of anxiety. However, marital status and structural aspects of social support were found to confer minimal influence on psychological adjustment. These findings provide support for the traditional conceptualisation of functional elements of social and marital support providing greater buffering against stressors compared to more structural elements. The research within this thesis also provides support for a biological trajectory between marital status and prognosis in CAD and ACS as significant marital status differentials were observed in HRV. This differential was particularly robust with regard to HF-HRV which has been purported as a marker of self-regulation, suggesting that married patients may exert greater self-regulatory effort during recovery. Risk stratification and intervention based upon elements of functional social support, marital satisfaction and marital status may improve adjustment, prognosis and, ultimately, outcome in CAD and ACS.

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# **APPENDIX I**

## **TIME 1 PATIENT QUESTIONNAIRE TRACE STUDY**

## Tracking Recovery after Coronary Events: The TRACE study.

### Time 1 Interview (In hospital)

Patient Study number: TR_ _ _	Patient name:
Hospital no.	Date of Birth
Date of Admission: Time:	Time of blood sample: (1)                      (2)
Date of Interview:	Interviewer:
Outside temperature on date of cardiac event (from Met. office):	
Date of discharge (if known):	
Patient's address:	GP name/address:
Patient's NHS number:	
Phone number: Landline: Mobile:	

### Details Of Acute Coronary Syndrome on admission      *Copy admission ECG*

Admission BP		Nitrate	
Admission pulse rate		Other	
ST elevation ?		Complications	
ST depression ?		Initial Outcome	
T wave inversion ?		For Angiogram ? (time/date)	
Bundle Branch Block ?		Angio result (time/date, no.of diseased vessels).	
Arrhythmia ? (AF/ VF/ VT)			
Territory (Inf/Ant/Post/Lat)		Treatment plan (date, med Tx, CABG, PTCA.	
Congestive Heart Failure (present/absent) ?			
Aspirin (given on admission)		Revascularisation details? (Include time/date)	
Heparin		Previous MI?	
Thrombolysis			



Separated      Living as Married      Other.....

If with a partner, B6. For how long have you been with your spouse / partner? \_\_\_\_\_ in years.

**Give patient next page to complete**

**B7. Which category do you feel best describes your ethnic origin?**

**ETHNIC GROUP (please tick the most relevant)**

**WHITE**

- White British
- White Irish
- Other White background.....

**MIXED**

- White and Black Caribbean
- White and Black African
- White and Asian
- Black and Asian
- Other mixed background.....

**ASIAN or ASIAN BRITISH**

- Indian
- Pakistani
- Bangladeshi
- Other Asian background.....

**BLACK or BLACK BRITISH**

- Black Caribbean
- African
- Other Black background.....

**CHINESE**

**OTHER ETHNIC GROUP**.....

---

**RELIGIOUS BELIEFS**

**CHRISTIAN**       **MUSLIM**       **HINDU**       **SIKH**

JEWISH       BUDDHIST       NONE       OTHER.....

---

**COUNTRY OF BIRTH**.....

B8. What educational qualifications do you have?

None .....  
School Certificate ..... CSE's .....  
GCSE's, O levels ..... A levels .....  
Degree ..... Other.....

B9. How old were you when you left formal education (school/college) ? .....

B10. With whom do you live (note how many people)?

Parents ..... Spouse ..... Friends .....  
Children ..... Other relatives ..... Rest/care home .....

B10.1 Can you count on anyone to give you emotional support (e.g. talking over problems to help you with a difficult decision)?

Yes                  No                  No need of help

(If Yes) How many people would give you this kind of support? .....

B.10.2 When you need some extra help, can you count on anyone to help with daily tasks like grocery shopping, house cleaning, cooking, telephoning, giving you a lift somewhere?

Yes                  No                  No need of help

(If Yes) How many people would give you this kind of support? .....

B11. Do you rent or own your own home? ... ..

B12. How many rooms are in your home (excluding bathroom, kitchen/utility room)? .....

B13. Do you have use of a car/van? Yes / No

B14. Were you employed at the time of your heart problem? Yes / No

B15. If so, what was the nature of your employment?

Job title .....

B16. Full time ..... Part time ..... Volunteer .....  
Disabled ..... Unemployed ..... Self employed .....

B17. If retired, what was your last major occupation? .....

B18. (If married) What is/was your spouse's occupation? .....

B19. What is your current source of income? (i.e. salary, benefits, pension savings/investments/ other)  
.....

B20. What is your approximate personal **yearly** income, **before tax** is deducted? (If retired, any incoming money, as well as pension). **Use card**

- |                   |                          |
|-------------------|--------------------------|
| Under £10,000     | <input type="checkbox"/> |
| £10,000 - £20,000 | <input type="checkbox"/> |
| £20,000 - £30,000 | <input type="checkbox"/> |
| £30,000 - £40,000 | <input type="checkbox"/> |
| Over £40,000      | <input type="checkbox"/> |

B21. What **total income** has your **household** received in the last 12 months? Please include your own income and that of others from any source, including wages, savings, investments, rent or property, and benefits. **Use card**

- Under £10,000
- £10,000 - £20,000
- £20,000 - £30,000
- £30,000 - £40,000
- Over £40,000

**YOUR HEALTH**

H1. Do you have: Diabetes Yes/No  
(If Yes) Do you take insulin? Yes/No  
High blood pressure? Yes/No  
High cholesterol in your blood? Yes/No

H2. Do you have any other health problems **at the moment** (relevant to heart problem and/or hormonal, immune, respiratory, eating disorders, etc)?

.....  
.....

H3. Have you had any other health problems in the **past 5 years**? e.g. arthritis, renal etc.

.....  
.....

H3.1 Have you ever had a heart condition? Yes/No

Details:.....  
.....

H4. When did you last have a cold or 'flu? .....

H5. Were you taking any medicines or pills before you were admitted to hospital? Yes/No

If Yes, what and for how long:

.....  
.....  
.....

H6. Has anyone in your family had heart disease? Yes/No

H7. If Yes, what kind of heart disease .....

H8. Did it cause the death of your relative(s)? Yes/No

H9. If Yes, at what age did they die? .....

H10. Do you smoke cigarettes, cigars or pipes (specify)? Yes / No Type:

H11 If "Yes", please specify how many per day, and for how long you have smoked

.....

H12 If not a current smoker, did you smoke in the past? Yes / No

H12.1 If "Yes", when did you quit smoking? .....

H12.2 Are you currently taking nicotine replacement therapy? Yes / No

H13. Do you drink alcohol? Yes / No

H14.1 If Yes, how many units per week on average do you drink? .....units per week  
*(1 Unit = ½ pint of beer, 1 glass of wine or 1 measure of spirit)*

H14.2 In the past 6 months have you taken any of the following drugs? If Yes, indicate average frequency.

Marijuana Yes/No ...../daily/weekly/monthly  
 Cocaine Yes/No ...../daily/weekly/monthly  
 Heroin Yes/No ...../daily/weekly/monthly  
 Amphetamine Yes/No ...../daily/weekly/monthly  
 Other Yes/No ...../daily/weekly/monthly  
 (details .....)

**EVENTS SURROUNDING YOUR HEART PROBLEM (Use 24 hour clock to record).**

E1 What **time** of the day or night, and on what E2 **date** did your heart problem occur?

.....

**(If not possible to establish time, abbreviate interview here i.e Skip down to S1)**

E3. Tell me about any symptoms that you experienced in the four days before you were admitted to hospital (type and duration) .....

.....

.....

.....

.....

Symptoms experienced **at onset**: (Interviewer tick off)

Pain		Non-pain	
Chest pain		sweating	
pain in arms/shoulders		shortness of breath (SOB)	
Jaw pain		paroxysmal nocturnal dyspnea (abrupt episode of difficulty breathing at night)	
Back pain		numbness/tingling in arms/hands	
Other (details) :		nausea/vomiting	
		dizziness/fainting/collapse	
		gastro-intestinal distress	


fatigue  
Other (details) :


E3.1 When your symptoms started how bad was your pain on a scale of 1 to 10?  
 0 1 2 3 4 5 6 7 8 9 10  
 (No pain) (worst pain ever)

E4. If it occurred at night were you asleep (it woke you up) or just awakening? .....

E5. On the day your symptoms occurred, what time did you wake up? .....

E6. What time do you normally wake up? Time..... No habitual time?.....

E7. Where were you when your heart problem occurred?

1. At home ..... 2. Outside ..... 3. Recreational activity .....

4. At work ..... 5. In a car .....

Get details .....

E8. What did you think was happening when your symptoms came on (ie did you think it was your heart or something else)?

E9 What action did you take after symptom onset? (e.g. self- medication, rest, wait and see).

E10 How long was it between the onset of your symptoms and deciding to seek help (approx) ?

E11 Who did you call (ambulance, GP, NHS Direct, family/friend) ?

E12a Was someone else present at this time? Who?.....

E12b Who called for help – you or someone else?.....

E13 How did you get to hospital?.....

E14 Can you tell me what time you called for help? .....

E15 What were your reasons for this delay in seeking help?

.....

E16 How long did you have to wait between deciding to seek help and receiving medical attention?

.....  
E17 What were the reasons for this delay in receiving medical attention?

.....  
.....

E18 Please describe what happened during the 24 hours before your heart problem occurred.

**I am now going to ask you about various behaviours and emotions that you may have experienced during certain time periods leading up to your heart problem.**

**During 2 hours pre-event:**

Think about the 2 hours before your heart problem. It was (day) and the time was .....(See E1 for details).

T1. Did you do any exercise or physical activity enough to make you out of breath during this time?

Yes/No

T1.1 If Yes, for how long did you do this activity? .....

T2 Did you take any recreational drugs during this time? Yes/No

T2.1 (If Yes) What did you take? .....

T3 Did anything unusual occur during this time, for example, had you eaten a very large meal; had you had a large quantity of alcohol? .....

.....

T4 If Yes, ask for estimated usual frequency ...../dy, wk, mth

T5 Were you irritated or angry during this time? Yes/No

T5.1 If Yes, show card. These are varying levels of irritation and anger. For each of these hours, how would you describe how irritated or angry you were....

(record highest **level** of anger reached, an estimate of **how long** the anger lasted, and the **reason** for the anger)

1<sup>st</sup> hour - .....

.....

.....

Reason.....

2<sup>nd</sup> hour - .....

.....

Reason.....

*Whatever highest level reported, make an additional note of length of time lower levels lasted. For example, if patient reports highest level as level 4, make an additional note of how long levels 3, 2 and 1 occurred.*

T6 Were you tense or stressed during this time? Yes/No

T6.1 If Yes, show card. These are varying levels of tension and stress. For each of these hours, how you would describe how tense or stressed you were....

(record highest **level** of stress reached, an estimate of **how long** the stress lasted, and the **reason** for the stress)

1<sup>st</sup> hour - .....

.....  
Reason.....

2<sup>nd</sup> hour - .....  
.....

Reason.....

*Whatever highest level reported, make an additional note of length of time lower levels lasted. For example, if patient reports highest level as level 4, make an additional note of how long levels 3, 2 and 1 occurred.*

T7 Were you sad or depressed during this time? Yes/No

T7.1 If Yes, show card. These are varying levels of sadness and depression. For each of these hours, how you would describe how sad or depressed you were....

(record highest **level** of depression reached, an estimate of **how long** the depression lasted, and the **reason** for the depression)

1<sup>st</sup> hour - .....  
.....

Reason.....

.....

2<sup>nd</sup> hour - .....  
.....

Reason.....

*Whatever highest level reported, make an additional note of length of time lower levels lasted. For example, if patient reports highest level as level 4, make an additional note of how long levels 3, 2 and 1 occurred.*

**During same 2 hours previous day:**

Now think about the same 2 hours the day before your heart problem; that was (day) between the times of ..... and .....

T8 Did you do any exercise or physical activity enough to make you out of breath during this time? Yes/No

T8.1 If Yes, for how long did you do this activity? .....

T9. Did you take any recreational drugs during this time? Yes/No

T9.1 (If Yes) What did you take? .....

T10 Did anything unusual occur during this time, for example, had you eaten a very large meal; had you had a large quantity of alcohol? .....

.....

T10.1 If Yes, ask for estimated usual frequency ...../dy, wk, mth

T11 Were you irritated or angry during this time? Yes/No

T11.1 If Yes, show card. These are varying levels of irritation and anger. For each of these hours, how you would describe how irritated or angry you were....

(record highest **level** of anger reached, an estimate of **how long** the anger lasted, and **reason** for the anger)

1<sup>st</sup> hour - .....

.....

Reason.....

2<sup>nd</sup> hour - .....

Reason.....

*Whatever highest level reported, make an additional note of length of time lower levels lasted. For example, if patient reports highest level as level 4, make an additional note of how long levels 3, 2 and 1 occurred.*

T12 Were you tense or stressed during this time? Yes/No

T12.1 If Yes, show card. These are varying levels of tension and stress. For each of these hours, how you would describe how tense or stressed you were....

(record highest **level** of stress reached, an estimate of **how long** the stress lasted, and the **reason** for the stress)

1<sup>st</sup> hour - .....

.....

Reason.....

2<sup>nd</sup> hour - .....

.....

Reason.....

*Whatever highest level reported, make an additional note of length of time lower levels lasted. For example, if patient reports highest level as level 4, make an additional note of how long levels 3, 2 and 1 occurred.*

T13 Were you sad or depressed during this time? Yes/No

T13.1 If Yes, show card. These are varying levels of sadness and depression. For each of these hours, how you would describe how sad or depressed you were.... (record highest **level** of depression reached, an estimate of **how long** the depression lasted, and the **reason** for the depression)

1<sup>st</sup> hour - .....

.....

Reason.....

2<sup>nd</sup> hour - .....

.....

Reason.....

S1 Now I'm going to ask you to think about the **past 6 months**, from (month) to (current month). Did anything particularly bad, upsetting or stressful happen during this time?

I'll now ask you about some specific situations.

S2 In the past **6 months** has your relationship with **your partner** been stressful? Yes/No

(If Yes, show card) How stressful has it been? 1 2 3 4

S3 In the past **6 months** has your relationship with **your family** been stressful? Yes/No

(If Yes, show card) How stressful has it been? 1 2 3 4

S4 In the past **6 months** has **work** been stressful? Yes/No

(If Yes, show card) How stressful has it been? 1 2 3 4

S5 Other than your heart problem, have you experienced **any illnesses** in the **past 6 months** that you have found stressful? Yes/No

(If Yes, show card) How stressful was that? 1 2 3 4

S6 In the past **6 months** have there been **any financial issues** that have been stressful? Yes/No

(If Yes, show card) How stressful was that? 1 2 3 4

S7 In the past 6 months have you felt more **tired/fatigued** than usual? Yes/No

That's the end of the structured interview.

\* Could you tell me whether you have a theory of your own about what triggered your heart problem (i.e. what brought it on) ?

Interviewer Impression

Any trigger? Yes / No / Maybe If Yes, what? .....

Did the patient frequently contradict him/herself or give information that s/he would have no way of knowing? Yes / No

Did the patient appear reluctant to answer questions and thus might not have given complete information?

Yes / No

Are there any missing data? Yes No If Yes, why?

.....

Any other comments of interest/importance (e.g. interesting stories, unclear issues)?

Notes

When recording narrative, in the case of any unusual types of events (e.g. Skydive, Public Speech), ask usual frequency.

\* Study Number: \_\_\_\_\_ \*

**Ask patient to complete as soon as possible (i.e. during hospital stay)**

**We are interested to find out more about your experiences when your heart symptoms came on. Listed below are a series of things that other patients say they have felt in this situation. Please indicate the extent to which each statement is true for you.**

E19. I was frightened when the symptoms came on.

Not at all true	Slightly true	Somewhat true	Very true	Extremely true
-----------------	---------------	---------------	-----------	----------------

E20. I thought that I might be dying when the symptoms came on.

Not at all true	Slightly true	Somewhat true	Very true	Extremely true
-----------------	---------------	---------------	-----------	----------------

E21. I found my cardiac event stressful.

