A Prospective Longitudinal Study of Repetitive Thought as a Vulnerability Factor for Depression in Patients with Coronary Heart Disease (CHD)

Submitted by Laura Victoria Baker, to the University of Exeter
as a thesis for the degree of Doctor of Clinical Psychology, May 2014

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Signature: ………………………………………………………………………..
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Depression and coronary heart disease: A systematic review of role of
Repetitive Thought as a mechanism

Trainee Name: Laura Baker
Primary Research Supervisor: Professor Edward Watkins
   Consultant Clinical Psychologist, Mood Disorders Centre
Secondary Research Supervisor: Professor Chris Dickens
   Consultant Psychiatrist, University of Exeter Medical School
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Abstract

*Background:* There is a bi-directional relationship between Coronary Heart Disease (CHD) and depression. In recent years, research has sought to understand the role of Repetitive Thought (RT) in the relationship between CHD and depression. It has been argued that RT could be a vulnerability factor or a perpetuating mechanism linking CHD and depression.

*Objectives:* This review summarises and synthesises the literature investigating RT and its relationship to depression in CHD populations.

*Method:* Systematic review of all literature to date using MEDLINE, PsycINFO, and Web of Knowledge databases with a narrative discussion.

*Results:* Seven relevant papers were included, consisting of cross-sectional and prospective studies.

*Conclusions:* There is a small amount of research that supports the notion of RT being involved in the relationship between depression and CHD. However the evidence is limited and how RT works as a mechanism or vulnerability factor remains unknown. Appropriate additional research is outlined.

*Keywords:* CHD, depression, repetitive thought, rumination.
Introduction

Rationale

**Depression and Coronary Heart Disease (CHD).** The ICD-10 defines depression to be when an individual experiences low mood, a reduction in levels of energy and activity, a reduced capacity for enjoyment or interest, and poor concentration (World Health Organisation, 2004). CHD includes angina, Acute Coronary Syndrome (ACS), Myocardial Infarction (MI) or heart failure and is thought to affect around 2.7 million people in the UK (NHS Choices, 2012).

CHD and depression have a co-morbid relationship (Goldston & Baillie, 2008). Reviews have concluded that depression is an independent risk factor for the onset of CHD (Lett et al., 2004), is associated with increased mortality (Barth, Schumacher, & Herrmann-Lingen, 2004) and adverse prognosis once CHD is established (Frasure-Smith & Lesperance, 2006; Wulsin, 2004).

Research shows varying rates of depression in CHD populations of between 10-25% (Rudisch & Nemeroff, 2003; Whooley, 2006). Depression severity varies depending on time since diagnosis; 10% of CHD outpatients have above moderate depression (Whooley et al., 2008), compared to 20% post-MI (Dickens et al., 2004; Lesperance & Frasure-Smith, 2000; Lett et al., 2004; Musselman, Evans, & Nemeroff, 1998). In comparison, the prevalence rate of depression in the general population is approximately 2.6% (NICE, 2009).

**CHD, Depression and Repetitive Thought (RT).** RT is defined as, “the process of thinking attentively, repetitively, or frequently about oneself and one’s world” (Segerstrom, Stanton, Alden, & Shortridge, 2003, p. 909). Watkins (2008) reviewed a number of cognitive mechanisms under the heading of RT, including: rumination, worry, repetitive thought, perseverative cognition, mental
stimulation, emotional processing, cognitive processing, reflection, problem solving, defensive pessimism, mind wandering, counterfactual and negative thought. RT includes depressive rumination, and is defined as “behaviors and thoughts that focus one's attention on one's depressive symptoms and on the implications of these symptoms” (Nolen-Hoeksema, 1991, p. 569). Depressive rumination has been identified as a final common mediator for a range of depression vulnerabilities including negative beliefs (Spasojević & Alloy, 2001), which has been experimentally demonstrated to increase negative thinking and cognitive distortions (Lyubomirsky & Nolen-Hoeksema, 1995; Rimes & Watkins, 2005). Prospective longitudinal research has found rumination predicted depression including the onset of new depressive episodes (Nolen-Hoeksema, 2000). Considerable evidence has shown that RT is implicated in the onset, duration, and maintenance of depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Segerstrom, Tsao, Alden, & Craske, 2000; Watkins, 2008).

Relevant theories linking RT and depression.

There are a number of potential relationships between CHD, depression and RT: e.g., (i) RT as common vulnerability factor for depression and CHD, (ii) RT as mediator of relationship between CHD and depression; (iii) RT as mediator of relationship between depression and CHD; (iv) RT as a moderator of relationship between depression and CHD, or (v) no causal relationships, but all factors associated with a common marker (e.g., neuroticism). To understand the CHD, depression and RT relationship it is important to consider relevant supported models which have been developed to understand the relationship between RT and depression.
Control Theory proposed that discrepancy between an individual’s perceived and desired rate of goal progress can result in rumination; which persists until the goal is either resolved or abandoned (Martin & Tesser, 1996). Consistent with this, experimental research demonstrated that thinking about unresolved goals increased ruminative thoughts relative to thinking about resolved goals (Roberts, Watkins, & Wills, 2013). Control theory would predict that RT would be prevalent in CHD as desired goals may suddenly become less attainable for individuals, resulting in the experience of multiple losses (e.g. in health, lifestyle, perceived mortality) which could increase rumination.

The response style theory (Nolen-Hoeksema, 1991), proposed that depressive rumination was a style of responding to distressing symptoms. Individuals repetitively focus on the symptoms of distress and the causes or consequences of the distress. The theory is well supported and evidence has shown rumination predicts the onset of depression (Nolen-Hoeksema, Wisco & Lyubmisky, 2008). The subsequent continued ruminative response is hypothesised to prolong depression. The theory should be applicable in all populations, including CHD where it could potentially account for elevated depression.

**Relevant theories and models linking CHD and RT.** Initially, Roberts (1989) proposed that cognitive mechanisms influence the relationship between depression and CHD: following an MI, individuals are hypothesised to experience cognitive distortions such as overgeneralisation, where general inferences about the self and the future are drawn on the basis of a single event (the MI), which, in turn, leads to guilt and depression. Overgeneralisation is a
characteristic of unhelpful abstract RT and a consequence of abstract rumination (Watkins, 2008).

Perseverative cognition is defined as “the repeated or chronic activation of the cognitive representation of one or more psychological stressors” (Brosschot, Gerin, & Thayer, 2006, p. 2), it is often referred to interchangeably with rumination and RT. Brosschot et al. (2006) argued that perseverative cognition takes place before, during, and after a stressful event. They proposed that perseverative cognition about a stressor creates a prolonged stress response and this causes sustained activation of physiological systems, including cardiovascular systems, which leads to worsened cardiac outcomes. Supporting experimental research found perseverative cognition delayed cardiac recovery (Verkuil, Brosschot, De Beurs, & Thayer, 2009). However, the evidence is limited by few experimental studies investigating the impact of RT on cardiac recovery. Existing studies have been conducted in non-cardiac populations and with induced rumination (Glynn, 2002). Therefore, they do not investigate the long-term implications of RT activating physiological systems. However, it is hypothesised that because evidence has shown rumination affects cardiac recovery, rumination may also be involved in CHD outcomes, though presently there is no robust evidence to support this (Kubzansky, Davidson, & Rozanski, 2005; Larsen & Christenfeld, 2009).

Larsen and Christenfeld (2009) extended Brosschot et al.’s (2005) earlier model to include depression. They hypothesised that perseverative cognition was related to both CHD and depression. They argued that inflexibility (cognitive, emotional, and physiological) leads to rumination, worry, low heart rate variability, reduced vagal tone and sympathetic arousal. These factors then
perpetuate one another and cause increased physiological arousal, worsening cardiac and depression outcomes. The model proposes that rumination could be one mechanism that elevates physiological arousal and impacts on CHD and depression outcomes.

Some reviews have investigated the involvement of cognitive processes in depression and CHD. They have concluded that the relationship between CHD and depression involves negative states or negative beliefs (French, Cooper & Weinman, 2006). One review identified five negative states (hopelessness, pessimism, rumination, anxiety, and anger) associated with depression and CHD (Kubzansky et al., 2005). Although this review did not specifically explore RT, it identified that rumination may well be involved. However, no studies involving rumination were conducted within the CHD population and due to the brevity of the review the results must be interpreted with some caution. Moreover, this was not a systematic review and is outdated following recent developments in the empirical data. This highlights a need for a current review to specifically address the relationship between RT, depression, and CHD.

Despite preliminary theoretical and empirical data implicating RT as a factor linking CHD and depression, to the knowledge of the researcher, to date, there have been no systematic reviews that have specifically examined the role of RT as a mechanism or risk factor in the relationship between CHD and depression. This review therefore questions whether there is sufficient evidence to support the idea that the relationship between CHD and depression could be mediated by the cognitive mechanism of RT. Consolidation of the research will inform the field of findings to date, outline inconsistencies in
findings, and highlight gaps in knowledge where further research would be beneficial.

**Objectives**

This systematic review aims to appraise the relevant literature in order to examine the evidence supporting a relationship between RT, CHD and depression. The review will seek to evaluate all literature which refers to RT as being involved in CHD and depression. It is interested in any mechanisms, or risk factors involved in the relationship between CHD and depression, and those that involve RT or closely related constructs. The review will focus on empirical evidence. Studies to be reviewed must be written in English and conducted with cardiac populations (CHD patients or following a MI). All studies included will be quantitative (cross-sectional, experimental, prospective, retrospective and clinical trials). The search included all empirical evidence published prior to April 2014. The search will not be restricted by interventions and comparators.

**Methods**

**Eligibility Criteria**

This systematic review was conducted using the PRISMA reporting protocol (Moher, Liberati, Tetzlaff, & Altman, 2009) as this allows for a standardised non-biased approach to the review. Population inclusion criteria included all studies with CHD and depression in an adult population. All settings such as outpatients, cardiac rehabilitation or inpatients were included. Outcome inclusion criteria included: (i) studies that had investigated both CHD and depression, (ii) were related to RT as outlined by Watkins (2008) or wider cognitive processes that would specifically include RT, e.g. dysfunctional
cognitions, (iii) focused on relationships or processes e.g. mechanisms for RT relationship with CHD and depression, (iv) studies with prospective, retrospective, experimental, cross-sectional designs, meta-analysis, and clinical trials, and (v) all searchable dates. The exclusion criteria was: (i) discussion papers, dissertations, books and media material, (ii) studies not published in English, (iii) any studies not conducted using an adult population of over 18 years, and (iv) any studies that focused on beliefs or general psychological or cognitive processes.

**Information Sources and Search Strategy**

In March 2014, MEDLINE, PsycINFO, and Web of Knowledge databases were systematically searched for relevant studies and reviews using the following string of search terms: [(Coronary heart disease or Myocardial infarction) and (risk or mechanisms or vulnerability or factor) and (ruminat* or worry or repetitive thought or perseverative cognition or mental stimulation or emotional processing or cognitive process or reflection or problem solving or defensive pessimism or mind wandering or counterfactual or negative thought) and depress*]. The terms used to search for RT were based on terms outlined in a previous systematic review (Watkins, 2008). Additionally, surrounding literature was revised for backward and forward citations from the selected papers to ensure all relevant papers were attained.

**Data Collection Process**

Data was extracted from the databases into EndNote X6 software. This allowed for archiving of articles for a second review to reduce human error and bias where possible. At the point of reviewing abstracts and to improve validity,
a second reviewer checked 50% of the studies to insure the inclusion and exclusion criteria were adhered to.

Risk of Bias in Studies

Each article was assessed for risk of bias. This included publication, reporting, selection, and measurement bias. Overall biases of the study such as study design, sample population and data collection methods were considered. Bias was assessed based on information provided in the study. Where data were available it was checked to ensure authors had indeed reflected the findings by reviewing statistical output, ensuring all potential studies were included in discussions and confirming that any key conflicting and supporting studies had been accurately reported. Results were checked to ensure they had been accurately reflected and reasonable conclusions had been drawn.

Results

Study Selection

Initial searches revealed 659 potentially relevant articles. After applying inclusion and exclusion criteria to the titles, 108 articles remained. Following a review of title and abstracts, a further 90 were eliminated as they did not fully meet the inclusion criteria. The majority of papers removed at this stage did not specifically focus on any cognitive process or focused on biological mechanisms. At the abstract stage in the search, 50% of the articles were additionally reviewed by an independent rater to establish the validity of the process. Excellent levels of consistency (100%) were found between the researcher and second rater with regard to the excluded or included studies, which ensured the validity of this process. There were potentially 18 papers remaining, but following a review of the full text articles, 12 were excluded for
not meeting the inclusion criteria. Some relevant but non-empirical papers were excluded, their citations were checked. Exclusions were made for articles that examined cognitive processes that were not closely associated with RT, e.g. cognitive factors or those focused on beliefs e.g. illness perceptions. An additional paper was found by reading around the literature and checking backward and forward citations. Seven appropriate empirical papers remained, which will be discussed within the review. Figure 1 below shows a flow diagram summarising the study selection.

Figure 1. Flow diagram of article selection through the different stages.
Synthesis of Results

Overview of selected articles. Table 1 provides an overview of the seven papers included in this review. There were four cross-sectional studies and three prospective studies. This narrative review will discuss the available empirical research by type of study; cross-sectional and prospective.
### Table 1.

