EXPLORING THE UTILITY OF THE METACOGNITIVE MODEL IN PREDICTING
AND PREVENTING EMOTIONAL DISTRESS AFTER CANCER

Thesis submitted in accordance with the requirements of the University of Liverpool
for the degree of Doctor of Philosophy

March 2015
Abstract

Why some people recover emotionally after diagnosis and treatment of cancer, while others experience persistent or recurrent symptoms of emotional distress is not well understood. The literature exploring predictors of persistent distress after cancer has not been able to explain this. In addition, predominant theoretical perspectives (based on the cognitive paradigm) have fallen short of being able to explain causal mechanisms that underlie the maintenance of distress. A more promising perspective is offered by the metacognitive model of emotional disorder. This model implicates beliefs about thinking (metacognitive beliefs) that drive a repetitive and problematic thinking style (the Cognitive Attentional Syndrome; CAS), as the key to understanding why such problems persist. The overarching aim of this thesis is to explore for the first time the utility of this model for understanding persistent emotional distress in cancer.

In order to achieve this, a series of linked empirical studies were conducted using data from a prospective cohort study of recently diagnosed breast and prostate cancer patients (n=206). Data were obtained at a pre-treatment baseline (T1) and twelve month follow-up (T2) using self-report questionnaires to assess emotional distress (HADS), illness perceptions (IPQ-R) and metacognitive beliefs and CAS processes (MCQ-30 / CAS-1).

The first study tested the validity of the MCQ-30 for use in cancer. Confirmatory and exploratory factor analyses provided evidence supporting the validity of the previously published 5-factor structure of the MCQ-30 in this population. In addition structural equation modelling (SEM) indicated that metacognitive beliefs were significantly associated with anxiety and depression as predicted, providing further evidence of concurrent validity.

Following this, three studies used hierarchical regression and SEM techniques to test theoretical predictions from the metacognitive model by exploring cross-sectional and prospective associations between maladaptive metacognitions and emotional distress as well
as testing whether metacognitive beliefs could explain more of the variance in emotional distress than could content of cognition (i.e. illness perceptions). The findings of these studies provided evidence supporting theoretical predictions that metacognitive beliefs cause and maintain distress by activating a style of inflexible responding to thoughts. The view that metacognitive beliefs may be more important in the development of emotional distress than the specific content of negative thoughts about cancer was also supported. Such findings suggest a potential to reduce anxiety by modifying metacognitive beliefs and processes as an alternative to more traditional cognitive approaches.

Finally, a small pilot study tested the potential of a single component of metacognitive therapy (MCT), Attention Training Technique, for reducing emotional distress in cancer. The findings did not provide clear evidence of benefit, but did indicate that intervention was effective, when undertaken appropriately, and was well received. This suggests there is promise in pursuing further development of interventions based on MCT in this population.
Table of Contents

Abstract i
List of Figures xi
List of Tables xiii
Acknowledgments xv
Prelude xvii

Chapter One: Introduction: Cancer and emotional distress

1.1 What is cancer? 2
1.2 Cancer: prevalence and outlook 2
1.3 Emotional distress after cancer? 4
1.4 Trajectory of emotional distress after cancer diagnosis 5
1.5 The impact of emotional distress after cancer 7
1.6 Interventions aimed at reducing emotional distress after cancer 8

Chapter Two: Predictors of persistent emotional distress after diagnosis of cancer: A literature review

2.1 Introduction 12
2.2 Method 12
2.3 Results 15
   2.3.1 Socio-demographic and clinical predictors: 21
   2.3.1.1 Age 21
   2.3.1.2 Gender 21
   2.3.1.3 Education and social class 22
   2.3.1.4 Treatment type 23
   2.3.1.5 Tumour characteristics 24
### 2.3.1.6 Physical health

2.3.1.7 Summary of socio-demographic and clinical predictors

2.3.2 Social and environmental predictors

2.3.2.1 Availability / characteristics of significant others and the social network

2.3.2.2 Social Support

2.3.2.3 Non-cancer-related difficulties and negative life events

2.3.2.4 Summary of social (environmental) predictors

2.3.3 Psychological predictors

2.3.3.1 Emotional distress

2.3.3.2 Self-esteem

2.3.3.3 Coping

2.3.3.4 Personality

2.3.3.5 Perceived control

2.3.3.6 Illness appraisal

2.3.3.7 Summary of psychological predictors

2.4 Conclusion

---

**Chapter Three: Current theoretical approaches to understanding emotional distress after cancer: An overview**

3.1 Cognitive model of adjustment to cancer

3.1.1 Coping theory

3.1.2 Schema theory

3.1.3 Evidence supporting the cognitive model of adjustment to cancer

3.2 Common Sense Model of self-regulation in health and illness

3.2.1 Evidence supporting the Common Sense Model in cancer
Chapter Four (Study one): Measuring metacognition in cancer: Validation of the Metacognitions Questionnaire 30 (MCQ-30).

4.1 Introduction

4.2 Methods
   4.2.1 Ethics Statement
   4.2.2 Participants
   4.2.3 Measures
   4.2.4 Procedure
   4.2.5 Data analysis

4.3 Results
   4.3.1 Sample
   4.3.2 Factorial Structure
   4.3.3 Convergent validity

4.4 Discussion
Chapter Five (Study two): The association of metacognitive beliefs with emotional distress after diagnosis of cancer

5.1 Introduction

5.2 Method

5.2.1 Measures

5.2.2 Data Analysis

5.3 Results

5.3.1 The association of metacognitive beliefs and distress

5.3.2 SEM - relationship between metacognitive beliefs and emotional distress

5.4. Discussion

5.4.1 The relationship between metacognitive beliefs and distress

5.4.2 Mediation of the relationship between metacognitive beliefs and distress by the CAS

5.4.3 Study implications, limitations and conclusions

Chapter Six (Study three): A prospective study of the association of metacognitive beliefs and processes with persistent emotional distress after diagnosis of cancer

6.1 Introduction

6.2 Method

6.2.1 Measures

6.2.2 Analysis

6.3 Results
6.3.1 Association of T1 illness perceptions with T2 anxiety, 106 depression and trauma
6.3.2 Association of T1 metacognitive beliefs with T2 anxiety, 109 depression and trauma
6.3.3 Predictive ability of T1 metacognitive beliefs over and above 112 demographic variables, T1 symptoms and content of thoughts about cancer

6.4 Discussion 114

Chapter Seven (Study four): Identifying causal predictors of emotional distress

12 months after cancer diagnosis: A Latent Growth Curve analysis

7.1 Introduction 119
7.2 Method 121
  7.2.1 Design 121
  7.2.2 Measures 121
  7.2.3 Analysis 122
7.3 Results 124
  7.3.1 Anxiety 124
  7.3.2 Depression 132
  7.3.3 Trauma 133
7.4 Discussion 134
  7.4.1 Hypothesis 1: Change in metacognitive beliefs over 12 months 134 will be associated with change in emotional distress
7.4.2 Hypothesis 2: The association between change in positive metacognitive beliefs and change in emotional distress is fully mediated by change in the CAS.

7.4.3 Hypothesis 3: The association between change in negative metacognitive beliefs and change in emotional distress is partially mediated by change in the CAS.

7.4.4 Study implications, limitations and conclusion

Chapter Eight (Study 5): Exploring the utility of Attention Training Technique (ATT) to reduce emotional distress in cancer patients: A case series

8.1 Introduction

8.2 Method

8.2.1 Design

8.2.2 Participants

8.2.3 Outcome measures

8.2.3.1 Weekly

8.2.3.2 Pre-treatment, Post-treatment and Follow-up

8.2.4 Intervention

8.2.5 Procedure

8.2.6 Analysis plan

8.3 Results

8.3.1 Session by session scores on outcome measures

8.3.2 Clinically significant change

8.3.3 Acceptability and feasibility of the intervention

8.4 Discussion
8.4.1 Is ATT an effective intervention for reducing emotional distress after cancer?

8.4.2 Is ATT a practical and acceptable intervention?

8.4.3 Limitations and conclusions

Chapter Nine: General Discussion & Conclusions

9.1 Re-orientation to thesis aims

9.2 Validity of the MCQ-30

9.3 Testing theoretical predictions from the metacognitive model of emotional disorder

9.3.1 Metacognitive beliefs will be associated with both current and future distress and negative beliefs will be the largest predictor

9.3.2 Metacognitive beliefs will predict additional variance in current and future emotional distress over and above previously implicated factors including; ‘content of cognition’ (i.e. Illness perceptions.) & baseline symptoms of distress

9.3.3 CAS processes such as worry and threat–focussed attention will mediate the relationship between metacognitive beliefs around diagnosis and current and future emotional distress

9.3.4 Problems with testing theoretical models

9.4 A final test - can an intervention designed to disrupt the CAS reduce emotional in cancer?

9.5 Recommendations for future research

References
## List of Appendices

<table>
<thead>
<tr>
<th>Appendix</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Case Series Study: Exit Interview Guide</td>
<td>191</td>
</tr>
<tr>
<td>B</td>
<td>Cook S.A; Salmon, P; Dunn, G; Fisher, P. (2014). Measuring metacognitions in cancer: Validation of the Metacognitions Questionnaire 30 (MCQ-30). <em>PlosOne</em>; 9 (9</td>
<td>193</td>
</tr>
<tr>
<td>C</td>
<td>Cook S.A; Salmon, P; Dunn, G; Holcombe, C; Cornford, P; Fisher, P. The association of metacognitive beliefs with emotional distress after diagnosis of cancer. (2015). <em>Health Psychology</em> 34 (3); 207-215</td>
<td>202</td>
</tr>
<tr>
<td>D</td>
<td>Cook S.A; Salmon, P; Dunn, G; Holcombe, C; Cornford, P; Fisher, P. (2015) A prospective study of the association between metacognitive beliefs and processes and persistent emotional distress after diagnosis of cancer. <em>Cognitive Therapy and Research</em> 39; 51-60</td>
<td>212</td>
</tr>
<tr>
<td>E</td>
<td>Prospective Cohort Study: Participant Feedback Leaflet</td>
<td>223</td>
</tr>
</tbody>
</table>
## List of Figures

**Figure 2.1** Flow diagram of literature search and sifting procedure  
14

**Figure 3.1** Cognitive model of adjustment to cancer  
50

**Figure 3.2** Common Sense Model of health and illness  
54

**Figure 3.3** The S-REF model of psychological disorder  
59

**Figure 3.4** Universal case formulation diagram  
61

**Figure 4.1** Structural equation model of the relationship between latent factors for the dimensions of the MCQ-30 and HADS anxiety and HADS depression  
78

**Figure 5.1** Hypothesised path model of the relationship between metacognitive beliefs and emotional distress.  
88

**Figure 5.2** Final path model of relationship of positive and negative metacognitive beliefs with anxiety, depression and PTSD symptoms, including mediation by worry (PSWQ)  
96

**Figure 5.3** Final path model of relationship between positive and negative metacognitive beliefs and anxiety, depression and trauma mediated by the CAS-I  
96

**Figure 7.1** Hypothesised LGC model of the relationship between change in metacognitive beliefs and change in emotional distress, mediated by change in worry  
123

**Figure 7.2a** Final LGC path model of relationship of change in metacognitive beliefs with change in anxiety symptoms, mediated by change in worry (PSWQ) over 12 months  
126

**Figure 7.2b** Final LGC path model of relationship of growth in metacognitive beliefs with growth in anxiety symptoms, mediated by growth in worry (CAS-I) over 12 months  
127

**Figure 7.3a** Final LGC path model of relationship of growth in metacognitive beliefs with growth in depression symptoms, mediated by growth in worry (PSWQ) over 12 months  
128

**Figure 7.3b** Final LGC path model of relationship of growth in metacognitive beliefs with growth in depression symptoms, mediated by growth in worry (CAS-I) over 12 months  
129
Figure 7.4a Final trimmed LGC path model of relationship of growth in metacognitive beliefs with growth in trauma symptoms, mediated by growth in worry (PSWQ) over 12 months

Figure 7.4b Final trimmed LGC path model of relationship of growth in metacognitive beliefs with growth in trauma symptoms, mediated by growth in worry (CAS-I) over 12 months

Figure 8.1 Emotional distress, worry and threat-focused attention scores across baseline, treatment and follow-up phases

Figure 8.2 MCQ-30 subscales & FCRI-Severity scale at pre-treatment, post-treatment and follow-up
List of Tables

Table 2.1 Sample characteristics of included studies 16

Table 2.2 Summary of study design and significant findings from included studies (grouped by outcome) 32

Table 2.3 Outcome measures used in included studies 47

Table 4.1 Sample Demographic and Clinical Characteristics 73

Table 4.2 Published scale structure and rotated (Geomin) factor loadings from EFA of the Metacognitions Questionnaire-30 at pre-treatment 75

Table 4.3 Descriptive data, internal consistency and inter correlations among the five latent MCQ-30 factors (CFA standardised solution) 76

Table 4.4 Fit indices for the pre-treatment and 12-month follow-up SEMs of the relationship between latent factors for the MCQ-30 and HADS anxiety and depression 77

Table 5.1 Final models of the variance in anxiety, depression and trauma explained by illness perceptions, after controlling for age & gender 91

Table 5.2 Final models of the variance in anxiety, depression and PTSD symptoms explained by metacognitive beliefs after controlling for; age & gender (Model 1), and age, gender & illness perceptions (Model 2). 93

Table 6.1 Distribution of anxiety, depression and trauma scores at both time-points 105

Table 6.2 Final models of the variance in T2 anxiety, depression and trauma predicted by T1 illness perceptions after controlling for age and gender (Analysis 1) and age, gender & T1 levels of symptoms (Analysis 2). R² change shows increment in variance explained when each set of variables was entered sequentially; beta, T and p are from the final model containing variables from all steps. 107

Table 6.3 Final models of the variance in T2 anxiety, depression and trauma predicted by T1 metacognitive beliefs after controlling for age and gender (Analysis 1) and age, gender & T1 levels of symptoms (Analysis 2). R² change shows increment in variance explained when each set of variables was entered sequentially; beta, T and p are from the final model containing variables from all steps. 110
Table 6.4 Final models of the variance in T2 anxiety, depression and trauma predicted by T1 metacognitive beliefs after controlling for age, gender, T1 level of symptoms and T1 illness perceptions (Analysis 3). $R^2$ change shows increment in variance explained by each step; beta, T and p are from the final model containing variables from all steps.

Table 8.1 Participants' exit interview ratings (0-10) of satisfaction with, and confidence in the intervention
Acknowledgments

This thesis is dedicated to my family; to Dave, Samuel and Benjamin Cook who have had to put up with it taking up so much of my time for so long, yet provided unflinching love, patience and support.

This research was conducted as part of a Population Health Scientist Fellowship funded by the Medical Research Council. I would like to gratefully thank the staff and patients of the Royal Liverpool & Broadgreen University NHS Hospital Trust for freely giving of their time and for supporting this research.