Not at all true	Slightly true	Somewhat true	Very true	Extremely true
-----------------	---------------	---------------	-----------	----------------

**Below is a list of words that describe feelings people have. Please read each one carefully, then circle the number which best describes the extent to which you have this feeling now.**

	Not at all	A little	Moderately	Quite a lot	Extremely
M1. Tense	0	1	2	3	4
M2. Feverish	0	1	2	3	4
M3. Worn out	0	1	2	3	4
M4. Angry	0	1	2	3	4
M5. Lively	0	1	2	3	4
M6. Confused	0	1	2	3	4
M7. Shaky	0	1	2	3	4
M8. Aching joints	0	1	2	3	4
M9. Sad	0	1	2	3	4
M10. Grouchy	0	1	2	3	4

M11. Active	0	1	2	3	4
M12. On edge	0	1	2	3	4
	Not at all	A little	Moderately	Quite a lot	Extremely
M13. Annoyed	0	1	2	3	4
M14. Energetic	0	1	2	3	4
M15. Hopeless	0	1	2	3	4
M16. Relaxed	0	1	2	3	4
M17. Resentful	0	1	2	3	4
M18. Unworthy	0	1	2	3	4
M19. Uneasy	0	1	2	3	4
M20. Can't concentrate	0	1	2	3	4
M21. Fatigued	0	1	2	3	4
M22. Nauseated	0	1	2	3	4
M23. Listless	0	1	2	3	4
M24. Nervous	0	1	2	3	4
M25. Lonely	0	1	2	3	4
M26. Muddled	0	1	2	3	4
M27. Furious	0	1	2	3	4
M28. Cheerful	0	1	2	3	4
M29. Exhausted	0	1	2	3	4
M30. Gloomy	0	1	2	3	4
M31. Sluggish	0	1	2	3	4
M32. Headache	0	1	2	3	4
M33. Weary	0	1	2	3	4
M34. Bewildered	0	1	2	3	4

M35. Alert	0	1	2	3	4
M36. Bitter	0	1	2	3	4
M37. Efficient	0	1	2	3	4
M38. Hungry	0	1	2	3	4
	Not at all	A little	Moderately	Quite a lot	Extremely
M39. Forgetful	0	1	2	3	4
M40. Guilty	0	1	2	3	4
M41. Vigorous	0	1	2	3	4
M42. Thirsty	0	1	2	3	4

<b>Factors that might have helped to cause my illness:</b>			
My illness is hereditary – it runs in my family	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Smoking played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by other medical problems	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Stress was a major factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Being overweight caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
High blood pressure was an important factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Diet played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
I became ill because I over-exerted myself	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
It was just by chance and bad luck that I became ill	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was caused by poor medical care in the past	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Lack of exercise was a cause of my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by tiredness and exhaustion	<b>No</b>	<b>Maybe</b>	<b>Yes</b>

Genetic factors (genes) caused my illness	No	Maybe	Yes
My state of mind played a major part in causing my illness	No	Maybe	Yes
Working too hard caused my illness	No	Maybe	Yes
A germ or virus caused my illness	No	Maybe	Yes

**Health before hospital admission**

The following questions are about your health and daily activities before you became ill and were admitted to hospital.

1. In general would you say your health was:

Excellent	Very Good	Good	Fair	Poor
-----------	-----------	------	------	------

2. The following questions are about the activities do during a typical day. Did **your health limit you** in these activities? If so how much?

- Moderate activities – such as moving a table, pushing a vacuum cleaner, bowling, or playing golf.

Yes, limited a lot	Yes, limited a little	No, not limited at all
--------------------	-----------------------	------------------------

- Climbing **several** flights of stairs.

Yes, limited a lot	Yes, limited a little	No, not limited at all
--------------------	-----------------------	------------------------

3. Before you were admitted to hospital, have you had any of the following problems with your work or other regular daily activities **as a result of your physical health**?

- **Accomplished less** than you would like.

Yes	No
-----	----

- Were limited in the **kind** of work or other activities.

Yes	No
-----	----

4. Before you were admitted to hospital, have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)?

- **Accomplished less** than you would like:

Yes	No
-----	----

- Did work or other activities less carefully than usual

Yes	No
-----	----

5. Before you were admitted to hospital, how much did **pain** interfere with your normal work (including both work outside the home and housework)?

Not at all	A little bit	Moderately	Quite a bit	Extremely
------------	--------------	------------	-------------	-----------

6. The next questions are about how you were feeling **before you were admitted to hospital**. For each question, please give the one answer that comes closest to the way you have been feeling.

How much of the time before you were admitted to hospital:

- Have you felt calm and peaceful?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Did you have a lot of energy?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Have you felt downhearted and low?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

7. Before you were admitted to hospital, how much of the time have your **physical health or emotional problems** interfered with your social activities (like visiting friends, relatives, etc.)?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

**Thank you very much for your help with this research.**

If you have completed this questionnaire after the research interview, please post it back to us using the freepost envelope provided to: TRACE Study, Psychobiology Group, Freepost WC5565, 1-19 Torrington Place, University College London, WC1E 6BT.

## APPENDIX II

### **TIME 2 PATIENT QUESTIONNAIRE**

**Tracking Recovery After Coronary Events (TRACE) study**  
**(Home Interview) Time 2**

Name: .....Date: .....Pt No: .....  
 Interviewer:.....

- Check that time 1 information about clinical procedures and treatment plan collected at time 1 are correct.
- Set up Actiheart and take Saliva sample 1.

**What medications are you currently taking? (Home Interview)**

Medication name:	Dose/ times per day	Do you ever forget, alter, stop, miss or take less than instructed of this medication?		Do you think there are any side effects? What?
		Yes	No	
1.				<u>Y/N</u>
2.				<u>Y/N</u>
3.				<u>Y/N</u>
4.				<u>Y/N</u>
5.				<u>Y/N</u>
6.				<u>Y/N</u>
7.				<u>Y/N</u>
8.				<u>Y/N</u>
9.				<u>Y/N</u>
10.				<u>Y/N</u>
11.				<u>Y/N</u>
12.				<u>Y/N</u>
13.				<u>Y/N</u>
14.				<u>Y/N</u>
15.				<u>Y/N</u>
16.				<u>Y/N</u>
17.				<u>Y/N</u>
18.				<u>Y/N</u>
19.				<u>Y/N</u>
20.				<u>Y/N</u>

- Have you experienced any pain or discomfort since you left hospital?  
 Yes/No  
 How would you describe that pain?

None	Mild	Discomforting	Distressing	Horrible	Excruciating
------	------	---------------	-------------	----------	--------------

Please explain:

Many people find a way of using their medicines which suits them. This may differ from the instructions on the label or from what their doctor has said.

We would like to ask you a few questions about how you use your medicines.

Here are some ways in which people have said that they use their medicines  
For each of the statements, please tick the box which best applies to you

		Always	Often	Sometimes	Rarely	Never
MARS1	I forget to take my medicines					
MARS21	I alter the dose of my medicines					
MARS3	I stop taking my medicines for a while					
MARS4	I decide to miss out a dose					
MARS5	I take less than instructed					

---

This part of the questionnaire consists of 21 groups of statements. After reading each group of statements carefully, circle the number (0, 1, 2, or 3) next to the one statement in each group which best describes the way you have been feeling since you were admitted to hospital, including today. If several statements within a group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1.    0    I do not feel sad.  
      1    I feel sad.  
      2    I am sad all the time and I can't snap out of it.  
      3    I am so sad or unhappy that I can't stand it.
  
2.    0    I am not particularly discouraged about the future.  
      1    I feel discouraged about the future.  
      2    I feel I have nothing to look forward to.  
      3    I feel that the future is hopeless and that things cannot improve.
  
3.    0    I do not feel like a failure.  
      1    I feel I have failed more than the average person.  
      2    As I look back on my life, all I can see is a lot of failures.  
      3    I feel I am a complete failure as a person.

4. 0 I get as much satisfaction out of things as I used to.  
 1 I don't enjoy things the way I used to.  
 2 I don't get real satisfaction out of anything anymore.  
 3 I am dissatisfied or bored with everything.
5. 0 I don't feel particularly guilty.  
 1 I feel guilty a good part of the time.  
 2 I feel guilty most of the time.  
 3 I feel guilty all of the time.
6. 0 I don't feel I am being punished.  
 1 I feel I may be punished.  
 2 I expect to be punished.  
 3 I feel I am being punished.
7. 0 I don't feel disappointed in myself.  
 1 I am disappointed in myself.  
 2 I am disgusted with myself.  
 3 I hate myself.
8. 0 I don't feel I am any worse than anybody else.  
 1 I am critical of myself for my weaknesses or mistakes.  
 2 I blame myself all the time for my faults.  
 3 I blame myself for everything bad that happens.
9. 0 I don't have any thoughts of killing myself.  
 1 I have thoughts of killing myself, but I would not carry them out.  
 2 I would like to kill myself.  
 3 I would kill myself if I had the chance.
10. 0 I don't cry any more than usual.  
 1 I cry more now than I used to.  
 2 I cry all the time now.  
 3 I used to be able to cry, but now I can't cry even though I want to.
11. 0 I am no more irritated now than I ever am.  
 1 I get annoyed or irritated more easily than I used to.  
 2 I feel irritated all the time now.  
 3 I don't get irritated at all by the things that used to irritate me.
12. 0 I have not lost interest in other people.  
 1 I am less interested in other people than I used to be.  
 2 I have lost most of my interest in other people.  
 3 I have lost all of my interest in other people.
13. 0 I make decisions about as well as I ever could.  
 1 I put off making decisions more than I used to.  
 2 I have greater difficulty in making decisions than before.  
 3 I can't make decisions at all any more.

14. 0 I don't feel I look any worse than I used to.  
 1 I am worried that I am looking old or unattractive.  
 2 I feel that there are permanent changes in my appearance that make me look unattractive.  
 3 I believe that I look ugly.
15. 0 I can work about as well as before.  
 1 It takes an extra effort to get started at doing something.  
 2 I have to push myself very hard to do anything.  
 3 I can't do any work at all.
16. 0 I can sleep as well as usual.  
 1 I don't sleep as well as I used to.  
 2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.  
 3 I wake up several hours earlier than I used to and cannot get back to sleep.
17. 0 I don't get more tired than usual.  
 1 I get tired more easily than I used to.  
 2 I get tired from doing almost anything.  
 3 I am too tired to do anything.
18. 0 My appetite is no worse than usual.  
 1 My appetite is not as good as it used to be.  
 2 My appetite is much worse now.  
 3 I have no appetite at all anymore.
19. 0 I haven't lost much weight, if any, lately.  
 1 I have lost more than 5 pounds.  
 2 I have lost more than 10 pounds.  
 3 I have lost more than 15 pounds.
- 19a I am purposely trying to lose weight by eating less. Yes \_\_\_\_\_ No \_\_\_\_\_
20. 0 I am no more worried about my health than usual.  
 1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.  
 2 I am very worried about physical problems and it's hard to think of much else.  
 3 I am so worried about my physical problems that I cannot think about anything else.
21. 0 I have not noticed any recent change in my interest in sex.  
 1 I am less interested in sex than I used to be.  
 2 I am much less interested in sex now.  
 3 I have lost interest in sex completely.

**Please circle**

Were you sexually active before your recent illness? Yes / No

Are you currently sexually active? Yes / No

**This part of the questionnaire is about your emotions and how you have been feeling since you were admitted to hospital. Read each item and tick the reply which comes closest to how you have been feeling since you were admitted to hospital.**

**I feel tense or 'wound-up':**

- 1. Most of the time
- 2. A lot of the time
- 3. Time to time, occasionally
- 4. Not at all

**I get a sort of frightened feeling as if something awful is about to happen:**

- 1. Very definitely and quite badly
- 2. Yes, but not too badly
- 3. A little, but it doesn't worry me
- 4. Not at all

**Worrying thoughts go through my mind:**

- 1. A great deal of the time
- 2. A lot of the time
- 3. From time to time but not too often
- 4. Only occasionally

**I can sit at ease and feel relaxed:**

- 1. Definitely
- 2. Usually
- 3. Not often
- 4. Not at all

**I get a sort of frightened feeling like 'butterflies' in the stomach:**

- 1. Not at all
- 2. Occasionally
- 3. Quite often
- 4. Very often

**I feel restless as if I have to be on the move:**

- 1. Very much indeed
- 2. Quite a lot
- 3. Not very much
- 4. Not at all

**I get sudden feelings of panic:**

- 1. Very often indeed
- 2. Quite often
- 3. Not very often
- 4. Not at all

## DISH + Saliva sample 2

The following questions will ask about how you have been feeling since you left hospital and the period shortly before you were admitted. Some people find these questions a bit unusual. The only reason that we ask is that we want to know how this illness might affect all areas of your life.

Since you have left hospital.....

### Loss of interest or pleasure in all

**1. Have you been feeling like you've lost interest in most things, or like you're not getting much pleasure from things you used to enjoy?**

yes
no

**2. If yes, have you been feeling like that most of the time? How long have you been feeling that way?**

0	no
1	thoughts & feelings of incapacity, more effort needed
2	loss of interest in activity, feels to have to push himself
3	decrease in actual time spent in activities or decrease in productivity
4	stopped working because of present illness

HRSD

duration: \_\_\_\_\_ (weeks/ days)

**3. Have you lost interest in spending time with other people or have you felt like avoiding people you usually like to visit?**

yes
no

**4. If yes, have you been feeling like that most of the time?**

4a. Rate social withdrawal

0	no
1	present some days
2	present most days
M	medical Sx
R	refused
U	unable to assess

4b. Rate anhedonia (loss of interest or pleasure in most activities)

0
1
2
M
R
U

no  
present some days  
present most days  
medical Sx  
refused  
unable to assess

2 = Depression Interview

duration: \_\_\_\_\_ (weeks/ days)

DSM-IV

---

**Depressed mood**

**5. What's your mood been like this week? Have you been feeling sad, depressed, empty etc. most of the time?**

0
1
2
3
4

no  
dysphoric, apparent only in PT's answers to questions  
dysphoric, PT talks spontaneously (without being asked about it)  
dysphoric, PT's answers and his/her facial expression, voice, posture, crying, etc.  
so severe that it is obvious, in virtually everything PT says and does

HRSD

**6. How long have you been feeling like that?**

0
1
2
M
R
U

no  
present some days  
present most days  
Medical Sx  
refused  
unable to assess

2 = Depression Interview

duration: \_\_\_\_\_ (weeks/ days)

DSM-IV

---

**If criteria met for anhedonia (item 4) or dysphoria (item 6) or BDI is positive go on. If negative but the impression exists, that the patient could be depressed go on, if not finish here and go to the history (page 16)**

# Depression Interview

## Appetite and weight change

### 7. How has your appetite been this past week?

0	no
1	loss of appetite but PT is eating without urging or encouragement
2	loss of appetite, urging or encouragement needed

HRSD

### 8. Has your appetite been like that most of the time? How long has it been that way?

0	normal
1	some days or weight changed but less than 5%
2	most days or weight changed but more than 5%
M	medical Sx
R	refused
U	unable to assess

DSM-IV

duration: \_\_\_\_\_

### 9. Have you lost or gained any weight lately? If yes how much? How long did it take?

0	no or only due to diet or illness
1	probable weight loss due to current depression
2	definite weight loss due to current depression

HRSD

weight gain in kg:

weight loss in kg:

duration: \_\_\_\_\_ (weeks/ days)

**Sleep**

**10. Have you had trouble falling asleep (takes more than 1/2 hour) at night this week?**

0
1
2

no  
occasional difficulty  
nightly difficulty

HRSD

**11. During the past week, have you been waking up in the middle of the night?**

yes
no

**12. If yes is that usually because you have to go to the bathroom, or for some other reason?**

0
1
2

no  
sleep is restless or disturbed during the night  
waking up during night and having difficulty falling back asleep

HRSD

**13. What time have you been waking up in the morning this week?**

Time:	
-------	--

**14. Is it too early, or is that the time that you want to wake up? (unable to go back sleep)**

0
1
2

no  
waking in early hours of the morning but goes back to sleep  
unable to fall asleep again

HRSD

**15. Have you been having trouble sleeping, sleeping too much etc. almost every day? How long has this been happening? (affect daytime functioning, extra sleep)**

0
1
2
M
R
U

no  
yes, but not causing daytime sleepiness or affecting daytime functioning  
yes, causing daytime sleepiness or affecting daytime functioning  
medical Sx  
refused  
unable to assess

DSM-IV

duration: \_\_\_\_\_ (weeks/ days) \_\_\_\_\_

**Fatigue or loss of energy**

**16. How has your energy level been this past week? Have you been feeling tired or fatigued this week?**

yes
no

**17. If yes, how bad has it been?**

0	normal
1	mild to moderate
2	severe, complains of associated symptoms e.g., aches and pains

HRSD

**18. Have you been feeling fatigued or low on energy most of the time? How long have you been feeling like that?**

0	no
1	present some days
2	present most days
M	medical Sx
R	refused
U	unable to assess

DSM-IV

duration: \_\_\_\_\_ (weeks/ days)

---

**Feelings of guilt and feelings of worthlessness**

**19. Have you been feeling guilty about anything?**

0	no
1	somewhat guilty, expresses self-reproach, thinks s/he has let other people down
2	very guilty or is ruminating about past errors or sinful deeds
3	PT believes that s/he is actually being punished in some way or delusional guilt
4	accusatory or denunciatory hallucinations

HRSD

**20. Over the last week, have you been criticizing, coming down pretty hard on yourself? Feeling worthless or inadequate? (general self-esteem)**

0	occasionally negative thoughts about self, but generally good self-esteem
1	fair self-esteem, sometimes critical of self
2	low self-esteem, frequently or strongly critical of self
M	medical Sx
R	refused
U	unable to assess

DSM-IV

**21. Have you been feeling guilty or worthless most of the time? How long have you been feeling like that?**

0	no
1	present some days
2	present most days
M	medical Sx
R	refused
U	unable to assess

DSM-IV

duration: \_\_\_\_\_ (weeks/ days)

**22. Have you been feeling hopeless most of the time? How long have you been feeling that way?**

0	not feeling hopeless
1	feels hopeless some days (duration= weeks)
2	feels hopeless most days (duration= weeks)
M	medical Sx (duration=weeks)
R	refused
U	unable to assess

DSM-IV

duration: \_\_\_\_\_ (weeks/ days)

---

**Thoughts of death and suicidal ideation**

**23. Have you had any thoughts of hurting or killing yourself?**

yes
no

**24. If yes, what have you been thinking about doing? Do you think you might actually do that? Have you made any plans to do this? How soon? Do you actually have the pills, weapon you'd need? Have you actually done anything to hurt yourself or to try to kill yourself?**

Rate severity of current suicidal features

0	no	
1	feels life is not worth living	
2	wishes s/he were dead	
3	actively thinking about, or planning/ preparing to attempt suicide, or has made a non-lethal suicidal gesture (e.g., taking a few pills)	
4	has actually attempted suicide this week	HRSD

Rate current suicidal features

0	no	
1	minimal suicidal ideation or behavior	
2	significant suicidal ideation or behavior	
M	medical Sx	
R	refused	
U	unable to assess	DSM-IV

duration: \_\_\_\_\_ (weeks/ days)

**Rate risk**

A	at imminent risk of attempting suicide within hours or days
B	at elevated risk of attempting suicide at some point

**Ability to concentrate and making decisions**

**25. Have you been having trouble concentrating, making decisions most of the time lately? How long has that been happening?**

0
1
2
M
R
U

no  
present some days  
present most days  
medical Sx  
refused  
unable to assess

DSM-IV

duration: \_\_\_\_\_ (weeks/ days)

**26. This past week, have you been worrying a lot? About big problems, or about little things that you don't ordinarily worry much about? If yes, like what, for example?**

0
1
2
3
4

no  
subjective tension and irritability  
worrying about minor matters  
worried, apparent in face and speech  
severely worried, fears expressed without questioning

HRSD

**27. In the past week, have you been feeling physically tense or nervous? If yes, how tense or nervous have you been? (bothersome symptoms)**

0
1
2
3
4

absent  
only apparent in PT's verbal answers to this item  
reports bothersome symptoms; may look tense or nervous  
reports severe symptoms; looks very tense or nervous  
is debilitated by nervousness

HRSD

**28. In the last week, how much have your thoughts been focused on your physical health or how your body is working?**

0
1
2
3
4

no  
somewhat worried or concerned about health  
preoccupied with worries or concerns about health, illness, or medical care  
very worried and preoccupied, or requests help in excess of need  
hypochondrical delusions

HRSD

**29. How has your interest in sex been this week? (different from usual interest)**

0
1
2

no  
mild loss of interest in sex  
severe loss of interest in sex

HRSD

### 30. Observations during interview

***rate current psychomotor retardation***

0	normal
1	slight retardation at interview
2	obvious retardation at interview
3	retardation so severe that PT is difficult to interview
4	PT is stuporous, unresponsive to most questions

HRSD

***rate current psychomotor agitation***

0	no
1	PT is edgy or mildly restless
2	PT is fidgety or uncomfortably restless
3	PT is overactive, unable to sit still
4	PT is strikingly agitated, E.G., relentlessly pacing, wringing hands, etc.