**Summary of the seven papers included in the review**

<table>
<thead>
<tr>
<th>Author</th>
<th>Design</th>
<th>Sample</th>
<th>Measures used</th>
<th>Main finding</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cross-sectional studies</strong></td>
<td></td>
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</tr>
<tr>
<td>Dickens, Coventry, Khara, Bower, Mansell, and Bakerly (2012)</td>
<td>Cross-sectional</td>
<td>190 patient with Long Term Conditions (LTCs)</td>
<td>PSWQ, HADS, WBSI, AAQ</td>
<td>22% of CHD patients were affected with symptoms of depression. They found participants with high worry, thought suppression and thought avoidance were more likely to suffer with depression when controlling for confounding variables.</td>
</tr>
<tr>
<td>Doyle, Mcgee, Conroy, and Delaney (2011b)</td>
<td>Cross-sectional</td>
<td>336 Acute Coronary Syndrome (ACS) patients</td>
<td>HADS-D MQ-10, LTE-Q, PES-AD, BJW-S, DS14</td>
<td>Vulnerabilities (cognitive, behavioural, personality D, and interpersonal) associated with depression in ACS patients. Psychological vulnerabilities were more strongly associated with depression than demographic or disease vulnerabilities.</td>
</tr>
<tr>
<td>Garnefski et al. (2009)</td>
<td>Cross-sectional</td>
<td>139 people following MI</td>
<td>CERQ, GOQ, HADS.</td>
<td>Cognitive coping strategies of rumination and catastrophising were significantly associated with higher depressive symptoms. Positive refocusing, goal re-engagement, were associated with lower depressive symptoms.</td>
</tr>
<tr>
<td>Martens et al. (2006)</td>
<td>Cross-sectional matched</td>
<td>120 (40 post MI depressed/ 40 post MI non-depressed. 40 psychiatric outpatients)</td>
<td>Clinical interview (SCID and CIDI), BDI, CCL-D</td>
<td>Depressive cognitions were higher in depressed patients post-MI than non-depressed patients post MI but not higher than clinically depressed psychiatric patients.</td>
</tr>
<tr>
<td><strong>Prospective Studies</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Denton, Rieckmann, Davidson, and Chaplin (2012)</td>
<td>Prospective longitudinal</td>
<td>387 Acute Coronary Syndrome</td>
<td>BDI-I, DAS-24, PES-E, LVEF, RST, DAS, RSS</td>
<td>Rumination independently predicted increased depression severity, above other vulnerabilities; it also interacted with poor dyadic adjustment to worsen severity of depression.</td>
</tr>
<tr>
<td>Doyle, Mcgee, Delaney, Motterlini, and Conroy (2011a)</td>
<td>Prospective longitudinal</td>
<td>375 Acute Coronary Syndrome (ACS) Patients</td>
<td>BDI-FS, HADS (depression subscale), LTE-Q, PES-AD, BJW-S, DS14</td>
<td>Identified that one year post ACS that depression was predicted by stressful life events, reinforcing events, cognitive distortions, personality for participants with persistent depression. For participants with sub-threshold depression, cognitive distortions and stressful life events were predictive of depression.</td>
</tr>
<tr>
<td>Garnefski and Kraaji (2010)</td>
<td>Prospective longitudinal</td>
<td>88 people following MI</td>
<td>CERQ, GOQ, and HADS.</td>
<td>After one year post-MI cognitive coping strategies (rumination, catastrophising, and positive refocusing) and goal adjustment strategies predicted 39% of the variance in depressive symptoms. Rumination and catastrophising at baseline was significantly associated with increased depressive symptoms at one year follow-up. Goal related coping at baseline was significantly associated with less depressive symptoms at one year follow up.</td>
</tr>
</tbody>
</table>

**Abbreviations:** CHD (Coronary Heart Disease), HADS (Hospital Anxiety and Depression Scale), BIPQ (Brief Illness Perceptions Questionnaire), IPQ (Illness Perceptions Questionnaire), CAQ (Cardiac Anxiety Questionnaire), SCL-90R (Symptom Checklist-90, revised version), MOSSAS (Medical Outcomes Study Specific Adherence scale), PCS SF-36 (Physical Component Score- Short Form-36, HRQol (Health Related Quality of Life), PSWQ (Penn State Worry Questionnaire), WBSI (White Bear Suppression Inventory), AAQ (Acceptance and Action Questionnaire), LTCs (Long-Term Health Condition’s), BDI-FS (Beck Depression Inventory – Fast Screen), LTE-Q (List of Threatening Experiences Questionnaire), PES-AD (Dysfunctional Attitude Scale), DAS (Dyadic Adjustment Scale), RSS (Ruminative Responses Scale).
Cross-sectional Studies. Overall, the cross-sectional studies show that there is a significant positive relationship between RT and constructs closely associated with RT (including perseverative cognition, rumination, cognitive distortions, depressive cognitions) and depression in CHD populations or post-MI (Dickens et al., 2012; Doyle et al., 2011b; Garnefski et al., 2009; Martens, 2006). The strength and importance of the relationship varies across the studies. Garnefski et al. (2009) investigated the relationship between goal adjustment (disengagement and reengagement) and cognitive coping variables (rumination/catastrophising, positive refocusing, planning, putting in perspective, positive appraisal and other blame) and depression in post-MI patients. They found a highly significant positive correlation between rumination and catastrophising and depression post-MI, with rumination having a greater effect. Rumination was significantly more strongly associated with depression than other cognitive coping processes. They also found a negative significant relationship, between goal reengagement and depression.

Dickens et al. (2012) investigated perseverative negative cognitive process in patients with long-term conditions (LTCs), including CHD. A relationship was found between RT and CHD and depression. Depression was 20% more likely with high worry, 10% more likely with thought suppression and 22% more likely with thought avoidance. Depression was strongly associated with thought suppression and thought avoidance. This provides evidence that perseverative negative cognition is associated with increased likelihood of depression in patients with LTCs, including CHD.

Martens et al. (2006) compared post-MI patients to a clinically depressed psychiatric population. They found significantly higher rates of depressive
cognitions in the post-MI depressed group in comparison to the post-MI non-depressed group, and higher rates again in the psychiatric population.

Doyle et al. (2011a) measured the association of a range of psychological vulnerabilities (cognition distortions, Type-D personality, and interpersonal events) with depression in patients with an Acute Coronary Syndrome (ACS). ACS is a specific heart condition which includes acute myocardial ischaemic states e.g. unstable angina (Grech & Ramsdale, 2003). They found the psychological vulnerabilities significantly predicted 22% of the variance for depression, with greater psychological vulnerability positively associated with increasing depression.

There is limited cross-sectional research, thus, all authors recommended additional prospective and longitudinal research to better understand depression, RT and CHD relationship and to better infer causal directions (Dickens et al., 2012; Doyle et al., 2011b; Garnefski et al., 2009; Martens et al., 2006).

The cross-sectional studies all had adequate sample sizes with the smallest being 120 participants. All the included studies failed to accurately measure cardiac symptoms or time since cardiac event. This could be a key confounding factor for both rates of depression and RT. Measurement of constructs varied greatly between the studies, thus conclusions should be drawn with caution. Doyle et al. (2011b) measured cognitive distortions using the Belief in a Just World Scale (BJW-S, Furnham, 2003), which is arguably not a comprehensive measure of cognitive distortions (Doyle et al., 2011b) or RT. The study would have benefited from using more specific measures which would allow for specific cognitive processes to have been identified e.g.
rumination or worry. Depression was measured on both the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) and Beck Depression Inventory- Fast Screen (BDI-FS: Beck, Steer, Ball, Ciervo, & Kabat, 1997); one item on the BDI-FS relating to suicide was omitted. The use of both measures was arguably unnecessary.

There are additional reasons for why the results from these studies should be interpreted with caution. Firstly, Dickens et al.’s (2012) sample consisted of LTC of which only 31.6% were patients with CHD; the authors did not conduct disease specific analyses. Furthermore, responses were only received from 66% of initially approached participants, which raises questions about the representativeness of those participants retained in the study and the generalisability of the findings. Secondly, Doyle et al.’s (2011b) study does not clearly define their understanding of cognitive distortions, or provide separate analysis for them. Cognitive distortions could include a range of constructs such as unhelpful thinking patterns, repetitive or ruminative thoughts or dysfunctional beliefs. Lastly, Martens et al. (2006) study compared post-MI patients with clinically depressed psychiatric patients. The study gives a limited inclusion and exclusion criteria, making it difficult to ascertain if all potential co-morbidities were adequately screened for and are not skewing the results. For example, patients were excluded for a diagnosis of psychosis but there is no mention of other disorders such as PTSD, which could have a profound impact on depression (Campbell et al., 2007) and RT.

**Prospective Studies.** The prospective studies expand the conclusions drawn from cross-sectional research. They found that RT predicted depression in cardiac populations (Denton et al., 2012; Doyle et al., 2011a; Garnefski &
Garnefski and Kraaji’s (2010) cross-sectional study in post-MI patients was continued prospectively for one-year to investigate changes from baseline measures. They found the specific cognitive processes of rumination, catastrophising, positive refocusing and goal adjustment predicted 39% of the variance in depression scores at a one-year follow-up. Rumination and catastrophising had a significant positive association with depression. Findings showed goal reengagement was associated with lower depressive symptoms at one year follow-up. However, only 88 of the 139 participants were followed-up and it perhaps therefore not necessarily a true representative of a post-MI population.

A three month prospective study found that rumination independently predicted depression severity in ACS (Denton et al., 2012) when controlling for baseline depression. This study included a large sample of 457 patients and retained 387 participants at three month follow-up, making the results reliable. Findings showed rumination independently predicted depression severity at three month follow-up. The study measured a range of vulnerabilities that could then be controlled for including cognitive, behavioural, interpersonal and cardiac severity.

Doyle et al.’s (2011a) cross-sectional study with ACS patients was continued prospectively for one-year. As would be expected they found baseline depression to be the largest predictor of depression at follow up. They also found that cognitive distortions were significant independent predictors of
depression at one year follow-up. Only 58% of participants were retained at one year follow-up, and the impact of this attrition is unknown; there is no evidence of a statistical analysis of respondents and non-respondents. Given the large attrition rate, such analyses would have been beneficial to rule out any bias and better understand the population, for example it may have been the more severely depressed, those who were physically unwell, or those with more cognitive distortions that dropped out.

In all of the prospective studies, patients were identified following a cardiac event, which meant that there was a lack of knowledge about pre-cardiac event rumination. Assessment of pre-cardiac event RT rumination would have determined if increased RT preceded the cardiac event or whether it was a consequence of the cardiac event and associated losses and unresolved goals (Martin & Tesser, 1996; Nolen-Hoeksema, 1991).

The prospective studies also attempted to measure cardiac symptoms. Denton et al. (2012) used the Left Ventricular Ejection Fraction (LVEF), which categorised patients into four groups (mild to severe). Garnefski and Kraaij’s (2010) study used the Physical Functioning Scale (McHorney, Ware, Lu, & Sherbourne, 1994), which assesses QoL, as opposed to specific cardiac symptoms. Although this is not a specific cardiac measure it allowed for physical health limitations to be controlled for. Doyle et al. (2011a) did not include a specific measure of cardiac symptoms, but they did categorise participants by type of cardiac event or specific diagnosis, which allowed for differences between diagnoses to be investigated.

Another methodological issue within the prospective research is a number of potential confounding factors were not measured and subsequently
controlled for. For example, social support, personality (Garnefski & Kraaji, 2010), QoL (Denton et al., 2012) and worry (Doyle et al., 2011a) were not assessed.

All of the prospective studies and cross-sectional studies, bar one (Martens et al., 2006), relied solely on self-report assessment measures. Potential disadvantages of self-report measures include response bias and incomplete measures. Potential advantages however are increased participation and less reliance on a research team to conduct assessments, making it more financially viable.

Discussion

Summary of Evidence

The studies outlined in this systematic review show that there is a cross-sectional (Dickens et al., 2012; Garnefski et al., 2009) and prospective (Denton et al., 2012; Garnefski & Kraaji, 2010) relationship between RT and depression in cardiac populations. This review highlights that there is very limited empirical research with a mere seven studies identified; therefore findings should be cautiously interpreted. Several prospective studies have found RT to be a predictive factor for depression in CHD (Denton et al., 2012; Garnefski & Kraaji, 2010); however, additional prospective longitudinal research is required to confirm these findings.

This review has found insufficient evidence to support models (Brosschot et al., 2006; Larsen & Christenfeld, 2009) that propose RT causes continued and prolonged activation of the physiological systems worsening CHD and depression. Further research is required to investigate the prolonged impact of RT on CHD and to expand on studies that have currently only used induced RT.
The research outlined in this review did not examine RT pre-cardiac event and this would be useful in understanding whether increased RT precedes a cardiac event or increases subsequent to a cardiac event (Denton et al., 2012). The current research does not enable inference of causal direction as prospective relationships could reflect a third factor predicting both depression and RT in CHD. Experimental manipulation of RT in CHD would provide more direct evidence of whether RT is a mechanism causing depression in CHD.

Given the emerging evidence that RT is involved in the relationship between depression and CHD, research into potential effective psychological interventions for RT in CHD could be beneficial and help to understand the causal role of RT in depression in CHD populations. Randomised controlled trials (RCTs) have shown enhanced depression care for CHD patients reduced depressive symptoms (Davidson et al., 2010) and psychological interventions improved depression outcomes (Whalley et al., 2011). Further RCTs are required to develop an evidence-based treatment for depression in cardiac populations (Davidson, Rieckmann, & Lesperance, 2004).

There are a number of methodological issues with the studies included in this review, most importantly with initial recruitment (Dickens et al., 2012) and attrition for longitudinal research (Doyle et al., 2011a). Going forward, research would benefit from improving recruitment rates (Dickens et al., 2012), and reducing attrition rates to gain a more representative CHD sample. In addition, findings may have greatly differed due to a variety of measurement tools being used; with almost all studies solely using self-report assessment.
Limitations

The conclusions of this review are limited by a lack of experimental, retrospective, prospective research or clinical trials available. This review has focused on RT; a wider review including other cognitive processes may add to the findings.

Conclusions

This systematic review of a small but emerging literature has confirmed a relationship between RT and depression in cardiac populations. It was unable to establish the specific role of RT as a mechanism or risk factor. Additional prospective longitudinal and experimental research is required to further support these conclusions.

This review has highlighted a need for research focussing on the role of RT on the physiological system in order to test the hypothesis that RT prolongs activation and worsens CHD. Research that investigates pre-cardiac event depression and RT would be advantageous to understand the specific role that RT could play.
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A Prospective Longitudinal Study of Repetitive Thought as a Vulnerability Factor for Depression in Patients with Coronary Heart Disease (CHD)

Trainee Name: Laura Baker

Primary Research Supervisor: Professor Edward Watkins
Consultant Clinical Psychologist, Mood Disorders Centre

Secondary Research Supervisor: Professor Chris Dickens
Consultant Psychiatrist, University of Exeter Medical School

Field Collaborators: Dr Manish Gandhi and Rebecca Chawner
Royal Devon and Exeter Hospital

Target Journal: Journal of Psychosomatic Research

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Abstract

**Objective:** Theoretical and empirical evidence suggests a relationship between Repetitive Thought (RT, e.g. rumination), and depression in patients with Coronary Heart Disease (CHD). To date, cross-sectional studies indicate that rumination is associated with depression in CHD, but additional prospective longitudinal research is required to determine if rumination predicts subsequent depression. This research therefore aimed to test the hypothesis that RT, specifically rumination, is a vulnerability factor for depression over time in a CHD population. It was predicted that RT at baseline would predict depression rates at three month follow-up after controlling for baseline depression and potential confounding factors.

**Methods:** Inpatients and outpatients with a diagnosis of CHD completed self-report questionnaires at baseline (N = 101) and at three month follow-up (N = 85). The data was analysed using a hierarchical multiple regression.

**Results:** Baseline rumination significantly predicted depression at the three month follow-up after controlling for baseline depression and potential confounding factors. Rumination accounted for 8.3% of the variance (p< .001). Subscales of brooding and reflection were also found to be individually predictive of follow-up depression explaining 4% of the variance (p< .005) and 7% of the variance (p< .001) respectively.

**Conclusion:** Findings are consistent with previous prospective and cross-sectional research that indicates that rumination plays a unique role in the maintenance of depression in CHD patients and is an identifiable vulnerability factor.
Introduction

Depression and Chronic Heart Disease (CHD)

The ICD-10 defines depression to be when a patient suffers from at least one of three key symptoms (low mood, reduction in energy, activity, capacity for enjoyment or interest), which should be present on most days, most of the time and for at least two weeks (World Health Organisation, 2004). CHD includes angina, Acute Coronary Syndrome (ACS), and Myocardial Infarction (MI) or heart failure (NHS Choices, 2012). CHD is also referred to as Coronary Artery Disease (CAD) and ischemic heart disease.