I would also like to thank the following people who have been directly involved in helping me complete my work:

A huge thank you to my supervisors at the University of Liverpool; Professor Peter Salmon and Dr Peter Fisher who have supported, encouraged, challenged and cajoled me every step of the way - I am full of appreciation for all that they have done, not only to help me complete this thesis but also to aid my professional development. In addition I would like to thank my external supervisor Professor Graham Dunn (The University of Manchester) for his patience, help and advice regarding the statistical analyses.

Aside from supervision, I am extremely thankful for, and grateful to, the following people who have provided practical assistance in completing the various study tasks:

- Ms Helen Ullmer who assisted with completing: SCID interviews, exit interviews and various administrative tasks (too numerous to mention).
- Ms Kirsten Atherton & Mr Chris Huntley who assisted with study recruitment and data acquisition.
- Ms Gemma Hayes, and also Ms Natasha Jermy, who assisted with updating the literature search.
- Ms Laura Hope-Stone for assisting with the ATT case series.
- Mr David Cook and Mr Dave Cook for braving the task of proof-reading.

Lastly but by no means least, I would like to thank my friends (particularly Adam, Laura and Mylo) who have kept me company, calmed me down and generally helped to keep me sane (ish) while writing up.
Declaration

I, Sharon Cook, declare that I am the author of this thesis, that, unless otherwise stated, all references cited have been consulted by me, that, unless otherwise stated, the work of which this thesis is a record has been done by myself and has not been previously accepted for a higher degree.

Sharon Cook
March 2015
Prelude

Thesis Overview

This thesis represents the first evaluation of the utility of the metacognitive model of emotional disorder for understanding persistent emotional distress after diagnosis of cancer. Following three introductory review chapters exploring the problem of persistent emotional distress in cancer, the existing literature on its predictors, and current theoretical approaches used to understand it, a new theoretical approach is proposed - The metacognitive model of emotional disorder. The remaining five chapters describe empirical studies conducted to test predictions from the metacognitive model in order to explore whether it is applicable in cancer and useful for understanding why emotional distress persists for some patients but not others.

The first introductory chapter provides a basic overview of cancer, as well as the prevalence, course and consequences of emotional distress among cancer patients and an overview of the psychotherapeutic interventions currently available for reducing distress. This introductory chapter is not intended to provide an exhaustive account; rather it is intended to set the scene for the thesis regarding the problem of persistent emotional distress after diagnosis of cancer and the need for greater understanding of the factors that underlie it – a problem which has been recognised worldwide as a significant clinical and research priority.

The second chapter continues setting the scene by reviewing the literature on potential predictors, available around the time of diagnosis, of persistent distress. For this chapter, and indeed for the thesis as a whole, the following working definition of persistent emotional distress is used:

‘a clinically significant or elevated level of emotional distress at any point greater than 12 months after receiving a diagnosis of cancer and/or starting primary treatment.’
This definition was decided upon based on the understanding that in cancer the majority of spontaneous psychological recovery occurs between 4-13 months of receiving a diagnosis (Helgeson, Snyder, & Seltman, 2004). Studies were selected for review where they tested predictors of persistent distress that were available around the time of diagnosis (i.e. within the first three months).

The final introductory chapter provides an overview of theoretical approaches that are typically used to understand emotional distress after diagnosis of cancer. These include the Cognitive model of adjustment to cancer (Moorey & Greer, 1989) and the Common Sense Model of self-regulation in health and illness (CSM: (Leventhal, Meyer, & Nerenz, 1980; Leventhal, Nerenz, & Steele, 1984). While again not intended to be an exhaustive review, this chapter highlights the main features of these approaches and the evidence available to support each model in cancer. Several limitations, for understanding persistent distress, are identified and an alternative model, the metacognitive model of emotional disorder, is proposed and described.

Before evaluating the metacognitive model in cancer, it is first necessary to establish that existing measurement tools are valid for assessing metacognitive beliefs and process in this population. Therefore, the first of the empirical studies, Study 1 presented in Chapter 4, uses confirmatory factor analysis and structural equation modelling to validate the Metacognitions Questionnaire (MCQ-30; (Wells & Cartwright-Hatton, 2004) in a sample of breast and prostate cancer patients.

The next three chapters (Chapters 5-7) each present studies (using the same sample as Study 1) that provide an empirical test of theoretical predictions from the metacognitive model. The first of these, Study 2 presented in Chapter 5, tests the prediction that metacognitive beliefs, specifically negative and positive beliefs about worry, are associated with emotional distress around the time of diagnosis; that they explain more of the variance
in emotional distress than content of cognition (i.e. illness perceptions); and that the relationship between metacognitive beliefs and emotional distress is mediated by worry- and/or threat-focussed attention. Following on from this cross-sectional study, Study 3, presented in Chapter 6, tests the prospective relationship between metacognitive beliefs around diagnosis and emotional distress 12 months later. This study uses hierarchical multiple regression to test whether metacognitive beliefs are able to explain additional variance in emotional distress over and above baseline symptoms of distress and content of cognition. The final study of the three, Study 4 presented in Chapter 7, addresses the limitations of regression analysis, identified in the preceding study, for identifying underlying causal processes when baseline distress is controlled. This study uses Latent Growth Curve modelling to test theoretical predictions that changes in metacognitive beliefs effects changes in emotional distress via changes in worry and/or threat-focussed attention.

The final empirical study of the thesis, Study 5, is presented in Chapter 8. This study is a small pilot study aimed at testing the potential of a single component of metacognitive therapy, Attention Training Technique (ATT), for reducing emotional distress after diagnosis and primary treatment for cancer. As such, it also provides an additional small-scale experimental test of the predicted relationships between metacognitive beliefs, worry and threat-focussed attention and emotional distress.
Chapter One

Introduction: Cancer and emotional distress
1.1 What is cancer?

Cancer is not one single disease, but a term used for a collection of diseases in which abnormal cells grow in an uncontrolled way, invading and destroying surrounding tissue and putting pressure on other bodily structures. If left untreated, cancer cells from primary tumours metastasise (spread) via the blood or lymphatic system to other parts of the body where they form secondary or metastatic tumours, and it is this ability that makes them particularly dangerous. There are as many different types of cancer as there are types of cells in the body and each may behave quite differently, causing different symptoms and responding differently to available treatment. The National Cancer Institute identifies five broad categories of cancer:

- Carcinoma (the most common category) – begins in the skin or tissues that line or cover internal organs
- Sarcoma – begins in connective or supportive tissues such as bone, cartilage, muscles and blood vessels.
- Leukaemia – begins in the blood-forming tissue (i.e. bone marrow) and causes large numbers of abnormal blood cells to be produced
- Lymphoma & Myeloma – begin in cells of the immune system
- Central nervous system cancers - begin in the tissue of the brain and spinal cord.

1.2 Cancer: prevalence and outlook

An estimated 12.7 million people were diagnosed with cancer worldwide in 2008 (Ferlay et al., 2010). Of these, 6.6 million were men among whom the most common diagnoses were cancers of the lung (16%) and prostate (14%). Among the six million women
diagnosed, breast cancer was by far the most common disease accounting for almost 1 in 4 of all diagnoses (23%). In the UK, the most commonly diagnosed cancers are prostate cancer, which accounts for almost a quarter of male cancers (24%), and breast cancer, almost a third of female cancers (31%).

Although mortality figures remain high (7.6 million people died from cancer worldwide in 2008 (Ferlay et al., 2010)), significant improvements in cancer care over the past decade have led to corresponding improvements in survival (Department of Health, Macmillan Cancer Support, & NHS Improvement, 2010; Institute of Medicine, 2007). It has previously been estimated that there are over 10 million long-term cancer survivors in the United States (Institute of Medicine, 2006) and more recently, around 2 million cancer survivors in the UK (Maddams et al., 2009) with numbers estimated to grow in excess of 3% per year. Three types of cancer (breast, prostate and colorectal) account for over half of all survivors in the UK, with the former two having estimated survival rates of greater than 85% (Department of Health et al., 2010).

Despite this improvement in prognosis, cancer diagnosis is still a diagnosis of a life-threatening disease and often has a profound emotional impact on the individual. Furthermore, it has been recognised that this impact does not end after treatment (Elliott et al., 2011) but can affect the lives of survivors for many years (Meyerowitz, Kurita, & D'Orazio, 2008). The importance of addressing psychosocial needs has figured prominently among concerns raised by cancer survivors both in the US (Reuben, 2004) and the UK (Department of Health et al., 2010). In a recent study across 66 cancer centres in the UK, psychological needs and fear of recurrence were the most common unmet need reported (Armes et al., 2009). In recognition of the continuing psychological impact of cancer on patients’ lives after treatment, official reports in both the UK and US have highlighted the importance of assessing and addressing patients’ psychological needs at key stages as a key
component in planning supportive care in both the treatment and survivorship phases (Holland, 1999; Institute of Medicine, 2007; National Institute for Clinical Excellence, 2004)

1.3 Emotional distress after cancer?

In 1975, Hans Selye (Selye, 1975) for the first time distinguished between two types of stress, ‘Eustress’ a stress which has positive outcomes, enhancing physical and/or mental functioning and ‘Distress’ a persistent stress, that fails to resolve through coping and adaptation and may lead to anxiety or withdrawal. More recently the term ‘distress’ has been defined by the National Comprehensive Cancer Network (NCCN) as ‘an unpleasant emotional experience of a psychological, social and/or spiritual nature that often interferes with the ability to cope effectively. It extends along a continuum, ranging from common and understandable feelings of vulnerability, sadness or fear to problems that can become disabling such as depression, anxiety, panic, social isolation and spiritual crisis’ (Holland, 1999). It was chosen as the term for describing patients’ negative responses to cancer for reasons of political correctness (considered less stigmatising than terms such as ‘psychiatric disorder’, or ‘psychosocial’ or ‘emotional’ problems, and more acceptable to patients) and because it was suitable for measurement by self-report, and has now been adopted worldwide (Holland, 1999).

In the literature, prevalence of emotional distress after cancer is usually defined in terms of prevalence of general distress, anxiety disorders and/or depression with estimates ranging from 15% to 50% (Derogatis et al., 1983; Strong et al., 2007). Distress is particularly common in the period after cancer diagnosis, with around half of all newly diagnosed patients reporting clinically significant levels of anxiety and/or depression (Henselmons et al., 2010; National Institute for Clinical Excellence, 2004). However, as survival rates continue to improve, it is increasingly recognised that long-term survivors remain at risk of clinically
significant distress. Over a third of patients in treatment or long-term follow-up report levels of distress, including anxiety and/or depression, that warrant intervention (Carlson et al., 2004). Annual prevalence of major depression or generalised anxiety disorder remains 22% in the fourth year after breast cancer diagnosis (Burgess et al., 2005), while life-time prevalence of cancer-related PTSD is 10-12% for breast cancer and 20% for other cancers (Andrykowski & Kangas, 2010). Furthermore, a USA population-based survey (Hoffman, McCarthy, Recklitis, & Ng, 2009) reported a 6% prevalence of psychiatric disorders amongst cancer survivors - double that in the non-cancer comparison group, even after controlling for socio-demographic and clinical correlates.

Consequently, psychological distress after cancer is recognised worldwide to be a significant problem. So much so that, in 2004, it was designated by the Canadian Strategy for Cancer Control (Bultz & Carlson, 2006), to be the ‘6th vital sign’ which should be used alongside the traditional biomedical indicators (temperature, respiration, heart rate, blood pressure and pain) to assess whether a patient’s functioning is sufficient to achieve ‘wellness’.

1.4 Trajectory of emotional distress after cancer diagnosis

Emotional distress around diagnosis should not be assumed to be evidence of psychopathology. For most people it represents a ‘normal’ and potentially adaptive stress response to a traumatic and threatening event, and would be expected to resolve spontaneously without the need for specialist help (Brennan, 2004; Salmon, 2000). For example, in a cohort of breast cancer patients, point prevalence of anxiety and/or depression cases dropped from 33% at diagnosis to 15% at one year (Burgess et al., 2005). In addition, a study (Helgeson et al., 2004) investigating trajectories of emotional distress in breast cancer over four years noted that most spontaneous improvement in mental health scores occurred in
the period between 4 and 13 months since diagnosis with little occurring across the remaining assessment points (Helgeson, Snyder, & Seltman, 2004). However, while many studies provide data that support this view, it is also recognised that there are groups of patients whose distress trajectories do not conform to such expected declines (Helgeson et al., 2004; Nosarti, Roberts, Crayford, McKenzie, & David, 2002).

A recent study (Henselmans et al., 2010) which assessed distress at five key points in the breast cancer journey (i.e. after diagnosis, after surgery, immediately after adjuvant treatment, and two and six months after the end of treatment), identified four groups with different trajectories of emotional distress across the study period. The first group (36%) experienced no significant distress at any point. The second group (33%) recovered emotionally and became indistinguishable from the first by six months after the end of treatment. The third group (15%) experienced persistent distress throughout the period, while the final group (15%) experienced a delayed emotional response, becoming distressed only after active treatment had ended. These trajectories are consistent with those found in previous studies of breast cancer patients, assessed immediately post-operatively and 12 months after surgery (Millar, Purushotham, McLatchie, George, & Murray, 2005), and in other cancer populations assessed at 3 and 15 months since diagnosis (Schroevers, Ranchor, & Sanderman, 2003a, 2003b). The interesting thing to note from these studies is that, while between 22% and 48% of patients were distressed at baseline, around a half to two-thirds of these improved psychologically (i.e. no longer met ‘case’ criteria) before the follow-up assessment. Conversely while most patients (52-78%) in each study were not ‘cases’ at baseline, between 7% and 29% of these met criteria by the follow-up assessment. Thus it cannot be assumed that patients who are emotionally distressed at diagnosis are necessarily vulnerable to persistent problems, or that those who appear emotionally well at this point will remain that way.
1.5 The impact of emotional distress after cancer

Studies conducted by cancer support charities such as Macmillan in the UK have indicated that unmet psychological needs figure prominently among the concerns raised by patients themselves, and that they pose a considerable burden to the individual’s quality of life and that of their families. Indeed in a survey of over 1500 people who had or were affected by cancer, 45% said that it was the emotional rather than the physical or practical effects of cancer which were the most difficult to deal with (Macmillan Cancer Support, 2010).