HRSD

***rate psychomotor behavior in total***

0	no
1	mild retardation or agitation observed, on some days only
2	retardation or agitation observed, on most days
M	medical Sx
R	refused
U	unable to assess

DSM-IV

***rate level of insight or lack of insight into depression***

0a	no
0b	is depressed, and is aware and <u>acknowledges</u> of being depressed
1	is depressed, but s/he <u>denies</u> being depressed or blames the symptoms on unlikely causes
2	is depressed, and is so severely depressed that s/he believes his/her current state is something other than (and perhaps much worse than) depression

HRSD

***Any major neuropsychiatric problems like paranoia, delusions, hallucinations, hypomania or mania, bizarre behavior, language deficits, dementia, confusion, lethargy?***

***Paranoia?***

0	no
1	suspicious
2	ideas of reference
3	delusions of reference and persecution

***Depersonalization and derealization (such as: feelings of unreality; nihilistic ideas)?***

0	no
1	mild
2	moderate
3	severe
4	incapacitating

## Psychiatric history

### 31. Have you ever been depressed before?

0	no
1	yes
R	refused
U	unable to assess

### 32. If yes, how many prior major depressive episodes lasting more than two weeks?

0	no
	number of probable major depressive episodes
R	refused
U	unable to assess

### 33. Which symptoms?

episode	symptoms	severity (minor:2-4 symptoms including dysphoria and/ or anhedonia: major:5-9 symptoms including d/a)
<b>1</b>	depressed mood	agitation or retardation
	anhedonia	fatigue or loss of energy
	appetite change	feeling worthless or guilty
	weight change	poor concentration or indecision
	sleep disturbance	suicidal ideation
<b>2</b>	depressed mood	agitation or retardation
	anhedonia	fatigue or loss of energy
	appetite change	feeling worthless or guilty
	weight change	poor concentration or indecision
	sleep disturbance	suicidal ideation
<b>3</b>	depressed mood	agitation or retardation
	anhedonia	fatigue or loss of energy
	appetite change	feeling worthless or guilty
	weight change	poor concentration or indecision
	sleep disturbance	suicidal ideation

more?

**34. How old were you the first time?**

	age at onset of first (prior) episode for major depression
N	not applicable (no prior episodes)
R	refused
U	unable to assess

**35. How old were you the last time?**

	age at onset of last episode for major depression
N	not applicable (less than 2 episodes)
R	refused
U	unable to assess

**36. Were you ever treated for depression during any of these times?**

yes
no

**37. Which treatment?**

A	Psychotherapy or counseling
B	antidepressant medication
C	ECT (Electro-convulsive or shock therapy)
D	Psychiatric hospitalization
E	others:

**38. Are you currently being treated for depression?**

yes	no
-----	----

**39. Which treatment?**

A	Psychotherapy or counseling
B	antidepressant medication

E
---

others:

**40. Have you ever been told by a psychiatrist that you have bipolar/ manic depression?**

0	no
1	yes
N	not applicable (less than 2 prior depressive episodes)
R	refused
U	unable to assess

**41. Has anyone in your immediate family ever been depressed for two weeks or longer?**

0	none
	number of affected first degree relatives with unipolar depression
R	refused
U	unable to assess

**42. Have you ever been treated for any other psychiatric disorder or emotional problem? (anxiety, PTSD, substance abuse etc.)**

yes
no

- Saliva sample 3

## Your reactions to your heart problem

**These questions relate to thoughts and/or feelings you may have experienced over the last few weeks since you had the acute heart symptoms which led to your hospital admission. Please circle whichever answer seems to apply closest you.**

1. Have you been trying not to think about or have feelings associated with your heart problem?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

2. Have you had upsetting thoughts or images that related to your heart problem and that came into your head when you didn't want them to?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

3. Have you felt very emotionally upset when reminded of the time your acute heart symptoms came on, such as becoming very scared, angry, sad?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

4. Have you been having physical reactions if reminded of when your heart symptoms first occurred, for example heart beating fast, breaking out in a cold sweat etc?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

5. Have you had the experience of reliving the time when your acute heart symptoms occurred, acting or feeling as if it were happening again?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

6. Have you been unable to remember any important parts of the time when your heart problem started and when you got into hospital?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

7. Have you been having problems falling or staying asleep?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

8. Have you been having bad dreams or nightmares about your heart problem?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

9. Have you been having difficulty concentrating, for example drifting in and out of conversations, losing track of the story on television, difficulty remembering what you have read?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

10. Have you been making efforts to avoid activities, situations or places that remind you of when the symptoms that led to your being admitted to hospital started?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

11. Have you felt distant or cut off from others?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

12. Have you been jumpier or more easily startled?

Not at all	A little bit	Half of the time	Almost always
------------	--------------	------------------	---------------

13. Have you been overly alert, for example checking to see who is around you?

Not at all	A little bit	Half of the time	Almost always
------------	--------------	------------------	---------------

14. Have you been more irritable or had outbursts of anger?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

15. Have you felt emotionally numb, for example, felt sad but couldn't cry, unable to have loving feelings?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

16. Have you found that you have not been interested in things you used to enjoy doing?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

17. Have you felt that any future plans or hopes have changed because of the heart problem that led to your going into hospital?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

The following is a list of activities that people often do during the week. Although for some people with several medical problems it is difficult to determine what it is that limits them, please go over the activities listed below and indicate how much limitation you have had due to chest pain, chest tightness, or angina since you left hospital.

Place an X in the box

	Activity	Severely Limited	Moderately Limited	Somewhat Limited	A Little Limited	Not Limited	Limited or did not do for other reasons.
SAQ1	Dressing yourself						
SAQ2	Walking indoors on level ground						
SAQ3	Showering						
SAQ4	Climbing a hill or a flight of stairs without stopping						
SAQ5	Gardening, vacuuming, or carrying groceries						
SAQ6	Walking more than a block at a brisk pace						
SAQ7	Running or jogging						
SAQ8	Lifting or moving heavy objects (e.g. furniture, children)						
SAQ9	Participating in strenuous sports (e.g. swimming tennis)						

Since you left hospital, on average, how many times have you had chest pain, chest tightness, or angina?

I get chest pain, chest tightness, or angina...

4 or more times per day	1-3 times per day	3 or more times per week but not every day	1-2 times per week	Less than once a week	None over the past 4 weeks
-------------------------	-------------------	--	--------------------	-----------------------	----------------------------

Since you left hospital, on average, how many times have you had to take nitros (nitroglycerin tablets) for your chest pain, chest tightness, or angina?

I take nitros...

4 or more times per day	1-3 times per day	3 or more times per week but not every day	1-2 times per week	Less than once a week	None over the past 4 weeks
-------------------------	-------------------	--	--------------------	-----------------------	----------------------------

How bothersome is it for you to take your pills for chest pain, chest tightness or angina as prescribed?

Very bothersome	Moderately bothersome	Somewhat bothersome	A little bothersome	Not bothersome at all	My doctor has not prescribed pills.
-----------------	-----------------------	---------------------	---------------------	-----------------------	-------------------------------------

**How satisfied are you that everything possible is being done to treat your chest pain, chest tightness or angina?**

Not at all satisfied	Mostly dissatisfied	Somewhat satisfied	Mostly satisfied	Highly satisfied
----------------------	---------------------	--------------------	------------------	------------------

**How satisfied are you with the explanations your doctor has given you about your chest pain, chest tightness, or angina?**

Not at all satisfied	Mostly dissatisfied	Somewhat satisfied	Mostly satisfied	Highly satisfied
----------------------	---------------------	--------------------	------------------	------------------

**Overall, how satisfied are you with the current treatment of your chest pain, chest tightness, or angina?**

Not at all satisfied	Mostly dissatisfied	Somewhat satisfied	Mostly satisfied	Highly satisfied
----------------------	---------------------	--------------------	------------------	------------------

The following questions are about your health and daily activities. Read each item and circle one answer for each question.

1. In general would you say your health is:

Excellent	Very Good	Good	Fair	Poor
-----------	-----------	------	------	------

2. The following questions are about the activities you might do during a typical day. Does **your health now limit you** in these activities? If so how much?

- Moderate activities – such as moving a table, pushing a vacuum cleaner, bowling, or playing golf.

Yes, limited a lot	Yes, limited a little	No, not limited at all
--------------------	-----------------------	------------------------

- Climbing **several** flights of stairs.

Yes, limited a lot	Yes, limited a little	No, not limited at all
--------------------	-----------------------	------------------------

3. Since you left hospital, have you had any of the following problems with your work or other regular daily activities **as a result of your physical health**?

- **Accomplished less** than you would like.

Yes	No
-----	----

- Were limited in the **kind** of work or other activities.

Yes	No
-----	----

4. Since you left hospital, have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)?

- **Accomplished less** than you would like:

Yes	No
-----	----

- Did work or other activities less carefully than usual

Yes	No
-----	----

5. Since you left hospital, how much did **pain** interfere with your normal work (including both work outside the home and housework)?

Not at all	A little bit	Moderately	Quite a bit	Extremely
------------	--------------	------------	-------------	-----------

6. These questions are about how you feel and how things have been with you **since you left hospital**. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the **time since you left hospital**:

- Have you felt calm and peaceful?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Did you have a lot of energy?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Have you felt downhearted and low?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

7. Since you left hospital, how much of the time has your **physical health or emotional problems** interfered with your social activities (like visiting friends, relatives, etc.)?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

**Thinking about the period before your recent illness,**

FV1 How many pieces of fruit – of any sort- do you eat on a typical day?

Average portions of fruit per day: \_\_\_\_\_

FV2 How often do you eat less than this average figure?

Never	Once a week	Twice a week	Three times a week	Four times a week	Five or more times a week
-------	-------------	--------------	--------------------	-------------------	---------------------------

How many portions of vegetables –excluding potatoes- do you eat on a typical day?

FV3 Average servings of vegetables per day:\_\_\_\_\_

FV4 How often do you eat less than this figure?

Never	Once a week	Twice a week	Three times a week	Four times a week	Five or more times a week
-------	-------------	--------------	--------------------	-------------------	---------------------------

**Thinking about the period before your recent illness,**

		Always/Almost Always	Sometimes	Rarely	Never	Do not eat
FT1	How often do you use skimmed or semi-skimmed milk instead of full-fat milk?					
FT2	When you use spread or cooking fat how often do you use a lower saturated fat option (e.g. sunflower, olive or rapeseed)					
FT3	When you eat meat, how often do you eat lean meat (chicken/turkey no skin, beef, pork lamb fat removed)					
FT4	When you eat ready meals how often do you eat a healthy option, controlled for calories, fat and					

	salt?					
--	-------	--	--	--	--	--

		Less than once a week/never	1-3	4-6	7 or more	
FT5	About how many times in a week do you eat cheese?					
FT6	About how many times in a week do you eat crisps or similar snacks (e.g. Doritos, Pringles)?					
FT7	About how many times in a week do you eat cakes, biscuits or puddings?					
FT8	About how many times a week do you eat meat products (e.g. sausages, pate, burgers not including those from fast-food outlets)?					
FT9	About how many times a week do you eat fast-food or take aways (e.g burgers, pizza, fried chicken, fish & chips, indian, chinese)?					

**In the month before you were admitted to hospital how often did you?**

		Not at all	1-3 days	4-7 days	8-14 days	15-20 days	21-31 days
JK1	Have trouble falling asleep?						
JK 2	Wake up several times per night?						
JK 3	Have trouble staying asleep (including waking far too early)?						
JK4	Wake up after your usual amount of sleep feeling tired and worn out?						
JK5	Have disturbed or restless sleep?						

**Physical activity: Before your recent illness?**

How many times per week did you do vigorous physical activity enough to make you out of breath?

**None            1            2            3            4            5            6+**

Please specify the activity .....

**Thinking about the days of the PAST WEEK.**

On average, for how long did you **walk** outside your home/workplace?  
(if you did not walk, please enter zero (0) in each box)

on each weekday

Hours	Minutes

on each weekend day

Hours	Minutes

On average, for how long did you cycle?  
(if you did not cycle, please enter zero (0) in each box)

for example 1 hour 30 minutes, **not** 90 minutes

on each weekday

Hours	Minutes

on each weekend day

Hours	Minutes

How would you describe your usual walking pace?

**(Please tick one box only)**

Slow pace (less than 3 mph)

1
---

Steady average pace

2
---

Brisk pace

3
---

Fast pace (over 4 mph)

4

- Saliva sample 4

**Questionnaire to leave with participant following home visit.**

Name: ..... Date: ..... Pt No: .....

**Medical Research Study (St George’s 2007)**

**Tracking Recovery after Acute Coronary Events:  
The TRACE study.**

Thank you very much for participating in this study of heart disease. In addition to the interview we have given you, we would like you to complete this questionnaire about your lifestyle, your attitudes and opinions, the way you feel about yourself and the way you feel about your heart problem. You may feel that some of the questions do not apply to you, but please answer each question with the answer that most closely fits the way you feel.

The answers you provide in this questionnaire will be kept **strictly confidential**. The information will go into the statistics for the study, and it will not be possible to identify you personally in any reports. Under no circumstances will any of the information you give us be made available to anyone else.

Most of the questions can be answered by circling the appropriate answer.

***For example:***

“It’s easy for me to relax.”

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

Please be sure to read the instructions to each section carefully.

Thank you very much for your participation. If you have any difficulty with any questions, please do not hesitate to contact us.

**This section of the questionnaire is concerned with how many people you see or talk to on a regular basis including family, friends, workmates, neighbours, etc. Please circle your answer to each question.**

1. Do you have children? 

Yes	No
-----	----

If Yes, how often do you see or talk on the phone to your children?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

2. Are either of your parents living? 

Yes	No
-----	----

If your mother is living, how often do you see or talk on the phone to her?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

If your father is living, how often do you see or talk on the phone to him?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

3. If you are married or living with your partner, are either of your in-laws (spouse's parents) living?

Yes	No
-----	----

If your mother-in-law is living, how often do you see or talk on the phone to her?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

If your father-in-law is living, how often do you see or talk on the phone to him?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

4. Are there other relatives who you feel close to?

Yes	No
-----	----

If Yes, how often do you see or talk on the phone to these relatives?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

5. Do you have friends who you feel close to (i.e., people you feel at ease with, can talk to about private matters, and can call on for help)?

Yes	No
-----	----

If Yes, how often do you see or talk on the phone to these friends?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

6. Do you belong to a church, temple, mosque or other religious group?

Yes	No
-----	----

If Yes, how often do you talk to members of this religious group?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

7. Do you attend any classes (school, university, technical training, or adult education) on a regular basis?

Yes	No
-----	----

If Yes, how often do you talk to fellow students or teachers?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

8. If you are currently working, how often do you talk to people (other than those you supervise) at work?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

		weeks		
--	--	-------	--	--

9. How often do you visit or talk to your neighbours?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

10. Are you currently involved in any regular volunteer work?

Yes	No
-----	----

If Yes, how often do you talk to people involved in this work?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

11. Do you belong to any non-religious groups? Examples include social clubs, recreational groups, trades unions, etc.

Yes	No
-----	----

If Yes, how often do you talk to fellow group members?