Research has indicated elevated co-morbidity between CHD and depression (Goldston & Baillie, 2008). A number of reviews have concluded that there is a causal bi-directional relationship between CHD and depression (Aromaa et al., 1994; Frasure-Smith & Lesperance, 2005, 2006; Goldston & Baillie, 2008; Kubzansky & Kawachi, 2000; Smith, 2001). Depression has a prevalence rate of 20% or higher post-MI (Dickens et al., 2004; Lesperance & Frasure-Smith, 2000; Lett et al., 2004; Musselman, Evans, & Nemeroff, 1998). Carney and Freedland (2008) reported that approximately 20% of patients with CHD have severe depression and 20% have minor depression. Depression has been found to be an independent (Ford, Mead, Chang, Cooper-Patrick, Wang, & Klag, 1998; Wulsin, 2004; Wulsin & Singal, 2003) and robust (Whooley & Wong, 2013) risk factor for worse cardiac outcomes in CHD. Research indicates that depression is a risk factor for mortality and increased cardiac symptoms in patients with CHD (Appels, 1997, 2002; Barefoot, Brummett, Helms, Mark, Siegler, & Williams, 2000; Barth, Schumacher, & Herrmann-Lingen, 2004; Dickens et al., 2008a; Frasure-Smith & Lesperance, 2003, 2005;
Retrospective studies have demonstrated that depression precedes CHD
(Dickens et al., 2005; Thiel, Parker, & Bruce, 1973), and it is proposed that
depression increases the risk of CHD onset by 1.5-2 times (Lett et al., 2004).
Recently research has proposed that Repetitive Thought (RT) could be a
mechanism linking CHD and depression together (Denton, Rieckmann,
Davidson, & Chaplin, 2012), building on work establishing RT as a risk factor for
depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008).

**Depression and RT**

There are a number of potential relationships between CHD, depression
and RT: e.g., (i) RT as common vulnerability factor for depression and CHD, (ii)
RT as mediator of relationship between CHD and depression; (iii) RT as
mediator of relationship between depression and CHD; (iv) RT as a moderator
of relationship between depression and CHD, or (v) no causal relationships, but
all factors associated with a common marker (e.g., neuroticism). To understand
the CHD, depression and RT relationship it is important to consider relevant
supported models which have been developed to understand the relationship
between RT and depression.

RT is defined as the “the process of thinking attentively, repetitively, or
frequently about oneself and one’s world” (Segerstrom, Stanton, Alden, &
constructs within RT, including: worry, positive and negative rumination, post-
event rumination, perseverative cognition, emotional and cognitive processing,
mental stimulation, planning, problem solving, rehearsal, reflection,
counterfactual thinking, defensive pessimism and habitual negative self-thinking
Depressive rumination is defined as “behaviors and thoughts that focus one's attention on one's depressive symptoms and on the implications of these symptoms” (Nolen-Hoeksema, 1991, p. 569). Nolen-Hoeksema (1991) proposed the response style theory which argued that depressive rumination is a mode for responding to distressing symptoms and it involves repetitively focusing on symptoms of distress and the causes or consequences of the distress. The continued ruminative response prolongs depression because individuals are focused on their negative emotional state and use negative biased thinking. Experimental research has identified that rumination is positively associated with negative affect (Watkins, 2004). Additionally, prospective research found rumination to be involved in the onset of depression (Nolen-Hoeksema, 2000). Rumination is an established factor that contributes to the onset, duration, severity and maintenance of depression (Nolen-Hoeksema et al., 2008; Segerstrom et al., 2000; Watkins, 2008). To date the effects of rumination on the development of depression have been studied in patients with depression or general community samples. However, it seems
likely that these consequences of RT would also extend to patients with CHD and potentially account for elevated depression.

Moreover, RT can also be understood in terms of Control Theory, which proposed that goal discrepancies result in rumination (Martin & Tesser, 1996). If there are discrepancies between an individual’s perceived and desired rate of goal progress (Carver & Scheier, 1990) they will ruminate about it. Unresolved goals will lead to rumination until the goal is resolved or abandoned. Rumination about goal attainment continues despite the likelihood of it being achievable and can be in order to make sense of an event (Martin & Tesser, 1996). Recent research supported this theory, finding that thinking about unresolved goals increased ruminative thoughts (Roberts, Watkins, & Wills, 2013).

**Is RT a linking mechanism between CHD and depression?**

RT is associated with depression in CHD populations (Denton et al., 2012; Garnefski & Kraaji, 2010). Because RT contributes to the onset and maintenance of depression, it is likely that this process occurs in CHD patients, consistent with the response styles theory. Moreover, for CHD patients following a life-changing event such as an MI or diagnosis of CHD, they are likely to be focusing on negative symptoms of distress and the causes or consequences of the distress (Nolen-Hoeksema, 1991). Therefore, CHD patients are likely to be ruminating about what they could have done differently, what will happen now and losses they have experienced e.g. in their ongoing health, life ambitions and mortality. It is then not surprising that emerging evidence has shown a link between RT and depression within a CHD population (Denton et al., 2012) and that rumination could be an important coping strategy (Garnefski & Kraaji, 2010). If additional prospective research of
CHD patients found rumination predicted change in depression levels over time it would support and extend the response styles theory to a CHD population, this will be tested in the current research. Additionally, when considering Control Theory (Carver & Scheier, 1990), there is a potential that the impact of a severe cardiac event could significantly alter goal attainment and leave unresolved goals, resulting in increased ruminative thoughts (Roberts et al., 2013).

Several theoretical models propose that RT could be involved in the onset and maintenance of CHD. Larsen and Christenfeld (2009) hypothesise that co-morbidity between CHD and depression could be a result of a state of cognitive and autonomic inflexibility. They note that rumination has been shown to impact physiological recovery and consequently cardiac health. Therefore, increased rumination, worry and obsessions, could increase sympathetic arousal leading to lower heart rate variability and reduced vagal tone. They hypothesise that rumination mediates physiological and psychological symptoms and outcomes by extending sympathetic arousal, and that this could become a perpetuating cycle. Thus, rumination could both cause and extend physiological arousal affecting both cardiac and depression outcomes. This theory suggests that rumination is a shared vulnerability factor for both depression and CHD. To test Larsen and Christenfeld’s theory patients would ideally need to be recruited pre-cardiac event and have a medical measure of cardiac symptoms; therefore this cannot be tested in the current study.

Larsen and Christenfeld’s (2009) hypothesis extended an earlier model by Brosschot, Gerin, and Thayer (2006); this model did not include the potential influence of depression but supports the hypothesis that RT plays an important
role in CHD. Brosschot et al. (2006) propose that perseverative cognition, defined as “the repeated or chronic activation of the cognitive representation of one or more psychological stressors” (p. 2) and manifested as worry or rumination, mediates health consequences by creating continued activation of physiological systems. Their model has been supported by experimental research, which found that perseverative cognition delayed cardiac recovery (Verkuil, Brosschot, De Beurs, & Thayer, 2009) and elevated blood pressure (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Glynn, 2002).

Research has linked RT and depression with CHD. Rumination and catastrophising are associated with higher depressive symptoms post-MI and at one year follow-up (Garnefski & Kraaji, 2010; Garnefski et al., 2009). Dickens et al. (2012) found a link between RT and CHD and depression. Depression was 20% more likely with high levels of worry, 10% more likely with thought suppression and 22% more likely with thought avoidance. Cognitive distortions have been found to be predictors of depression in CHD (Doyle, Mcgee, Delaney, Motterlini, & Conroy, 2011a; Doyle, Mcgee, Conroy, & Delaney, 2011b; Stafford, Jackson, & Berk, 2009), indicating that cognitive process play a role in depression and CHD. The research outlined indicates a relationship between RT, CHD and depression, but the specific mechanism that RT may play and extent of the mechanism has not clearly been established.

To our knowledge, only one prospective longitudinal study has specifically examined RT as a mechanism or vulnerability factor for depression in CHD (Denton et al., 2012). After controlling for baseline depression and cardiac disease severity, cognitive, behavioural, interpersonal vulnerabilities including rumination were longitudinally associated with depression severity
following an Acute Coronary Syndrome (ACS). ACS refers to a range of acute myocardial ischaemic states, which can include unstable angina, non-ST segment elevation myocardial infarction, and ST segment elevation infarction (Grech & Ramsdale, 2003), ACS is a specific diagnosis that would come under a broader heading of CHD. This study was conducted in the USA, it included both men and women admitted with ACS and patients were assessed within 7 days of admission. They had an overall longitudinal retention rate of 85%, which equated to 387 participants. The study categorised patients as depressed or non-depressed, excluding patients with a Beck Depression Inventory (BDI; Beck, Steer & Carbin, 1988) score of 5-9. This could have impacted on overall representation of the ACS population. The study found depression severity at three month follow-up was independently predicted by rumination (specifically brooding). This research indicates that rumination may play a role in worsening depression in non-depressed and previously depressed patients following ACS. Given the emerging nature of the research, further prospective longitudinal research is needed to investigate if rumination is a predictive factor for depression in cardiac populations.

**CHD, depression and Quality of Life (QoL)**

QoL is significantly associated with depressive symptoms in cardiac patients (Ruo, Rumsfeld, Hlatky, Liu, Browner, & Whooley, 2003). Pragodpol and Ryan’s (2013) review of Health Related Quality of Life (HRQoL) in a CHD population, found HRQoL was predicted by socio-demographic, clinical and psychosocial factors, and concluded that depression was a negative predictor of HRQoL. Foxwell, Morley, and Frizelle (2013) found illness perceptions predicted QoL and mood across CHD. Dickens, Cherrington, and Mcgowan (2011) found that depression was associated with HRQoL in patients with CHD;
it was mediated by negative illness perceptions, cardiac anxiety, and awareness of somatic symptoms. The more patients with CHD thought about their illness and their symptoms the worst their HRQoL. Additionally, cardiac patient’s illness perception schemata was closely associated with depressive symptoms and QoL, irrelevant of socio-demographic background and diagnostic category (Le Grande et al., 2012). Cardiac patients’ recovery from surgery has also been found to be influenced by their pre-surgery illness beliefs (Juergens, Seekatz, Moosdorf, Petrie, & Rief, 2010). There are no known studies that have specifically investigated the role of RT on QoL or HRQoL in patients with CHD. Further research investigating this would need to control for depression given the association that has been identified.

To date, the direction of causality between rumination and depression in CHD has not been definitively established (Glassman, 2008). Further research is needed to specifically understand the mechanisms linking CHD and depression (Poole, Dickens, & Steptoe, 2011) and whether any specific risk factors or mechanisms such as rumination can be identified as increasing depression rates in CHD patients. Hence, this study will test the hypothesis that rumination is a mechanism that contributes to increased depression in CHD, by examining whether RT at baseline predicts increases in depression symptoms over time. This study aims to replicate the recent research by Denton et al. (2012) to see if the role of rumination in depression and in CHD can be more definitively established. It will extend Denton et al.’s (2012) study in the following ways: 1) by conducting it in the UK, 2) with a broader cardiac population which will include CHD, ACS and post-MI patients, 3) by including all patients irrelevant of their baseline depression score to reflect a more representative CHD population, and 4) to include both inpatients and
outpatients at a range of treatment stages. The study will aim to gather evidence for relevant models and theories (Brosschot et al., 2006; Carver & Scheier, 1990; Larsen & Christenfeld, 2009; Nolen-Hoeksema, 1991). Based on this hypothesis, we predicted that RT (rumination) at baseline will predict higher rates of depression at three month follow-up when controlling for depression and cardiac QoL at baseline.

In addition two secondary hypotheses were also investigated. A strong association between CHD, depression and QoL has been established (Ruo et al., 2003). Furthermore reviews have found illness perceptions predicted QoL and depression across CHD (Foxwell, Morley, & Frizelle, 2013), which suggests there are cognitive processes involved in the relationship between CHD, QoL, and depression. One hypothesis is that continued rumination about patients’ illness perceptions maintains or worsens depression and impacts on their perceived QoL. Previous research has found that RT, specifically counterfactual thinking, worsened QoL in health domains in women having breast implants (Parker, Middleton & Kulik, 2002) and negative thoughts have be found to moderate and partially mediate the influence of pain intensity on mental QoL in patients with haemophilia (Elander, Robinson, Mitchell & Morris, 2009). There is thus evidence that RT is potentially associated with QoL outcomes in health conditions. There are no known studies that have investigated whether over time rumination maintains or worsens QoL in patients with CHD. It would also be important to control for the effects of depression. Therefore, the second hypothesis of this study will test if rumination is a mechanism that contributes to worsened cardiac QoL in CHD. We predicted that RT (rumination) at baseline will predict higher rates of cardiac QoL at three month follow-up when controlling for depression and cardiac QoL at baseline.
In addition to measuring general QoL, it is also important to see if there is a prospective relationship between cardiac specific QoL and rumination over time. Although cardiac QoL may overlap with general QoL, it may also tap into more specific issues within this population. Additionally, cardiac QoL measures such as the Seattle Angina Questionnaire (SAQ) have been argued to be valid measures of cardiac symptoms (Spertus et al., 1995). Measurement of cardiac symptoms therefore tests whether rumination at baseline worsens both cardiac and depression outcomes over time, consistent with the predictions of theories which propose that RT causes and maintains both depression and cardiac symptoms in a CHD population (Brosschot et al, 2006; Larsen & Christenfeld, 2009). Therefore the final hypothesis will test if rumination is a mechanism that contributes to worsened QoL in CHD. We predicted that RT (rumination) at baseline will predict worse QoL at three month follow-up when controlling for depression, QoL, and cardiac QoL at baseline.

Method

Design

This was a prospective longitudinal study with a three month follow-up. This questionnaire based study aimed to examine the relationship between variables including: depression, RT, cardiac QoL and QoL measured by self-report questionnaires in a CHD population.

Participants and recruitment procedure

A total of 101 participants (77 men and 24 women) were recruited from Royal Devon and Exeter Hospital (RD&E), Cardiology Unit through cardiac admissions and the cardiac rehabilitation group. Inclusion criteria at recruitment was that participants were (i) aged between 18-85 years of age, modal age was
66-75 years old, (ii) patients with CHD (including post-MI or with a diagnosis of CHD or angina), with a diagnosis confirmed by a Consultant Cardiologist and Specialist Cardiac Nurse who signposted suitable patients, (iii) inpatients or outpatients at RD&E hospital presenting for cardiac care, (iv) were English native speakers, to maintain the validity of the questionnaires, and (v) had given informed consent to participate. Exclusion criteria included (i) neurological illness or head injuries, (ii) current psychosis or schizophrenia, or (iii) a learning difficulty. The exclusion criteria were assessed by the researcher during the initial contact and using information provided by the medical staff.

Potential participants were initially approached by cardiac staff whilst they were inpatients or at follow-up cardiac rehabilitation group. If they were willing to discuss potential participation they were signposted to the researcher. The researcher spent time with the participants to talk through the information sheet, consent forms, provide an opportunity to ask any questions and assess their suitability using the inclusion and exclusion criteria. Participants were offered twenty-four hours to consider their consent. If participants met the criteria and wanted to participate they were allocated a participant pack (see appendix B), consisting of the consent form, consent to contact sheet and study questionnaire in their preferred format (an emailed on-line link or paper copy with stamped addressed envelope). 101 participants completed the initial questionnaire (82 on paper; 19 via online survey using LimeSurvey, 2012).

Three months later participants completed an identical set of questionnaires to those that were completed at baseline, sent by their preferred method (email, post). If participants had not returned the questionnaire within two weeks they were contacted by the researcher and prompted to return it.
Eighty-five of the participants completed the three month follow-up questionnaire. Of the 16 non-responders, three had died during the three month follow up. The overall retention rate was 82.83%. Where items were omitted on the questionnaires, the researcher followed up to confirm and complete the forms, as appropriate.

**Ethical approval and considerations**

The study was approved by the National Health Service (NHS) Research Ethics Committee in Exeter (ref: 13/SW/0062), the Department of Psychology at the University of Exeter (ref: 2012/846) and the RD&E research and development department (ref: 1401879). See appendix C for further details.