As well as the impact on quality of life, it is recognised that neglecting psychological distress can also exacerbate illness and increase health care costs. In a recent systematic review, evidence collated from 25 independent studies conducted worldwide indicated that mortality rates were 26% higher among cancer patients reporting depressive symptoms (based on 14 studies) and 39% higher among patients with major or minor depressive disorder (based on 3 studies) (Satin, Linden, & Phillips, 2009). However, it should be noted that this finding relates to ‘all-cause’ mortality and as yet no direct effect of depression on cancer-specific development or mortality has been demonstrated (Garssen, 2011; Schneider & Moyer, 2010). In addition, studies have also shown that when patients' emotional needs remain unresolved, they are more likely to use community health or accident and emergency services (Carlson & Bultz, 2004) and place higher demands on scarce provider time and resources (Bultz & Carlson, 2006). Indeed, a recent estimate of the cost of extended bed days due to preventable psychological illness in cancer patients at one UK NHS trust was £366,000 per year (Macmillan Cancer Support, 2010).

Consequently, it is suggested that providing appropriate and timely psychological and emotional support can not only improve patients’ long-term quality of life but also save
money for health and social care providers and provide benefit for the wider community in terms of enabling cancer patients to return to work, as well as their community and social activities (Macmillan Cancer Support, 2011).

1.6 Interventions aimed at reducing emotional distress after cancer

In recognition of the continuing impact of emotional distress after cancer, health policies have recommended that all patients undergo systematic psychological assessment at key points from diagnosis, and have prompt access to psychological support. However, in reality it is often the case that specialist help is limited and few patients have access to it. Thus most psychological care that is provided is offered reactively, i.e. at the time of emotional crisis (Zabora et al., 2001).

Psychological interventions offered in cancer can take many forms, with the four most commonly used approaches being: relaxation training, psychoeducation, individual cognitive behavioural therapy (CBT), and group or individual supportive therapy (Carlson L, 2003). It is often suggested that such psychological interventions show positive benefits for cancer patients across a range of psychosocial, physical and survival outcomes. However, in reality systematic reviews and meta-analytic studies of CBT and other psychotherapeutic interventions in cancer have produced mixed results. In 2002, a rigorous systematic review (Newell, Sanson-Fisher, Savolainen, & Pro, 2002), excluding trials of low internal validity, concluded that only tentative recommendations were possible regarding the benefit of psychological therapies for improving cancer outcomes. This review found some limited evidence: that group therapy, psycho-education, counselling and CBT could improve psychosocial outcomes; that relaxation training and guided imagery could improve physical side effects; and that psychological therapies in general could improve immune system outcomes. However, for all outcomes the reviewers reported there was too little research of
sufficient methodological rigour to make any strong recommendations supporting these therapies. Furthermore, meta-analytic studies in cancer (Faller et al., 2013) have concluded that small to moderate effect sizes are typical. The earliest of these (Sheard & Maguire, 1999) reported that when estimating robust effects of psychological interventions, these ranged from negligible for depression (0.19), to moderate (0.36) for anxiety. Similarly, a more recent study reported that initial moderate to large effects of psychological treatments for anxiety and depression (Naaman, Radwan, Fergusson, & Johnson, 2009) in breast cancer were not robust to study quality. Overall effects were reduced by almost 50% to -0.26 for anxiety, and by 75% to -0.24 for depression when just the studies with high internal validity were considered. Collectively these studies indicate that there is considerable room for improvement in psychotherapeutic effectiveness.

Conclusions that can be drawn regarding the efficacy of psychological therapies for reducing emotional distress in cancer are limited not only by the methodological quality of reviews but also by the wide variety of interventions employed, outcomes assessed, and variation in timing of intervention delivery. It is difficult to reach conclusions across studies where those studies are in effect asking different questions. In order to generate useful and meaningful guidance on how to help cancer patients manage emotional distress we need to move away from the imprecise question of, ‘do psychological interventions work in cancer?’, in favour of testing specific components or therapies that target clearly defined outcomes. Only then can comparisons be made and conclusions drawn across studies.

Given that we know a significant number of patients are likely to experience clinically significant levels of emotional distress at some point in their cancer journey a more ethical approach would be to identify those ‘at risk’ and provide some form of preventative intervention at the outset. However, the different trajectories of distress after diagnosis and the small effect sizes found by the meta-analyses described above indicate that current
interventions are unlikely to be considered cost-effective if delivered to all patients on the basis of cancer morbidity alone (Sheard & Maguire, 1999). In addition to being unnecessary for many patients, there is also the possibility that early, and indiscriminate, intervention may exacerbate rather than ameliorate emotional distress for some. Evidence from the post-traumatic stress disorder literature (Litz, Gray, Bryant, & Adler, 2002; Rose, Bisson, & Wessely, 2003) suggests that early interventions, such as psychological debriefing, can for some people aggravate distress by undermining confidence at a time of personal vulnerability and by disrupting normal processes of coping and psychological adjustment. In line with this, a recent study conducted at Liverpool (Baker et al., 2012) found that some newly diagnosed cancer patients were reluctant to discuss emotional needs and were hostile to a psycho-educational intervention designed to help them recognise, understand and self-manage emotional distress, complaining that it compounded their problems. Consequently, in order to potentially provide such support, we first need to know more about the factors that underlie vulnerability to emotional distress after cancer so that intervention can be targeted effectively.
Chapter Two

Predictors of persistent emotional distress after diagnosis of cancer:

A literature review
2.1. Introduction

The need to identify factors early in the cancer trajectory that underlie patients’ vulnerability to persistent distress after cancer was highlighted in Chapter 1. The aim of this chapter is to explore whether such factors can be reliably identified from the literature. Over the last 10-15 years a substantial body of research has arisen out of the focus on psychological morbidity in cancer. However, much of this has been aimed at quantifying prevalence and improving early detection of emotional distress, rather than identifying the factors that cause and maintain it.

Cross-sectional studies have identified clinical, socio-demographic and psychological factors that are associated with emotional distress after cancer. However, such studies are of limited use for identifying causal factors as they are unable to provide information about their relationship with future levels of distress. In contrast, prospective studies, while still falling short of proving cause, are able to identify factors that predict vulnerability to persistent distress by demonstrating an association between baseline factors and future levels of distress, and as such they provide greater insight into potential causal relationships.

Consequently, a review of the literature was undertaken focussing specifically on prospective studies that sought to identify predictors of longer-term or persistent distress (i.e. at least 12 months since diagnosis) from those available early in the cancer journey (i.e. within three months of diagnosis), on the basis that these might indicate factors that potentially cause and maintain emotional distress that could be targeted by therapy.

2.2. Method

The literature search used the EBSCO database, which searches across five different medical, nursing and psychology databases incl: Medline full text, Psychinfo, PsychARTICLES, CINAHL plus, AHMED. The search combined the term ‘cancer’ with
terms relating to emotional distress (including: emotional distress or psychological distress or anxiety, depress* or posttraumatic stress or PTSD or psychological morbidity or psych*, adjustment or emotional adjustment or mood or adjustment disorder or acute stress disorder or fear of recurrence (all fields)) and those commonly used to describe the focus of relevant studies (predict* or risk factors, caus* or vulnerability (all fields)). Studies were restricted to adult populations (NOT: adolescent cancer or child* cancer or paed*carers or palliative (abstract)) and to those with a primary cancer diagnosis (NOT: genetic testing, genetic screening (abstract)); (NOT: palliative, metastatic cancer, advanced cancer, survival, mortality (title)). No date restriction was applied, although only English language articles were included. In addition, the references of all of the full articles screened in ‘Sift 5’ (see Figure 1) were searched to identify any additional studies that may have been missed by the database search.

This search yielded 11,080 papers. After removing the duplicates, an Endnote title search was used to exclude papers where the primary focus was not relevant to the search topic (i.e. Cardio, Arthritis, Stroke, Obesity, Diabetes, Childhood, Adolescent, Parent, Mammogram, Caregiver, Genetic Menopause, Mortality, Terminal, HIV, AIDS, Non-cancer, Benign, Fatigue, Smoking). Two further sifts were conducted by hand (using titles & abstracts, respectively) to identify papers reporting primary studies with a prospective or cross-sectional (with historical predictors) study design, where predictor variables were assessed before or within three months of cancer diagnosis and used to predict persistent distress or emotional distress a minimum of 12 months after diagnosis. Where this information could not be ascertained from the abstract the articles were obtained for detailed scrutiny.

The literature search and sifting procedure is detailed in Figure 1 below.
Figure 2.1 Flow diagram of literature search and sifting procedure

Records identified through EBSCO (PsycARTICLES, PsycINFO, CINAHL Plus, MEDLINE, AHMED)

Refs exported to ENDNOTE (N = 11,080)

(Sift 1) Duplicates removed (retained N = 7586)

Records excluded (N = 3494)

(Sift 2) Endnote keyword search (retained N = 5059)

Records excluded (N = 2527)

(Sift 3) Title screened (retained = 945)

Records excluded (N = 4114)

(Sift 4) Abstract screened (retained N = 113)

Records excluded (N = 832)

(Sift 5) Full articles screened

Records excluded (N = 75)

Articles retained (Table 1) (N = 38)
A standardised data extraction protocol was applied in order to evaluate the evidence supporting different factors (available around the time of cancer diagnosis) as predictors of persistent distress. Data extracted included: general study details (author, date, country); participants’ details (age, gender, cancer diagnosis); study design and methodology (sample size and attrition, outcome and predictor variables, timing of baseline and follow-up assessments, and analysis); and a summary of the reported findings (i.e. betas/odds ratios (where available) or % variance explained). Details of each study’s sample characteristics are presented in Table 2.1, while details of study design and reported findings (grouped by distress outcome) are presented in Table 2.2. A glossary of the outcomes measures used in the included studies is provided in Table 2.3. The evidence is grouped, evaluated and summarised in turn for each of three categories of predictors: socio-demographic and clinical predictors; social and environmental predictors and psychological predictors. The findings from each summary are drawn together and discussed in a concluding paragraph.

### 2.3. Results

A total of 38 articles reporting 34 primary studies were reviewed. The sample characteristics for each study are presented in Table 2.1.

Most studies were conducted in Europe (n = 21); eight in North America (USA & Canada) and the remaining five in Australia, Japan and Korea. All except two (Andrykowski & Cordova, 1998; Grassi, Malacarne, Maestri, & Ramelli, 1997) were prospective cohort studies. These two exceptions were cross-sectional but tested whether premorbid psychiatric history and retrospective self-report of emotional response around the time of diagnosis could predict depression at 12 months post-diagnosis.
Table 2.1: Sample characteristics of included studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Cohort</th>
<th>Diagnosis</th>
<th>% Female</th>
<th>Time 1 (N)</th>
<th>Time 2 (N)</th>
<th>Age Mean (SD)</th>
<th>Age Median (Range)</th>
<th>Country</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dean 1987</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>122</td>
<td>111</td>
<td>48.7</td>
<td>(20-60)</td>
<td>UK</td>
</tr>
<tr>
<td>Ramirez et al, 1995</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>102</td>
<td>91</td>
<td>-</td>
<td>56 (24-69)</td>
<td>UK</td>
</tr>
<tr>
<td>Grassi et al 1997</td>
<td>Cross-sectional</td>
<td>Mix</td>
<td>80</td>
<td>113</td>
<td>-</td>
<td>52.3 (11.5)</td>
<td>(20-67)</td>
<td>Eur</td>
</tr>
<tr>
<td>Andrykowski &amp; Cordova 1998</td>
<td>Cross-sectional</td>
<td>BC</td>
<td>100</td>
<td>82</td>
<td>-</td>
<td>56.6 (10.5)</td>
<td>(37-85)</td>
<td>USA</td>
</tr>
<tr>
<td>Carver et al, 1998</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>66</td>
<td>61</td>
<td>52.9 (11.2)</td>
<td>(28-76)</td>
<td>USA</td>
</tr>
<tr>
<td>Tjemsland et al, 1998</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>Not stated</td>
<td>106</td>
<td>-</td>
<td>50 (33-70)</td>
<td>Eur</td>
</tr>
<tr>
<td>Hammerlid et al, 1999</td>
<td>Prospective</td>
<td>HN</td>
<td>28</td>
<td>357</td>
<td>215</td>
<td>63</td>
<td>(18-88)</td>
<td>Eur</td>
</tr>
<tr>
<td>Bleiker et al, 2000</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>244</td>
<td>200</td>
<td>51.9 (10.5)</td>
<td>(29-75)</td>
<td>Eur</td>
</tr>
<tr>
<td>de Leeuw et al 2000†</td>
<td>Prospective</td>
<td>HN</td>
<td>21</td>
<td>204</td>
<td>155</td>
<td>59 (10.8)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>de Graeff et al 2000†</td>
<td>Prospective</td>
<td>HN</td>
<td>20</td>
<td>204</td>
<td>153</td>
<td></td>
<td>(29-76)</td>
<td>Eur</td>
</tr>
<tr>
<td>De Leeuw 2001†</td>
<td>Prospective</td>
<td>HN</td>
<td>22</td>
<td>204</td>
<td>171/139/123</td>
<td>59 (10.6)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Ranchor et al, 2002</td>
<td>Prospective</td>
<td>Mix</td>
<td>42</td>
<td>167</td>
<td>99</td>
<td>73.4 (7.46)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Stanton et al 2002</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>80</td>
<td>70</td>
<td>52.6 (11.94)</td>
<td>30-80</td>
<td>USA</td>
</tr>
<tr>
<td>Mehta et al, 2003</td>
<td>Prospective</td>
<td>PC</td>
<td>0</td>
<td>519</td>
<td>259</td>
<td>64.8 (4.8)</td>
<td>-</td>
<td>USA</td>
</tr>
<tr>
<td>Shroever &amp; Sanderman, 2003 **</td>
<td>Prospective</td>
<td>Mix</td>
<td>73</td>
<td>475</td>
<td>403</td>
<td>58 (14.3)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Shroever &amp; Sanderman, 2003 **</td>
<td>Prospective</td>
<td>Mix</td>
<td>73</td>
<td>475</td>
<td>403</td>
<td>58 (14.3)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Uchitomi et al, 2003</td>
<td>Prospective</td>
<td>LC</td>
<td>40</td>
<td>262</td>
<td>212</td>
<td>62.1 (10.8)</td>
<td>63.5 (22-83)</td>
<td>Japan</td>
</tr>
<tr>
<td>Schou et al, 2004</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>195</td>
<td>165</td>
<td>56 (10.3)</td>
<td>21-78</td>
<td>Eur</td>
</tr>
<tr>
<td>Aarstad et al, 2005</td>
<td>Prospective</td>
<td>HN</td>
<td>0</td>
<td>27</td>
<td>27</td>
<td>59.9 (1.3)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Burgess et al, 2005</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>202</td>
<td>170</td>
<td>48.5 (7.8)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Millar et al, 2005</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>371</td>
<td>279</td>
<td>59.4 (10.9)</td>
<td>29-98</td>
<td>Eur</td>
</tr>
<tr>
<td>Karnell et al, 2006</td>
<td>Prospective</td>
<td>HN</td>
<td>32</td>
<td>235</td>
<td>148</td>
<td>-</td>
<td>-</td>
<td>USA</td>
</tr>
<tr>
<td>Steginga et al, 2006</td>
<td>Prospective</td>
<td>PC</td>
<td>0</td>
<td>111</td>
<td>104</td>
<td>61.54 (8.13)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Gustavsson-Lilius et al, 2007</td>
<td>Prospective</td>
<td>Mix</td>
<td>68</td>
<td>349</td>
<td>123</td>
<td>58 (8.6)</td>
<td>34-76</td>
<td>Eur</td>
</tr>
<tr>
<td>Study</td>
<td>Cohort</td>
<td>Diagnosis</td>
<td>% Female</td>
<td>Time 1 (N)</td>
<td>Time 2(N)</td>
<td>Age Mean (SD)</td>
<td>Age Median (range)</td>
<td>Country</td>
</tr>
<tr>
<td>----------------------------</td>
<td>--------</td>
<td>-----------</td>
<td>----------</td>
<td>------------</td>
<td>-----------</td>
<td>---------------</td>
<td>-------------------</td>
<td>---------</td>
</tr>
<tr>
<td>Lebel et al, 2008</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>146</td>
<td>86</td>
<td>61-7 (10.8)</td>
<td>37-88</td>
<td>Can</td>
</tr>
<tr>
<td>Barez et al, 2009</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>129</td>
<td>101</td>
<td>48.03 (8.4)</td>
<td>25-65</td>
<td>Eur</td>
</tr>
<tr>
<td>Den Oudsten et al, 2009</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>223</td>
<td>144</td>
<td>58.7 (9.4)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Risvedt &amp; Trinkaus, 2009</td>
<td>Prospective</td>
<td>RC</td>
<td>44</td>
<td>123</td>
<td>80</td>
<td>67.5 (12)</td>
<td>29-88</td>
<td>USA</td>
</tr>
<tr>
<td>Couper et al, 2010</td>
<td>Prospective</td>
<td>PC</td>
<td>0</td>
<td>211</td>
<td>175</td>
<td>66.2 (8.3)</td>
<td>43-92</td>
<td>Aus</td>
</tr>
<tr>
<td>Scharlloo et al, 2010</td>
<td>Prospective</td>
<td>HN</td>
<td>24</td>
<td>177</td>
<td>95</td>
<td>59.6 (10.8)</td>
<td>36-84</td>
<td>Eur</td>
</tr>
<tr>
<td>Elkit &amp; Blum, 2011</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>81</td>
<td>64</td>
<td>56.3 (9.1)</td>
<td>41-89</td>
<td>Eur</td>
</tr>
<tr>
<td>Lee et al, 2011</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>299</td>
<td>206</td>
<td>-</td>
<td>(20-79)</td>
<td>Korea</td>
</tr>
<tr>
<td>Carlson et al 2013***</td>
<td>Prospective</td>
<td>BC</td>
<td>43</td>
<td>877</td>
<td>505</td>
<td>62.3 (14.1)</td>
<td>-</td>
<td>Can</td>
</tr>
<tr>
<td>Enns et al, 2013***</td>
<td>Prospective</td>
<td>Mix</td>
<td>43</td>
<td>1196</td>
<td>480</td>
<td>60.4 (13.3)</td>
<td>-</td>
<td>Can</td>
</tr>
<tr>
<td>Lockeefeer &amp; de Vries, 2013</td>
<td>Prospective</td>
<td>BC</td>
<td>100</td>
<td>227</td>
<td>163</td>
<td>58.9 (9.3)</td>
<td>-</td>
<td>Eur</td>
</tr>
<tr>
<td>Neilson et al, 2013</td>
<td>Prospective</td>
<td>HN</td>
<td>16</td>
<td>101</td>
<td>37</td>
<td>63</td>
<td>(37-85)</td>
<td>Aus</td>
</tr>
<tr>
<td>Adachi et al, 2014</td>
<td>Prospective</td>
<td>HN</td>
<td>22</td>
<td>116</td>
<td>78</td>
<td>61.2 (11.4)</td>
<td>20-85</td>
<td>Japan</td>
</tr>
<tr>
<td>Kohler et al, 2014</td>
<td>Prospective</td>
<td>PC</td>
<td>0</td>
<td>390</td>
<td>329</td>
<td>65.3 (6.4)</td>
<td>-</td>
<td>Eur</td>
</tr>
</tbody>
</table>