Never	Once a month	Once every two weeks	Once a week	Every day
-------	--------------	----------------------	-------------	-----------

**These questions are about the support that you get from other people. Please circle your answer to each question.**

1. Is there someone available to whom you can count on to listen to you when you need to talk?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

2. Is there someone available to you to give you good advice about a problem?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

3. Is there someone available to you who shows you love and affection?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

4. Is there someone available to help with daily chores?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

5. Can you count on anyone to provide you with emotional support (talking over problems or helping you make a difficult decision)?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

6. Do you have as much contact as you would like with someone you feel close to, someone in whom you can trust and confide in?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

7. Is there someone available who reminds you to take your medication?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

8. Is there someone available who reminds you or helps you to eat a healthy diet?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

9. Is there someone available who reminds you or helps you to take some exercise?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

**Below are a number of statements that people often use to describe themselves. Please read each statement and then circle the appropriate number next to that statement to indicate your answer. There are no right or wrong answers: Your own impression is the only thing that matters.**

**0=FALSE 1=RATHER FALSE 2=NEUTRAL 3=RATHER TRUE 4=TRUE**

- 
- <sup>1</sup> I make contact easily when I meet people -----     **0 1 2 3 4**
- <sup>2</sup> I often make a fuss about unimportant things -----      **0 1 2 3 4**
- <sup>3</sup> I often talk to strangers -----     **0 1 2 3 4**
- <sup>4</sup> I often feel unhappy -----     **0 1 2 3 4**

- 5 I am often irritated -----     **0 1 2 3 4**
- 6 I often feel inhibited in social interactions -----     **0 1 2 3 4**
- 7 I take a gloomy view of things -----     **0 1 2 3 4**
- 8 I find it hard to start a conversation -----     **0 1 2 3 4**
- 9 I am often in a bad mood -----     **0 1 2 3 4**
- 10 I am a closed kind of person -----      **0 1 2 3 4**
- 11 I would rather keep other people at a distance-----     **0 1 2 3 4**
- 12 I often find myself worrying about something -----     **0 1 2 3 4**
- 13 I am often down in the dumps -----     **0 1 2 3 4**
- 14 When socializing, I don't find the right things  
to talk about -----     **0 1 2 3 4**
-

**Below are some statements that describe people's beliefs and attitudes and the way they might react to some situations. If the statement applies to you or describes you in general, circle TRUE. If the statement does not describe you, circle FALSE.**

1.	When someone does me a wrong I feel I should pay them back if I can, just for the principle of the thing.	TRUE	FALSE
2.	I have often had to take orders from someone who did not know as much as I did.	TRUE	FALSE
3.	I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.	TRUE	FALSE
4.	It takes a lot of argument to convince most people of the truth.	TRUE	FALSE
5.	I think most people would lie to get ahead.	TRUE	FALSE
6.	Someone has it in for me.	TRUE	FALSE
7.	Most people are honest chiefly because they are afraid of being caught.	TRUE	FALSE
8.	Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it.	TRUE	FALSE
9.	I commonly wonder what hidden reason another person may have for doing something nice for me.	TRUE	FALSE
10.	It makes me impatient when people ask my advice or otherwise interrupt me when I am working on something important.	TRUE	FALSE
11.	I feel that I have often been punished without cause	TRUE	FALSE
12.	Some of my family have habits that bother and annoy me very much.	TRUE	FALSE
13.	My relatives are nearly all in sympathy with me.	TRUE	FALSE
14.	My way of doing things is apt to be misunderstood by others.	TRUE	FALSE
15.	I don't blame anyone for trying to grab everything they can get in this world.	TRUE	FALSE
16.	No one cares much what happens to you.	TRUE	FALSE
17.	I can be friendly with people who do things which I consider wrong.	TRUE	FALSE
18.	It is safer to trust nobody.	TRUE	FALSE
19.	I do not blame a person for taking advantage of people who leave themselves open to it.	TRUE	FALSE
20.	I have often felt that strangers were looking at me critically	TRUE	FALSE
21.	Most people make friends because friends are likely to be useful to them.	TRUE	FALSE

22.	I am sure I am being talked about	TRUE	FALSE
23.	Most people inwardly dislike putting themselves out to help other people.	TRUE	FALSE
24.	I tend to be on my guard with people who are somewhat more friendly than I had expected.	TRUE	FALSE
25.	People often disappoint me.	TRUE	FALSE
26.	I am not easily angered.	TRUE	FALSE
27.	I have often met people who were supposed to be experts who were no better than I.	TRUE	FALSE
28.	I would certainly enjoy beating criminals at their own game.	TRUE	FALSE
29.	I have at times had to be rough with people who were rude or annoying.	TRUE	FALSE
30.	People generally demand more respect for their own rights than they are willing to allow for others.	TRUE	FALSE
31.	There are certain people whom I dislike so much that I am inwardly pleased when they are catching it for something they have done.	TRUE	FALSE
32.	I am often inclined to go out of my way to win a point with someone who has opposed me.	TRUE	FALSE
33.	The man who had most to do with me when I was a child (such as my father, stepfather, etc.) was very strict with me .	TRUE	FALSE
34.	I have often found people jealous of my good ideas, just because they had not thought of them first.	TRUE	FALSE
35.	I do not try to cover up my poor opinion or pity of people so that they won't know how I feel.	TRUE	FALSE
36.	I have frequently worked under people who seem to have things arranged so that they get credit for good work but are able to pass off mistakes onto those under them.	TRUE	FALSE
37.	I strongly defend my own opinions as a rule.	TRUE	FALSE
38.	Sometimes I am sure that other people can tell what I am thinking.	TRUE	FALSE
39.	A large number of people are guilty of bad sexual conduct	TRUE	FALSE

## YOUR VIEWS ABOUT YOUR ILLNESS

Listed below are a number of symptoms that you may or may not have experienced since your illness. Please indicate by circling Yes or No, whether you have experienced any of these symptoms since your illness, and whether you believe that these symptoms are related to your illness.

	I have experienced this symptom <i>since my illness</i>		This symptom is <i>related to</i> <i>my illness</i>		
	Yes	No	Yes	No	
Pain	Yes	No	_____	Yes	No
Sore Throat	Yes	No	_____	Yes	No
Nausea	Yes	No	_____	Yes	No
Breathlessness	Yes	No	_____	Yes	No
Weight Loss	Yes	No	_____	Yes	No
Fatigue	Yes	No	_____	Yes	No
Stiff Joints	Yes	No	_____	Yes	No
Sore Eyes	Yes	No	_____	Yes	No
Wheeziness	Yes	No	_____	Yes	No
Headaches	Yes	No	_____	Yes	No
Upset Stomach	Yes	No	_____	Yes	No
Sleep Difficulties	Yes	No	_____	Yes	No
Dizziness	Yes	No	_____	Yes	No
Loss of Strength	Yes	No	_____	Yes	No

We are interested in your own personal views of how you now see your current illness. Please indicate how much you agree or disagree with the following statements about your illness by ticking the appropriate box.

	VIEWS ABOUT YOUR ILLNESS	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IPQ1	My illness will last a short time					
IPQ2	My illness is likely to be permanent rather than temporary					
IPQ3	My illness will last for a long time					
IPQ4	This illness will pass quickly					

	VIEWS ABOUT YOUR ILLNESS	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IPQ5	I expect to have this illness for the rest of my life					
IPQ6	My illness is a serious condition					
IPQ7	My illness has major consequences on my life					
IPQ8	My illness does not have much effect on my life					
IPQ9	My illness strongly affects the way others see me					
IPQ10	My illness has serious financial consequences					
IPQ11	My illness causes difficulties for those who are close to me					
IPQ12	There is a lot which I can do to control my symptoms					
IPQ13	What I do can determine whether my illness gets better or worse					
IPQ14	The course of my illness depends on me					
IPQ15	Nothing I do will affect my illness					
IPQ16	I have the power to influence my illness					
IPQ17	My actions will have no affect on the outcome of my illness					
IPQ18	My illness will improve in time					
IPQ19	There is very little that can be done to improve my illness					
IPQ20	My treatment will be effective in curing my illness					
IPQ21	The negative effects of my illness can be prevented (avoided) by my treatment					
IPQ22	My treatment can control my illness					
IPQ23	There is nothing which can help my condition					

	VIEWS ABOUT YOUR ILLNESS	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IPQ24	The symptoms of my condition are puzzling me					
IPQ25	My illness is a mystery to me					
IPQ26	I don't understand my illness					
IPQ27	My illness doesn't make any sense to me					
IPQ28	I have a clear picture or understanding of my condition					
IPQ29	The symptoms of my illness change a great deal from day to day					
IPQ30	My symptoms come and go in cycles					
IPQ31	My illness is very unpredictable					
IPQ32	I go through cycles in which my illness gets better and worse.					
IPQ33	I get depressed when I think about my illness					
IPQ34	When I think about my illness I get upset					
IPQ35	My illness makes me feel angry					
IPQ36	My illness does not worry me					
IPQ37	Having this illness makes me feel anxious					
IPQ38	My illness makes me feel afraid					

**What do you think caused your heart problem?**

**Serious heart disease may be caused by many different factors. We would like to find out what factors you think were involved with your own illness. Listed below are a series of factors that patients in the past have thought helped to cause their heart disease symptoms. Please think about each item, then circle the answer that indicates how much you agree or disagree with each statement.**

<b>Factors that might have helped cause my illness:</b>			
My illness is hereditary – it runs in my family	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Smoking played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by other medical problems	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Stress was a major factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Being overweight caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
High blood pressure was an important factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Diet played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
I became ill because I over-exerted myself	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
It was just by chance and bad luck that I became ill	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was caused by poor medical care in the past	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Lack of exercise was a cause of my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by tiredness and exhaustion	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Genetic factors (genes) caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My state of mind played a major part in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Working too hard caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
A germ or virus caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>

**These questions concern the way you feel about your heart problem. Please indicate the extent you agree with each of the following statements. Circle one answer for each statement. Please try to be as accurate and honest as you can and try not to let your answers to one question influence your answers to another question. There are no right or wrong answers.**

1. I was not at all afraid when my symptoms first occurred.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

2. I am a carefree, jovial person.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

3. I was not at all afraid when I learned that I had had a heart problem.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

4. I do not fear dying at all.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

5. I very seldom take unnecessary risks.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

6. My friends worry much more about my well-being than I do.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

7. I seldom change the way I describe my heart problem to others, no matter who they are.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

8. I am very calm even when faced with serious difficulties.

Strongly disagree	Disagree	Agree	Strongly agree
-------------------	----------	-------	----------------

**To what extent do you feel confident that you can do the different things you need to do to take care of your heart condition, regardless of whether or not you actually do them?**

**Please look at each of these items in the list below and give a number from 0 to 10 to indicate how confident you are that you can do the following:**

0 <b>Not at all confident</b>	1	2	3	4	5	6	7	8	9	10 <b>Extremely confident</b>
--------------------------------------	---	---	---	---	---	---	---	---	---	--------------------------------------

1. Take your medication when you are supposed to \_\_\_\_\_/10
2. Keep to a healthy diet \_\_\_\_\_/10
3. Maintain a healthy body weight \_\_\_\_\_/10
4. Get enough exercise \_\_\_\_\_/10
5. Pace yourself to avoid exercising too strenuously \_\_\_\_\_/10
6. Control symptoms of your health condition (e.g., chest pain, breathlessness) by adjusting your activity, medications, or diet \_\_\_\_\_/10
7. Avoid using tobacco and alcohol \_\_\_\_\_/10
8. Get enough sleep each night \_\_\_\_\_/10
9. Avoid stressful situations \_\_\_\_\_/10
10. Get medical advice when you need it \_\_\_\_\_/10

**Think about your closest relationship (e.g. with a family member or friend, but not your partner). Considering what you put into this relationship compared to what you get out of it and what this person puts in compared to what he or she gets out of it, how does your relationship stack up?**

- +3:** I am getting a much better deal than this person
- +2:** I am getting a somewhat better deal.
- +1:** I am getting a slightly better deal.
- 0:** We are both getting an equally good or bad deal.
- 1:** This person is getting a slightly better deal.
- 2:** This person is getting a somewhat better deal.
- 3:** This person is getting a much better deal than I am.

**The following are ways people react to various difficult, stressful, or upsetting situations. Please circle a number from 1 to 5 for each item. Indicate how much you engage in these types of activities when you encounter a difficult, stressful, or upsetting situation.**

**Typically I.....**

**1. Take some time off and get away from the situation**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**2. Focus on the problem and see how I can solve it**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**3. Blame myself for having gotten into this situation**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**4. Treat myself to a favorite food or snack**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**5. Feel anxious about not being able to cope**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**6. Think about how I solved similar problems**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**7. Visit a friend**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**8. Determine a course of action and follow it**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**9. Buy myself something**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**10. Blame myself for being too emotional about the situation**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**11. Work to understand the situation**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**12. Become very upset**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**13. Take corrective action immediately**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**14. Blame myself for not knowing what to do**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**15. Spend time with a special person**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**16. Think about the event and learn from my mistakes**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**17. Wish that I could change what had happened or how I felt**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**18. Go out for a snack or meal**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**19. Analyze my problem before reacting**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**20. Focus on my general inadequacies**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**21. Phone a friend**

1 not at all	2	3	4	5 very much
--------------	---	---	---	-------------

**Remove this page if participant has no partner.**

**If you have a spouse or partner please answer the following questions?**

**"Every relationship has its good and bad aspects. How satisfied are you with the following aspects of yours?"**

**a. Amount of time you and your partner spend together**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**b. Communication between you and your partner**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**c. Similar interests**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**d. Similar lifestyles**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**e. Sexual activity**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**f. Similar temperament**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**g. Agreement on financial matters**

---

**Considering what you put into your relationship with your partner compared to what you get out of it and what your partner puts in compared to what he or she gets out of it, how does your relationship stack up?**

- +3:** I am getting a much better deal than my partner
  - +2:** I am getting a somewhat better deal
  - 0:** We are both getting an equally good or bad deal.
  - 1:** My partner is getting a slightly better deal.
  - 2:** My partner is getting a somewhat better deal.
  - 3:** My partner is getting a much better deal than I am.
-

**The following statements concern your attitudes and opinions. Please indicate the extent you agree with each of the following statements. Please circle one answer for each statement. There are no right or wrong answers.**

1. In uncertain times, I usually expect the best.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

2. It's easy for me to relax.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

3. If something can go wrong for me, it will.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

4. I'm always optimistic about my future.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

5. I enjoy my friends a lot.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

6. It's important for me to keep busy.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

7. I hardly ever expect things to go my way.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

8. I don't get upset too easily.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

9. I rarely count on good things happening to me.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

10. Overall, I expect more good things to happen to me than bad.

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

Patients who have experienced a heart problem sometimes feel that having a heart problem makes contributions to their lives, as well as causing problems. Indicate how much you agree with each of the following statements, using these response options below.

**My heart problem...**

**1. Has led me to be more accepting of things.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**2. Has taught me how to adjust to things I cannot change.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**3. Has helped me take things as they come.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**4. Has brought my family closer together.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**5. Has made me more sensitive to family issues.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**6. Has taught me that everyone has a purpose in life.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**7. Has shown me that all people need to be loved.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**8. Has made me realize the importance of planning for my family's future.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**9. Has made me more aware and concerned for the future of all human beings.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**10. Has taught me to be patient.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**11. Has led me to deal better with stress and problems.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**12. Has led me to meet people who have become some of my best friends.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**13. Has contributed to my overall emotional and spiritual growth.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**14. Has helped me become more aware of the love and support available from other people.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**15. Has helped me realise who my real friends are.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**16. Has helped me become more focused on priorities, with a deeper sense of purpose in life.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**17. Has helped me become a stronger person, more able to cope effectively with future life challenges.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

That is the end of the questionnaire. Please check that you have answered all of the questions. If you had any difficulty with any of the questions we can call you to discuss it. Thank you very much for taking the time to make this important contribution to our study of emotions and heart disease. We will be in touch with you regarding the next stage of the study.

**Please return this questionnaire with the saliva samples and diary in the freepost envelope. If you have misplaced the envelope you can return the questionnaire, saliva samples and diary to the following address, there is no need to use a stamp:**

**TRACE  
Psychobiology group (3<sup>rd</sup> floor)  
Department of Epidemiology & Public Health  
UCL, 1-19 Torrington Place  
FREEPOST WC5565  
London WC1E 6BT**

## APPENDIX III

### **TIME 3 PATIENT QUESTIONNAIRE TRACE STUDY**

## Tracking Recovery After Coronary Events (TRACE) study

Time 3

Date questionnaire mailed:.....

Name: .....Date: .....Pt No: .....

Thank you very much for participating in this study of psychological experience & acute coronary events. You may feel that some of the questions do not apply to you, but please answer each question with the answer that most closely fits the way you feel.

The answers you provide in this questionnaire will be kept **strictly confidential**. The information will go into the statistics for the study, and it will not be possible to identify you personally in any reports. Under no circumstances will any of the information you give us be made available to anyone else.

Most of the questions can be answered by circling the appropriate answer or placing an X or a tick in the box..

***For example:***

“It’s easy for me to relax.”

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

Please be sure to read the instructions to each section carefully.

Thank you very much for your participation. If you have difficulty with any questions, please ask the researcher.

Many people find a way of using their medicines which suits them. This may differ from the instructions on the label or from what their doctor has said.

We would like to ask you a few questions about how you use your medicines.