All participants were provided with details of support organisations and contacts. Participants were also informed that if they were identified as having above moderate depression that their general practitioner (GP) would be informed. Where consent was obtained, letters outlining the study were sent to participants GPs.

**Measures**

All measures were consolidated into one questionnaire pack to improve usability. A demographic questionnaire asked about age range, gender, ethnicity, marital status, employment status, smoking status (former, current, never), alcohol consumption (units in past week), perceived social support, hours exercised, diet, BMI and health conditions. It also captured information pertaining to the CHD such as the duration of diagnosis, number of acute events, and the date of last acute event.
The Patient Health Questionnaire-(PHQ-9). The PHQ-9 (Kroenke, Spitzer, & Williams, 2001), is a widely used brief self-report nine item questionnaire, which measures severity of depression. Symptoms of depression are rated from “not at all” to “nearly every day”. Higher scores indicate more severe depression (range 0-27) with scores interpreted with respect to severity of depression: minimal (0-4), mild (5-9), moderate (10-14), moderately severe (15-19) or severe (20-27). It can be used as a diagnostic tool (Martin, Rief, Kraiberg, & Braehler, 2006; Spitzer, Kroenke, Williams, & The Patient Health Questionnaire Primary Care Study, 1999) or screening tool. The PHQ-9 is a valid and reliable brief measure of the severity for depression, with Cronbach’s alpha of 0.89 (Kroenke et al., 2001). In this study Cronbach’s alpha had a high reliability of 0.92. It has been found to be a superior measure to the Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) for detecting severe depression (Lowe, 2004). It is widely used in medical settings (Gilbody, Richards, Brealey, & Hewitt, 2007) and for screening depression in CHD patients (Lichtman et al., 2008) where it has also been found to be diagnostically superior (Haddad et al., 2013) to the HADS.

Rumination Response Scale of the Response Styles Questionnaire (RRS). The RRS (Treynor, Gonzalez, & Nolen-Hoeksema, 2003), is a twenty-two item measure which assesses depressive rumination. Participants rate the frequency that they use ruminative strategies from “almost never” to “almost always”, higher scores indicate higher levels of rumination (range 22-88). It contains two sub scales that specifically assess reflection and brooding. The RRS has been found to have good reliability with a Cronbach’s alpha of 0.89 (Nolen-Hoeksema & Morrow, 1991). In this study Cronbach’s alpha had a high reliability of 0.97. Good reliability has been replicated by later studies (Roelofs,
Muris, Huibers, Peeters, & Arntz, 2006). It has been used in previous cardiac research (Denton et al., 2012).

**Seattle Angina Questionnaire (SAQ).** The SAQ (Spertus et al., 1995), is a 19 item self-administered questionnaire. The SAQ measures health related quality of life and is specific to cardiac symptoms. Questions are grouped into five sub-categories which include physical limitation, anginal stability, anginal frequency, treatment satisfaction and disease perception and are scored by percentage (range 0-500). The SAQ has been found to be a valid measure of cardiac symptoms (Spertus et al., 1995). It has acceptable re-test reliability with Cronbach’s alpha ranging from 0.65 to 0.96 (Dougherty, Dewhurst, Nichol, & Spertus, 1998), in this study there was a Cronbach’s alpha of 0.66. The SAQ is specifically aimed at patients with angina; it was therefore explained to participants that if they do not have chest pains then they should relate the questions to their specific CHD symptoms.

**Health Related Quality of Life- Short Form-12 (SF12).** The SF-12 (Ware, Kosinski, & Keller, 1996) is a twelve item self-report health related quality of life measure. It measures eight concepts of health status: physical functioning, role limitations due to physical health problems, bodily pain, general health, vitality (energy/fatigue), social functioning, role limitations due to emotional problems, and mental health. The measure was scored using scoring software (Quality Metrics, V.4.5). The SF-12 has been found to be a valid and reliable measure (Globe, Levin, Chang, Mackenzie, & Azen, 2002). It has a Cronbach’s alpha ranging from 0.68 to 0.94 (Gandek et al., 1998), in this study a high Cronbach’s alpha of 0.88 was found. It is also a robust measure for use with CHD patients (Melville, Lari, Brown, Young, & Gray, 2003).
**New York Heart Association classification (NYHA).** The NYHA is used to classify heart failure in order to inform diagnosis and treatment (Swedberg et al., 2005). The NYHA is widely used and has been found to be a valid and reliable measure both clinically and for the purposes of research (Bennett, Riegel, Bittner, & Nichols, 2002). The NYHA has a Cronbach’s alpha of 0.94 (Moser & Riegel, 2000). The NYHA has four criterion: class I: no symptoms and limitation in ordinary activities; class II: mild symptoms and slight limitation in ordinary activities; class III: marked limitation in activities; class IV: severe limitations. This classification measure was the most widely used at the recruitment site and therefore the most available and reliable measure of cardiac severity for this sample.

**Results**

**Preliminary Analysis**

**Descriptive Statistics.** A summary of the sample demographic statistics are shown in Table 1. As is typical with a CHD population, there are more men than women and there are higher rates of participants with a BMI outside the healthy range (64-71%) in comparison to the general population (61.3%; Department of Health, 2010). There is also a diabetes prevalence rate of 19.9%, which is above the national average of 4.6% (Diabetes UK, 2013). Half of participants also stated that they had an additional health condition other than CHD. The modal age range was 66-75, with 51.5% of participants retired. Reflecting the demographics of the recruitment area (Exeter), 98% of participants identified themselves as White British.
Table 1.

Sample demographic characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Full sample recruited (N=101)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in years (range)</td>
<td>10 (36-45) 34 (66-75)</td>
</tr>
<tr>
<td></td>
<td>27 (56-65) 18 (76-85)</td>
</tr>
<tr>
<td>Gender (n)</td>
<td>77 (Male) 24 (Female)</td>
</tr>
<tr>
<td>Employment n (%)</td>
<td></td>
</tr>
<tr>
<td>Employed full time/part-time</td>
<td>33 (32.7)/ 7 (6.9)</td>
</tr>
<tr>
<td>Retired</td>
<td>52 (51.5)</td>
</tr>
<tr>
<td>Full-time homemaker or carer</td>
<td>3 (3.0)</td>
</tr>
<tr>
<td>Marital status n (%)</td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>73 (72.3)</td>
</tr>
<tr>
<td>Single</td>
<td>3 (3.0)</td>
</tr>
<tr>
<td>Co-habiting/Civil union</td>
<td>6 (5.9)/ 1 (1.0)</td>
</tr>
<tr>
<td>Widowed</td>
<td>8 (7.9)</td>
</tr>
<tr>
<td>Divorced</td>
<td>10 (9.9)</td>
</tr>
<tr>
<td>Ethnicity n (%)</td>
<td></td>
</tr>
<tr>
<td>White British</td>
<td>99 (98.0)</td>
</tr>
<tr>
<td>Other</td>
<td>1 (1.0)</td>
</tr>
<tr>
<td>Not stated</td>
<td>1 (1.0)</td>
</tr>
<tr>
<td>Smoking status n (%)</td>
<td></td>
</tr>
<tr>
<td>Never smoked</td>
<td>31 (30.1)</td>
</tr>
<tr>
<td>Former smoker</td>
<td>62 (61.4)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>8 (7.9)</td>
</tr>
<tr>
<td>Alcohol consumed n (%)</td>
<td></td>
</tr>
<tr>
<td>No/Yes</td>
<td>33 (32.7)/67 (66.3)</td>
</tr>
<tr>
<td>Exercise n (%)</td>
<td></td>
</tr>
<tr>
<td>No/Yes</td>
<td>18 (17.8)/82 (81.2)</td>
</tr>
<tr>
<td>Diabetes n (%)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>80 (79.2)</td>
</tr>
<tr>
<td>Type 1</td>
<td>5 (5.0)</td>
</tr>
<tr>
<td>Type 2</td>
<td>15 (14.9)</td>
</tr>
<tr>
<td>Missing data</td>
<td>1 (1.0)</td>
</tr>
<tr>
<td>Other health conditions n (%)</td>
<td></td>
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<tr>
<td>No</td>
<td>48 (47.5)</td>
</tr>
<tr>
<td>Yes</td>
<td>48 (47.5)</td>
</tr>
<tr>
<td>Missing data</td>
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</tr>
<tr>
<td>Body Mass Index (BMI) n (%)</td>
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<td>Healthy weight</td>
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<tr>
<td>Overweight</td>
<td>32 (32.1)</td>
</tr>
<tr>
<td>Obese</td>
<td>26 (26.0)</td>
</tr>
<tr>
<td>Severely obese/Morbidly obese</td>
<td>3 (3.0)/4 (4.0)</td>
</tr>
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<td>Missing data</td>
<td>7 (6.9)</td>
</tr>
<tr>
<td>Number of acute cardiac events n (%)</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>11 (10.9)</td>
</tr>
<tr>
<td>1</td>
<td>48 (47.5)</td>
</tr>
<tr>
<td>2</td>
<td>15 (14.9)</td>
</tr>
<tr>
<td>≥3</td>
<td>16 (15.9)</td>
</tr>
</tbody>
</table>
Mean, standard deviations (SD) and ranges for the key variables (PHQ, RRS, SAQ and SF12) at baseline and follow-up were reported (Table 2). Examining the scores, there is a reduction in rumination (RRS), depression (PHQ), and QoL (SAQ) scores from baseline to follow-up and an increase in cardiac QoL (SF-12). The largest portion (60%) of participants had minimal depression. However, 21% of participants received depression scores within the moderate to severe range, consistent with previous research which demonstrated post-MI 21% suffered with depression (Dickens et al., 2004).

Table 2. 
Summary of key variables ranges, means, and SD’s.

<table>
<thead>
<tr>
<th>Measure</th>
<th>N</th>
<th>Range</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
<th>SD</th>
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<tbody>
<tr>
<td></td>
<td>T1</td>
<td>T2</td>
<td>T1</td>
<td>T2</td>
<td>T1</td>
<td>T2</td>
</tr>
<tr>
<td>PHQ</td>
<td>101</td>
<td>84</td>
<td>27</td>
<td>26</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>27</td>
<td>26</td>
<td>6.70</td>
<td>5.11</td>
<td>4.15</td>
<td>6.70</td>
</tr>
<tr>
<td>RRS</td>
<td>101</td>
<td>62</td>
<td>56</td>
<td>44</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
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<td>84</td>
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<td>35.91</td>
<td>13.67</td>
<td>33.38</td>
<td>12.91</td>
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<tr>
<td>SAQ</td>
<td>100</td>
<td>79</td>
<td>408</td>
<td>377</td>
<td>87</td>
<td>123</td>
</tr>
<tr>
<td></td>
<td>495</td>
<td>500</td>
<td>335</td>
<td>367</td>
<td>88.51</td>
<td>73.46</td>
</tr>
<tr>
<td>SF12</td>
<td>97</td>
<td>81</td>
<td>675</td>
<td>663</td>
<td>25</td>
<td>37.5</td>
</tr>
<tr>
<td></td>
<td>700</td>
<td>700</td>
<td>445</td>
<td>445</td>
<td>168.02</td>
<td>167.68</td>
</tr>
</tbody>
</table>

Note. T1 (Baseline), T2 (Follow-up), Depression (PHQ), full rumination measure (RRS), Cardiac QoL (SAQ), QoL (SF-12).

Tests of normality and outliers. Boxplots were used to identify and explore outliers. Histograms were created for the continuous key variables and visually examined for skewness and kurtosis and converted to z-scores (standard residuals) as recommended by Field (2013). Some participants were significantly different from the normal distribution on measures of depression and rumination at baseline and follow-up (Appendix D1). Due to the nature of the hypotheses, it was important that the depression and rumination scores were truly representative of the sample. Therefore the data was left in its
original state and was not subject to any transformation. Additionally, the analyses of correlation and multiple regression are robust to deviations from normality so the data met this assumption adequately. Parametric assumptions of the regression were checked prior to the evaluation of the model as recommended by Field (2013).

**Preliminary analysis.** Pearson correlations between all variables were investigated in order to examine relationships and identify any confounding variables. Key variables measuring depression (PHQ), rumination (RRS), cardiac QoL (SAQ), QoL (SF12) baseline score were correlated with their corresponding follow-up score. All key variables were also correlated with each other at $p < .001$ level, see Table 3.

Previous research into depression and CHD has identified that age, gender, and social support can be confounding variables (Brosschot et al., 2006). Correlation matrix (Table 3 below) shows age was found to be significantly correlated with rumination (RRS), $r(99) = -.232$, $p < .05$ and time since diagnosis $r(99) = .227$, $p < .05$. Social support was significantly correlated with rumination (RRS), $r(96) = -.354$, $p < .001$. BMI was significantly correlated with age $r(93) = -.255$, $p < .05$, and depression (PHQ) $r(93) = .295$, $p < .005$. A one-way ANOVA was conducted to test the association of gender on rumination. There was a statistically significant difference, $F(1,99) = 5.158$, $p < .05$, finding that women ($Mean = 41.33, SD = 16.42$) had higher rumination scores than men ($Mean = 34.22, SD = 12.34$), consistent with the wider literature (Nolen-Hoeksema & Jackson, 2001). Age, gender, social support, and BMI are potential confounding variables and were therefore controlled for in the later regression analyses.
Table 3.

**Correlation matrix of variables**

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>BMI</th>
<th>TSD</th>
<th>SS</th>
<th>PHQ T1</th>
<th>PHQ T2</th>
<th>RRS T1</th>
<th>RRS T2</th>
<th>SF12 T1</th>
<th>SF12 T2</th>
<th>SAQ T1</th>
<th>SAQ T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.136</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
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<td>.184</td>
<td>1</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time since diagnosis</td>
<td>.227 *</td>
<td>.079</td>
<td>.033</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Social support</td>
<td>-.083</td>
<td>-.199</td>
<td>.049</td>
<td>-.057</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHQ score T1</td>
<td>-.144</td>
<td>.089</td>
<td>.295 **</td>
<td>.095</td>
<td>-.098</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PHQ score T2</td>
<td>-.201</td>
<td>.006</td>
<td>.434 **</td>
<td>.047</td>
<td>-.210</td>
<td>.727 **</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RRS T1</td>
<td>-.232</td>
<td>.223</td>
<td>.180</td>
<td>.050</td>
<td>-.354</td>
<td>.687 **</td>
<td>.729 **</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RRS T2</td>
<td>-.248</td>
<td>.075</td>
<td>.292 **</td>
<td>-.098</td>
<td>-.316</td>
<td>.698 **</td>
<td>.821 **</td>
<td>.865 **</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SF12 T1</td>
<td>-.056</td>
<td>-.139</td>
<td>-.185</td>
<td>.128</td>
<td>.135</td>
<td>-.597 **</td>
<td>-.469 **</td>
<td>-.510 **</td>
<td>-.432 **</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SF12 T2</td>
<td>-.111</td>
<td>-.019</td>
<td>-.283 **</td>
<td>-.229</td>
<td>.084</td>
<td>-.583 **</td>
<td>-.674 **</td>
<td>-.476 **</td>
<td>-.536 **</td>
<td>.756 **</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>SAQ T1</td>
<td>-.003</td>
<td>-.022</td>
<td>-.093</td>
<td>-.020</td>
<td>.100</td>
<td>-.487 **</td>
<td>-.341 **</td>
<td>-.362 **</td>
<td>-.297 **</td>
<td>.673 **</td>
<td>.552 **</td>
<td>1</td>
</tr>
<tr>
<td>SAQ T2</td>
<td>-.201</td>
<td>-.036</td>
<td>-.164</td>
<td>-.203</td>
<td>.091</td>
<td>-.367 **</td>
<td>-.323 **</td>
<td>-.316 **</td>
<td>.551 **</td>
<td>.679 **</td>
<td>.496 **</td>
<td>1</td>
</tr>
</tbody>
</table>

Note. Coding of items: Age (0= 18-25, 1= 26-35, 2= 36-45, 3= 46-55, 4= 56-65, 5= 66-75, 6= 76-85, 7= 85 and over), gender (0= male, 1= female), time since diagnosis (number of days since diagnosis), social support (0= none of the time, 1= a little of the time, 2= some of the time, 3= most of the time, 4= all of the time). * Correlation is significant at the 0.05 level (2-tailed), ** Correlation is significant at the 0.01 level (2-tailed), T1 (baseline), T2 (follow-up).