N.B: BC – breast cancer; Mix – mixed diagnoses; HN – Head & Neck Cancer; PC – prostate cancer; LC – lung cancer; RC – rectal cancer; UK – United Kingdom; Eur - Europe; USA – United States of America; Aus – Australia; Can – Canada
The reviewed studies were predominantly of breast cancer patients (n=16) (Andrykowski & Cordova, 1998; Bárez, Blasco, Fernández-Castro, & Viladrich, 2009; Bleiker, Pouwer, van der Ploeg, Leer, & Adèr, 2000; Burgess et al., 2005; Carver et al., 1998; Dean, 1987; Den Oudsten, Van Heck, Van der Steeg, Roukema, & De Vries, 2009; Elklit & Blum, 2011; Lebel, Rosberger, Edgar, & Devins, 2008; Lee et al., 2011; Lockefeer & De Vries, 2013; Millar et al., 2005; Ramirez, Richards, Jarrett, & Fentiman, 1995; Schou, Ekeberg, Ruland, Sandvik, & Karesen, 2004; Stanton, Danoff-Burg, & Huggins, 2002; Tjemsland, Søreide, & Malt, 1998), although head & neck, (n=7) (Aarstad, Aarstad, Heimdal, & Olofsson, 2005; Adachi et al., 2014; de Graeff et al., 2000; de Leeuw et al., 2000, 2001; Hammerlid et al., 1999; Karnell, Funk, Christensen, Rosenthal, & Magnuson, 2006; Neilson et al., 2013; Scharloo et al., 2010), prostate (n=4) (Couper et al., 2010; Köhler et al., 2014; Mehta, Lubeck, Pasta, & Litwin, 2003; Steginga & Occhipinti, 2006), lung (n=1)(Uchitomi et al., 2003), rectal (n=1) (Ristvedt & Trinkaus, 2009), and heterogeneous cancer populations (n=5) (Carlson, Waller, Groff, Giese-Davis, & Bultz, 2013; Enns et al., 2013; Grassi et al., 1997; Gustavsson-Lilius, Julkunen, Keskivaara, & Hietanen, 2007; Ranchor et al., 2002; Schroevers et al., 2003a, 2003b) were also included. The mean ages of samples studied ranged from 48 to 73 years. Reflecting the cancer populations investigated, most studies (n=18) (Andrykowski & Cordova, 1998; Bárez et al., 2009; Bleiker et al., 2000; Burgess et al., 2005; Carver et al., 1998; Dean, 1987; Den Oudsten et al., 2009; Elklit & Blum, 2011; Grassi et al., 1997; Lebel et al., 2008; Lee et al., 2011; Lockefeer & De Vries, 2013; Millar et al., 2005; Ramirez et al., 1995; Schou et al., 2004; Schroevers et al., 2003a, 2003b; Stanton et al., 2002; Tjemsland et al., 1998) reported on samples that were entirely or mostly female.

Of the 32 prospective studies, one had a pre-morbid baseline (Ranchor et al., 2002), two completed baseline assessments just prior to receiving a diagnosis (Den Oudsten et al., 2009; Lockefeer & De Vries, 2013), seven (Aarstad et al., 2005; Enns et al., 2013; Gustavsson-
Lilius et al., 2007; Hammerlid et al., 1999; Lee et al., 2011; Scharloo et al., 2010; Schou et al., 2004) immediately after diagnosis, thirteen before primary treatment started (Adachi et al., 2014; Carver et al., 1998; Couper et al., 2010; de Graeff et al., 2000; de Leeuw et al., 2000, 2001; Dean, 1987; Karnell et al., 2006; Köhler et al., 2014; Mehta et al., 2003; Neilson et al., 2013; Ramirez et al., 1995; Stanton et al., 2002; Steginga & Occhipinti, 2006; Tjemsland et al., 1998) and eight after primary treatment finished (Bárez et al., 2009; Bleiker et al., 2000; Elklit & Blum, 2011; Lebel et al., 2008; Millar et al., 2005; Ristvedt & Trinkaus, 2009; Schroevers et al., 2003a, 2003b; Uchitomi et al., 2003). The one remaining study (Burgess et al., 2005) interviewed patients five months after diagnosis to retrospectively assess the occurrence of predictor variables within the period from one month pre-diagnosis to four months post-diagnosis.

The predominant outcome assessed across studies was depression (n = 23) (Aarstad et al., 2005; Adachi et al., 2014; Burgess et al., 2005; Carlson et al., 2013; Couper et al., 2010; de Graeff et al., 2000; de Leeuw et al., 2000, 2001; Dean, 1987; Den Oudsten et al., 2009; Enns et al., 2013; Grassi et al., 1997; Gustavsson-Lilius et al., 2007; Hammerlid et al., 1999; Karnell et al., 2006; Lee et al., 2011; Lockefer & De Vries, 2013; Neilson et al., 2013; Ramirez et al., 1995; Schou et al., 2004; Schroevers et al., 2003a, 2003b; Uchitomi et al., 2003). Ten of these studies also assessed anxiety (Burgess et al., 2005; Carlson et al., 2013; Couper et al., 2010; Dean, 1987; Enns et al., 2013; Gustavsson-Lilius et al., 2007; Hammerlid et al., 1999; Neilson et al., 2013; Ramirez et al., 1995; Schou et al., 2004). No studies assessed anxiety in isolation. Nine of these 23 papers predicted anxiety and/or depression ‘caseness’ at follow-up or change in ‘caseness’ while the remaining 14 predicted the severity, or change in severity of, symptoms.

Generic emotional distress/functioning was assessed across twelve papers (Bárez et al., 2009; Carver et al., 1998; de Graeff et al., 2000; Köhler et al., 2014; Lebel et al., 2008; Millar...
et al., 2005; Ranchor et al., 2002; Ristvedt & Trinkaus, 2009; Scharloo et al., 2010; Stanton et al., 2002; Steginga & Occhipinti, 2006; Uchitomi et al., 2003) and fear of recurrence in two (Mehta et al., 2003; Stanton et al., 2002). Finally, six papers predicted symptoms of trauma (Andrykowski & Cordova, 1998; Bleiker et al., 2000; Elklit & Blum, 2011; Lebel et al., 2008; Ristvedt & Trinkaus, 2009; Tjemsland et al., 1998).

Twenty-four studies (Adachi et al., 2014; Bleiker et al., 2000; Carver et al., 1998; Couper et al., 2010; de Graeff et al., 2000; de Leeuw et al., 2000; Dean, 1987; Den Oudsten et al., 2009; Elklit & Blum, 2011; Enns et al., 2013; Grassi et al., 1997; Gustavsson-Lilius et al., 2007; Hammerlid et al., 1999; Köhler et al., 2014; Millar et al., 2005; Neilson et al., 2013; Ramirez et al., 1995; Ranchor et al., 2002; Schou et al., 2004; Schroevers et al., 2003b; Stanton et al., 2002; Steginga & Occhipinti, 2006; Tjemsland et al., 1998; Uchitomi et al., 2003) assessed point prevalence of the outcome between 12 and 18 months after baseline, five (de Leeuw et al., 2001; Lockefer & De Vries, 2013; Mehta et al., 2003; Ristvedt & Trinkaus, 2009; Scharloo et al., 2010) after two years, and two (Aarstad et al., 2005; Lebel et al., 2008) after five or more years. In addition, four studies (Bárez et al., 2009; Carlson et al., 2013; Lee et al., 2011; Schroevers et al., 2003a) assessed predictors of change in depression over the follow-up period, one study (Burgess et al., 2005) assessed predictors of one or more episodes of anxiety or depression between two and five years after diagnosis and one study assessed predictors of persistent depression (defined as scores of 10 or higher on two or more BDIs administered at least six months apart) (Karnell et al., 2006).
2.3.1 Socio-demographic and clinical predictors:

2.3.1.1 Age.

Age was a significant predictor in only four papers. Of these, one study (Burgess et al., 2005) reported that, between two and five years post-diagnosis of breast cancer, younger age predicted one or more episodes of anxiety or depression (SCID-III). However, the statistic presented (younger age Hazards Ratio 0.96) suggested the opposite to be the case, thus preventing any conclusions being drawn from this study. The remaining three studies also reported younger age to be a significant predictor. In two of these studies age, entered as a continuous variable, predicted trauma symptoms (IES) (Tjemsland et al., 1998) and emotional distress (POMS total) (Stanton et al., 2002) one year after breast cancer surgery, while in the final one age, entered as a categorical variable, predicted anxiety (HADS-A) 18 months after diagnosis of head & neck cancer (Neilson et al., 2013).

2.3.1.2 Gender.

In the ten studies with mixed-gender samples (Carlson et al., 2013; de Graeff et al., 2000; de Leeuw et al., 2000, 2001; Enns et al., 2013; Gustavsson-Lilius et al., 2007; Hammerlid et al., 1999; Neilson et al., 2013; Ranchor et al., 2002; Ristvedt & Trinkaus, 2009; Schroevers et al., 2003a, 2003b; Uchitomi et al., 2003) only two found gender to be a significant predictor. In head and neck cancer patients, female gender predicted emotional distress (QLQ-C30-EF)(de Graeff et al., 2000) but not depression (CES-D)(de Graeff et al., 2000; de Leeuw et al., 2000, 2001) twelve months after the start of treatment. However, by two and three years post-treatment, female gender did predict depression in this sample (de Leeuw et al., 2001). The second study, which employed a heterogeneous sample (Enns et al., 2013), also reported that female gender predicted ‘occasional’ (defined as exceeding clinical cut-offs on the PSS
CAN at one or more time-points) or ‘continuous’ (defined as exceeding PSSCAN clinical cut-offs at all time-points) anxiety or depression during the 12 months since diagnosis.

2.3.1.3. Education and social class.

Only two other demographic variables (educational level and social class) were reported in any study to be prospectively related to distress twelve or more months after diagnosis. In one mixed diagnoses cohort, patients with more education (not clearly defined) reported a greater decrease in depression over time (between three and 15 months since diagnosis) (Schroevers et al., 2003a). The authors postulate that patients with a higher level of education have greater access to social support, and in turn greater feelings of control, optimism and self-esteem (Schroevers et al., 2003a). An alternative explanation is that patients with a higher level of education are more able to understand and integrate the information they are given, facilitating better adjustment over time. However, this paper only considered clinical and demographic factors in isolation. Therefore it is not known whether educational level would remain related to the course of depression if other social or psychological factors were controlled. In a study of lung cancer patients (Uchitomi et al., 2003), lower education level (junior high school or less) predicted depression 12 months after treatment. The other 10 studies found no effect of education on depression and/or anxiety (Carlson et al., 2013; Den Oudsten et al., 2009; Enns et al., 2013; Gustavsson-Lilius et al., 2007; Lockefer & De Vries, 2013; Schou et al., 2004), emotional distress (Lebel et al., 2008; Ranchor et al., 2002) or trauma symptoms (Andrykowski & Cordova, 1998; Ristvedt & Trinkaus, 2009).