Here are some ways in which people have said that they use their medicines  
For each of the statements, please tick the box which best applies to you

		Always	Often	Sometimes	Rarely	Never
MARS1	I forget to take my medicines					
MARS21	I alter the dose of my medicines					
MARS3	I stop taking my medicines for a while					
MARS4	I decide to miss out a dose					
MARS5	I take less than instructed					

This part of the questionnaire consists of 21 groups of statements. After reading each group of statements carefully, circle the number (0, 1, 2, or 3) next to the one statement in each group which best describes the way you have been feeling during the past two weeks, including today. If several statements within a group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1.    0      I do not feel sad.  
      4      I feel sad.  
      5      I am sad all the time and I can't snap out of it.  
      6      I am so sad or unhappy that I can't stand it.
  
2.    0      I am not particularly discouraged about the future.  
      4      I feel discouraged about the future.  
      5      I feel I have nothing to look forward to.  
      6      I feel that the future is hopeless and that things cannot improve.
  
3.    0      I do not feel like a failure.  
      4      I feel I have failed more than the average person.  
      5      As I look back on my life, all I can see is a lot of failures.  
      6      I feel I am a complete failure as a person.
  
4.    0      I get as much satisfaction out of things as I used to.  
      4      I don't enjoy things the way I used to.  
      5      I don't get real satisfaction out of anything anymore.  
      6      I am dissatisfied or bored with everything.
  
5.    0      I don't feel particularly guilty.  
      4      I feel guilty a good part of the time.  
      5      I feel guilty most of the time.  
      6      I feel guilty all of the time.

6. 0 I don't feel I am being punished.  
4 I feel I may be punished.  
5 I expect to be punished.  
6 I feel I am being punished.
7. 0 I don't feel disappointed in myself.  
4 I am disappointed in myself.  
5 I am disgusted with myself.  
6 I hate myself.
8. 0 I don't feel I am any worse than anybody else.  
4 I am critical of myself for my weaknesses or mistakes.  
5 I blame myself all the time for my faults.  
6 I blame myself for everything bad that happens.
9. 0 I don't have any thoughts of killing myself.  
4 I have thoughts of killing myself, but I would not carry them out.  
5 I would like to kill myself.  
6 I would kill myself if I had the chance.
10. 0 I don't cry any more than usual.  
4 I cry more now than I used to.  
5 I cry all the time now.  
6 I used to be able to cry, but now I can't cry even though I want to.
11. 0 I am no more irritated now than I ever am.  
4 I get annoyed or irritated more easily than I used to.  
5 I feel irritated all the time now.  
6 I don't get irritated at all by the things that used to irritate me.
12. 0 I have not lost interest in other people.  
4 I am less interested in other people than I used to be.  
5 I have lost most of my interest in other people.  
6 I have lost all of my interest in other people.
13. 0 I make decisions about as well as I ever could.  
4 I put off making decisions more than I used to.  
5 I have greater difficulty in making decisions than before.  
6 I can't make decisions at all any more.
14. 0 I don't feel I look any worse than I used to.  
4 I am worried that I am looking old or unattractive.  
5 I feel that there are permanent changes in my appearance that make me look unattractive.  
6 I believe that I look ugly.
15. 0 I can work about as well as before.  
4 It takes an extra effort to get started at doing something.  
5 I have to push myself very hard to do anything.  
6 I can't do any work at all.

16. 0 I can sleep as well as usual.  
 4 I don't sleep as well as I used to.  
 5 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.  
 6 I wake up several hours earlier than I used to and cannot get back to sleep.
17. 0 I don't get more tired than usual.  
 4 I get tired more easily than I used to.  
 5 I get tired from doing almost anything.  
 6 I am too tired to do anything.
18. 0 My appetite is no worse than usual.  
 4 My appetite is not as good as it used to be.  
 5 My appetite is much worse now.  
 6 I have no appetite at all anymore.
19. 0 I haven't lost much weight, if any, lately.  
 4 I have lost more than 5 pounds.  
 5 I have lost more than 10 pounds.  
 6 I have lost more than 15 pounds.
- 19a I am purposely trying to lose weight by eating less. Yes \_\_\_\_\_ No \_\_\_\_\_
20. 0 I am no more worried about my health than usual.  
 4 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.  
 5 I am very worried about physical problems and it's hard to think of much else.  
 6 I am so worried about my physical problems that I cannot think about anything else.
21. 0 I have not noticed any recent change in my interest in sex.  
 4 I am less interested in sex than I used to be.  
 5 I am much less interested in sex now.  
 6 I have lost interest in sex completely.

Are you currently sexually active?

Yes / No

**What is your weight now? Please write weight in either Kg or lbs.**

Kg	Stone/lbs
----	-----------

**Smoking**

I have always been a non-smoker	Yes	No
I used to smoke but not now	Yes	No
I stopped smoking after my heart problem but have started again	Yes	No
I am a smoker at the moment	Yes	No
The number of cigarettes I smoke per day		

**Would you now tell me which comes closest to how you have been feeling in the past 7 days?**

**I feel tense or 'wound-up':**

- 5. Most of the time
- 6. A lot of the time
- 7. Time to time, occasionally
- 8. Not at all

**I get a sort of frightened feeling as if something awful is about to happen:**

- 5. Very definitely and quite badly
- 6. Yes, but not too badly
- 7. A little, but it doesn't worry me
- 8. Not at all

**Worrying thoughts go through my mind:**

- 5. A great deal of the time
- 6. A lot of the time
- 7. From time to time but not too often
- 8. Only occasionally

**I can sit at ease and feel relaxed:**

- 5. Definitely
- 6. Usually
- 7. Not often
- 8. Not at all

**I get a sort of frightened feeling like 'butterflies' in the stomach:**

- 5. Not at all
- 6. Occasionally
- 7. Quite often
- 8. Very often

**I feel restless as if I have to be on the move:**

- 5. Very much indeed
- 6. Quite a lot
- 7. Not very much
- 8. Not at all

**I get sudden feelings of panic:**

- 5. Very often indeed
- 6. Quite often
- 7. Not very often
- 8. Not at all

## Your reactions to your heart problem

**These questions relate to thoughts and/or feelings you may have experienced over the last few months since you had the acute heart symptoms which led to your hospital admission. Please circle whichever answer seems to apply closest you.**

1. Have you been trying not to think about or have feelings associated with your heart problem?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

2. Have you had upsetting thoughts or images that related to your heart problem and that came into your head when you didn't want them to?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

3. Have you felt very emotionally upset when reminded of the time your acute heart symptoms came on, such as becoming very scared, angry, sad?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

4. Have you been having physical reactions if reminded of when your heart symptoms first occurred, for example heart beating fast, breaking out in a cold sweat etc?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

5. Have you had the experience of reliving the time when your acute heart symptoms occurred, acting or feeling as if it were happening again?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

6. Have you been unable to remember any important parts of the time when your heart problem started and when you got into hospital?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

7. Have you been having problems falling or staying asleep?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

8. Have you been having bad dreams or nightmares about your heart problem?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

9. Have you been having difficulty concentrating, for example drifting in and out of conversations, losing track of the story on television, difficulty remembering what you have read?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

10. Have you been making efforts to avoid activities, situations or places that remind you of when the symptoms that led to your being admitted to hospital started?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

11. Have you felt distant or cut off from others?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

12. Have you been jumpier or more easily startled?

Not at all	A little bit	Half of the time	Almost always
------------	--------------	------------------	---------------

13. Have you been overly alert, for example checking to see who is around you?

Not at all	A little bit	Half of the time	Almost always
------------	--------------	------------------	---------------

14. Have you been more irritable or had outbursts of anger?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

15. Have you felt emotionally numb, for example, felt sad but couldn't cry, unable to have loving feelings?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

16. Have you found that you have not been interested in things you used to enjoy doing?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

17. Have you felt that any future plans or hopes have changed because of the heart problem that led to your going into hospital?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

The following questions are about your health and daily activities. Read each item and circle one answer for each question.

1. In general would you say your health is:

Excellent	Very Good	Good	Fair	Poor
-----------	-----------	------	------	------

2. The following questions are about the activities you might do during a typical day. Does **your health now limit you** in these activities? If so how much?

- Moderate activities – such as moving a table, pushing a vacuum cleaner, bowling, or playing golf.

Yes, limited a lot	Yes, limited a little	No, not limited at all
--------------------	-----------------------	------------------------

- Climbing **several** flights of stairs.

Yes, limited a lot	Yes, limited a little	No, not limited at all
--------------------	-----------------------	------------------------

3. Have you had any of the following problems with your work or other regular daily activities **as a result of your physical health**?

- **Accomplished less** than you would like.

Yes	No
-----	----

- Were limited in the **kind** of work or other activities.

Yes	No
-----	----

4. Have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)?

- **Accomplished less** than you would like:

Yes	No
-----	----

- Did work or other activities less carefully than usual

--	--

Yes	No
-----	----

5. How much did **pain** interfere with your normal work (including both work outside the home and housework)?

Not at all	A little bit	Moderately	Quite a bit	Extremely
------------	--------------	------------	-------------	-----------

6. These questions are about how you feel and how things have been with you **since you left hospital**. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the **time since you left hospital**:

- Have you felt calm and peaceful?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Did you have a lot of energy?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Have you felt downhearted and low?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

7. Since you left hospital, how much of the time has your **physical health or emotional problems** interfered with your social activities (like visiting friends, relatives, etc.)?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

**Thinking about the last month,**

FV1 How many pieces of fruit – of any sort- do you eat on a typical day?

Average portions of fruit per day: \_\_\_\_\_

FV2 How often do you eat less than this average figure?

Never	Once a week	Twice a week	Three times a week	Four times a week	Five or more times a week
-------	-------------	--------------	--------------------	-------------------	---------------------------

How many portions of vegetables –excluding potatoes- do you eat on a typical day?

FV3 Average servings of vegetables per day: \_\_\_\_\_

FV4 How often do you eat less than this figure?

Never	Once a week	Twice a week	Three times a week	Four times a week	Five or more times a week
-------	-------------	--------------	--------------------	-------------------	---------------------------

**Thinking about the last month,**

		Always/Almost Always	Sometimes	Rarely	Never	Do not eat
FT1	How often do you use skimmed or semi-skimmed milk instead of full-fat milk?					
FT2	When you use spread or cooking fat how often do you use a lower saturated fat option (e.g. sunflower, olive or rapeseed)					
FT3	When you eat meat, how often do you eat lean meat (chicken/turkey no skin, beef, pork lamb fat removed)					
FT4	When you eat ready meals how often do you eat a healthy option, controlled for calories, fat and salt?					

**Thinking about the last month,**

		Less than once a week/never	1-3	4-6	7 or more	
FT5	About how many times in a week do you eat cheese?					
FT6	About how many times in a week do you eat crisps or similar snacks (e.g. Doritos, Pringles)?					
FT7	About how many times in a week do you eat cakes, biscuits or puddings?					
FT8	About how many times a week do you eat meat products (e.g. sausages, pate, burgers not including those from fast-food outlets)?					
FT9	About how many times a week do you eat fast-food or take aways (e.g burgers, pizza, fried chicken, fish & chips, indian,					

**How often in the past month did you?**

		Not at all	1-3 days	4-7 days	8-14 days	15-20 days	21-31 days
JK1	Have trouble falling asleep?						
JK 2	Wake up several times per night?						
JK 3	Have trouble staying asleep (including waking far too early)?						
JK4	Wake up after your usual amount of sleep feeling tired and worn out?						
JK5	Have disturbed or restless sleep?						

**Physical activity:**

How many times per week do you do vigorous physical activity enough to make you out of breath?

**None            1            2            3            4            5            6+**

Please specify the activity .....

**Alcohol**

Do you drink alcohol? Yes / No

If Yes, how many units per week on average do you drink? .....units per week  
*(1 Unit = ½ pint of beer, 1 glass of wine or 1 measure of spirit)*

**Thinking about the days of the PAST WEEK.**

On average, for how long did you **walk** outside your home/workplace? (if you did not walk, please enter zero (0) in each box)

on each weekday

Hours	Minutes

on each weekend day

Hours	Minutes

How would you describe your usual walking pace?

**(Please tick one box only)**

Slow (less than 3mph)

1
---

Steady average pace

2
---

Brisk pace

3

Fast pace (over 4mph)

4

**These questions are about the support that you get from other people. Please circle your answer to each question.**

1. Is there someone available to whom you can count on to listen to you when you need to talk?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

2. Is there someone available to you to give you good advice about a problem?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

3. Is there someone available to you who shows you love and affection?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

4. Is there someone available to help with daily chores?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

5. Can you count on anyone to provide you with emotional support (talking over problems or helping you make a difficult decision)?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

6. Do you have as much contact as you would like with someone you feel close to, someone in whom you can trust and confide in?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

7. Is there someone available who reminds you to take your medication?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

8. Is there someone available who reminds you or helps you to eat a healthy diet?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

9. Is there someone available who reminds you or helps you to take some exercise?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

### YOUR VIEWS ABOUT YOUR ILLNESS

Listed below are a number of symptoms that you may or may not have experienced since your illness. Please indicate by circling Yes or No, whether you have experienced any of these symptoms since your illness, and whether you believe that these symptoms are related to your illness.

	I have experienced this symptom <i>since my illness</i>		This symptom is <i>related to my illness</i>		
	Yes	No	Yes	No	
Pain	Yes	No	_____	Yes	No
Sore Throat	Yes	No	_____	Yes	No
Nausea	Yes	No	_____	Yes	No
Breathlessness	Yes	No	_____	Yes	No
Weight Loss	Yes	No	_____	Yes	No
Fatigue	Yes	No	_____	Yes	No
Stiff Joints	Yes	No	_____	Yes	No
Sore Eyes	Yes	No	_____	Yes	No
Wheeziness	Yes	No	_____	Yes	No
Headaches	Yes	No	_____	Yes	No
Upset Stomach	Yes	No	_____	Yes	No
Sleep Difficulties	Yes	No	_____	Yes	No
Dizziness	Yes	No	_____	Yes	No
Loss of Strength	Yes	No	_____	Yes	No

We are interested in your own personal views of how you now see your current illness. Please indicate how much you agree or disagree with the following statements about your illness by ticking the appropriate box.

	VIEWS ABOUT YOUR ILLNESS	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IPQ1	My illness will last a short time					
IPQ2	My illness is likely to be permanent rather than temporary					
IPQ3	My illness will last for a					

	long time					
IPQ4	This illness will pass quickly					
	<b>VIEWS ABOUT YOUR ILLNESS</b>	<b>STRONGLY DISAGREE</b>	<b>DISAGREE</b>	<b>NEITHER AGREE NOR DISAGREE</b>	<b>AGREE</b>	<b>STRONGLY AGREE</b>
IPQ5	I expect to have this illness for the rest of my life					
IPQ6	My illness is a serious condition					
IPQ7	My illness has major consequences on my life					
IPQ8	My illness does not have much effect on my life					
IPQ9	My illness strongly affects the way others see me					
IPQ10	My illness has serious financial consequences					
IPQ11	My illness causes difficulties for those who are close to me					
IPQ12	There is a lot which I can do to control my symptoms					
IPQ13	What I do can determine whether my illness gets better or worse					
IPQ14	The course of my illness depends on me					
IPQ15	Nothing I do will affect my illness					
IPQ16	I have the power to influence my illness					
IPQ17	My actions will have no affect on the outcome of my illness					
IPQ18	My illness will improve in time					
IPQ19	There is very little that can be done to improve my illness					
IPQ20	My treatment will be effective in curing my illness					
IPQ21	The negative effects of my illness can be prevented (avoided) by my treatment					
IPQ22	My treatment can control my illness					

IPQ23	There is nothing which can help my condition					
	<b>VIEWS ABOUT YOUR ILLNESS</b>	<b>STRONGLY DISAGREE</b>	<b>DISAGREE</b>	<b>NEITHER AGREE NOR DISAGREE</b>	<b>AGREE</b>	<b>STRONGLY AGREE</b>
IPQ24	The symptoms of my condition are puzzling me					
IPQ25	My illness is a mystery to me					
IPQ26	I don't understand my illness					
IPQ27	My illness doesn't make any sense to me					
IPQ28	I have a clear picture or understanding of my condition					
IPQ29	The symptoms of my illness change a great deal from day to day					
IPQ30	My symptoms come and go in cycles					
IPQ31	My illness is very unpredictable					
IPQ32	I go through cycles in which my illness gets better and worse.					
IPQ33	I get depressed when I think about my illness					
IPQ34	When I think about my illness I get upset					
IPQ35	My illness makes me feel angry					
IPQ36	My illness does not worry me					
IPQ37	Having this illness makes me feel anxious					
IPQ38	My illness makes me feel afraid					

### What do you think caused your heart problem?

**Serious heart disease may be caused by many different factors. We would like to find out what factors you think were involved with your own illness. Listed below are a series of factors that patients in the past have thought helped to cause their heart disease symptoms. Please think about each item, then circle the answer that indicates how much you agree or disagree with each statement.**

<b>Factors that might have helped cause my illness:</b>			
My illness is hereditary – it runs in my family	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Smoking played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by other medical problems	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Stress was a major factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Being overweight caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
High blood pressure was an important factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Diet played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
I became ill because I over-exerted myself	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
It was just by chance and bad luck that I became ill	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was caused by poor medical care in the past	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Lack of exercise was a cause of my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by tiredness and exhaustion	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Genetic factors (genes) caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My state of mind played a major part in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Working too hard caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
A germ or virus caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>

If you have a spouse or partner please answer the following questions?

"Every relationship has its good and bad aspects. How satisfied are you with the following aspects of yours?"