The follow-up questionnaire was completed by eighty-five participants. In the sixteen participants that did not complete the follow-up, three are known to have died and one participant’s health meant that they were too poor to complete the questionnaire. Further analyses (t-test, chi-square) tested if there were any differences at baseline between those participants who completed the follow-up versus those who did not. No significant differences were found.
between participants that completed follow-up and those that did not

$$X^2 (4) = 6.59, p > .05, t(99) = 3.14, p > .05.$$  

The data collected for the NYHA only allow patients to be categorised into four categories; within the current dataset all participants ended up in one of two categories, which meant this data was not appropriate to use in the analysis. The SAQ will be used as it has been argued to be a valid measure of cardiac symptoms (Spertus et al., 1995) and it provides more information than the few discrete categories that the NYHA provides. Therefore the SAQ is more likely to show if there is any variation in cardiac symptoms over time for this sample.

**Main analyses**

*Principal hypothesis: RT (rumination) at baseline will predict higher rates of depression at three month follow-up when controlling for depression and cardiac QoL at baseline.*

To test the principal hypothesis, a hierarchical multiple regression was conducted with depression (PHQ) at follow-up as the dependent variable. Baseline depression (PHQ) was entered at the first step in the regression equation; cardiac QoL and potential confounding variables of age, BMI, gender, and social support were added at step two, and then the independent variable of RT (RRS) was added at step three. The model met parametric assumptions as recommended by Field (2013).

The overall model was significant, $$F(7, 78) = 29.456, p < .001.$$  As expected, baseline depression significantly predicted depression three months later, accounting for 61% of the variance. Consistent with the hypothesis, rumination at baseline was a significant additional predictor of depression at
follow-up after controlling for baseline depression, age, gender, BMI, social support and cardiac QoL, accounting for an additional 8.3% of the variance, $F(1, 71) = 22.135, p<.001$, with a positive directional effect. Table 4 shows the individual predictors that contributed significantly to explain depression scores at three month follow-up.

**Table 4.**
Results of hierarchical multiple regression for the principal hypothesis

<table>
<thead>
<tr>
<th>Model</th>
<th>B</th>
<th>SE (B)</th>
<th>Beta</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Constant</td>
<td>.693</td>
<td>.479</td>
<td>.783***</td>
<td>11.031</td>
</tr>
<tr>
<td>Total PHQ (T1)</td>
<td>.671</td>
<td>.061</td>
<td>.479</td>
<td>11.031</td>
</tr>
<tr>
<td>2. Constant</td>
<td>-.402</td>
<td>3.634</td>
<td>-1.101</td>
<td>8.261</td>
</tr>
<tr>
<td>Total PHQ score</td>
<td>.599</td>
<td>.72</td>
<td>.699***</td>
<td>8.261</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>.001</td>
<td>.05</td>
<td>.013</td>
<td>.169</td>
</tr>
<tr>
<td>Age</td>
<td>.048</td>
<td>.327</td>
<td>.11</td>
<td>.146</td>
</tr>
<tr>
<td>Gender</td>
<td>-.248</td>
<td>.999</td>
<td>-.022</td>
<td>-.316</td>
</tr>
<tr>
<td>BMI</td>
<td>.234</td>
<td>.084</td>
<td>.213**</td>
<td>2.799</td>
</tr>
<tr>
<td>Support available</td>
<td>-.598</td>
<td>.339</td>
<td>-.122</td>
<td>-1.766</td>
</tr>
<tr>
<td>3. Constant</td>
<td>-.1096</td>
<td>3.515</td>
<td>-1.101</td>
<td>3.404</td>
</tr>
<tr>
<td>Total PHQ score</td>
<td>.304</td>
<td>.089</td>
<td>.355***</td>
<td>3.404</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>-.001</td>
<td>.004</td>
<td>-.011</td>
<td>-.158</td>
</tr>
<tr>
<td>Age</td>
<td>.215</td>
<td>.290</td>
<td>.48</td>
<td>.327</td>
</tr>
<tr>
<td>Gender</td>
<td>-.942</td>
<td>.803</td>
<td>-.073</td>
<td>-.173</td>
</tr>
<tr>
<td>BMI</td>
<td>.241</td>
<td>.073</td>
<td>.219**</td>
<td>3.277</td>
</tr>
<tr>
<td>Support available</td>
<td>-.228</td>
<td>.308</td>
<td>-.047</td>
<td>-.740</td>
</tr>
<tr>
<td>Total RRS (T1)</td>
<td>.202</td>
<td>.043</td>
<td>.460***</td>
<td>4.705</td>
</tr>
</tbody>
</table>

*Note.* Depression (PHQ), Cardiac QoL (SAQ), rumination measure (RRS) and body mass index (BMI). Adjusted $R^2 = .607$ for step 1, $R^2 = .636$ for step 2, $\Delta R^2 = .083$ for step 3 ($p < .001$). * $p < .05$, ** $p < .01$, *** $p < .001$.

The relationship between depression over time and rumination was examined in Figure 1. The scatterplot shows that as baseline rumination increases, there is a greater depression at follow-up, after controlling for baseline depression.
Previous research (Denton et al., 2012) had found that the subscales brooding and rumination of the RRS explained different variance in depression at three month follow-up. These subscales were also tested using separate hierarchical multiple regressions identical to above but entering the reflection or brooding subscales instead of the full RRS score.

For reflection, the overall model was significant, $F(7, 78) = 27.745$, $p < .001$. Depression at baseline significantly predicted depression three months later, accounting for 61% of the variance. Reflection at baseline was a significant additional predictor of depression at follow-up after controlling for baseline depression, age, gender, BMI, social support and cardiac QoL.
accounting for an additional 7.0% of the variance, $F(1, 71)= 18.110, p<.001$, with positive directional effect. Table 5 shows the individual predictors that contributed significantly to explain depression scores at three month follow-up.

Table 5.
Results of hierarchical multiple regression for the principal hypothesis for reflection

<table>
<thead>
<tr>
<th>Model</th>
<th>B</th>
<th>SE (B)</th>
<th>Beta</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Constant</td>
<td>693</td>
<td>.479</td>
<td>1.446</td>
<td></td>
</tr>
<tr>
<td>Total PHQ (T1)</td>
<td>.671</td>
<td>.061</td>
<td>.783***</td>
<td>11.031</td>
</tr>
<tr>
<td>2. Constant</td>
<td>-4.002</td>
<td>3.634</td>
<td>-1.101</td>
<td></td>
</tr>
<tr>
<td>Total PHQ score</td>
<td>.399</td>
<td>.072</td>
<td>.699***</td>
<td>8.261</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>.001</td>
<td>.005</td>
<td>.013</td>
<td>.169</td>
</tr>
<tr>
<td>Age</td>
<td>.048</td>
<td>.327</td>
<td>.011</td>
<td>.146</td>
</tr>
<tr>
<td>Gender</td>
<td>-.248</td>
<td>.899</td>
<td>-.022</td>
<td>-.316</td>
</tr>
<tr>
<td>BMI</td>
<td>.234</td>
<td>.084</td>
<td>.213**</td>
<td>2.799</td>
</tr>
<tr>
<td>Support available</td>
<td>-.598</td>
<td>.339</td>
<td>-.122</td>
<td>-1.766</td>
</tr>
<tr>
<td>Total PHQ score</td>
<td>.404</td>
<td>.080</td>
<td>.472***</td>
<td>5.083</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>.000</td>
<td>.004</td>
<td>-.003</td>
<td>-.046</td>
</tr>
<tr>
<td>Age</td>
<td>.308</td>
<td>.301</td>
<td>.069</td>
<td>1.024</td>
</tr>
<tr>
<td>Gender</td>
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<td>.852</td>
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<td>-1.679</td>
</tr>
<tr>
<td>BMI</td>
<td>.218</td>
<td>.075</td>
<td>.199**</td>
<td>2.900</td>
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<tr>
<td>Support available</td>
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<td>.307</td>
<td>-.086</td>
<td>-1.359</td>
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<tr>
<td>RRS-Reflection</td>
<td>.743</td>
<td>.175</td>
<td>.375***</td>
<td>4.256</td>
</tr>
</tbody>
</table>

Note. Depression (PHQ), cardiac QoL (SAQ), rumination reflection subscale (RRS-reflection) and body mass index (BMI). Adjusted $R^2 = .607$ for step 1, $R^2 = .636$ for step 2, $\Delta R^2 = .070$ for step 3 ($p < .001$). * $p < .05$, ** $p < .01$, *** $p < .001$

For the subscale of brooding the overall model was significant, $F(4, 78) = 24.298, p<.001$. Depression at baseline significantly predicted depression three months later, accounting for 61% of the variance. Brooding at baseline was a significant additional predictor of depression at follow-up after controlling for baseline depression, age, gender, BMI, social support and cardiac QoL, accounting for an additional 4.0% of the variance, $F(1, 71) = 10.004, p<.005$, with a positive directional effect. Table 6 shows the individual predictors that contributed significantly to explain depression scores at three month follow-up.
Table 6.

Hierarchical multiple regression for the principal hypothesis for brooding

<table>
<thead>
<tr>
<th>Model</th>
<th>β</th>
<th>SE (B)</th>
<th>Beta</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Constant</td>
<td>.693</td>
<td>.479</td>
<td>.479</td>
<td>1.446</td>
</tr>
<tr>
<td>Total PHQ (T1)</td>
<td>.671</td>
<td>.061</td>
<td>.783***</td>
<td>11.031</td>
</tr>
<tr>
<td>2. Constant</td>
<td>-4.002</td>
<td>3.634</td>
<td>-1.101</td>
<td></td>
</tr>
<tr>
<td>Total PHQ score</td>
<td>.599</td>
<td>.72</td>
<td>.699***</td>
<td>8.261</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>.001</td>
<td>.005</td>
<td>.013</td>
<td>.169</td>
</tr>
<tr>
<td>Age</td>
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<td>.146</td>
</tr>
<tr>
<td>Gender</td>
<td>-.248</td>
<td>.899</td>
<td>-.022</td>
<td>-.316</td>
</tr>
<tr>
<td>BMI</td>
<td>.234</td>
<td>.084</td>
<td>.213**</td>
<td>2.799</td>
</tr>
<tr>
<td>Social support</td>
<td>-.598</td>
<td>.339</td>
<td>-.122</td>
<td>-1.766</td>
</tr>
<tr>
<td>3. Constant</td>
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<td>-2.221</td>
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</tr>
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<td>Total PHQ score</td>
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<td>.085</td>
<td>.515***</td>
<td>5.218</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>-.001</td>
<td>.005</td>
<td>-.018</td>
<td>-.248</td>
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<tr>
<td>Age</td>
<td>.142</td>
<td>.310</td>
<td>.032</td>
<td>.458</td>
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<td>Gender</td>
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<td>.853</td>
<td>-.047</td>
<td>-.705</td>
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<td>BMI</td>
<td>.245</td>
<td>.079</td>
<td>.223**</td>
<td>3.103</td>
</tr>
<tr>
<td>Social support</td>
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<td>.333</td>
<td>-.060</td>
<td>-.885</td>
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<tr>
<td>RRS-Brooding</td>
<td>.501</td>
<td>.158</td>
<td>.281**</td>
<td>3.163</td>
</tr>
</tbody>
</table>

Note. Depression (PHQ), cardiac QoL (SAQ), rumination brooding subscale (RRS-Brooding) and body mass index (BMI). Adjusted $R^2$ = .607 for step 1, $R^2$ = .636 for step 2, $\Delta R^2$ = .04 for step 3 ($p < .005$). * $p < .05$, ** $p < .01$, *** $p < .001$

Hypothesis 2: RT (rumination) at baseline will predict higher rates of cardiac QoL three month follow-up when controlling for depression and cardiac QoL symptoms at baseline.

Hypothesis 2 was tested using a hierarchical multiple regression, with cardiac QoL (SAQ) at follow-up, the dependent factor in this model. Baseline cardiac QoL was entered in the first step of the regression equation, baseline depression (PHQ), and potential confounding variables of age, gender, BMI, and social support were added at step two, lastly, RT (RRS) was added at step three.

The overall model was significant, $F(7,73) = 6.619$, $p<.001$. As expected, baseline cardiac QoL significantly predicted cardiac QoL three months later, accounting for 28% of the variance. Contrary to the hypothesis, rumination at
baseline did not significantly predict any additional variance after controlling for baseline depression, age, gender, BMI and social support, $F(1, 66) = 1.485$, $p > .05$. Summary of the model can be seen in Table 7.

Table 7.  
*Results of hierarchical multiple regression for hypothesis 2*

<table>
<thead>
<tr>
<th>Model</th>
<th>B</th>
<th>SE (B)</th>
<th>Beta</th>
<th>$t$</th>
</tr>
</thead>
<tbody>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Constant</td>
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<td>33.491</td>
<td>5.710</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>.516</td>
<td>.095</td>
<td>.539***</td>
<td>5.436</td>
</tr>
<tr>
<td>2.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Constant</td>
<td>367.137</td>
<td>71.215</td>
<td>5.155</td>
</tr>
<tr>
<td>Total SAQ</td>
<td>.443</td>
<td>.106</td>
<td>.463***</td>
<td>4.182</td>
</tr>
<tr>
<td>Total PHQ score</td>
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<td>-.189</td>
<td>-1.596</td>
</tr>
<tr>
<td>Age</td>
<td>-19.449</td>
<td>6.429</td>
<td>-.304**</td>
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<td>.482***</td>
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<td>-.325**</td>
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<tr>
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<td>-.009</td>
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<td>-1.219</td>
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*Note.* Depression (PHQ), cardiac QoL (SAQ), rumination measure (RRS) and body mass index (BMI). Adjusted $R^2 = .281$ for step 1, $R^2 = .399$ for step 2, $\Delta R^2 = .005$ for step 3 ($p > .05$). * $p < .05$, ** $p < .01$, *** $p < .001$

**Hypothesis 3:** RT (rumination) at baseline will predict QoL at three month follow-up when controlling for depression, QoL, and cardiac QoL at baseline.

Hypothesis 3 was also tested using a hierarchical multiple regression, with QoL (SF12) at follow-up, the dependent factor in this model. Baseline QoL (SF12) was entered in the first step in the regression equation, baseline depression (PHQ), cardiac QoL at baseline and potential confounding variables of age, gender, BMI, and social support were added at step two, and RT (RRS) was added at step three. The model met parametric assumptions as previously described and in line with Field (2013).
The overall model was significant, $F(8, 75) = 18.375, p < .001$. As expected, baseline QoL significantly predicted QoL three months later, accounting for 58% of the variance. Contrary to the hypothesis, rumination at baseline did not significantly predict any additional variance after controlling for baseline depression, age, gender, BMI and social support, $F(1, 67) = 2.468, p > .05$. Summary of the model can be seen in Table 8.