Finally, lower social class (not clearly defined) was found to significantly predict psychiatric status 12 months after mastectomy for breast cancer (Dean, 1987). This variable was not assessed in any other of the included studies.
2.3.1.4 Treatment type.

Most studies (7/9) (Andrykowski & Cordova, 1998; Bárez et al., 2009; Bleiker et al., 2000; Burgess et al., 2005; Dean, 1987; Lockefeer & De Vries, 2013; Schou et al., 2004) exploring treatment type as a predictor of distress after breast cancer found no effect of either surgery type or adjuvant therapy. Of the two studies that did find an effect, one (Den Oudsten et al., 2009) found that having undergone breast conserving surgery rather than mastectomy or no surgery at all, predicted depression 12 months (de Leeuw et al., 2001) after diagnosis. The other (Tjemsland et al., 1998) found no effect of surgery type but did report that having radiotherapy versus not having radiotherapy predicted reduced trauma symptoms (IES) at 12 months. There is no obvious explanation for these findings. Indeed, they are counterintuitive as one might expect patients receiving more extensive surgery or radiotherapy in addition to surgery to fare worse. The authors postulate that these findings may be due to a subset of patients becoming concerned that breast conservation will not completely eradicate their cancer (Den Oudsten et al., 2009) or that daily contact with others in similar circumstances during radiotherapy treatment gives patients a greater opportunity to work through their concerns (Tjemsland et al., 1998). However, in both studies the amount of variance explained is small. This, taken together with the greater number of studies reporting no effect, suggests that that these findings could be attributable to measurement error.

Similarly, in other cancer populations, only two of nine studies (Carlson et al., 2013; de Graeff et al., 2000; de Leeuw et al., 2000, 2001; Enns et al., 2013; Grassi et al., 1997; Karnell et al., 2006; Mehta et al., 2003; Neilson et al., 2013; Ristvedt & Trinkaus, 2009; Schroevers et al., 2003a, 2003b; Uchitomi et al., 2003) that explored treatment as a predictor reported an effect. De Graeff et al (de Graeff et al., 2000) reported that combination therapy versus single
treatment modality predicted emotional distress (QLQ-C30 –EF) and depression (CES-D) twelve months after treatment for head & neck cancer. However, in this analysis, treatment was considered only as part of a constructed group variable (combining site, stage and treatment). Therefore, it cannot be assumed that treatment was responsible for the observed effect despite the authors’ assertion that it was. Patients receiving combination therapy were more likely to be female and had more advanced disease than those requiring single treatment (either surgery or radiotherapy). Furthermore, in two further papers reporting on the same prospective cohort (de Leeuw et al., 2000, 2001), treatment did not predict depression (CES-D) when considered in isolation. In a more recent study with a mixed diagnosis cohort (Enns et al., 2013), receipt of chemotherapy predicted occasional anxiety or depression (i.e. exceeding clinical PSSCAN cut-offs on one or more assessment in the 12-months since diagnosis) but not continuous distress (i.e. exceeding PSSCAN cut-offs at every assessment). This finding may arise because some, but not all, of the assessment periods are likely to correspond with the stage at which patients are receiving chemotherapy, the side effects of which often cause a considerable physical and emotional burden during treatment but which is alleviated once treatment ends. In support of this explanation, a second paper reporting the same study (Carlson et al., 2013) found no predictive effect of chemotherapy on overall reduction in anxiety or depression over the 12 months since diagnosis. It did find, however, that not having surgery predicted greater improvement in depression over time, and patients with higher levels of depression or anxiety around diagnosis reported a greater reduction in distress over time if they had not received radiotherapy.

2.3.1.5 Tumour characteristics

Most studies assessing tumour-related characteristics (i.e. stage, size, site) found they did not predict long-term distress (Bleiker et al., 2000; Burgess et al., 2005; Den Oudsten et al.,
2009; Grassi et al., 1997; Gustavsson-Lilius et al., 2007; Hammerlid et al., 1999; Karnell et al., 2006; Mehta et al., 2003; Ristvedt & Trinkaus, 2009; Scharloo et al., 2010; Schou et al., 2004; Uchitomi et al., 2003). One exception to this was a study in a mixed diagnosis sample (Schroevers et al., 2003a), which found that patients with lower stage disease had a greater reduction in depression (CES-D) over time (between three and 15 months since diagnosis). In line with this, a study in breast cancer (Andrykowski & Cordova, 1998) found that more advanced disease at diagnosis predicted more extensive trauma symptoms (PCL-C) among post-operative patients assessed between six months and six years after surgery. Another study to report an effect was a three-year prospective study of head and neck cancer patients (de Graeff et al., 2000; de Leeuw et al., 2000, 2001). The authors stated that they expected cancer stage to influence post-treatment distress only as a result of physical morbidity caused by the treatment it dictates (de Graeff et al., 2000). In line with this, cancer stage predicted depression (CES-D) 12 months after treatment in one paper (de Leeuw et al., 2000) when it was entered first in the regression (alongside treatment type), but not in a later paper (de Leeuw et al., 2001) where it was entered after controlling for treatment type and recurrence, and alongside all other pre-treatment variables. This later paper also found that cancer stage failed to predict depression at two years, although it did contribute a small amount (2%) to the total variance (65%) explained at three years. Such inconsistency suggests it may not be a robust predictor.

2.3.1.6 Physical health

Five studies found measures of physical health status that predicted persistent emotional distress. Three breast cancer studies found effects of pre-diagnosis physical health including: self-reports of being on long term medication in the ten years before diagnosis which predicted trauma (IES) 12 months after diagnosis (Tjemsland et al., 1998), and high
pre-diagnosis fatigue which predicted depression (CES-D) at 12 months (Den Oudsten et al., 2009), and two years after diagnosis (Lockefer & De Vries, 2013) even after controlling for baseline depression. In addition, lower pre-treatment physical functioning (KPS) predicted worse emotional functioning (QLQ-C30-EF) 12 months after treatment for head and neck cancer (de Graeff et al., 2000), while post-operative sleep and health complaints predicted more intrusive thoughts (IES) 18 months after surgery for breast cancer (Bleiker et al., 2000). However, in contrast to these findings, a further five studies that considered similar indicators of baseline physical health found no predictive effects. These indicators included: level of physical function (KPS) at diagnosis (Hammerlid et al., 1999); pre-operative menopausal status (Dean, 1987); single items measuring post-operative perceived health (Lebel et al., 2008); single items measuring post-operative general health, physical function and pain (Millar et al., 2005); and scales measuring post-operative physical functioning (as measured by the QLQ-C30) (Bárez et al., 2009).

2.3.1.7. Summary of socio-demographic and clinical predictors

In summary, although it has often been suggested that baseline demographic and clinical factors may help to identify individuals vulnerable to long-term or persistent distress, the findings are inconsistent. The lack of evidence for cancer and treatment-specific variables as predictors of emotional distress supports the view that emotional distress is more likely to be predicted by factors relating to the individual rather than the disease. Socio-demographic risk factors for distress after diagnosis of cancer are reported to be similar to those in the general population (Burgess et al., 2005). However, while some of the studies described above support this view, the predominance of negative findings make it unwise to regard any of the demographic variables as reliable predictors of persistent distress. Finally, as demographic
and clinical variables can rarely be modified, they can only be markers of vulnerability. They offer little insight into how such distress may be reduced or prevented.

2.3.2 Social and environmental predictors

Seventeen studies explored social factors within the first three months of diagnosis as potential predictors of persistent distress.

2.3.2.1 Availability / characteristics of significant others and the social network

One early study (Dean, 1987) reported that 12 months after mastectomy married women were more likely than single women to be cases (anxiety or depression) on the Present State Examination, which suggests that the presence of a ‘significant other’ is not necessarily beneficial. In line with this a study of head and neck cancer patients (Hammerlid et al., 1999) also found that living alone did not predict anxiety or depression cases on the HADS. Other studies with either breast or heterogeneous cancer populations found no effect of marital status / living with a partner (Carlson et al., 2013; Den Oudsten et al., 2009; Enns et al., 2013; Lockefer & De Vries, 2013). Just one study explored partner characteristics as a potential predictor of persistent distress. Specifically they assessed the relationship between ‘sense of coherence’ (SOC)¹ and emotional distress and found that, while partners’ SOC predicted their own depression (BDI) and anxiety (EMAS-State) 14 months after diagnosis, this had no cross-over effect on the patients’ (Gustavsson-Lilius et al., 2007). Finally, one study (de Leeuw et al., 2001) reported that a smaller (formal) social network predicted head and neck cancer patients who became depressed (CES-D) at one year, and a smaller (informal) social network predicted those who became depressed at three years post-treatment, although the individual contribution to variance explained was small (7% at one year, 1% at three years).

¹ Sense of coherence, defined as a global orientation based on an individual’s perception that: (1) a stressful event is structured, predictable and explicable; (2) the resources are available to meet the demands of the event; and (3) the demands and challenges are worth the investment (Antonovsky, 1987)
2.3.2.2 Social Support

The concept of social support refers to an individual’s satisfaction with their perceived ‘available’ or ‘received’ support. Several dimensions of support have been identified, including: emotional support (expressing concern or listening to the individual); appraisal support (giving assurance of an individual’s intrinsic worth as a human being, allowing opportunities for social comparison or providing feedback on the efficacy of a task performed); informational support (giving advice and direction); and instrumental support (giving actual physical assistance) (House, Umberson, & Landis, 1988). Four out of eleven studies investigating a prospective relationship between social support and distress found that a lack of some specific types of support predicted later anxiety and/or depression. In breast cancer, a lack of a confiding relationship (which may be equated with emotional support) around diagnosis predicted anxiety and/or depression cases (SCID) two to five years later after controlling for baseline distress (Burgess et al., 2005), and deterioration in emotional support over the 12 months since diagnosis was associated with deterioration in depression (Lee et al., 2011). In head and neck cancer, after controlling for baseline depression, lack of received and available emotional support (de Leeuw et al., 2001) and lack of available appraisal support (de Leeuw et al., 2000, 2001) pre-treatment predicted depression (CES-D) one year later. In addition, lack of available instrumental support and a lack of openness to discuss cancer within the nuclear family predicted depression two years after treatment (de Leeuw et al., 2001), and a lack of received emotional support predicted depression three years after treatment (de Leeuw et al., 2001). Finally, in a mixed diagnosis cohort a lack of problem-focussed support (which may be equated with informational support) three months after diagnosis and more negative interactions with others predicted depression (CES-D) 15
months after diagnosis after controlling for depression at baseline (Schroevers et al., 2003b). However, in contrast to the other studies mentioned above, a lack of perceived emotional support was not predictive. These differences in the specific types of support which predict reduced distress may reflect changing social support needs over time since diagnosis. However, since the contribution to variance explained in distress across all studies and at different stages in the cancer journey is small (1-4%), this inconsistency may just as likely be due to measurement error. Finally, in contrast to the four studies described above, and contrary to what might be expected, one study in a heterogeneous population of cancer patients found that higher levels of supportive interactions before diagnosis predicted emotional distress (GHQ-12) twelve months after diagnosis (Ranchor et al., 2002). The remaining six studies failed to find any predictive effects of social support either for depression (Adachi et al., 2014; Dean, 1987; Den Oudsten et al., 2009) or emotional distress (Bleiker et al., 2000; Lebel et al., 2008; Tjemsland et al., 1998). This discrepancy with the previous studies may be due to differences in the measures used. The measures used in studies which found an effect were those that clearly distinguished different types of social support and, in particular, assessed perceived emotional support without prescribing the source of that support. In contrast, studies that found no effect either used more general social support scales or subscales (Adachi et al., 2014; Den Oudsten et al., 2009; Lebel et al., 2008), were restricted to asking about partner support (Tjemsland et al., 1998), or asked about social interactions (Bleiker et al., 2000) (Den Oudsten et al., 2009; Ranchor et al., 2002), impaired social, work or family functioning (Dean, 1987; Tjemsland et al., 1998).

2.3.2.3 Non-cancer-related difficulties and negative life events

Breast cancer survivors who reported more non-cancer related difficulties before treatment were more likely to experience borderline or case anxiety and/or depression (on the
SCID) in the longer term (two to five years post diagnosis) (Burgess et al., 2005). However, in contrast, having experienced more severe (Burgess et al., 2005) or negative life events (Tjemsland et al., 1998) before cancer diagnosis or having had previous cancer or other illness-related experiences (Schou et al., 2004) were not predictive. This discrepancy may reflect measurement issues. Asking about life events and illness experiences implies discrete events in the past, which may not have any lasting consequences, meaning the individual may be unaffected at the time of diagnosis. By contrast, ‘non-cancer related difficulties’ may be more likely to refer to ongoing problems which may be compounded by receiving a diagnosis of cancer.

2.3.2.4 Summary of social (environmental) predictors

There is little evidence to support any of the social or environmental variables assessed as reliable predictors of longer-term emotional distress.

Burgess and colleagues (Burgess et al., 2005) concluded that the social/environmental risk factors for depression and anxiety after breast cancer are the same as those for the general female population (i.e. lack of social support and non-cancer related difficulties). That is, social support affects psychological well-being regardless of the presence or absence of threat (Thoits, 1982, 1995). In support of this view, one study (Schroevers et al., 2003a, 2003b) reported that the relationship between social support and depression (CES-D) was similar between patients and population controls, with one difference: that the lack of problem-focused emotional support three months after diagnosis was more strongly related to depression 15 months later among patients than among population controls.