**b. Amount of time you and your partner spend together**

Moderately satisfied   
Very satisfied

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**b. Communication between you and your partner**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**c. Similar interests**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**d. Similar lifestyles**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**e. Sexual activity**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**f. Similar temperament**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**g. Agreement on financial matters**

- Not at all satisfied
- Not too satisfied

**To what extent do you feel confident that you can do the different things you need to do to take care of your heart condition, regardless of whether or not you actually do them?**

**Please look at each of these items in the list below and give a number from 0 to 10 to indicate how confident you are that you can do the following:**

0 <b>Not at all confident</b>	1	2	3	4	5	6	7	8	9	10 <b>Extremely confident</b>
--------------------------------------	---	---	---	---	---	---	---	---	---	--------------------------------------

- 11. Take your medication when you are supposed to \_\_\_\_\_/10
- 12. Keep to a healthy diet \_\_\_\_\_/10
- 13. Maintain a healthy body weight \_\_\_\_\_/10
- 14. Get enough exercise \_\_\_\_\_/10
- 15. Pace yourself to avoid exercising too strenuously \_\_\_\_\_/10
- 16. Control symptoms of your health condition (e.g., chest pain, breathlessness) by adjusting your activity, medications, or diet \_\_\_\_\_/10
- 17. Avoid using tobacco and alcohol \_\_\_\_\_/10
- 18. Get enough sleep each night \_\_\_\_\_/10
- 19. Avoid stressful situations \_\_\_\_\_/10
- 20. Get medical advice when you need it \_\_\_\_\_/10

**Patients who have experienced a heart problem sometimes feel that having a heart problem makes contributions to their lives, as well as causing problems. Indicate how much you agree with each of the following statements, using these response options below.**

**My heart problem...**

**1. Has led me to be more accepting of things.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**2. Has taught me how to adjust to things I cannot change.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**3. Has helped me take things as they come.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**4. Has brought my family closer together.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**5. Has made me more sensitive to family issues.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**6. Has taught me that everyone has a purpose in life.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**7. Has shown me that all people need to be loved.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**8. Has made me realize the importance of planning for my family's future.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**9. Has made me more aware and concerned for the future of all human beings.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**10. Has taught me to be patient.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**11. Has led me to deal better with stress and problems.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**12. Has led me to meet people who have become some of my best friends.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**13. Has contributed to my overall emotional and spiritual growth.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**14. Has helped me become more aware of the love and support available from other people.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**15. Has helped me realise who my real friends are.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**16. Has helped me become more focused on priorities, with a deeper sense of purpose in life.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**17. Has helped me become a stronger person, more able to cope effectively with future life challenges.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

That is the end of the questionnaire. Please check that you have answered all of the questions. If you have any difficulty with any of the questions, please call us on the number below. Thank you very much for taking the time to make this important contribution to our study of emotions and heart disease. We will be in touch with you regarding the next stage of the study.

**Please return this questionnaire in the freepost envelope. If you have misplaced the envelope you can return the questionnaire to the following address, there is no need to use a stamp:**

**TRACE  
Psychobiology group (3<sup>rd</sup> floor)  
Department of Epidemiology & Public Health  
UCL, 1-19 Torrington Place  
FREEPOST WC5565  
London WC1E 6BT**

**020 7679 1804**

## APPENDIX IV

### **TIME 4 PATIENT QUESTIONNAIRE TRACE STUDY**

## Tracking Recovery After Coronary Events (TRACE) study

Time 4

Date questionnaire mailed:.....

Name: .....Date (please fill in): .....Pt No:.....

Thank you very much for participating in this study of psychological experience & acute coronary events. You may feel that some of the questions do not apply to you, but please answer each question with the answer that most closely fits the way you feel.

The answers you provide in this questionnaire will be kept **strictly confidential**. The information will go into the statistics for the study, and it will not be possible to identify you personally in any reports. Under no circumstances will any of the information you give us be made available to anyone else.

Most of the questions can be answered by circling the appropriate answer or placing an X or a tick in the box..

***For example:***

“It’s easy for me to relax.”

Strongly disagree	Disagree	Neutral	Agree	Strongly agree
-------------------	----------	---------	-------	----------------

Please be sure to read the instructions to each section carefully.

Thank you very much for your participation. If you have difficulty with any questions, please do not hesitate to contact us.

Many people find a way of using their medicines which suits them. This may differ from the instructions on the label or from what their doctor has said.

We would like to ask you a few questions about how you use your medicines.

Here are some ways in which people have said that they use their medicines  
For each of the statements, please tick the box which best applies to you

		Always	Often	Sometimes	Rarely	Never
MARS1	I forget to take my medicines					
MARS21	I alter the dose of my medicines					
MARS3	I stop taking my medicines for a while					
MARS4	I decide to miss out a dose					
MARS5	I take less than instructed					

This part of the questionnaire consists of 21 groups of statements. After reading each group of statements carefully, circle the number (0, 1, 2, or 3) next to the one statement in each group which best describes the way you have been feeling during the past two weeks, including today. If several statements within a group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1.    0    I do not feel sad.  
      7    I feel sad.  
      8    I am sad all the time and I can't snap out of it.  
      9    I am so sad or unhappy that I can't stand it.
  
2.    0    I am not particularly discouraged about the future.  
      7    I feel discouraged about the future.  
      8    I feel I have nothing to look forward to.  
      9    I feel that the future is hopeless and that things cannot improve.
  
3.    0    I do not feel like a failure.  
      7    I feel I have failed more than the average person.  
      8    As I look back on my life, all I can see is a lot of failures.  
      9    I feel I am a complete failure as a person.
  
4.    0    I get as much satisfaction out of things as I used to.  
      7    I don't enjoy things the way I used to.  
      8    I don't get real satisfaction out of anything anymore.  
      9    I am dissatisfied or bored with everything.
  
5.    0    I don't feel particularly guilty.  
      7    I feel guilty a good part of the time.  
      8    I feel guilty most of the time.  
      9    I feel guilty all of the time.

6. 0 I don't feel I am being punished.  
7 I feel I may be punished.  
8 I expect to be punished.  
9 I feel I am being punished.
7. 0 I don't feel disappointed in myself.  
7 I am disappointed in myself.  
8 I am disgusted with myself.  
9 I hate myself.
8. 0 I don't feel I am any worse than anybody else.  
7 I am critical of myself for my weaknesses or mistakes.  
8 I blame myself all the time for my faults.  
9 I blame myself for everything bad that happens.
9. 0 I don't have any thoughts of killing myself.  
7 I have thoughts of killing myself, but I would not carry them out.  
8 I would like to kill myself.  
9 I would kill myself if I had the chance.
10. 0 I don't cry any more than usual.  
7 I cry more now than I used to.  
8 I cry all the time now.  
9 I used to be able to cry, but now I can't cry even though I want to.
11. 0 I am no more irritated now than I ever am.  
7 I get annoyed or irritated more easily than I used to.  
8 I feel irritated all the time now.  
9 I don't get irritated at all by the things that used to irritate me.
12. 0 I have not lost interest in other people.  
7 I am less interested in other people than I used to be.  
8 I have lost most of my interest in other people.  
9 I have lost all of my interest in other people.
13. 0 I make decisions about as well as I ever could.  
7 I put off making decisions more than I used to.  
8 I have greater difficulty in making decisions than before.  
9 I can't make decisions at all any more.
14. 0 I don't feel I look any worse than I used to.  
7 I am worried that I am looking old or unattractive.  
8 I feel that there are permanent changes in my appearance that make me look unattractive.  
9 I believe that I look ugly.
15. 0 I can work about as well as before.  
7 It takes an extra effort to get started at doing something.  
8 I have to push myself very hard to do anything.  
9 I can't do any work at all.

16. 0 I can sleep as well as usual.  
 7 I don't sleep as well as I used to.  
 8 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.  
 9 I wake up several hours earlier than I used to and cannot get back to sleep.
17. 0 I don't get more tired than usual.  
 7 I get tired more easily than I used to.  
 8 I get tired from doing almost anything.  
 9 I am too tired to do anything.
18. 0 My appetite is no worse than usual.  
 7 My appetite is not as good as it used to be.  
 8 My appetite is much worse now.  
 9 I have no appetite at all anymore.
19. 0 I haven't lost much weight, if any, lately.  
 7 I have lost more than 5 pounds.  
 8 I have lost more than 10 pounds.  
 9 I have lost more than 15 pounds.
- 19a I am purposely trying to lose weight by eating less. Yes \_\_\_\_\_ No \_\_\_\_\_
20. 0 I am no more worried about my health than usual.  
 7 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.  
 8 I am very worried about physical problems and it's hard to think of much else.  
 9 I am so worried about my physical problems that I cannot think about anything else.
21. 0 I have not noticed any recent change in my interest in sex.  
 7 I am less interested in sex than I used to be.  
 8 I am much less interested in sex now.  
 9 I have lost interest in sex completely.

Are you currently sexually active?

Yes / No

**What is your weight now? Please write weight in either Kg or lbs.**

Kg	Stone/lbs
----	-----------

**Smoking**

I have always been a non-smoker	Yes	No
I used to smoke but not now	Yes	No
I stopped smoking after my heart problem but have started again	Yes	No
I am a smoker at the moment	Yes	No
The number of cigarettes I smoke per day		

**Would you now tell me which comes closest to how you have been feeling in the past 7 days?**

**I feel tense or 'wound-up':**

- 9. Most of the time
- 10. A lot of the time
- 11. Time to time, occasionally
- 12. Not at all

**I get a sort of frightened feeling as if something awful is about to happen:**

- 9. Very definitely and quite badly
- 10. Yes, but not too badly
- 11. A little, but it doesn't worry me
- 12. Not at all

**Worrying thoughts go through my mind:**

- 9. A great deal of the time
- 10. A lot of the time
- 11. From time to time but not too often
- 12. Only occasionally

**I can sit at ease and feel relaxed:**

- 9. Definitely
- 10. Usually
- 11. Not often
- 12. Not at all

**I get a sort of frightened feeling like 'butterflies' in the stomach:**

- 9. Not at all
- 10. Occasionally
- 11. Quite often
- 12. Very often

**I feel restless as if I have to be on the move:**

- 9. Very much indeed
- 10. Quite a lot
- 11. Not very much
- 12. Not at all

**I get sudden feelings of panic:**

- 9. Very often indeed
- 10. Quite often
- 11. Not very often
- 12. Not at all

## Your reactions to your heart problem

**These questions relate to thoughts and/or feelings you may have experienced over the last few months since you had the acute heart symptoms which led to your hospital admission. Please circle whichever answer seems to apply closest you.**

1. Have you been trying not to think about or have feelings associated with your heart problem?

Not at all	Once in a while	Somewhat	Very much
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2. Have you had upsetting thoughts or images that related to your heart problem and that came into your head when you didn't want them to?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
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3. Have you felt very emotionally upset when reminded of the time your acute heart symptoms came on, such as becoming very scared, angry, sad?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

4. Have you been having physical reactions if reminded of when your heart symptoms first occurred, for example heart beating fast, breaking out in a cold sweat etc?

Not at all	Once in a while	Somewhat	Very much
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5. Have you had the experience of reliving the time when your acute heart symptoms occurred, acting or feeling as if it were happening again?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

6. Have you been unable to remember any important parts of the time when your heart problem started and when you got into hospital?

Not at all	Once in a while	Somewhat	Very much
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7. Have you been having problems falling or staying asleep?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
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8. Have you been having bad dreams or nightmares about your heart problem?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
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9. Have you been having difficulty concentrating, for example drifting in and out of conversations, losing track of the story on television, difficulty remembering what you have read?

Not at all	Once in a while	Somewhat	Very much
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10. Have you been making efforts to avoid activities, situations or places that remind you of when the symptoms that led to your being admitted to hospital started?

Not at all	Once in a while	Somewhat	Very much
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11. Have you felt distant or cut off from others?

Not at all	Once in a while	Somewhat	Very much
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12. Have you been jumpier or more easily startled?

Not at all	A little bit	Half of the time	Almost always
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13. Have you been overly alert, for example checking to see who is around you?

Not at all	A little bit	Half of the time	Almost always
------------	--------------	------------------	---------------

14. Have you been more irritable or had outbursts of anger?

Not at all	Once per week or less	2 – 4 times per week	5 or more times per week
------------	-----------------------	----------------------	--------------------------

15. Have you felt emotionally numb, for example, felt sad but couldn't cry, unable to have loving feelings?

Not at all	Once in a while	Somewhat	Very much
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16. Have you found that you have not been interested in things you used to enjoy doing?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

17. Have you felt that any future plans or hopes have changed because of the heart problem that led to your going into hospital?

Not at all	Once in a while	Somewhat	Very much
------------	-----------------	----------	-----------

The following questions are about your health and daily activities. Read each item and circle one answer for each question.

1. In general would you say your health is:

Excellent	Very Good	Good	Fair	Poor
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2. The following questions are about the activities you might do during a typical day. Does **your health now limit you** in these activities? If so how much?

- Moderate activities – such as moving a table, pushing a vacuum cleaner, bowling, or playing golf.

Yes, limited a lot	Yes, limited a little	No, not limited at all
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- Climbing **several** flights of stairs.

Yes, limited a lot	Yes, limited a little	No, not limited at all
--------------------	-----------------------	------------------------

3. Have you had any of the following problems with your work or other regular daily activities **as a result of your physical health**?

- **Accomplished less** than you would like.

Yes	No
-----	----

- Were limited in the **kind** of work or other activities.

Yes	No
-----	----

4. Have you had any of the following problems with your work or other regular daily activities **as a result of any emotional problems** (such as feeling depressed or anxious)?

- **Accomplished less** than you would like:

Yes	No
-----	----

- Did work or other activities less carefully than usual

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Yes	No
-----	----

5. How much did **pain** interfere with your normal work (including both work outside the home and housework)?

Not at all	A little bit	Moderately	Quite a bit	Extremely
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6. These questions are about how you feel and how things have been with you **since you left hospital**. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the **time since you left hospital**:

- Have you felt calm and peaceful?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Did you have a lot of energy?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

- Have you felt downhearted and low?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
-----------------	------------------	------------------------	------------------	--------------------------	------------------

7. Since you left hospital, how much of the time has your **physical health or emotional problems** interfered with your social activities (like visiting friends, relatives, etc.)?

All of the time	Most of the time	A good bit of the time	Some of the time	A little bit of the time	None of the time
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**Thinking about the last month,**

FV1 How many pieces of fruit – of any sort- do you eat on a typical day?

Average portions of fruit per day: \_\_\_\_\_

FV2 How often do you eat less than this average figure?

Never	Once a week	Twice a week	Three times a week	Four times a week	Five or more times a week
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How many portions of vegetables –excluding potatoes- do you eat on a typical day?

FV3 Average servings of vegetables per day: \_\_\_\_\_

FV4 How often do you eat less than this figure?

Never	Once a week	Twice a week	Three times a week	Four times a week	Five or more times a week
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**Thinking about the last month,**

		Always/Almost Always	Sometimes	Rarely	Never	Do not eat
FT1	How often do you use skimmed or semi-skimmed milk instead of full-fat milk?					
FT2	When you use spread or cooking fat how often do you use a lower saturated fat option (e.g. sunflower, olive or rapeseed)					
FT3	When you eat meat, how often do you eat lean meat (chicken/turkey no skin, beef, pork lamb fat removed)					
FT4	When you eat ready meals how often do you eat a healthy option, controlled for calories, fat and salt?					

**Thinking about the last month,**

		Less than once a week/never	1-3	4-6	7 or more	
FT5	About how many times in a week do you eat cheese?					
FT6	About how many times in a week do you eat crisps or similar snacks (e.g. Doritos, Pringles)?					
FT7	About how many times in a week do you eat cakes, biscuits or puddings?					
FT8	About how many times a week do you eat meat products (e.g. sausages, pate, burgers not including those from fast-food outlets)?					
FT9	About how many times a week do you eat fast-food or take aways (e.g burgers, pizza, fried chicken, fish & chips, indian,					

**How often in the past month did you?**

		Not at all	1-3 days	4-7 days	8-14 days	15-20 days	21-31 days
JK1	Have trouble falling asleep?						
JK 2	Wake up several times per night?						
JK 3	Have trouble staying asleep (including waking far too early)?						
JK4	Wake up after your usual amount of sleep feeling tired and worn out?						
JK5	Have disturbed or restless sleep?						

**Physical activity:**

How many times per week do you do vigorous physical activity enough to make you out of breath?

**None            1            2            3            4            5            6+**

Please specify the activity .....

**Alcohol**

Do you drink alcohol? Yes / No

If Yes, how many units per week on average do you drink? .....units per week  
*(1 Unit = ½ pint of beer, 1 glass of wine or 1 measure of spirit)*

**Thinking about the days of the PAST WEEK.**

On average, for how long did you **walk** outside your home/workplace? (if you did not walk, please enter zero (0) in each box)

on each weekday

Hours	Minutes

on each weekend day

Hours	Minutes

How would you describe your usual walking pace?

**(Please tick one box only)**

Slow (less than 3mph)

1
---

Steady average pace

2
---

Brisk pace

3

Fast pace (over 4mph)

4

**These questions are about the support that you get from other people. Please circle your answer to each question.**

1. Is there someone available to whom you can count on to listen to you when you need to talk?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
------------------	----------------------	------------------	------------------	-----------------

2. Is there someone available to you to give you good advice about a problem?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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3. Is there someone available to you who shows you love and affection?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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4. Is there someone available to help with daily chores?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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5. Can you count on anyone to provide you with emotional support (talking over problems or helping you make a difficult decision)?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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6. Do you have as much contact as you would like with someone you feel close to, someone in whom you can trust and confide in?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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7. Is there someone available who reminds you to take your medication?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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8. Is there someone available who reminds you or helps you to eat a healthy diet?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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9. Is there someone available who reminds you or helps you to take some exercise?