Table 8.

*Results of hierarchical multiple regression for hypothesis 3*

<table>
<thead>
<tr>
<th>Model</th>
<th>B</th>
<th>SE (B)</th>
<th>Beta</th>
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<td>Total SF12</td>
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</table>

*Note.* QoL (SF-12), Depression (PHQ), cardiac QoL (SAQ), rumination measure (RRS) and body mass index (BMI). Adjusted $R^2 = .583$ for step 1, $R^2 = .642$ for step 2, $\Delta R^2 = .008$ for step 3 ($p < .005$). * $p < .05$, ** $p < .01$, *** $p < .001$
Discussion

The aim of this study was to test whether RT (rumination) prospectively predicts symptoms of depression over a three month period in a CHD population. As predicted, and consistent with previous comparable research (Denton et al., 2012), the results demonstrate that RT, specifically rumination, is a vulnerability factor for increased depression in CHD. Moreover, rumination at baseline is an independent and significant predictor of depression at a three month follow-up after controlling for baseline depression and other possible confounding factors. The primary hypothesis was supported as the data showed a positive relationship in which increasing baseline rumination is associated with greater depression at three month follow-up. This indicates that rumination may be a maintaining factor in severity of depression.

The study supports the notion that rumination has a predictive effect on depression (Ciesla & Roberts, 2007; Nolen-Hoeksema et al., 2008). Previous cross-sectional research has demonstrated a link between RT, depression and CHD (Dickens et al., 2012; Doyle et al., 2011a), which highlighted that RT could be a vulnerability factor for depression in CHD populations; the current study supports and extends this to a predictive prospective relationship.

The findings of this study and Denton et al.’s (2012) study provide convergent evidence that rumination is a risk factor for increased or maintained depression in cardiac patients. This extends the robust findings for rumination as a risk factor for depression in the general population (Nolen-Hoeksema, 2000; Stafford et al., 2009) to a CHD population. The study aimed to replicate and extend the recent study by Denton et al. (2012) of an ACS population,
which found rumination brooding independently predicted depression severity at three month follow-up, explaining 1.2% of the variance. The current study found that both the subscales of brooding and rumination independently predicted depression at three month-up follow up; 4% and 7% respectively. Additionally, when the full rumination measure (RRS) was applied it explained 8.3% of the total variance of depression at three month follow-up when controlling for baseline depression and confounding variables. These results are consistent with the previous longitudinal research. This study added to previous research (Denton et al., 2012) in its finding that both subscales of brooding and reflection predicted depression at three month follow-up, rather than just brooding as previously found, and with greater predictive variance than previously shown.

The differences between the findings in the two studies should be noted and could be accounted for by methodological and analytic differences. Firstly, the current study controlled for a number of possible confounding factors revealed in the preliminary analyses (gender, age, BMI, and social support) which differed from Denton et al. (2012). Secondly, different possible confounding variables were controlled for in the two studies: Denton et al. (2012) measured and controlled for psychosocial vulnerabilities, but did not control for QoL and Cardiac QoL, which was significantly correlated with depression and rumination in the current data. Some constructs were measured differently, including depression and cardiac severity, which may also account for some variance. The Denton et al. (2012) study excluded participants with a BDI of 5-9, whereas the current study included all participants irrelevant of depression score at baseline, which arguably provided a more representative CHD sample. The participant samples varied between
the two studies. The current study was conducted in the UK which has a different healthcare system to the US and could have influenced participants, cardiac health, QoL and psychological wellbeing. The current study also recruited from a wider diagnostic category of CHD opposed to a specific diagnosis (ACS), this could explain some of the variation in the findings. Moreover, the current study recruited both inpatients and outpatients at varying stages of treatment and time since CHD diagnosis. Although time since diagnosis was not a confounding factor, the flexibility in the recruitment process may have had an impact and delivered a more representative CHD sample. Lastly, there are differences between the current study and the Denton et al. (2012) findings for the rumination subscales (brooding and reflection). The current study showed that both sub-scales of rumination are associated with depression, whereas Denton et al. (2012) only found that brooding was associated. An example of a brooding thought that post-MI patients’ might have is ‘What have I done to deserve this?’ Brooding is the more pathological component of rumination so it is more likely to be found in the more depressed patients. However, in the current study reflection was also found to be predictive of depression over time. This is consistent with the wider literature, which finds that although brooding is more pathological, reflection can have pathological effects (Watkins, 2008). It is also possible that the current study’s findings are within the confidence interval given that Denton et al.’s (2012) study had a much larger sample; this would support replication of the study to support the findings.

The findings show that rumination plays an important role in predicting depression outcomes (Nolen-Hoeksema, 2000) and this study has extended this to a CHD population. It supports the Nolen-Hoeksema (1991) Response
Style Theory, which proposed that a continued rumination response prolongs depression.

The findings are not consistent with the Brosschot et al.’s (2006) model and it may be that more extensive and physiological assessment of cardiac health would have determined a relationship. Furthermore in order to support a hypothesis that RT prolongs a stress response which worsens cardiac health and can contribute to the onset of cardiac problems, participants would have needed to have been assessed before and after cardiac events, which was not possible in the current study. The current study does however show RT is a factor involved in depression in patients with CHD. Larsen and Christenfeld’s (2009) theory which extends Brosschot et al.’s (2006) model to include depression is partially supported by the findings. It has been shown that rumination extends and predicts depression in CHD and evidence has found rumination affects cardiac recovery (Verkuil et al., 2009). Further research with accurate measurement of CHD symptoms is required to investigate this hypothesis fully; however, this study goes some way in identifying that rumination could play a role in the relationship between CHD and depression.

The study also tested two secondary hypotheses, investigating if RT at baseline was predictive of QoL or cardiac QoL at three month follow-up when controlling for baseline QoL or cardiac QoL, baseline depression, and confounding factors. However, despite overall significant models, both secondary hypotheses were unsupported and rumination was not an independent predictor of QoL. Although QoL and HRQoL have been found to be associated with depression in cardiac populations (Ruo et al., 2003; Pragodpol and Ryan, 2013), the findings of this study are not consistent with the
hypothesis that rumination is predictive of general QoL or Cardiac QoL. Therefore these findings are not supportive of theories which propose that rumination maintains and prolongs cardiac symptoms (Larsen and Christenfeld, 2009; Brosschot et al.’s, 2006). However, this could reflect the relatively low power of this sample given the sample size, or that the measures of cardiac symptoms are not that sensitive. The findings of this study only support an association between rumination and depression in CHD patients.

The results of the current study have important clinical implications, in both medical and psychological settings. In a medical setting it supports the notion that depression screening in cardiac populations is necessary to identify the 21% of cardiac patients with above moderate levels of depression and raise practitioners’ awareness of this (Larsen, Vestergaard, Søndergaard, & Christensen, 2013). Identifying CHD patients with depression is important given the relationship between mortality in cardiac populations and depression (Barth et al., 2004; Dickens et al., 2008b). If additional research found rumination to be a key mechanism then this could also be screened for so that those with elevated longitudinal risk could be identified. Patients with high rumination scores would be at increased risk of depression; and beneficial psychological treatments could be rumination-focused cognitive-behavioural therapy (Watkins et al., 2011). Specific psychological interventions for patients with CHD are recommended (Dickens et al., 2013); tailored and preventive treatments could potentially reduce levels of depression and concurrently improve cardiac outcomes. Reducing the prevalence of depression in medical settings could be beneficial as patients with co-morbid major depression and a chronic illness have been found to cost between 50-100% more in primary care than non-
depressed patients, despite socioeconomic status and the medical condition being controlled for (Katon, 2011).

**Limitations and recommendations for future directions**

There are several limitations to this study. Firstly our assessment of depression, RT, QoL and cardiac QoL relied on self-report assessment. Ideally depression would have been assessed via clinical interview and psychometric assessment which would have provided a more reliable diagnosis and risk assessment. Additionally, for measurement of cardiac severity, rather than being categorised, it could have been assessed for via symptom checklists or biological measures. However, the study did not have the capacity to complete this with the sample, and it may have served to reduce the number of willing participants. Therefore, the PHQ-9 was chosen as a valid and reliable measure of the severity for depression (Kroenke et al., 2001). It is widely used in medical settings (Gilbody et al., 2007) and for screening depression in CHD patients (Lichtman et al., 2008). Cardiac severity was measured using the NYHA, a widely used categorisation; however, it resulted in the majority of participants being in one of two categories; a more precise measure would have been advantageous. Secondly, in order to provide a robust analysis a large sample was required and subsequently it consisted of a broad range of diagnoses within CHD, e.g. a heterogenous sample. Replication of the current research by dividing participants using their specific diagnosis (e.g. post-MI or ACS) could explore similarities and differences between this and previous comparable research (Denton et al, 2012).
Similarly to previous research in this area, 21% of participants were found to be suffering with above moderate depression (Dickens et al., 2004). The overall mean depression levels were mild and reduced to minimal at follow-up, and there was also a reduction in rumination. This is indicative of a reduction in depression and rumination with the passing of time.

To enable flexibility during recruitment, the participants recruited were both inpatients and outpatients which resulted in great variation of time since diagnosis. Time since diagnosis was not significantly correlated to any key variables. However, it could have influenced the level of rumination and depression participants experienced. It is thought that depression after a cardiac event is dependent on pre-depression level and the severity of the cardiac event (Dickens et al., 2005). Depression severity has also been found to be related to length of stay in hospital post-MI (Dickens, 2005). Therefore the relationship between depression and CHD is complicated and duration in hospital and pre-diagnosis depression could be factors that would ideally be controlled for. Participants recruited immediately post-MI could have increases in rumination because of the sudden life event, with rumination thought to be affected by more severe negative events (Moberly & Watkins, 2008). However, it could be argued that those recruited later, during cardiac rehabilitation, may have had more time to realise the impact of their cardiac condition and the reduced likelihood of attaining goals (Carver & Scheier, 1990), which may have increased rumination. Further qualitative research could investigate specific ruminative cognitions and patients’ experience at different treatment points to contextualise the relationship between rumination and depression in a cardiac population.
The time-constraints surrounding the project meant only one three month follow-up period was attainable. However, additional follow-up periods at six and twelve months could provide additional understanding of the longer term implications of rumination on depression in a CHD population. Additional longitudinal data would have the potential to better investigate the impact of rumination on cardiac symptoms e.g. through future cardiac event, hospital admission or deaths.

Participants’ historical information was not gathered so it is not known whether participants were receiving a psychological or medical intervention, if they were previously depressed or had a psychiatric history. This could impact on levels of depression and rumination and could not be controlled for. If this study was replicated it would be helpful to gather participants’ historical data at recruitment.

A gap in the literature is also whether rumination or depression can predict the onset of CHD or if they are a consequence of CHD. A larger cohort study that recruited participants’ pre-CHD could explore this in more detail. In line with best practice, a randomised controlled trial of psychological interventions for cardiac patients could be beneficial to develop an evidence-based treatment for depression in cardiac populations (Davidson, Rieckmann, & Lesperance, 2004).

Given the findings in this study show that the overall mean population had mild depression, the results can only be applied generally to patients with CHD rather than to those with clinical depression. There is limited comparable research, as such additional investigation is needed to support these findings. Additionally, further prospective longitudinal research that is more medically
focused could explore the effects of rumination on physiological changes, over-time, on mortality (Larsen & Christenfeld, 2009) and how this relates to depression.

**Conclusion**

In conclusion, the results of the study contribute to a growing body of literature exploring potential mechanisms that link CHD and depression together. To our knowledge this is the second study that has examined whether rumination is involved in the development of depressive symptoms in CHD patients using a prospectively longitudinal methodology, and the first of such studies to be conducted in the UK. The findings of this study support previous research that demonstrated the relationship between depression and RT, but the study has extended this finding to a CHD population. Due to the limited nature of research investigating RT in CHD and depression, the findings should be interpreted with caution but importantly should provide a foundation for further research.

What is still unknown is whether rumination not only has an impact on depression but also on cardiac outcomes by affecting physiological systems. Future research may be able to substantiate a hypothesis of rumination mediating both psychological as well as physiological outcomes.
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Appendices

A. Glossary of terms of RT

B. Participant pack
   1. Information sheet
   2. Consent form
   3. Consent to contact sheet
   4. Study questionnaire

C. Ethics documentation
   1. NHS ethical approval (agreed and substantive amendment)
   2. School ethics
   3. R&D approval

D. Extended data analysis
   1. Power analysis
   2. Extended data management
      i. Missing data
      ii. Tests of normality and outliers

E. Copy of instructions for authors for the Journal of Psychosomatic Research

F. Dissemination statement
Appendix A: Glossary of Terms

**Counterfactual thinking** - imagined mental representations of alternative versions of the past that would have improved the outcome now i.e. “if I had only …”

**Defensive pessimism** - this is when people set low expectations about the future and reflect on and rehearse possible situations to plan what might happen or go wrong and then how it could be prevented.

**Depressive rumination** - this is when people ruminate and think over their depressive symptoms.

**Emotional and cognitive processing** - this is when people actively thinking about a stressor, and thinking about it implications.

**Habitual negative self-thinking** - this is when people negative thinking has become like a habit and almost not conscious.

**Reflection** - this is chronic self-consciousness, it includes exploring unique, or alternative self-perception.

**Perseverative cognition** - it is thought of a key feature in worry and rumination. Brosschot (2006) model suggested repeated cognitions about a stress prolong the psychological problem or stressor and also prolong physiological responses. This prolonged stress response can then go onto to development disease i.e. cardiovascular.

**Planning, problem solving and mental stimulation** - this is using cognitive coping strategies, such as anticipatory coping, planning, rehearsal, and problem solving.

**Positive rumination** - this looks at how much somebody ruminates on positive emotions such as ‘happiness’.

**Post-event rumination** - people ruminate about past events, this could be what happened or what they did or said.

**Worry** - people think about the future and things they are uncertain about, people often try and problem solve future events such as catastrophes, risks and unseen events.

Adapted from Watkins et al. (2003).
Appendix B- Participant pack

A Prospective Longitudinal Study Repetitive Thought as a Vulnerability Factor for Depression in Coronary Heart Disease (CHD) Sufferers

Participant Information Sheet

I would like to invite you to take part in my research. My name is Laura Baker and I am a Trainee Clinical Psychologist with the University of Exeter. I am conducting research at the Royal Devon and Exeter Trust. My research hopes to understand the link between Coronary Heart Disease and depression.

Before deciding whether or not you would like to participate, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully.

Please feel free to contact me directly (contact details are given below) if there is anything that is not clear, or if you would like more information. It is important that you understand that you do not have to take part in the research and that if you do agree to participate you are free to withdraw at any time without having to give a reason.

Thank you for taking the time to read this.

Summary of the study

This study aims to look at the link between depression and Coronary Heart Disease. It is particularly interested in understanding if there are particular thinking patterns linking together depression and Coronary Heart Disease. If you agree to participate, you will be asked to fill out questionnaires which will take appropriately 20-30 minutes. You will then be asked to fill out the same questionnaires again 3 months later. The questionnaires will then either be posted to Rebecca Chawner (Cardiology Nurse Specialist) at the Royal Devon and Exeter Hospital with the provided stamped addressed envelope, or you will be able to complete them on-line. I am hoping to recruit 110 participants for my study. The study has been reviewed and passed by the National Research Ethics Service (NRES) Committee South West-Exeter and by the Royal Devon and Exeter Trust research and development department.