Furthermore, when effects are found it is difficult to establish whether reduced social support causes persistent distress or whether baseline distress produces this effect by reducing the individuals’ ability to access support. In all of the studies that found a positive effect of
social support variables after controlling for baseline distress (Burgess et al., 2005; de Leeuw et al., 2000, 2001; Schroevers et al., 2003b) baseline distress was the largest predictor. This suggests that psychological variables may be more useful predictors of persistent distress than social or environmental ones.
Table 2.2 Summary of study design and significant findings from included studies (grouped by outcome (DV))

<table>
<thead>
<tr>
<th>Study</th>
<th>Cancer Diagnosis</th>
<th>Time 1</th>
<th>T2 (N months later)</th>
<th>Dependant Variable (DV)</th>
<th>DV controlled@T1</th>
<th>Analysis</th>
<th>Medical /demographic Independent Variable</th>
<th>Social /Environmental independent Variable</th>
<th>Psychological Independent Variable</th>
<th>Significant Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>DV - ANXIETY/DEPRESSION CASE</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dean 1987</td>
<td>Br</td>
<td>Pre-op</td>
<td>12</td>
<td>Anx or Dep case (PSE)</td>
<td>Y</td>
<td>Stepwise LR</td>
<td>Menopausal status, Trt</td>
<td>Marital status, Social class, confidant</td>
<td>Pre-op case (RDC/GHQ) Coping style Psy Trt Attitude</td>
<td>Lower social class OR 4.57 Pre-op case OR 4.37 Perimenopausal OR 8.9 Prev Psy Trt OR 7.56 Marital status (single)OR 5.85 (results not clear)</td>
</tr>
<tr>
<td>Ramirez et al 1995</td>
<td>Br</td>
<td>Pre-op</td>
<td>12</td>
<td>Anx or Dep case (PSE)</td>
<td>N</td>
<td>ROC</td>
<td>None</td>
<td>None</td>
<td>ED (HADS&gt;10)</td>
<td>HADS &gt;10 identified 83% cases</td>
</tr>
<tr>
<td>Hammerlid et al 1999</td>
<td>HN</td>
<td>Diag</td>
<td>12</td>
<td>Anx or Dep case (HADS)</td>
<td>Y</td>
<td>LR</td>
<td>KPS, Age, Gender, TSite, TStage</td>
<td>Living status</td>
<td>Baseline Anx or Dep case</td>
<td>Anx or Dep case at diagnosis - no data provided</td>
</tr>
<tr>
<td>Shroevers et al, 2003</td>
<td>Mix</td>
<td>Post- Trt (3 month post-diag)</td>
<td>15</td>
<td>Change in Dep case status over time (CES-D)</td>
<td>N</td>
<td>Repeated measures Anova</td>
<td>TSite, TStage, Trt, Age, Gender, Marstat, Educ</td>
<td>None</td>
<td>None</td>
<td>Greater reduction in Dep with lower stage disease (stage 1 vs Stage 2 or higher) F [2,332], p&lt;.05 and higher education F [3,332], p&lt;.01</td>
</tr>
<tr>
<td>Schou et al 2004</td>
<td>Br</td>
<td>Diag</td>
<td>12</td>
<td>Dep case (HADS)</td>
<td>Y</td>
<td>LR</td>
<td>Educ, Tgrade,Trt</td>
<td>None</td>
<td>Optimism /pessimism, +VE Trt expectation, Anxi/Dep, Coping</td>
<td>Dep Case: low Opt OR = 0.83; Anxious preoccupation OR = 3.2 Anx Case: low Opt OR = 0.86; Anx OR = 2.71; fatalism OR = 3.16</td>
</tr>
<tr>
<td>Study</td>
<td>Design</td>
<td>Post-Treatment</td>
<td>Duration</td>
<td>Diagnosis</td>
<td>Measures</td>
<td>Confounding Factors</td>
<td>Analysis</td>
<td>Findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------</td>
<td>----------------</td>
<td>----------</td>
<td>-----------</td>
<td>----------</td>
<td>---------------------</td>
<td>----------</td>
<td>----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uchitomi et al, 2003</td>
<td>LC</td>
<td>Post-Trt (1 mth)</td>
<td>12</td>
<td>MD Case(SCID)</td>
<td>LR (backward)</td>
<td>Age, Gender, Educ, Pre-op smoking, Pre-op TStage, Trt, dyspnea, Forced expiratory volume (FEV)</td>
<td>Marital status, Pre-morbidity &amp; post Trt MD, post Trt ED</td>
<td>MD: Post-Trt MDD OR = 2.1, Educ OR = 2.4, ED: post-Trt distress ( \beta = 0.47 ), Pre-Trt MDD ( \beta = 0.18 )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Burgess et al, 2005</td>
<td>Br</td>
<td>-1 – 4 months post diag</td>
<td>24-60</td>
<td>MD / GAD Case (SCID)</td>
<td>LR -</td>
<td>Age, Tsize, Lymph nodes, Histology, Adj Trt</td>
<td>Confiding relationship, Severe life events &amp; difficulties</td>
<td>Premorbid Psychiatric Trt, GAD/MD since diag</td>
<td>Confiding relationship HR = 1.43; Younger HR = 0.96; Severe diff HR = 1.54; GAD or MD since diag HR = 1.55</td>
<td></td>
</tr>
<tr>
<td>Karnell et al, 2006</td>
<td>HN</td>
<td>Pre -Trt</td>
<td>3-12</td>
<td>Dep Case (BDI) for 6 month period</td>
<td>LR</td>
<td>TSite, TStage, Trt</td>
<td>Social disruption</td>
<td>Dep</td>
<td>Dep OR = 1.76</td>
<td></td>
</tr>
<tr>
<td>Enns et al, 2013***</td>
<td>Mix</td>
<td>Diagnos is</td>
<td>12</td>
<td>Anx Case, Dep Case (PSS CAN)</td>
<td>LR</td>
<td>Age, Gender, Income, Educ, Cancer type, Trt</td>
<td>Marital status,</td>
<td>Anx: Gender OR = 0.44, Chemotherapy OR = 2.66, Head &amp; Neck OR = 4.10; Dep: Chemotherapy OR = 2.24</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>DV - ANXIETY/DEPRESSION</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grassi et al, 1997</td>
<td>Mix</td>
<td>N/A</td>
<td>12-14</td>
<td>Dep (HDRS)</td>
<td>MR (Cross-sectional)</td>
<td>T stage, radio, Karnovsky Performance Status (KPS)</td>
<td>Social Sup Neg life events</td>
<td>Maladjustment (Diag), Psychiatric history (pre-morbid) Locus of Control</td>
<td>Maladjustment (diag) ( \beta = 0.55 ), KPS ( \beta = 0.25 ), Soc Sup ( \beta = 0.19 ), Psychiatric History(Pre) ( \beta = 0.16 )</td>
<td></td>
</tr>
<tr>
<td>De Leeuw et al, 2000**</td>
<td>HN</td>
<td>Pre-Trt</td>
<td>12</td>
<td>Dep (CES-D)</td>
<td>Hierarchical MR (stepwise)</td>
<td>TStage Trt, Age, Gender, Symptoms, General health, Physical functioning</td>
<td>Received/available support, perceived social network</td>
<td>Coping, Locus of Control, Dep</td>
<td>TStage R^2 = 0.4, Dep R^2 = 0.2, Available supp R^2 = 0.07, Social network R^2 = 0.04, Gen Health R^2 = 0.02</td>
<td></td>
</tr>
<tr>
<td>De Graeff et al</td>
<td>HN</td>
<td>Pre-Trt</td>
<td>12</td>
<td>EF (QLQ-C30-EF)</td>
<td>MR</td>
<td>Gender, Age</td>
<td></td>
<td>Dep</td>
<td>EF</td>
<td></td>
</tr>
<tr>
<td>Year</td>
<td>Study</td>
<td>Design</td>
<td>Time Period</td>
<td>Dep (CES-D)</td>
<td>Method</td>
<td>Group (site, stage, Trt), Karnovsky Performance Status (KPS), symptoms</td>
<td>Received/available support, perceived social network openness to discussion</td>
<td>Dep, Coping, Locus of Control</td>
<td>Physical function, Symptoms, Trt, Recur, Tstage, Age, Gender</td>
<td>Dep</td>
</tr>
<tr>
<td>------------</td>
<td>------------</td>
<td>--------</td>
<td>-------------</td>
<td>-------------</td>
<td>--------</td>
<td>------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>2000</td>
<td>De Leuw et al 2001</td>
<td>HN</td>
<td>Pre-Trt</td>
<td>12, 24,36</td>
<td>Dep (CES-D)</td>
<td>Y (stepwise)</td>
<td>MR (stepwise)</td>
<td>Physical function, Symptoms, Trt, Recur, Tstage, Age, Gender</td>
<td>Received/available support, perceived social network openness to discussion</td>
<td>Dep, Coping, Locus of Control</td>
</tr>
<tr>
<td>2001</td>
<td>Shroovers 2003</td>
<td>Mix</td>
<td>Post-Trt (3 month post-diag)</td>
<td>15</td>
<td>Dep (CES-D)</td>
<td>Y (Stepwise)</td>
<td>MR (Stepwise)</td>
<td>Sociodemog (not stated), Group membership (patient vs. control)</td>
<td>Social support</td>
<td>Dep, Self-esteem</td>
</tr>
<tr>
<td>2005</td>
<td>Aarstad et al 2005</td>
<td>HN</td>
<td>Diag</td>
<td>72+</td>
<td>Dep (BDI)</td>
<td>Y</td>
<td>Partial Correlation</td>
<td>None</td>
<td>None</td>
<td>Anx; Dep; Humour; Neuroticism (controlled)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mix</td>
<td>Diag</td>
<td>14</td>
<td>Anx (EMAS-State)</td>
<td>Y</td>
<td>SEM - path</td>
<td>Gender, Educ, Partner sense of SOC (life as Anx: Anx $\beta = 0.32$, in</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: The table represents a summary of research findings related to depression (Dep) and its predictors. The studies include various samples and methodologies, focusing on different aspects such as clinical trials, observational studies, and surveys. The results highlight the importance of considering various factors like Karnovsky Performance Status (KPS), gender, group membership, and symptomatology in understanding depression outcomes. The table also notes the role of emotional and social support variables, physical function, and coping strategies as significant predictors in different contexts.*
<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Design</th>
<th>Follow-up</th>
<th>Outcome</th>
<th>Predictor</th>
<th>Beta 1</th>
<th>Beta 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>al 2007</td>
<td></td>
<td>Analysis</td>
<td>TStage</td>
<td>Coherence (SOC)</td>
<td>Predictable/manageable/meaningful Anxiety, Dep</td>
<td>Direct effect of T1 SOC via T2 SOC $\beta = -0.30$, via T1 Anx $\beta = -0.15$</td>
<td>Direct effect of T1 SOC via T2 SOC $\beta = -0.30$, via T1 Anx $\beta = -0.22$</td>
</tr>
<tr>
<td>Den Oudsten et al 2009</td>
<td></td>
<td>Br Pre-diag 12</td>
<td>Dep (CES-D)</td>
<td>Y MR (stepwise)</td>
<td>Age, Empstat, Educ, Surgery, Adj Trt, Tstage, Tsize, Fatigue, Pain &amp; discomfort</td>
<td>Social support family status, Personality, Dep, trait Anx, Self-esteem, Body image, Cognitive function</td>
<td>Fatigue $\beta = 0.28$, Neuroticism $\beta = 0.16$, Surgery $\beta = 0.17$, Agreeableness $\beta = -0.15$ Dep 0.22</td>
</tr>
<tr>
<td>Couper et al 2010</td>
<td></td>
<td>Pro Pre-Trt 12</td>
<td>Dep Anx (BSI)</td>
<td>Y Y Hierarchical MR</td>
<td>HRQoL.</td>
<td>Dep, Anx, Coping</td>
<td>Dep: Dep $\beta = 0.48$, QoL-vitality $\beta = 0.24$, Fatalism $\beta = 0.13$ Anx: anx $\beta = 0.62$, QoL-vitality $\beta = -0.19$</td>
</tr>
<tr>
<td>Lee et al 2011</td>
<td></td>
<td>Br Diag 0-12 month</td>
<td>Deteriorated Dep (Zung-SDS)</td>
<td>N Hierarchical LR (Cross-sectional)</td>
<td>Age, Co morbidity, smoking, Menopausal status, Deteriorated finances, Radiotherapy, Deteriorated role functioning</td>
<td>Deteriorated emotional support</td>
<td>None No T1 Sig predictors. Deteriorated emotional support OR = 3.4, Deteriorated finances OR = 2.9, Deteriorated role functioning = 2.3</td>
</tr>
<tr>
<td>Carlson et al 2013***</td>
<td></td>
<td>Mix 1 month since diag 12</td>
<td>Improved Dep Improved Anx (PSSCAN)</td>
<td>Y Y MR (improved DV)</td>
<td>Age, Gender, Source of income, Educ, Ethnic/cultural background Cancer diag, Trt</td>
<td>Psychosocial resources, marital, livstat, Anx, Dep</td>
<td>Improved Dep: Dep $\beta = -0.56$, No surgery $\beta = 0.08$, Improved Anx: Anx $\beta = -0.42$</td>
</tr>
<tr>
<td>Lockefer &amp;</td>
<td></td>
<td>Br Pre-24</td>
<td>Dep (CES-D)</td>
<td>Y Hierarchical</td>
<td>Age Educ, partner, Dep, trait Anx</td>
<td>Trait anxiety $\beta = 0.37$,</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Design</td>
<td>Time</td>
<td>Measure</td>
<td>Model Type</td>
<td>Variables</td>
<td>Outcomes</td>
<td>β Values</td>
</tr>
<tr>
<td>------------------------</td>
<td>--------</td>
<td>-------</td>
<td>---------</td>
<td>------------</td>
<td>-----------------------------------------</td>
<td>----------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Defries 2013</td>
<td>diag</td>
<td>MR</td>
<td>empstat, Diag, Chemo, Radio, HT, Fatigue, Sleep quality</td>
<td>children, Fatigue</td>
<td>MR</td>
<td>Fatigue β = 0.23</td>
<td></td>
</tr>
<tr>
<td>Neilson et al 2013</td>
<td>HN</td>
<td>Pre–Trt</td>
<td>Dep Anx (HADS)</td>
<td>N</td>
<td>Multi-level mixed effects linear regression</td>
<td>Time, Age, Gender, Chemo, Pain, Symptoms, Trt</td>
<td>livstat, Dep: Symptoms β = - .24 Anx: Age β = 0.54, symptoms β = -0.09e</td>
</tr>
<tr>
<td>Adachi et al 2014</td>
<td>HN</td>
<td>Pre-op</td>
<td>Dep (HADS)</td>
<td>Y</td>
<td>MR (stepwise)</td>
<td>Gender, Facial disfigurement</td>
<td>Social support, Dep, Coping, Trauma</td>
</tr>
<tr>
<td>Andrykowski &amp; Cordova 1998</td>
<td>Br</td>
<td>N/A</td>
<td>Trauma (PCL-C)</td>
<td>N</td>
<td>Hierarchical MR (stepwise) Cross-sectional</td>
<td>Educ, TStage at diag, Age at diag, Comorbidity, Surgery, Chemo, Current tamoxifen,</td>
<td>Social Support, Dep history, Pre-morbid stressors</td>
</tr>
<tr>
<td>Tjemsland et al 1998</td>
<td>Br</td>
<td>Pre-op</td>
<td>Trauma (IES)</td>
<td>Y</td>
<td>MR</td>
<td>Age, Adj Trt, Health problem / Medications in last 10yrs, Recurrence</td>
<td>Work/social/family function, Lack of crisis support</td>
</tr>
<tr>
<td>Bleiker et al 2000</td>
<td>Br</td>
<td>Post-op</td>
<td>Intrusion Avoidance (IES)</td>
<td>Y</td>
<td>MR (Backward)</td>
<td>Age, Surgery, Lymph node s, Adj Trt, Sleep, Health complaints (SCL-90)</td>
<td>Life events, Perceived social supp (SEC)</td>
</tr>
<tr>
<td>Elkit &amp; Blum</td>
<td>Br</td>
<td>Post op</td>
<td>Trauma (HTQ)</td>
<td>N</td>
<td>Hierarchical</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>
### DV - EMOTIONAL DISTRESS/FUNCTIONING