None of the time	A little of the time	Some of the time	Most of the time	All of the time
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**We are interested in your own personal views of how you now see your recent heart problems.**

**Please indicate how much you agree or disagree with the following statements about your heart problems by ticking the appropriate box.**

	VIEWS ABOUT YOUR ILLNESS	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IPQ1	My illness will last a short time					
IPQ2	My illness is likely to be permanent rather than temporary					
IPQ3	My illness will last for a long time					
IPQ4	This illness will pass quickly					
IPQ5	I expect to have this illness for the rest of my life					
IPQ6	My illness is a serious condition					
IPQ7	My illness has major consequences on my life					
IPQ8	My illness does not have much effect on my life					
IPQ9	My illness strongly affects the way others see me					
IPQ10	My illness has serious financial consequences					
IPQ11	My illness causes difficulties for those who are close to me					
IPQ12	There is a lot which I can do to control my symptoms					
IPQ13	What I do can determine whether my illness gets better or worse					
IPQ14	The course of my illness depends on me					
IPQ15	Nothing I do will affect my illness					
IPQ16	I have the power to influence my illness					
IPQ17	My actions will have no affect on the outcome of my illness					

IPQ18	My illness will improve in time					
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	VIEWS ABOUT YOUR ILLNESS	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
IPQ19	There is very little that can be done to improve my illness					
IPQ20	My treatment will be effective in curing my illness					
IPQ21	The negative effects of my illness can be prevented (avoided) by my treatment					
IPQ22	My treatment can control my illness					
IPQ23	There is nothing which can help my condition					
IPQ24	The symptoms of my condition are puzzling me					
IPQ25	My illness is a mystery to me					
IPQ26	I don't understand my illness					
IPQ27	My illness doesn't make any sense to me					
IPQ28	I have a clear picture or understanding of my condition					
IPQ29	The symptoms of my illness change a great deal from day to day					
IPQ30	My symptoms come and go in cycles					
IPQ31	My illness is very unpredictable					
IPQ32	I go through cycles in which my illness gets better and worse.					
IPQ33	I get depressed when I think about my illness					
IPQ34	When I think about my illness I get upset					
IPQ35	My illness makes me feel angry					
IPQ36	My illness does not worry me					

IPQ37	Having this illness makes me feel anxious					
IPQ38	My illness makes me feel afraid					

### What do you think caused your heart problem?

**Serious heart disease may be caused by many different factors. We would like to find out what factors you think were involved with your own illness. Listed below are a series of factors that patients in the past have thought helped to cause their heart disease symptoms. Please think about each item, then circle the answer that indicates how much you agree or disagree with each statement.**

<b>Factors that might have helped cause my illness:</b>			
My illness is hereditary – it runs in my family	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Smoking played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by other medical problems	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Stress was a major factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Being overweight caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
High blood pressure was an important factor in my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Diet played a major role in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
I became ill because I over-exerted myself	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
It was just by chance and bad luck that I became ill	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was caused by poor medical care in the past	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Lack of exercise was a cause of my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My illness was brought on by tiredness and exhaustion	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Genetic factors (genes) caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
My state of mind played a major part in causing my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
Working too hard caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>
A germ or virus caused my illness	<b>No</b>	<b>Maybe</b>	<b>Yes</b>

If you have a spouse or partner please answer the following questions?

"Every relationship has its good and bad aspects. How satisfied are you with the following aspects of yours?"

**c. Amount of time you and your partner spend together**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**b. Communication between you and your partner**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**c. Similar interests**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**d. Similar lifestyles**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**e. Sexual activity**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**f. Similar temperament**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

**g. Agreement on financial matters**

- Not at all satisfied
- Not too satisfied
- Moderately satisfied
- Very satisfied

To what extent do you feel confident that you can do the different things you need to do to take care of your heart condition, regardless of whether or not you actually do them?

Please look at each of these items in the list below and give a number from 0 to 10 to indicate how confident you are that you can do the following:

0 Not at all confident	1	2	3	4	5	6	7	8	9	10 Extremely confident
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- 21. Take your medication when you are supposed to \_\_\_\_\_/10
- 22. Keep to a healthy diet \_\_\_\_\_/10
- 23. Maintain a healthy body weight \_\_\_\_\_/10
- 24. Get enough exercise \_\_\_\_\_/10
- 25. Pace yourself to avoid exercising too strenuously \_\_\_\_\_/10
- 26. Control symptoms of your health condition (e.g., chest pain, breathlessness) by adjusting your activity, medications, or diet \_\_\_\_\_/10
- 27. Avoid using tobacco and alcohol \_\_\_\_\_/10
- 28. Get enough sleep each night \_\_\_\_\_/10
- 29. Avoid stressful situations \_\_\_\_\_/10
- 30. Get medical advice when you need it \_\_\_\_\_/10

Patients who have experienced a heart problem sometimes feel that having a heart problem makes contributions to their lives, as well as causing problems. Indicate how much you agree with each of the following statements, using these response options below.

**My heart problem...**

**1. Has led me to be more accepting of things.**

Not at all	A little	Moderately	Quite a bit	Extremely
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**2. Has taught me how to adjust to things I cannot change.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**3. Has helped me take things as they come.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**4. Has brought my family closer together.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**5. Has made me more sensitive to family issues.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**6. Has taught me that everyone has a purpose in life.**

Not at all	A little	Moderately	Quite a bit	Extremely
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**7. Has shown me that all people need to be loved.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**8. Has made me realize the importance of planning for my family's future.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**9. Has made me more aware and concerned for the future of all human beings.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**10. Has taught me to be patient.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**11. Has led me to deal better with stress and problems.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**12. Has led me to meet people who have become some of my best friends.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**13. Has contributed to my overall emotional and spiritual growth.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**14. Has helped me become more aware of the love and support available from other people.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**15. Has helped me realise who my real friends are.**

Not at all	A little	Moderately	Quite a bit	Extremely
------------	----------	------------	-------------	-----------

**16. Has helped me become more focused on priorities, with a deeper sense of purpose in life.**

Not at all	A little	Moderately	Quite a bit	Extremely
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**17. Has helped me become a stronger person, more able to cope effectively with future life challenges.**

Not at all	A little	Moderately	Quite a bit	Extremely
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**Are you currently taking the following medications, please tick**

Aspirin

Statin e.g. Simvastatin (Zocor), Atorvastatin (Lipitor)

Clopidogrel e.g. Plavix

Beta-blocker e.g. Metoprolol (Lopressor, Toprol), Bisoprolol (Emcor), Atenolol (Tenormin)

Ace-inhibitor e.g. Ramipril (Tritace), Losartan (Cozaar), Lisinopril (Zestril)

Omacor (Omega-3)

GTN spray or tablets (taken under the tongue)

**Please list any other medications you are currently taking:**

8.
9.
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20.

That is the end of the questionnaire. Please check that you have answered all of the questions. If you have any difficulty with any of the questions, please call us on the number below. Thank you very much for taking the time to make this important contribution to our study of emotions and heart disease.

**Please return this questionnaire in the freepost envelope. If you have misplaced the envelope you can return the questionnaire to the following address, there is no need to use a stamp:**

**TRACE  
Psychobiology group (3<sup>rd</sup> floor)  
Department of Epidemiology & Public Health  
UCL, 1-19 Torrington Place  
FREEPOST WC5565  
London WC1E 6BT**

**020 7679 1804**

## **APPENDIX V**

### **CHAPTER 6**

#### **TIME 4 PSYCHOLOGICAL DISTRESS ANALYSES – TRACE STUDY**

## 6.12 Psychological distress after ACS at Time 4

The mean scores for the BDI and HADS-A at Time 4 are depicted in Table 6.13

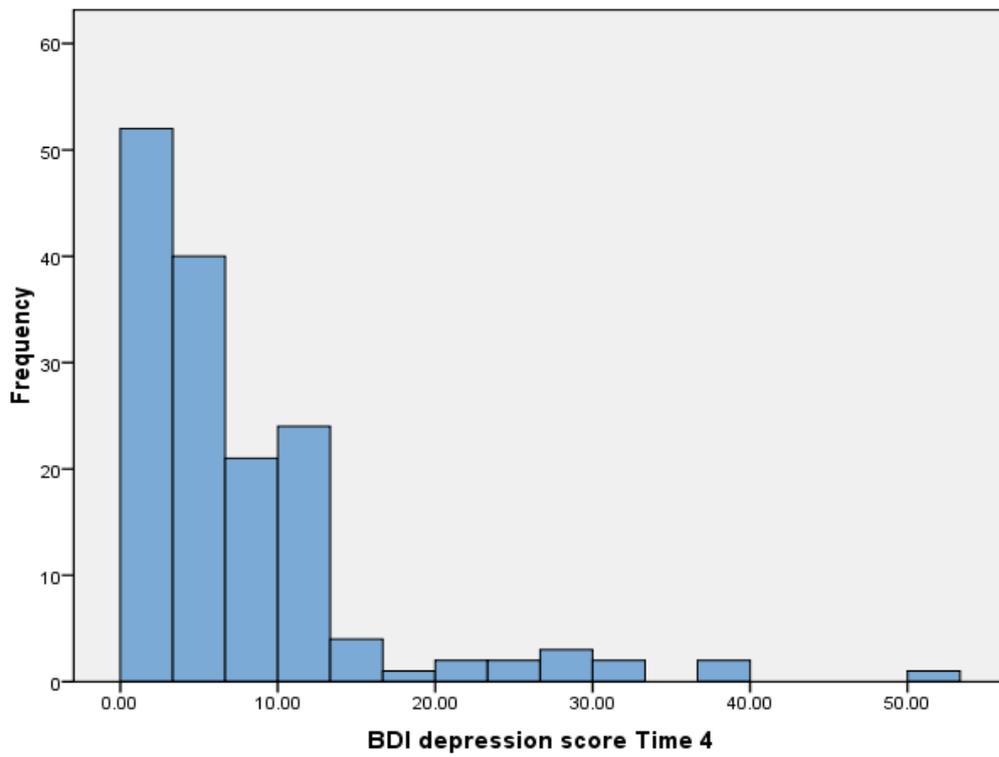
**Table 6.54 Mean depression (BDI) and anxiety (HADS) score at Time 4**

	BDI	HADS
<b>Mean (SD)</b>	7.62 (8.12)	4.43 (4.28)
<b>Range</b>	0 – 51	0 – 20
<b>N</b>	155	154

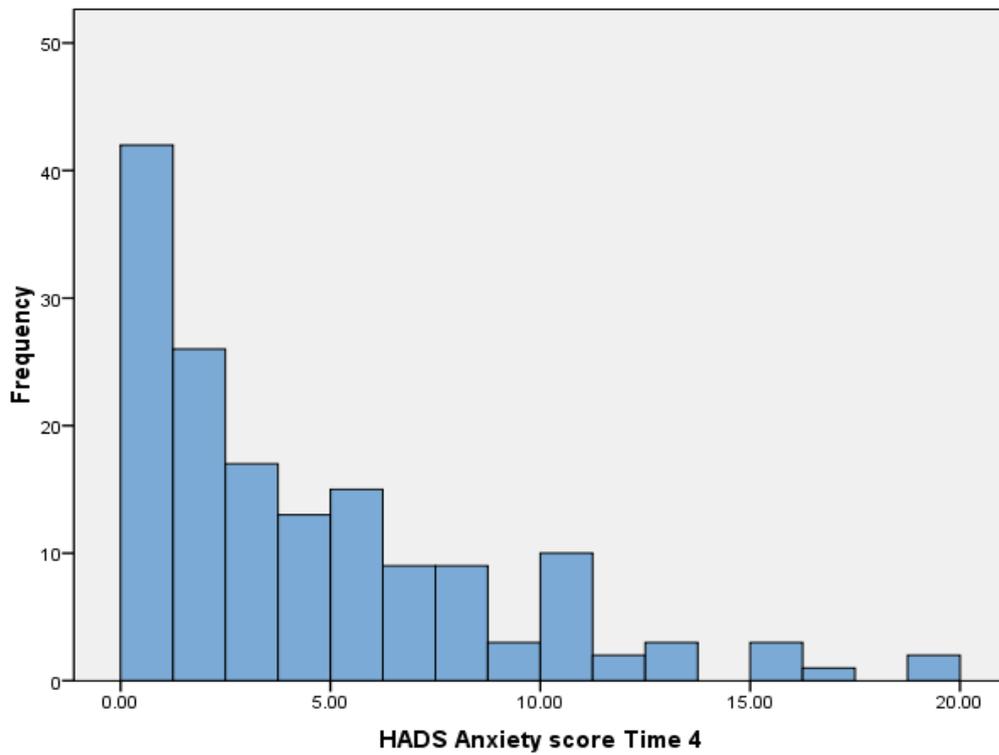
All Time 3 psychological response scores were highly correlated with the corresponding Time 4 scores and there were no significant differences between any of the Time 3 and 4 psychological response scores. All Time 2 psychological scores were also highly correlated with the respective Time 4 psychological scores. There was a significant increase in mean BDI score ( $t(135) = -2.72, p < 0.05$ ) between Time 2 and 4. There was also a significant increase in the number of patients scoring above the clinical threshold for depression from Time 2 – Time 3 - Time 4 ( $X^2(2, 108) = 9.75, p < 0.05$ ). No change was noted in the number of patients scoring above the threshold for anxiety.

The score frequency and distribution of depression and anxiety scores at Time 4 are presented in Figures 6.15 and 6.16, and are very similar to those found at Time 3. The depression and anxiety scores were highly positively correlated suggesting significant comorbidity with 25 (16%) patients exceeding the threshold criteria for significant anxiety and depression. Consonant with Time 2 and 3 scores, both score sets were positively skewed and both included a number of outliers including 3 extreme outliers for the depression scores. The 5% trimmed mean (6.55) is lower than the mean (7.62) depression score indicating an influence of outliers on the mean score. The 5% trimmed mean (4.03) of the anxiety scores is not different from the mean (4.43) indicating no undue influence from outliers.

**Figure 6.15 Score frequency for BDI depression at Time 4**



**Figure 6.16 Score frequency for BDI depression at Time 4**



### **6.12.1 The influence of demographic and clinical variables on psychological distress at Time 4**

In order to determine the influence of demographic and clinical variables collected at Time 1 on depression and anxiety scores at Time 4, two different analyses were conducted using BDI depression scores and HADS-Anxiety scores as both continuous and categorical outcomes in order to ensure that the skewed nature of the anxiety and depression score distributions and the influence of outliers on depression scores was accounted for. A series of one way between group's analyses of covariance were conducted to identify any demographic or clinical factors that may influence depression and anxiety scores at Time 4. Continuous anxiety and depression scores were the dependent variables, age and gender were entered as covariates and the independent variables were ethnicity (white/non-white), marital status (married/unmarried), employment status (employed/not employed), educational status (basic/secondary/degree), Time 2 depression score, deprivation index (low/moderate/high), history of depression (yes/no), the presence of diabetes (yes/no), prior heart disease (yes/no) and GRACE score (low/moderate/high). Depression at Time 4 did not significantly vary according to any of the selected demographic or clinical factors. Patients who had higher depression scores at Time 2 were significantly more likely to have higher depression scores at Time 3 ( $F(31, 102) = 5.52, p < 0.05, \text{partial } \eta^2 = 0.63$ ). Patients who had higher depression scores at time 3 were also significantly more likely to have higher depression scores at Time 4 ( $F(33, 86) = 29.21, p < 0.05, \text{partial } \eta^2 = 0.92$ ).

A multiple regression analysis was conducted using Time 4 depression scores as the dependent variable and age, gender, depression score at Time 2 and depression score at Time 3 as the independent variables. The model explained a significant proportion of variance in depression scores ( $R^2 = 0.39, F(3, 132) = 28.52, p < 0.05$ ) with patient depression score at Time 2 being the largest independent predictor (Table 6.14). Gender was also a borderline significant independent predictor. Repeating the model without Time 2 depression score did not reveal any new significant predictors.

**Table 6.55 Demographic and clinical predictors of depression at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-41.09	-7.01 – 4.82		-0.37	0.715
Age	0.00	-0.09 – 0.09	0.00	0.03	0.975
Gender	2.71	0.00– 5.42	0.14	1.98	0.050
Time 2 depression score*	0.86	0.67 – 1.05	0.61	8.87	0.001

\*Significant independent predictor

Anxiety at Time 4 did not significantly vary according to age, ethnicity, marital status, educational level, employment status, deprivation status, depression history prior to ACS, GRACE score or whether the patient reported diabetes or a previous heart condition. Patients with higher anxiety scores at Time 2 were found to have higher anxiety at Time 4 ( $F(18, 115) = 6.43, p < 0.05, \text{partial } \eta^2 = 0.502$ ). Patients with higher anxiety scores at Time 3 were also found to have higher anxiety at time 4 ( $F(18, 103) = 16.03, p < 0.05, \text{partial } \eta^2 = 0.737$ ). Female patients reported significantly higher anxiety than male patients at Time 4 ( $F(1, 152) = 4.36, p < 0.05, \text{partial } \eta^2 = 0.028$ ).