What is the purpose of the study?

We know that people with Coronary Heart Disease are more likely to suffer with depression. However it is not known if depression increases the likelihood of somebody suffering with Coronary Heart Disease or if suffering with Coronary Heart Disease increases people’s likelihood of suffering with depression. In order to understand this relationship, it is important to think about mechanisms that could be linking them together. This research investigates if cognitive mechanisms such as repetitive thoughts (rumination and worry) might be involved in this link. The study includes a 3 month follow-up as this will provide information about whether the way people with Coronary Heart Disease think has an impact on Coronary Heart Disease and depression over time.

Why have I been invited?

You have been chosen because you have a diagnosis of Coronary Heart Disease.

Do I have to take part?

It is completely up to you whether or not to take part. If you do decide to take part, please sign and return the ‘consent to contact’ sheet which is enclosed with this pack or contact me (with the details below). I will then contact you, to arrange sending you a questionnaire pack by your chosen method (post, email or on-line survey). If you decide you would like to take part, you are still free to withdraw at anytime without giving a reason.
If you are unsure about taking part you may find it helpful to discuss your decision with other people such as friends, family or your GP.

**What will happen to me if I take part?**

If you decide to take part in the study it will involve filling out one questionnaire pack when you join the study and filling out a second one 3 months later. Each pack will take approximately 20-30 minutes. We will agree your preferred method to send you the second questionnaire (on-line survey, email, post). If the questionnaire has not been returned after two weeks then I will contact you to offer a polite reminder. I will agree your preferred reminder method (phone, text, email, post) with you in advance. You will be given a copy of this information sheet and a signed copy of your consent form to keep. We will also inform your GP of your participation by letter. If I realise that you do not meet the criteria for the study then I will withdraw your participation.

In some cases, to gather all of the information that I am hoping to collect I may need to access medical records. I will ask for your consent to this and I have been given permission to access medical records by Royal Devon and Exeter Trust. If you wish for your records not to be accessed that is fine and you can still take part in the study.

**What do I have to do in order to take part?**

If you received this information sheet from your Cardiac Nurse and you would like to take part, please sign and return the ‘consent to contact’ sheet in the stamped addressed envelope. I will then contact you, to arrange sending you a questionnaire pack by your chosen method (post, email or on-line survey).

If you are reading this information already with the questionnaire pack then, please fill in the ‘consent to contact’ sheet and ‘participant consent’ sheet. Then please fill in the questionnaires enclosed in the pack and return them with the consent forms, in the stamped addressed envelope. You will be informed when I have received your questionnaires and arrange how to send your second pack 3 months later. It is important for you to be assured that all information will be treated confidentially and all data will be anonymous.

**What are the possible disadvantages and risks of taking part?**

The questionnaires measure depression, worry, and rumination. Some of the questions may be difficult and you may find them upsetting. Information about support services available are listed below.

**What are the possible benefits of taking part?**

It is hoped that the information gathered in this study may help to provide more information about the link between Coronary Heart Disease and depression, and that this will contribute to better screening, treatment and interventions for depression in patients with Coronary Heart Disease.

**What would happen if we were concerned about your safety?**

When you join the study a letter will be sent to your GP informing them of your participation and giving them a brief outline of the study. One of the questionnaires you will complete may show that you may be suffering from depression. If this were to happen, I would take appropriate steps to ensure your wellbeing. I would inform your cardiologist and GP, by letter or telephone call.

**What if there is a problem?**

Any complaint about the way you have been dealt with during the study will be addressed. Complaints should be in relation to adverse effects from the study and not about your treatment by the Cardiology Unit, which instead should be addressed with the Royal Devon and Exeter Trust. If you have a concern about any aspect of this study, you should ask to speak to the researchers who will do their best to answer your questions 07581 048672. If you remain unhappy and wish to complain formally, you can do this through the NHS Complaints Procedure. Details can be obtained from http://www.nhs.uk/choiceintheNHS/Rightsandpledges/complaints.

In the very unlikely event that something does go wrong and you are harmed during the research and this is due to someone’s negligence then you may have grounds for a legal action
for compensation against the University of Exeter, but you may have to pay your legal costs. The normal National Health Service complaints mechanisms will still be available to you.

**Will my taking part in this study be kept confidential?**

Data will be kept confidential. Your responses from the questionnaire will be anonymised and you will be assigned a number code. Personal information will be stored separately to the raw data. I will be the only person that has access to this and it will be securely stored in a password protected database. Personal data will not be kept once the study has been completed. It is only required so that I can contact you to collect the second set of data and disseminate the study’s results. Data will be securely stored in a locked filing cabinet and electronic data will be password protected. Anonymised raw data from the study may be stored for up to five years. If you decide you would like to withdraw from the study your data will be destroyed. Your GP will be informed of your participation in the study.

**What will happen to results of the study?**

I am hoping to publish the study in an academic journal; no identifiable information will be included. Results of the research will be sent to all participants.

**Who is organising and funding the research?**

I am a paid employee of the NHS. I am completing my doctorate in Clinical Psychology at the University of Exeter, which has been funded by the NHS. This study is being conducted as part of my academic qualification and I do not receive any additional funding or payment for carrying it out. My research is being supervised by Professor Edward Watkins and Professor Chris Dickens who are employed by the University of Exeter.

**Who has reviewed the study?**

All research in the NHS is looked at by an independent group of people called a Research Ethics Committee to protect your safety, rights, wellbeing and dignity. This study has been reviewed and given favourable opinion by NRES committee South West-Exeter.

**Further information and contact details**

If you would like any advice about participating in research you can contact Consumers for Ethics in Research (CERES), this is an organisation that offers information and advice on research in the NHS. CERES, PO Box 1365, London, N16 0BW Email: info@ceres.org.uk

Visit: http://www.ceres.org.uk/about.htm

Support services available:

- The Samaritans, website: [http://www.samaritans.org](http://www.samaritans.org), helpline: 08457 90 90 90
- British Heart Foundation, website: [www.bhf.org.uk](http://www.bhf.org.uk), heart helpline: 0300 330 3311
- NHS Choices website [www.bhf.org.uk](http://www.bhf.org.uk) provides information and guidance of heart disease

If you have any further questions please feel free to talk to Laura Baker, the study’s Chief Investigator. Address: The Cardiology Department, Royal Devon & Exeter NHS Foundation Trust, Barrack Road, Exeter, EX2 5DW.

Tel: 07581 048672  Email: chdresearchexeter@gmail.com
A Prospective Longitudinal Study Repetitive Thought as a Vulnerability Factor for Depression in Coronary Heart Disease (CHD) Sufferers

Consent Form

Name of Researcher: Laura Baker

Patient Identification Number for this trial:

Please initial all boxes

1. I confirm that I have read and understand the information sheet dated April 2013 (Version 2) for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason, without my medical treatment being affected.

3. I understand that relevant sections of my medical notes and data collected during the study may be looked at by individuals from the University of Exeter, from regulatory authorities or from the NHS Trust, where it is relevant to my taking part in this research. I give permission for these individuals to have access to my records.

4. I understand that my personal details will be kept secure place and any information that is entered onto a computer will be password protected. There will be no identifiable details used in the research write-up and/or publication.

5. I agree to my GP being informed of my participation in the study.

6. I agree that the researchers may contact me to remind me to return my questionnaires after two weeks if they have not been received. The research will contact me by an agreed method (phone, text, email, post).

7. I agree to take part in the above study.

_________________________  ________________________  ________________________
Name of Participant      Date                   Signature

_________________________  ________________________  ________________________
Name of Person           Date                   Signature
A Prospective Longitudinal Study Repetitive Thought as a Vulnerability Factor for Depression in Coronary Heart Disease (CHD) Sufferers

Consent to Contact Sheet

1. I have read and understand the information sheet provided.
2. I consent to be contacted by Laura Baker to discuss the possibility of taking part in the above research.
3. I understand I can contact Laura Baker to discuss any aspect of the research before taking part and I am not committed to take part until I decide to.
4. I am aware that I have the right to withdraw from the study at any time.
5. I understand that participation in the study will not affect my cardiac treatment or any other treatment I receive in anyway.

Name: ...................................................................................

Contact details:
Address: ...................................................................................
..............................................................................................
..............................................................................................
..............................................................................................

Telephone no: .................................................................
Mobile no: .................................................................
Email: ........................................................................

Preferred contact type (telephone, text, email, post):
........................................................................

Signature: ........................................................................
Date: ........................................................................
A Prospective Longitudinal Study Repetitive Thought as a Vulnerability Factor for Depression in Coronary Heart Disease (CHD) Sufferers

Participant Questionnaire

Please complete as many of the questions below as you feel comfortable to do so, please tick where appropriate. This information will be kept anonymous and confidential.

Name ..........................................................

Address ........................................................................................................................................
........................................................................................................................................

Contact Number ..............................................

GP Surgery...................................................... GP Name
..........................................................................................

Age: 18-25 □ 26-35 □ 36-45 □ 46-55 □ 56-65 □ 66-75 □ 76-85 □ 85 and over □

Sex : Male □ Female □

Employment status:

<table>
<thead>
<tr>
<th>Employment status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Employed full-time (30 hours or more per week)</td>
</tr>
<tr>
<td>Employed Part-time</td>
</tr>
<tr>
<td>Unemployed</td>
</tr>
<tr>
<td>Full-time student</td>
</tr>
<tr>
<td>Retired</td>
</tr>
<tr>
<td>Full-time homemaker or carer</td>
</tr>
</tbody>
</table>
Ethnicity:

<table>
<thead>
<tr>
<th>White</th>
<th>Mixed</th>
</tr>
</thead>
<tbody>
<tr>
<td>British</td>
<td>White and Black Caribbean</td>
</tr>
<tr>
<td>Irish</td>
<td>White and Black African</td>
</tr>
<tr>
<td>Any other White background</td>
<td>White and Asian</td>
</tr>
<tr>
<td></td>
<td>Any other mixed background</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Asian or Asian British</th>
<th>Black or British Black</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indian</td>
<td>Caribbean</td>
</tr>
<tr>
<td>Pakistani</td>
<td>African</td>
</tr>
<tr>
<td>Bangladeshi</td>
<td>Any other Black background</td>
</tr>
<tr>
<td>Any other Asian background</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Other ethnic groups</th>
<th>Not stated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chinese</td>
<td></td>
</tr>
<tr>
<td>Any other ethnic group</td>
<td></td>
</tr>
</tbody>
</table>

Marital status:

- Married
- Single
- Co-habiting
- Civil union
- Widowed
- Divorced

Smoking status:

- Never smoked
- Former smoker
- Current smoker * 

*If you are a current smoker, please state how many cigarettes on average you smoke per week
### Alcohol consumption:

| Do you drink alcohol? | Yes □  No □ | If Yes how many units did you drink in the past week? ........
|----------------------|-------------|-------------------------------------------------------------
|                      |             | (see guide below)                                            |
|                      |             | Pint of large/beer/cider – 3 units                           |
|                      |             | Small glass of wine (125ml)- 1.5                             |
|                      |             | Bottle of wine (750ml)- 10 units                             |
|                      |             | Single sprit mixer- 1 unit                                   |

### Exercise:

| Do you exercise? | Yes □  No □ | If Yes, please state how many hours for each type of exercise you completed in the last week?
|------------------|-------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------
|                  |             | Mild Exercise (e.g. walking) ......                                                                                                                                                                                                              |
|                  |             | Moderate Exercise (e.g. fast walking, cycling, swimming, playing tennis) ......                                                                                                                                                                |
|                  |             | Vigorous Exercise (e.g. running/jogging, fast swimming, aerobics) ......                                                                                                                                                                         |

### How many fruit and vegetables to you consume each day .......

### Do you suffer from any other health conditions?

<table>
<thead>
<tr>
<th>Do you suffer from any other health conditions?</th>
<th>Yes □  No □</th>
</tr>
</thead>
<tbody>
<tr>
<td>If Yes please state which conditions.............</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Do you suffer from diabetes:

<table>
<thead>
<tr>
<th>Do you suffer from diabetes:</th>
<th>Please Tick one box</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Yes- Type 1 Diabetes</td>
<td></td>
</tr>
<tr>
<td>Yes- Type 2 Diabetes</td>
<td></td>
</tr>
</tbody>
</table>
What is your weight? Stone….. Pounds….. or ……. KG

What is your height? Feet……. Inches…… or Meters …….Centimetres..

When were you diagnosed with Coronary Heart Disease?  __/__/__

How many acute cardiac events have you had? ……..

When was your last acute cardiac event? ……. 

Social Support

| Are you currently married or living with a partner? | Yes ☐   No ☐ |

Is there someone available to whom you can count on to listen to you when you need to talk?: (please tick one below)

- None of the time ☐
- A little of the time ☐
- Some of the time ☐
- Most of the time ☐
- All of the time ☐
PHQ9
Over the **last 2 weeks**, how often have you been bothered by any of the following problems?

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>Several days</th>
<th>More than half</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Little interest or pleasure in doing things</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Feeling down, depressed, or hopeless</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Trouble falling asleep, staying asleep, OR sleeping too much</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Feeling tired or having little energy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Poor appetite OR overeating</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Feeling bad about yourself OR feeling that you are a failure OR have let yourself or your family down</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Trouble concentrating on things such as reading a newspaper or watching television</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Moving or speaking so slowly that other people could have noticed OR the opposite, being so fidgety or restless that you have been moving around a lot more than usual</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Thoughts that you would be better off dead or thoughts of hurting yourself in some way</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**PSWQ**
Rate each of the following statements on a scale of 1 (‘not at all typical of me’) to 5 (‘very typical of me’). Please do not leave any items blank.

<p>| | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>If I do not have enough time to do everything, I do not worry about it</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>My worries overwhelm me</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>I do not tend to worry about things</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Many situations make me worry</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>I know I should not worry about things, but I just cannot help it</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>When I am under pressure I worry a lot</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>I am always worrying about something</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>I find it easy to dismiss worrisome thoughts</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>As soon as I finish one task, I start to worry about everything else I have to do</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>I never worry about anything</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>When there is nothing more I can do about a concern, I do not worry about it anymore</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>I have been a worrier all my life</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>I notice that I have been worrying about things</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Once I start worrying, I cannot stop</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>I worry all the time</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>I worry about projects until they are done</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>
### Responses to Depression (RSQ)

People think and do many different things when they feel down, sad or depressed. Please read each of the items below and indicate whether you never, sometimes, often, or always think or do each one when you feel down, sad or depressed. Please indicate what you *generally* do, not what you think you should do.