<table>
<thead>
<tr>
<th>Year</th>
<th>Design</th>
<th>Time</th>
<th>Measure</th>
<th>Level</th>
<th>Factors</th>
<th>Variables</th>
<th>p-value or Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>2011</td>
<td>post diag</td>
<td></td>
<td>MR</td>
<td></td>
<td>defence style,</td>
<td>Emotional coping, Avoidance, Negative affectivity</td>
<td>Negative affectivity $\beta = 0.55$</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Body image, Appearance</td>
<td>Body integrity concern</td>
<td>None sig</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>body image, Appearance</td>
<td>Body integrity concern</td>
<td>None sig</td>
</tr>
<tr>
<td>Carver et al 1998</td>
<td>Br</td>
<td>Pre-Trt</td>
<td>12</td>
<td>ED (Affects Balance Scale)</td>
<td>N</td>
<td>MR</td>
<td>Age</td>
</tr>
<tr>
<td>Ranchor et al 2002</td>
<td>Mix</td>
<td>Pre-morbid</td>
<td>12</td>
<td>ED (GHQ-12)</td>
<td>Y</td>
<td>Hierarchical MR</td>
<td>Age, Gender, Educ</td>
</tr>
<tr>
<td>Stanton et al, 2002</td>
<td>Br</td>
<td>Pre-op</td>
<td>12</td>
<td>ED (POMS) FOR (Fear of Recurrence Scale)</td>
<td>Y</td>
<td>Hierarchical MR</td>
<td>Age</td>
</tr>
<tr>
<td>Millar et al 2005</td>
<td>Br</td>
<td>Post-op</td>
<td>12</td>
<td>ED (GHQ-28)</td>
<td>Y</td>
<td>Hierarchical MR (Stepwise)</td>
<td>Age, Deprivation, General health, Physical function, Pain</td>
</tr>
<tr>
<td>Steginga &amp; Occhipinti 2006</td>
<td>Pr</td>
<td>Pre-Trt</td>
<td>12</td>
<td>Decisional distress (Decisional conflict scale)</td>
<td>Y</td>
<td>Hierarchical MR</td>
<td>None</td>
</tr>
<tr>
<td>Lebel et al, 2008**</td>
<td>Br</td>
<td>Post-Trt</td>
<td>72</td>
<td>ED (POMS) Trauma (IES)</td>
<td>Y</td>
<td>Hierarchical MR (stepwise)</td>
<td>Age, Educ, 2nd cancer, Perceived health</td>
</tr>
<tr>
<td>Barez et al 2009</td>
<td>Br</td>
<td>Post-op</td>
<td>Chan</td>
<td>ED (HADS &amp; POMS)</td>
<td>Y</td>
<td>LGC</td>
<td>Age, Trt,</td>
</tr>
</tbody>
</table>

37
| Risvedt (Ristvedt & Trinkaus, 2009) & Trinkaus 2009 | Rectal | Post-op | 24-60 | EF (FACT) Trauma (IES) | N | LR | Gender, Age, Educ, TStage, Ostomy, Faecal incontinence | None | Trait anxiety | EF: Trait anxiety OR 1.45, Trauma: Faecal Incontinence OR 1.05 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Scharloo et al 2010 | HN | Diag | 24 | EF (QLQ-C30 - EF) | Y | Hierarchical MR (Forced / stepwise) | Age, Tstage | None | Illness perceptions, EF | EF: Trait anxiety OR 1.45, Trauma: Faecal Incontinence OR 1.05 |
| Kohler et al, 2014 | Pr | Pre-Trt | 12 post surgery | ED (HADS) | Y | MR | Concurrent urinary symptoms and erectile dysfunction | None | ED | ED = 0.48, Concurrent urinary symptom OR 0.39 |
| Mehta et al 2003 | Pr | Pre-Trt | NOT clear of prospective or cross-sectional | Fear of Cancer Recurrence (FCR) | N | MR | Age, Clinical characteristics, Trt, HRQoL (SF-36) Symptoms | None | None | QoL-physical R²c = 0.27, QoL-mental R²c = 0.04 (no data) |

**N.B:** Diag = diagnosis; Op = Operation/surgery; Trt = Treatment; MD = Major Depressive Disorder; GAD = Generalised Anxiety Disorder; Anx = Anxiety; Dep = Depression; Trauma = Trauma symptoms; EF = Emotional Functioning; ED = Emotional distress; Opt = Optimism; ROC = Receiver Operating Curve; MR = Multiple regression, LR = Logistic regression; R²c - R² change; β = Beta; OR = Odds ratio; HR = Hazards Ratio; R = Correlation; Only coefficients sig p<.05 are shown; HRQoL = Health related Quality of Life; Tstage = Tumour stage; Tsize = Tumour size; TSite = Tumour site; Recur = cancer recurrence; Adj Trt = adjuvant treatment; Chemo = Chemotherapy, Radio = radiotherapy, HT = Hormone Therapy; Educ = Education; Marstat = Marital status, Livstat = Living arrangements/status; Empstat = Employment status; Grey font = Cross-sectional
2.3.3 Psychological predictors

2.3.3.1 Emotional distress

Most of the studies reviewed (27/34) examined whether baseline measures of distress (around or within three months of diagnosis) predicted distress at follow-up.

In most cases, the same measure of distress was used for both the baseline and follow-up assessments and this was the largest or only significant predictor. There were a few exceptions to this where baseline levels of distress did not predict follow-up distress. Two studies in breast cancer found that pre-diagnosis (Lockeefeer & De Vries, 2013) and pre-operative (Schou et al., 2004) depression (CES-D & HADS respectively) did not predict depression at follow-up (24 and 12 months later respectively). Although in the former study (Lockeefeer & De Vries, 2013), depression two years post-diagnosis was predicted by trait anxiety assessed just prior to diagnosis. In another study (Lebel et al., 2008), post-treatment emotional distress (POMS) did not predict emotional distress six years after surgery for breast cancer. Lastly, although depression after diagnosis of head and neck cancer initially predicted depression (BDI), six years later this was no longer the case after baseline neuroticism was controlled (Aarstad et al., 2005).

Five studies (Andrykowski & Cordova, 1998; Grassi et al., 1997; Ramirez et al., 1995; Ristvedt & Trinkaus, 2009; Tjemsland et al., 1998) used a different measure of distress to predict persistent distress, instead of the outcome measure and without controlling for it at baseline. Of these, three found a positive effect. One cross-sectional study reported that maladjustment at diagnosis and pre-morbid psychiatric history predicted depression (HDRS) in a heterogeneous cancer population twelve months after diagnosis (Grassi et al., 1997). The other two found that trait anxiety (assessed post-operatively) predicted emotional functioning (FACT-G - EF) between two and five years after surgery for rectal cancer, (Ristvedt & Trinkaus, 2009), and that pre-operative emotional distress (HADS) predicted psychiatric
‘caseness’ (PSE) 12 months after surgery for breast cancer (Ramirez et al., 1995). In contrast, the remaining two studies (Andrykowski & Cordova, 1998, Tjemsland et al., 1998), found that pre-morbid psychiatric diagnosis (depression and PTSD respectively) did not predict persistent trauma symptoms (PCL-C and IES respectively) after surgery for breast cancer.

These findings taken together provide fairly compelling evidence to support the view that baseline distress is a reliable predictor of persistent distress, especially where the same measure is used at both assessment points.

2.3.3.2 Self-esteem

Two studies included self-esteem as a predictor. One (Den Oudsten et al., 2009) found no effect of pre-diagnosis self-esteem on 12-month depression (CES-D) in breast cancer patients. However, the other study in a mixed cohort (Schroevers et al., 2003b) found that negative, but not positive, self-esteem assessed three months after diagnosis predicted 12-month depression (CES-D). However, the data for this latter finding was not reported. Therefore, it cannot be concluded that there is any reliable evidence to support the role of self-esteem as a predictor of persistent distress.

2.3.3.3 Coping

Ten studies (Adachi et al., 2014; Couper et al., 2010; de Leeuw et al., 2000, 2001; Dean, 1987; Elklit & Blum, 2011; Lebel et al., 2008; Millar et al., 2005; Schou et al., 2004; Stanton et al., 2002; Steginga & Occhipinti, 2006) looked at whether coping predicted distress at follow-up. Studies employed different measures of coping, all of which aimed to assess the cognitive and behavioural strategies used to manage the stress of cancer.
Five studies across breast (Dean, 1987; Elklit & Blum, 2011; Millar et al., 2005), prostate (Steginga & Occhipinti, 2006), and head and neck (Adachi et al., 2014) cancer found no effect of pre-operative, or immediately post-operative, coping. Another study, in head and neck cancer, reported results that were inconsistent. In one paper (de Leeuw et al, 2000) the authors reported that pre-treatment coping did not predict depression (CES-D) twelve months later, whereas in another (de Leeuw et al 2001) they reported that coping through religion explained 2% of the variance in depression 12 months after treatment, while palliative coping explained 2% of the variance after three years. It is likely that this discrepancy in results is due to small differences in the way the regression analyses were conducted between papers. Nonetheless it suggests that pre-treatment coping was not a reliable predictor of persistent post-treatment depression in this sample.

Coping did predict persistent emotional distress (after controlling for baseline distress) in the remaining four studies. Pre-treatment fatalism predicted depression (BSI) 12-months later among prostate cancer patients, although no aspect of coping predicted anxiety (BSI) (Couper et al., 2010). Pre-operative ‘acceptance coping’ and interaction of pre-operative hope with several aspects of coping, including turning to religion, ‘problem focussed’ coping and seeking social support predicted less emotional distress (POMS) 12 months after diagnosis of breast cancer (Stanton et al., 2002), while, in another study, fatalism at diagnosis predicted greater anxiety (HADS), and helpless/hopeless coping at diagnosis predicted greater depression (HADS) among breast cancer patients 12-months after surgery (Schou et al., 2004). More surprising is the finding that ‘positive problem solving’ three months after diagnosis of breast cancer predicted greater emotional distress (POMS) six years later (Lebel et al., 2008). The authors of this study speculated that this approach to coping predicted distress because of a detrimental effect of the pressure to find positive outcomes and ‘think positive’ that many cancer patients encounter early in the cancer journey. Participants in this
study were being followed up six years after a randomised trial of a coping skills
intervention. While the trial found no intervention effect, it is possible that being encouraged
towards ‘positive problem solving’ at an early stage may encourage overly optimistic
expectations and discourage acceptance of the reality of the disease and its implications.
Such approaches could be counterproductive, potentially interfering with normal adjustment
processes, especially if the patient experiences further disease- or treatment-related
difficulties.

Due to the inconsistency of findings, the considerable differences in the ways coping was
operationalised and the varying approaches used in design and analysis between studies
(including different timing of assessment points, and different predictors/controls included in
the analysis) it must be concluded that the evidence for coping as a predictor of persistent
emotional distress is at best inconclusive.

2.3.3.4 Personality

Eight studies (Bleiker et al., 2000; Den Oudsten et al., 2009; Lebel et al., 2008; Millar et
al., 2005; Ranchor et al., 2002; Schou et al., 2004; Steginga & Occhipinti, 2006; Tjemsland et
al., 1998) explored various personality factors. Optimism/pessimism was assessed in four
studies using the Life Orientation Test (LOT). In newly diagnosed breast cancer patients
(Schou et al., 2004) after controlling for baseline distress, pessimism predicted cases of
anxiety or depression (HADS) 12 months later. The authors claim that pessimism was the
strongest individual predictor for both outcomes at 12 months and that the effects of
pessimism were mediated by fatalism and helpless/hopeless coping respectively. However,
the basis for drawing the former conclusion is not clear from the data presented. Furthermore,
in contrast to this study, three other studies - one which investigated pre-treatment optimism
amongst prostate cancer patients (Staginga & Occhipinti, 2006) and two that investigated
post-operative optimism in breast cancer (Bleiker et al., 2000; Lebel et al., 2008) - found no effect on distress outcomes after controlling for baseline distress.

In contrast, all four of the studies that assessed baseline neuroticism (using either the Neuroticism-Extraversion-Openness-Five Factor Inventory (NEO-FFT) or the Eysenck Personality Inventory – Neuroticism (EPI-N)) found that it predicted 12-month outcomes after controlling for baseline distress including: emotional distress (GHQ) (Millar et al., 2005; Ranchor et al., 2002); depression (CES-D) (Den Oudsten et al., 2009); and trauma (IES) (Tjemsland et al., 1998). In addition, a study of patients with newly diagnosed head and neck cancer (Aarstad et al., 2005) reported that controlling for neuroticism reduced to non-significant the correlation between baseline depression and depression (BDI) at six years while it considerably strengthened the positive correlation between baseline humour (defined as a sensitivity to humorous messages and tendency to enjoy comical situations) and long-term depression. Unfortunately the independent contribution of neuroticism as a predictor of depression at six years was not reported.

These findings indicate that some aspects of personality are more reliable predictors of persistent distress than others. There is currently no evidence to support the role of optimism in predicting persistent distress, although there is some consistent support for the role of neuroticism.

2.3.3.5 Perceived control

Three studies (Bárez et al., 2009; de Leeuw et al., 2000, 2001; Gustavsson-Lilius et al., 2007) examined patients’ perceived control as a potential predictor of distress at follow-up. In one study of early breast cancer patients (Bárez et al., 2009), perceived control was a latent variable constructed from the mean scores of scales assessing: fighting spirit and helplessness; self–efficacy to overcome breast cancer related concerns; and personal
competence in interacting effectively with the environment. In this study, higher perceived control one week after surgery predicted faster improvement in emotional distress (latent variable derived from the combined scores of the POMS and the HADS) over the subsequent year, and change in perceived control over time was also associated with a corresponding change in emotional distress. In another study (Gustavsson-Lilius et al., 2007), this time of mixed diagnosis patients, a similar construct - sense of sense of coherence (SOC; see definition on page 27), assessed at diagnosis was negatively associated with anxiety and depression 14 months later even after controlling for baseline distress. Finally, a study in head and neck cancer patients (de Leeuw et al., 2000, 2001) investigated whether cancer locus of control (defined as a sense of internal control about the cause and course of cancer) predicted long term depression (CES-D) one to three years after treatment (19/21) but found no effect. These findings once again are difficult to interpret as the construct of ‘perceived control’ is operationalised and assessed quite differently across studies. Nonetheless there is little evidence to support the view that perceived control is a reliable predictor of persistent distress.