Based on the findings of these ANCOVA analyses, a multiple regression analysis was conducted using Time 4 anxiety scores as the dependent variable and age, gender and Time 2 anxiety score as the independent variables. The model explained a significant proportion of variance in anxiety scores ( $R^2 = 0.37, F(3, 132) = 26.14, p < 0.05$ ) with Time 2 anxiety score and gender being the only significant independent predictor (Table 6.15). Repeating the model without Time 2 anxiety score did not reveal any new findings. None of the variables included in any of the Time 4 models showed multicollinearity according to variance inflation factor and tolerance values.

**Table 6.56 Demographic and clinical predictors of anxiety at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	t	Sig.
Constant	-0.61	-3.89 – 2.68		-0.37	0.715
Age	0.00	-0.05 - 0.05	0.00	0.03	0.977
Gender*	1.75	0.26 – 3.24	0.16	2.32	0.022
Time 2 anxiety score*	0.62	0.47 – 0.77	0.58	8.27	0.000

\* Significant independent predictor

As utilised at both Time 2 and Time 3 psychological response scores, binary variables were also created for the Time 4 depression and anxiety scores based on the cut off threshold ( $\geq 10$  BDI,  $\geq 8$  HADS) to create two status categories for each scale: non-depressed versus depressed, non-anxious versus anxious. Mean scores, sample sizes and % for non-depressed/depressed and non-anxious/anxious groups for depression and anxiety are described in Table 6.16.

**Table 6.57 Mean depression and anxiety scores by depression and anxiety status at Time 4**

	<i>Mean (SD)</i>	<i>N</i>	<i>%N</i>
<b><i>BDI Depression T4</i></b>			
Non-depressed	4.03 (2.72)	113	73
Depressed	17.51 (9.74)	41	27
Total	7.62 (8.12)	154	100
<b><i>HADS Anxiety T4</i></b>			
Non-anxious	2.62 (2.14)	122	79
Anxious	11.26 (3.20)	33	21
Total	4.43 (4.28)	155	100

Logistic regression was performed to assess the influence of demographic and clinical factors on the likelihood that patients would report depression above the cut off threshold at Time 4. The model contained eleven categorical independent variables (gender, ethnicity, marital status, educational level, employment status, depression history, depression status at Time 2, deprivation level, previous CHD, and presence of diabetes) and two continuous independent variables (age and GRACE score). The full model containing all the predictors was statistically significant ( $X^2 (14, 131) = 29.94, p < 0.05$ ) indicating that the model was able to distinguish between patients who did and did not report significant depression (Table 6.17). The full model explained 30.4% of the variance in depression status and correctly classified 80.2% of cases. The only independent predictor of depression status at Time 4 was depression status at Time 2 with an odds ratio of 8.96 suggesting that patients who scored over the threshold for depression at Time 2 were nearly 9 times more likely to score over the threshold at Time 4.

**Table 6.58 Logistic regression predicting likelihood of depression at Time 4**

<b>Variable</b>	<b>Categories</b>	<b>Adjusted odds ratio</b>	<b>95% C.I.</b>	<b>P</b>
Age	<i>Annual increase</i>	0.94	0.86 to 1.02	0.15
Gender	<i>Male</i>	1		
	<i>Female</i>	3.13	0.90 to 10.96	0.074
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	0.45	0.10 to 2.06	0.30
	<i>High</i>	1.13	0.20 to 6.51	0.89
Marital status	<i>Married</i>	1		
	<i>Not married</i>	0.57	0.17 – 1.92	0.36
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	1.41	0.36 – 5.52	0.63
Education	<i>Basic</i>	1		
	<i>Secondary</i>	0.84	0.29 – 2.39	0.74
	<i>Degree</i>	0.55	0.11 – 2.70	0.46
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	1.22	0.36 – 4.17	0.75
Previous CHD	<i>No</i>	1		
	<i>Yes</i>	2.10	0.61 – 7.21	0.24
Diabetes	<i>No</i>	1		
	<i>Yes</i>	0.74	0.16 – 3.46	0.71
Depression history*	<i>No</i>	1		
	<i>Yes</i>	3.07	1.08 – 8.73	0.035
T2 Depression status *	<i>Not depressed</i>	1		
	<i>Depressed</i>	8.96	2.45 – 32.74	0.13
GRACE score	<i>Score increase</i>	1.04	1.00 – 1.08	0.074

\* Significant independent predictor

Logistic regression was also performed to assess the influence of demographic and clinical factors on the likelihood that patients would report anxiety above the cut off threshold at Time 4. The model contained ten categorical independent variables (gender, ethnicity, marital status, educational level, employment status, depression history, anxiety status at

Time 2, deprivation level, previous CHD, and presence of diabetes) and two continuous independent variables (age and GRACE score). The full model containing all the predictors was statistically significant ( $X^2 (14, 131) = 37.77, p < 0.05$ ) indicating that the model was able to distinguish between patients who did and did not report anxiety (Table 6.18). The model explained 40% of the variance in anxiety status and correctly classified 84.7% of cases. Anxiety status at Time 2 was the only significant independent predictor in the model with an odds ratio of 16.90 indicating that patients who scored over the threshold for anxiety at Time 2 were nearly 17 times more likely to score over the threshold at Time 4. Depression history was the only other significant predictor with an odds ratio of 4.23 suggesting that patients with a history of depression were over 4 times more likely to report significant anxiety at Time 4.

**Table 6.59 Logistic regression predicting likelihood of anxiety at Time 4**

<b>Variable</b>	<b>Categories</b>	<b>Adjusted odds ratio</b>	<b>95% C.I.</b>	<b>P</b>
Age	<i>Annual increase</i>	1.01	0.92 to 1.11	0.83
Gender	<i>Male</i>	1		
	<i>Female</i>	2.54	0.64 to 10.01	0.18
Social deprivation	<i>Low</i>	1		
	<i>Intermediate</i>	0.30	0.06 – 1.56	0.90
	<i>High</i>	0.06	0.00 – 1.18	0.15
Marital status	<i>Married</i>	1		
	<i>Not married</i>	2.16	0.55 – 8.43	0.27
Ethnicity	<i>White</i>	1		
	<i>Non-white</i>	2.12	0.45 – 10.02	0.34
Education	<i>Basic</i>	1		
	<i>Secondary</i>	1.47	0.42 – 5.09	0.55
	<i>Degree</i>	0.20	0.02 – 2.44	0.21
Employment	<i>Employed</i>	1		
	<i>Not employed</i>	3.04	0.70 – 13.09	0.13
Previous CHD	<i>No</i>	1		
	<i>Yes</i>	3.71	0.86 – 16.07	0.079
Diabetes	<i>No</i>	1		
	<i>Yes</i>	1.04	0.18 – 5.98	0.97
Depression history*	<i>No</i>	1		
	<i>Yes</i>	4.23	0.1.17 – 15.37	0.03
Anxiety status at Time 2*	<i>Not anxious</i>	1		
	<i>Anxious</i>	16.90	4.07 – 70.16	0.001
GRACE score	<i>Score increase</i>	1.02	0.98 – 1.06	0.41

\*Significant independent predictors

## **APPENDIX VI**

### **CHAPTER 7**

#### **TIME 4 QUALITY OF LIFE DESCRIPTIVE ANALYSES – TRACE STUDY**

### Quality of life at Time 4

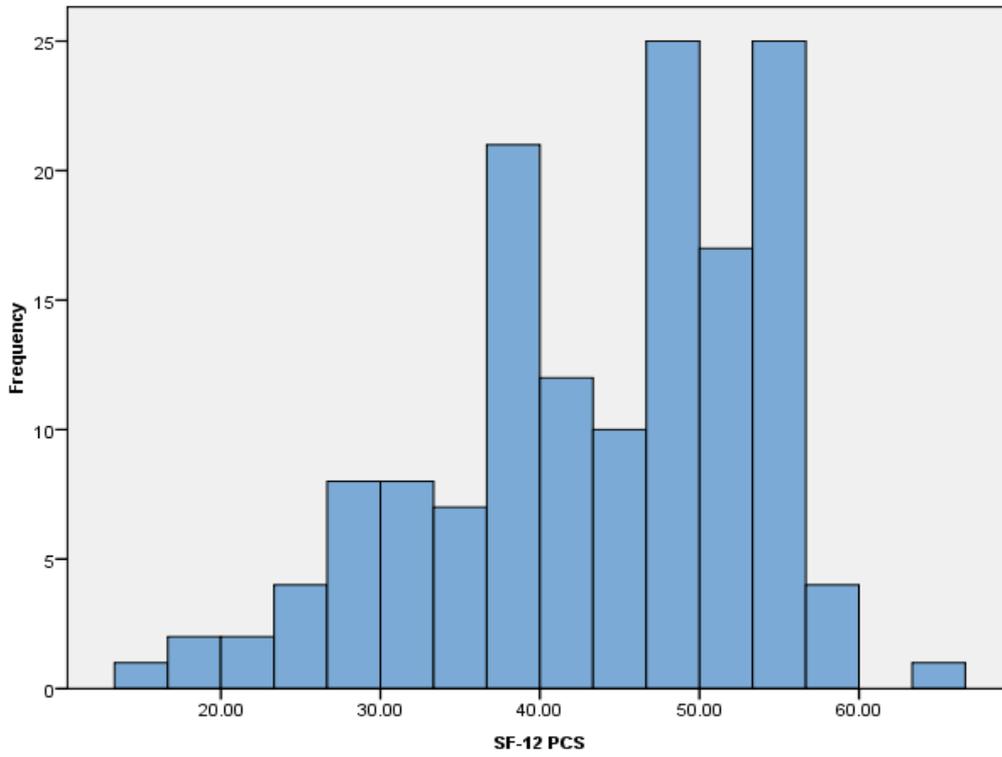
The mean scores for the SF-12 PCS and MCS at Time 4 (and Time 2 and 3 for comparison) are depicted in Table 7.33. The Time 4 scores indicate slightly below average physical health and average mental health quality of life. There was a significant increase in SF-12 PCS scores ( $t(126) = -4.72, p < 0.05$ ) between Time 2 and Time 4, but not between Time 3 and Time 4 indicating an overall improvement in physical health quality of life within the 12 months following ACS with the majority of this improvement occurring in the first 6 months. There was no significant change in SF-12 MCS score between Time 2 and Time 4, nor between Time 3 and Time 4. Time 4 SF-12 PCS and MCS scores were negatively correlated ( $r(147) = -0.20, p < 0.05$ ).

**Table 7.33 Mean SF-12 scores at Time 2, Time 3 and Time 4**

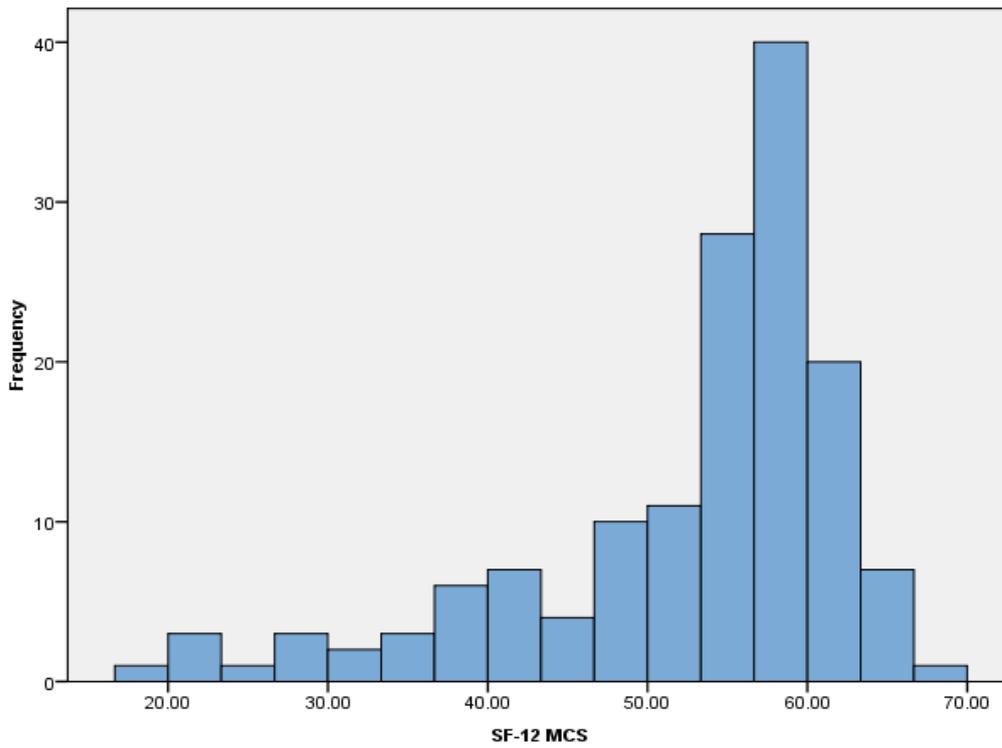
	Time 2		Time 3		Time 4	
	SF-12 PCS	SF-12 MCS	SF-12 PCS	SF-12 MCS	SF-12 PCS	SF-12 MCS
<b>Mean (SD)</b>	40.20 (9.56)	53.07 (9.89)	44.06 (10.23)	52.72 (10.20)	43.83 (10.10)	52.62 (10.22)
<b>Range</b>	13.68 – 61.19	15.17 – 67.55	14.21 – 58.96	15.39 – 65.48	16.23 – 64.41	18.85 – 68.41
<b>N</b>	209	209	146	146	147	147

The score frequency and distribution of SF-12 PCS and MCS at Time 4 are presented in Figures 7.15 and 7.16. The distributions were similar to those observed at Time 3 with the MCS being highly positively skewed with a number of outliers. The 5% trimmed mean (53.51) was not substantially different from the mean indicative of no undue influence from these outliers. The PCS scores were slightly positively skewed with no outliers.

**Figure 7.15 Score distribution for SF-12 PCS at Time 4**



**Figure 7.16 Score distribution for SF-12 MCS at Time 4**



The same ANCOVA analysis utilised for the Time 2 and Time 3 SF-12 data was run using SF-12 PCS and MCS at Time 4 as the dependent variables. ANCOVA analysis revealed that patients with a history of CHD had significantly lower physical quality of life at Time 4 than patients with no CHD history ( $F(1, 143) = 17.83, p < 0.05, \text{partial } \eta^2 = 0.11$ ). Patients with higher GRACE scores at Time 1 (indicative of more severe ACS) were also significantly more likely to report poorer physical quality of life at Time 4 than patients with lower GRACE scores ( $F(2, 142) = 4.28, p < 0.05, \text{partial } \eta^2 = 0.06$ ). Married patients reported better mental health quality of life at Time 4 than unmarried patients ( $F(1, 143) = 5.73, p < 0.05, \text{partial } \eta^2 = 0.04$ ).

Based on the findings of these ANCOVA analyses, a multiple regression analysis was conducted using Time 4 SF-12 PCS as the dependent variable and age, gender, previous CHD, GRACE score and Time 2 PCS score as the independent variables. The model explained a significant proportion of variance in PCS scores ( $R^2 = 0.26, F(5, 121) = 8.27, p < 0.05$ ) with Time 2 PCS score and previous CHD being the only significant independent predictors (Table 7.34). The model remained significant with the omission of Time 2 PCS and patient history of previous CHD was remained a significant independent predictor ( $\beta = -0.31, p < 0.05$ ).

**Table 7.34 Demographic and clinical predictors of SF-12 PCS at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	T	Sig.
Constant	48.25	35.56 – 60.94		7.53	0.001
Age	-0.11	-0.35 - 0.14	-0.12	-0.86	0.389
Gender	0.15	-4.55 – 4.85	0.01	0.06	0.949
Previous CHD*	-4.80	-9.29 – -0.30	-0.18	-2.11	0.037
GRACE score	-0.07	-0.19 – 0.04	-0.18	-1.26	0.212
T2 PCS*	0.25	0.07 – 0.43	0.24	2.79	0.006

\* Significant independent predictor

Multiple regression analysis was also conducted using Time 4 SF-12 MCS as the dependent variable and age, gender, marital status and Time 2 MCS score as the independent variables. The model explained a significant proportion of variance in MCS scores ( $R^2=0.47$ ,  $F(4, 122) = 26.92$ ,  $p<0.05$ ) with Time 2 MCS being the only significant independent predictors (Table 7.35). The model remained significant with the removal of Time 2 MCS score with marital status ( $\beta=-0.20$ ,  $p<0.05$ ) identified as a significant predictor and age as a near significant predictor ( $\beta=0.15$ ,  $p=0.059$ ). None of the variables included in either regression model showed multicollinearity according to variance inflation factor and tolerance values.

**Table 7.35 Demographic and clinical predictors of SF-12 MCS at Time 4**

	$\beta$	95% C.I for $\beta$	Standardised $\beta$	T	Sig.
Constant	16.35	5.89 – 26.80		3.10	0.002
Age	0.08	-0.03 – 0.18	0.09	1.39	0.166
Gender	-2.77	-6.53 – 1.00	-0.10	-1.46	0.148
Marital status	-0.91	-3.67 – 1.85	-0.04	-0.65	0.516
T2 MCS*	0.66	0.52 – 0.80	0.64	9.47	0.001

\* Significant independent predictor

As was found for the Time 2 and Time 3 scores, correlational analysis revealed that PCS and MCS were significantly negatively correlated with BDI depression and HADS anxiety scores at Time 3 (Table 7.36) indicating the close association between the experience of psychological distress and poor health quality of life.

**Table 7.36 Correlations between psychological distress and quality of life measures at Time 4**

	T4 SF-12 PCS	T4 SF-12 MCS
<b>T4 BDI depression</b>	-.398*	-.802*
<b>T4 HADS anxiety</b>	-.293*	-.754*

\*correlation is significant  $p<0.001$