<table>
<thead>
<tr>
<th>Almost Never</th>
<th>Sometimes</th>
<th>Often</th>
<th>Almost Always</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tbody>
</table>

1. Think about how alone you feel.
2. Think “I won’t be able to do my job/work because I feel so bad”
3. Think about your feelings of fatigue and achiness
4. Think about how hard it is to concentrate
5. Think about how passive and unmotivated you feel
6. Analyse recent events to try and understand why you are depressed.
7. Think about how you don’t seem to feel anything anymore
8. Think “Why can’t I get going?”
9. Think “Why do I always react this way?”
10. Go away by yourself and think about why you feel this way
11. Write down what you are thinking about and analyse it
12. Think about a recent situation, wishing it would have gone better
13. Think “Why do I have problems other people don’t have?”
14. Think about how sad you feel
<table>
<thead>
<tr>
<th></th>
<th>Almost</th>
<th>Never</th>
<th>Sometimes</th>
<th>Often</th>
<th>Almost</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>15.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Think about all your shortcomings, failings, faults and mistakes</td>
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<tr>
<td>16.</td>
<td></td>
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<tr>
<td>Think about how you don't feel up to doing anything</td>
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<td>17.</td>
<td></td>
<td></td>
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<tr>
<td>Analyse your personality to try and understand why you are depressed</td>
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<tr>
<td>18.</td>
<td></td>
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<tr>
<td>Go someplace alone to think about your feelings</td>
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<td>19.</td>
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<tr>
<td>Think about how angry you are with yourself</td>
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<td>20.</td>
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<tr>
<td>Listen to sad music</td>
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<td>21.</td>
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<tr>
<td>Isolate yourself and think about the reasons why you feel sad</td>
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<td>22.</td>
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<tr>
<td>Try to understand yourself by focusing on your depressed mood</td>
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<tr>
<td>23.</td>
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<tr>
<td>Think “What am I doing to deserve this?”</td>
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<tr>
<td>24.</td>
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<tr>
<td>Think “I won’t be able to concentrate if I keep feeling this way”</td>
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<td>25.</td>
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<tr>
<td>Think “Why can’t I handle things better?”</td>
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</tr>
</tbody>
</table>
The Seattle Angina Questionnaire

1. 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 over the past 4 weeks. If you do not suffer from angina then please relate the responses to your specific problems.

<table>
<thead>
<tr>
<th>Activity</th>
<th>Extremely Limited</th>
<th>Quite a bit Limited</th>
<th>Moderately Limited</th>
<th>Slightly Limited</th>
<th>Not at all Limited</th>
<th>Limited for other reasons or did not do the activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dressing yourself</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Walking indoors on level ground</td>
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<tr>
<td>Showering</td>
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<tr>
<td>Climbing a hill or a flight of stairs without stopping</td>
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<tr>
<td>Gardening, vacuuming, or carrying groceries</td>
<td></td>
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<tr>
<td>Walking more than a block at a brisk pace</td>
<td></td>
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<tr>
<td>Running or jogging</td>
<td></td>
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<tr>
<td>Lifting or moving heavy objects (e.g. furniture, children)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Participating in strenuous sports (e.g. swimming, tennis)</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. Compared with 4 weeks ago, how often do you have chest pain, chest tightness, or angina when doing your most strenuous activities?

I have had chest pain, chest tightness, or angina...

<table>
<thead>
<tr>
<th>Much more often</th>
<th>Slightly more often</th>
<th>About the same</th>
<th>Slightly less often</th>
<th>Much less often</th>
<th>I have had no chest pain over the last 4 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3. Over the past 4 weeks, on average, how many times have you had chest pain, chest tightness, or angina?

I have had 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

4. Over the past 4 weeks, on average, how many times have you had to take nitroglycerin (nitroglycerin tablets or spray) for your chest pain, chest tightness, or angina?

I have taken nitroglycerin...

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

5. How bothersome is it for you to take your pills for chest pain, chest tightness, or angina as prescribed?

Extremely bothersome Quite a bit bothersome Moderately bothersome Slightly bothersome Not bothersome at all My doctor has not prescribed pills

6. How satisfied are you that everything possible is being done to treat your chest pain, chest tightness, or angina?

Not satisfied at all Mostly dissatisfied Somewhat satisfied Mostly satisfied Completely satisfied

7. How satisfied are you with the explanations your doctor has given you about your chest pain, chest tightness, or angina?

Not satisfied at all Mostly dissatisfied Somewhat satisfied Mostly satisfied Completely satisfied

8. Overall, how satisfied are you with the current treatment of your chest pain, chest tightness, or angina?

Not satisfied at all Mostly dissatisfied Somewhat satisfied Mostly satisfied Completely satisfied
9. Over the **past 4 weeks**, how much has your *chest pain, chest tightness, or angina* limited your enjoyment of life?

<table>
<thead>
<tr>
<th>Extreme limitation</th>
<th>Quite a bit limited</th>
<th>Moderately limited</th>
<th>Slightly limited</th>
<th>Not limited at all</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all</td>
<td>Slightly</td>
<td>Moderately</td>
<td>Quite a bit</td>
<td>Extremely</td>
</tr>
</tbody>
</table>

10. If you had to spend the rest of your life with your *chest pain, chest tightness, or angina* the way it is right now, how would you feel about this?

<table>
<thead>
<tr>
<th>Not satisfied at all</th>
<th>Mostly dissatisfied</th>
<th>Somewhat satisfied</th>
<th>Mostly satisfied</th>
<th>Completely satisfied</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all</td>
<td>Slightly</td>
<td>Moderately</td>
<td>Quite a bit</td>
<td>Extremely</td>
</tr>
</tbody>
</table>

11. How often do you think or worry that you may have a heart attack or die suddenly?

<table>
<thead>
<tr>
<th>Can't stop thinking or worrying</th>
<th>Often think or worry</th>
<th>Occasionally think or worry</th>
<th>Rarely think or worry</th>
<th>Never think or worry</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not at all</td>
<td>Slightly</td>
<td>Moderately</td>
<td>Quite a bit</td>
<td>Extremely</td>
</tr>
</tbody>
</table>
SF-12

This survey asks for your views about your health. This information will help you keep track of how you feel and how well you are able to do your usual activities. Thank you for completing this survey!

For each of the following questions, please tick the circle that best describes your answer.

1) In general, would you say your health is:

- Excellent  [ ]
- Very good  [ ]
- Good  [ ]
- Fair  [ ]
- Poor  [ ]

2) The following questions are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?

- Yes, limited a lot  [ ]
- Yes, limited a little  [ ]
- No, not limited at all  [ ]

a. Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf  [ ]

b. Climbing several flights of stairs  [ ]

3) During the past 4 weeks, how much of the time have you had any of the following problems with your work or other regular daily activities as a result of your physical health?

- All of the time  [ ]
- Most of the time  [ ]
- Some of the time  [ ]
- A little of the time  [ ]
- None of the time  [ ]

a. Accomplished less than you would like  [ ]

b. Were limited in the kind of work or other activities  [ ]

4) During the past 4 weeks, how much of the time 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)?

- All of the time  [ ]
- Most of the time  [ ]
- Some of the time  [ ]
- A little of the time  [ ]
- None of the time  [ ]

a. Accomplished less than you would like  [ ]

b. Did work or activities less carefully than usual  [ ]

5) During the past 4 weeks, 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 during
During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting friends, relatives, etc.)? For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the past 4 weeks...

<table>
<thead>
<tr>
<th>Question</th>
<th>All of the time</th>
<th>Most of the time</th>
<th>Some of the time</th>
<th>A little of the time</th>
<th>None of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you felt calm and peaceful?</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Did you have a lot of energy?</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Have you felt downhearted and depressed?</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

7) During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting friends, relatives, etc.)?

<table>
<thead>
<tr>
<th>Question</th>
<th>All of the time</th>
<th>Most of the time</th>
<th>Some of the time</th>
<th>A little of the time</th>
<th>None of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Have you felt calm and peaceful?</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Did you have a lot of energy?</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Have you felt downhearted and depressed?</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

Thank you for filling in this questionnaire
Appendix C- Ethics documentation

Health Research Authority

NRES Committee South West - Exeter
Bristol Research Ethics Committee Centre
Whitefriars
Level 3
Block B
Lewins Mead
Bristol
BS1 2NT
Tel: 0117 342 1332
Fax: 0117 342 0445

26 September 2013

Miss Laura Baker
Doctorate in Clinical Psychology
University of Exeter
Flat 4, 60 Oakfield Road
Clifton
Bristol
BS8 2BG

Dear Miss Baker

Study title: A PROSPECTIVE LONGITUDINAL STUDY OF REPETITIVE THOUGHT AS A VULNERABILITY FACTOR FOR DEPRESSION IN CORONARY HEART DISEASE (CHD) SUFFERERS

REC reference: 13/SW/0062
Amendment number: 1
Amendment date: 23 August 2013
IRAS project ID: 114459

The above amendment was reviewed on 09 September 2013 by the Sub-Committee in correspondence.

Ethical opinion

The members of the Committee taking part in the review gave a favourable ethical opinion of the amendment on the basis described in the notice of amendment form and supporting documentation.

Approved documents

The documents reviewed and approved at the meeting were:

<table>
<thead>
<tr>
<th>Document</th>
<th>Version</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Notice of Substantial Amendment (non-CTIMPs)</td>
<td>3.5</td>
<td>23 August 2013</td>
</tr>
<tr>
<td>Covering Letter</td>
<td></td>
<td>23 August 2013</td>
</tr>
<tr>
<td>GP letter - 1</td>
<td>4</td>
<td>23 August 2013</td>
</tr>
</tbody>
</table>
Membership of the Committee

The members of the Committee who took part in the review are listed on the attached sheet.

R&D approval

All investigators and research collaborators in the NHS should notify the R&D office for the relevant NHS care organisation of this amendment and check whether it affects R&D approval of the research.

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

We are pleased to welcome researchers and R & D staff at our NRES committee members' training days – see details at http://www.hra.nhs.uk/hra-training/

13/SW/0062: Please quote this number on all correspondence

Yours sincerely

[Signature]

p.p. Dr Denise Sheehan
Chair

Enclosures: List of names and professions of members who took part in the review

Copy to: Ms Lynda Garcia, Royal Devon & Exeter NHS Foundation Trust (l.garcia@nhs.net)
Ms Gill Seymour (g.m.seymour@exeter.ac.uk)
Royal Devon and Exeter
NHS Foundation Trust

Miss Laura Baker
Doctorate in Clinical Psychology
University of Exeter
Flat 4, 60 Oakfield Road
Clifton
Bristol
BS8 2BG

6 June 2013

Dear Laura

Study Title: A prospective longitudinal study of repetitive thought as a vulnerability factor for depression in coronary heart disease sufferers.
R&D Number: 1401879
MREC Ref: 13/SW/0062

I have reviewed the Trust R&D file for the above named study, which has received approval from the appropriate regulatory bodies, and I am happy to give approval on behalf of the Royal Devon & Exeter NHS Foundation Trust (RD&E).

The documents approved for use in this study are those approved by ethics, these are detailed on a separate sheet.

As named Investigator for this research that is being undertaken at the RD&E, it is your responsibility to manage and conduct this study in accordance with:

- The requirements of the Research Governance Framework for Health and Social Care (2006) and Medicines for Human Use (Clinical Trials) Regulations 2004 (if applicable).
- ICH-GCP (Good Clinical Practice) – It is mandatory for those staff who will be consenting participants into this study to have undertaken GCP and to ensure it is updated every 2 years.
- The Data Protection Act 1998 which details the eight principles of 'good information handling'.
- R&D Standard Operating Procedures (SOPs) and Trust policies which are available on the Trust intranet site.

As Lead Investigator for this research, you are required to ensure study specific duties are appropriately delegated and clearly documented on the study Delegation Log. This guarantees clarity of roles and must be signed and dated by each individual on the study and yourself as Lead Investigator.

Safety Reporting
Guidance on the classification of Adverse Events/Reactions (AEs/ARs) / Serious Adverse Events/Reactions (SAEs/SARs) and Suspected Unexpected Serious Adverse Reactions (SUSARs) and the requirements for reporting to the sponsor can be found in the study protocol. For RD&E sponsored studies this is also detailed in the sponsorship letter. All safety events that involve RD&E

R&D Trust Approval Letter (excluding No Ethics and Tissue Bank)
V1.1 09/05/2013

Chairman: James Brent   Chief Executive: Angela Pedder
To: Laura Baker  
From: Cris Burgess  
CC: Ed Watkins & Chris Dickens  
Re: Application 2012/546 to Ethics Committee  
Date: 11 August 2014

The School of Psychology Ethics Committee met on recently and your proposal was discussed. The Committee raised a number of conditions of agreement to this application being accepted. You would be expected to address these before beginning the research but sight of the evidence is not required by the Committee and the project has been approved in principle for the duration of your study.

The conditions are as follows:

- Management approval is sought and granted for each NHS/non-NHS host organisation involved in the research.

In any correspondence with the Ethics Committee about this application, please quote the reference number above or decisions may be delayed.

Yours sincerely,

Cris Burgess  
Chair of School Ethics Committee
Appendix D- Extended Results

**Power Analysis.** A priori power calculation was determined using G* Power 3.1 software (Faul, Erdfelder, Lang, & Buchner, 2007). The regression calculation was based on the assumption that up to four variables may need to be controlled for following the correlation. It allowed for one tested predictor and up to eight total predictors, for 95% power, with a medium effect size of 0.15, alpha of 0.05, 89 participants were required. In a comparable study, Denton (2012), they had 387 participants with significance <0.001. The crosslagged correlation between rumination and depression was 0.49, indicating a large effect size. On this basis, the current study is suitably powered.

**Extended Data Management.**

*Missing data.* In order to utilise all participants data, participants were still included if they had missing data on a specific measure but they would not be included in any analysis which required that measure. If participants measures could not be scored due to missing data the entry was set as missing data. Descriptive data of all variables was examined to ensure all data was within expected ranges, *means* and *SD*.

*Tests of normality and outliers.* Boxplots were used to identify and explore outliers. Histograms were created for the continuous key variables and visually examined for skewness and kurtosis. Skewness and kurtosis were also calculated and converted to z-scores (standard residuals) as recommended by Field (2013) to ensure 99% of z scores were within the range of -3.29 and +3.29. Z scores outside of this range indicated participants' variables were
significantly different from normal distribution. This was the case for measures of baseline and follow-up depression and rumination (see below).

Figure D 1. Box-plot to show outliers on measure of depression at baseline.
Figure D 2. Box-plot to show outliers on measure of depression at follow-up.

Figure D 3. Box-plot to show outliers on measure of rumination at baseline.

Figure D 4. Box-plot to show outliers on measure of rumination at follow-up.
Parametric assumptions of the regression were checked prior to evaluation of the model as suggested by Field (2013). Multicollinearity was checked by looking at the correlation table to ensure that predictor variables did not correlate above .90 and checking variance inflation factor (VIF) was close to one. Durbin-Watson was checked to examine independence of the residuals and ensuring values were between one and three.

Histograms and P-Plots were used to examine normality or the residuals, with no major deviations apparent. Mahalanobis distance measures the extent to which cases are multivariate outliers, and should not exceed the critical value for the number of predictors in each analysis (Barnett, 1978); this assumption was met with all data. Casewise diagnostics showed only 1% of cases were identified, which is below what would be expected for this model. Cooks distance measures the overall influence of one case on the model as this was below one in all cases, it suggests the model is predictive despite any outliers.
Appendix E- Instructions for authors

Before You Begin

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Appendix F: Dissemination statement

The results of this study will be disseminated to interested parties through feedback, journal publication and presentation.

**Dissemination to participants and NHS services.**

As stated on the participant information sheet participants will be informed of the results of the study. Participants will be provided with details of who to contact, should they require further information. Additionally the cardiac staff involved in the research will be provided with a summary of the findings. The NHS research ethics committee at Exeter and RD&E Research and Development team will be sent a summary of the findings of the study and will be informed that the study is now complete.

**Journal Publication**

It is expected that the study will be submitted for publication with the Journal of Psychosomatic Research (Impact factor 3.37).

**Presentation**

On 9th June 2014, my research findings were presented to an academic audience, for peer review, as part of the Doctorate in Clinical Psychology at the University of Exeter.

The findings will also be orally presented to the Lifetime service in Bath, a specialist paediatric service which work closely with children and young people with complex cardiac conditions.