2.3.3.6 Illness appraisal

A final psychological predictor that has been explored across several studies is the individual’s appraisal of their illness. Two studies assessed patients’ appraisal of their cancer using the Illness Perception Questionnaire-Revised (IPQ-R) (Millar et al., 2005; Scharloo et al., 2010), and two used single items to assess patients’ appraisal of cancer as a challenge, threat or loss (Lebel et al., 2008; Schou et al., 2004). Only one study (Millar et al., 2005) using the IPQ-R found any effect of appraisal on distress at follow-up, reporting that greater perceived symptom burden (as measured by the IPQ-R illness identity scale) in the days after surgery predicted worse emotional distress (GHQ-28) 12 months later. In contrast, a study of
head and neck cancer patients found that none of the illness perception subscales (IPQ-R) at diagnosis were able to predict emotional distress two years later (QLQ-C30 – EF) (Scharloo et al., 2010), although several, including ‘illness identity’, ‘consequences’ and ‘behavioural attributions’ predicted other aspects of health-related quality of life (including physical, role and social functioning and global health). Once again, as with the section above, the differences between studies in how appraisal was assessed makes it difficult to compare across studies. However, this aside, as it stands it must be concluded that there is currently no evidence to support the view that patients’ baseline appraisal of their cancer predicts persistent distress.

2.3.3.7 Summary of psychological predictors.

In summary, it can be seen that of the psychological variables assessed, only baseline distress and neuroticism were consistently found to predict persistent emotional distress across studies. For the other psychological variables, particularly those where the findings were inconsistent across studies (i.e. coping), it is difficult to draw conclusions. However, based on the current evidence it seems unlikely that these are important predictors.

The finding that distress around, or within three months of, diagnosis predicts follow-up distress when using the same measure merely tells us that, for most of the patients experiencing longer-term distress, this is a problem that has been maintained from the start of their cancer journey. This lends support to guidelines recommending psychological assessment and appropriate intervention at the earliest stages of the cancer journey. It does not, however, provide any insight into how or why distress is maintained in some patients and not others. The finding by Aarstad et al (Aarstad et al., 2005) that neuroticism reduced to non-significant the effect of depression at diagnosis on depression at six years in head and
neck cancer patients is intriguing and may imply that it is the enduring characteristics of the individual that are key, rather than more transient emotional responses to the cancer.

2.4 Conclusion

Identifying baseline ‘risk’ or ‘vulnerability’ factors that predict persistent emotional distress after cancer has been largely unsuccessful. There is no consistent evidence to support any demographic, medical or social variables as potential predictors. The only psychological variable for which there is substantial supportive evidence - baseline emotional distress - is of limited clinical utility as it remains unclear why some people who are distressed at baseline experience persistent problems and others do not. There is clearly still a need for greater understanding of the causal mechanisms that underlie the development and maintenance of emotional distress after cancer. If we could identify the psychological processes that give rise to and maintain distress after diagnosis of cancer, we could better detect those who may have problems adjusting with usual care alone (Lynch, Steginga, Hawkes, Pakenham, & Dunn, 2008) and could develop interventions that target these processes in order to reduce vulnerability to persistent distress.
<table>
<thead>
<tr>
<th>Measure</th>
<th>Abbreviation</th>
<th>Outcome assessed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present State Examination</td>
<td>PSE</td>
<td>Anxiety/ Depression Cases</td>
</tr>
<tr>
<td>Structured Clinical Interview</td>
<td>SCID</td>
<td>Major Depressive Disorder/ Generalized Anxiety Disorder</td>
</tr>
<tr>
<td>The Psychological Screen for Cancer</td>
<td>PSSCAN</td>
<td>Anxiety / Depression Cases</td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Scale</td>
<td>HADS</td>
<td>Anxiety / Depression / Emotional Distress</td>
</tr>
<tr>
<td>The Center for Epidemiologic Studies Depression Scale</td>
<td>CES-D</td>
<td>Depression</td>
</tr>
<tr>
<td>Beck Depression Inventory</td>
<td>BDI</td>
<td>Depression</td>
</tr>
<tr>
<td>Hamilton Depression Rating Scale</td>
<td>HDRS</td>
<td>Depression</td>
</tr>
<tr>
<td>The Zung Self-Rating Depression Scale</td>
<td>Zung - SDS</td>
<td>Depression</td>
</tr>
<tr>
<td>Brief Symptom Inventory</td>
<td>BSI</td>
<td>Anxiety / Depression</td>
</tr>
<tr>
<td>Endler Multidimensional Anxiety Scales</td>
<td>EMAS-State</td>
<td>Anxiety</td>
</tr>
<tr>
<td>Post-traumatic Stress Disorder Checklist – Civilian version</td>
<td>PCL-C</td>
<td>Trauma Symptoms</td>
</tr>
<tr>
<td>Impact of Events Scale</td>
<td>IES</td>
<td>Trauma Symptoms</td>
</tr>
<tr>
<td>Harvard Trauma Questionnaire</td>
<td>HTQ</td>
<td>Trauma Symptoms</td>
</tr>
<tr>
<td>Profile of Mood State</td>
<td>POMS</td>
<td>Emotional Distress</td>
</tr>
<tr>
<td>General Health Questionnaire</td>
<td>GHQ- 28 / GHQ 12</td>
<td>Emotional Distress</td>
</tr>
<tr>
<td>The European Organization for Research and Treatment of Cancer (EORTC) Core Quality of Life Questionnaire – Emotional Functioning Scale</td>
<td>QLQ-C30 – EF</td>
<td>Emotional Functioning</td>
</tr>
<tr>
<td>Functional Assessment of Cancer Therapy-General – Emotional Functioning Scale</td>
<td>FACT-G - EF</td>
<td>Emotional Functioning</td>
</tr>
<tr>
<td>Affects Balance Scale</td>
<td>ABS</td>
<td>Emotional Distress</td>
</tr>
<tr>
<td>Decisional Conflict Scale</td>
<td>DCS</td>
<td>Decisional Distress</td>
</tr>
<tr>
<td>Fear of Recurrence Scale</td>
<td>FCR</td>
<td>Fear of Cancer Recurrence</td>
</tr>
<tr>
<td>Fear of Recurrence Scale</td>
<td>FOR</td>
<td>Fear of Cancer Recurrence</td>
</tr>
</tbody>
</table>
Chapter Three

Current theoretical approaches to understanding emotional distress after cancer:

An overview
Current views and models of adjustment in cancer have developed from ideas derived from both clinically orientated research and health psychology theory. As a result, several different models are evident in the literature. However, they are all based on the central tenet of the cognitive paradigm; that is, it is our interpretation of an event, rather than the objective consequences of it, that is central to our emotional and behavioral response. The most well-known models include: the cognitive model of adjustment to cancer (Moorey & Greer, 2002) and the Common-Sense Model of illness representations (Leventhal et al., 1980; Leventhal et al., 1984).

3.1 Cognitive model of adjustment to cancer

The cognitive model of adjustment to cancer (see Figure 3.1 below) is a clinically derived model. It was developed more than 20 years ago but has been influential in guiding psychological treatment for emotional distress in cancer ever since, particularly by providing the theoretical basis for the most well known adaptation of CBT in oncology - Adjuvant Psychological Therapy (APT).
The model itself is not specified in detail beyond the schematic shown above. Instead the authors draw heavily on coping theory and Beck’s schema theory in order to explain and understand emotional distress after cancer.

3.1.1 Coping theory

Lazarus and Folkman’s work on stress and coping (Lazarus & Folkman, 1984) is cited as a major influence on the cognitive model of adjustment as it contributes greatly to our understanding of the role of appraisal in mobilising coping behaviours (Moorey & Greer, 2002). In this work, Lazarus and Folkman defined psychological stress (or distress) as ‘a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being’.

---

Figure 3.1: Cognitive model of adjustment to cancer (Moorey & Greer, 2002)
(Lazarus & Folkman, 1984) p. 19). They identified two distinct forms of appraisal which together determine the individual’s response to an event or stressor: primary appraisal of the stressor itself; and secondary appraisal of the individual’s resources to manage it. Therefore, in the case of receiving a diagnosis of cancer when the disease is appraised as a challenge that can be met, individuals may feel positive and optimistic about their future. However, if it is appraised as a foregone conclusion or defeat that they can do anything about, they may feel that the prognosis is hopeless and the future bleak.

In cancer, the resulting patterns of thoughts feelings and behaviours have been categorised into five adjustment or coping styles (M. Watson et al., 1988), which are assumed to represent relatively stable attitudes and ways of behaving, similar to personality traits (Brennan, 2001)

- **Fighting spirit** – the individual sees cancer as a challenge over which they can exert some control, they take an active role in recovery
- **Avoidance or denial** – the individual denies the impact of the cancer, whether or not they can manage the diseases is irrelevant, they engage in behaviour to minimize its impact
- **Fatalism** – the individual interprets cancer as a threat over which they have no control, they adopt a coping style of passive acceptance
- **Helpless/Hopeless** - the individual appraises cancer as a threat with impending loss over which they have no control, they behave as if the negative outcome has already occurred
- **Anxious preoccupation** – the individual perceives cancer as a major threat causing an unpredictable future, they are unsure of their ability to manage the situation and engage in worry and reassurance seeking.
3.1.2 Schema theory

As in Beck’s Schema theory, appraisal within the cognitive model of adjustment to cancer is seen as being influenced by early experience and prior knowledge, as this shapes an individual’s beliefs and perceptions about cancer, its implications and their ability to cope. This theory emphasises that cancer is appraised in two areas; as a potential threat to the individual’s schemata (i.e. cognitive structures that guide the screening, encoding, organizing, storing and retrieving of information (Beck & Clark, 1988)) about survival and mortality and also to their self-schema.

It is suggested that, for individuals who become distressed, core positive beliefs about survival, the self and the world around are challenged or even shattered by a diagnosis of cancer and core negative beliefs activated, resulting in an overall negative appraisal of the situation and thus a negative emotional response. Schema theory asserts that once such a negative view is established it then has the potential to become self-perpetuating due to the tendency for negative schemata to bias information processing in order to preserve the negative view. This bias occurs via systematic logical errors in thinking or cognitive distortions, such as: all-or-nothing thinking, selective abstraction, arbitrary inference, overgeneralization, labelling and magnification/minimisation (Beck et al 1979). Negative automatic thoughts (NATS) are generated that, seeming accurate and realistic to the individual at the time, influence their emotional responses both directly and indirectly as part of a complex interplay between thoughts, feelings, behaviour and physiological responses (see Figure 3.1). Although not necessarily always at the forefront of the individual’s attention, such NATS are available to consciousness and it is these, together with the associated emotions and maladaptive coping styles (described above), that are the focus of Adjuvant Psychological Therapy (APT) (Moorey & Greer, 2002).
3.1.3 Evidence supporting the cognitive model of adjustment to cancer

As stated above, the cognitive model of adjustment in cancer was developed predominantly to describe and guide the clinical application of CBT to people with cancer (Moorey & Greer, 2002). As such, the model itself has not been formally tested. Instead, support for the model is usually derived from research conducted into the effectiveness of CBT in oncology settings. However, as noted in Chapter 1, the results of this research are inconclusive.

Researchers have also sought support for the model through conducting research aimed at identifying specific ‘coping strategies’ that, in the cognitive model (see Figure 3.1), are thought to mediate the relationship between NATS and emotional distress. However, as described in Chapter 2, there is currently scarce evidence to support the role of any specific coping style as a reliable predictor of persistent distress after cancer. Indeed, it is argued that as cancer involves many different and changing challenges across its course, it is unlikely that just one type of coping style is helpful in all contexts and for all people (Brennan, 2001). Consequently, more recent research has moved away from focussing solely on coping towards understanding more about the individual’s appraisal of their cancer and how this guides selection of coping strategies. In particular, research has concentrated on whether the nature of the relationship between specific illness perceptions, coping and emotional well-being can add to our understanding of what causes emotional distress, and why it is maintained. Much of this work has been conducted using the framework of Leventhal’s Common Sense Model of self-regulation in health and illness (CSM (Leventhal et al., 1984)).
3.2 Common Sense Model of self-regulation in health and illness

Leventhal’s Common Sense Model (CSM) of self-regulation in health and illness ((Leventhal et al., 1980; Leventhal et al., 1984)) is a well-established and tested theoretical model (see (Hagger & Orbell, 2010) for a review) derived from health psychology research (see Figure 3.2). It focuses specifically on how individuals appraise their illness and how this influences their coping and subsequent emotional response. Consequently, it may be argued that it complements, rather than contradicts, the cognitive model of adjustment described above by specifically exploring how the variation in patients’ perception of their illness relates to coping style, and how this relates to emotional outcomes.

Figure 3.2: Common Sense Model (CSM) of health and illness (Hagger & Orbell, 2010)

First outlined by Leventhal and colleagues in the 1980s, (Leventhal et al., 1980; Leventhal et al., 1984) the CSM is conceptualised as a self-regulatory model whereby
individuals attempt to minimise the impact of ill-health and return to a state of ‘normality’. It is particularly attractive to health professionals because it views patients as active problem solvers, who may be helped to achieve better outcomes by facilitating a more adaptive understanding of their condition (Wearden & Peters, 2008). The CSM is organised into three stages: initially, it is suggested that an individual faced with ill-health develops two sets of mental representations, a cognitive representation or interpretation of the nature of the health threat (similar to the primary appraisal described above) and an emotional representation (i.e. fear). These representations then act in parallel to guide coping responses aimed at regulating both the threat itself and the individual’s emotional response to it. In the third stage, the success or failure of these coping strategies is continually monitored and the resulting appraisal (similar to secondary appraisal described above) modifies the initial representations and/or the individual’s selection of coping strategies.

Leventhal suggested that individuals develop cognitive representations in response to both abstract and concrete sources of information (Leventhal et al., 1980). In terms of the content of these representations, it is suggested that patients organise their thinking about illness around five key dimensions (Hagger & Orbell, 2010; Weinman, Petrie, & Horne, 1996): Illness identity (knowledge and beliefs about symptoms attributed to the illness); Consequences (perceived effects and outcomes of the illness); Timeline (beliefs about the likely duration of the illness); Control/Cure (beliefs about the ability to control /cure the illness); and Causes (beliefs about aetiology). A sixth dimension – coherence (perceived understanding of the illness and its implications) was added later (Moss-Morris et al., 2002). A meta-analytic review (Hagger & Orbell, 2010) of studies examining the stability of the original dimensions and their association with emotional distress outcomes across 23 diseases found that, as would be predicted, consequences, timeline and identity were positively related to distress, while control/cure was negatively related.
3.2.1 Evidence supporting the Common Sense Model in cancer.

Cross-sectional studies have confirmed associations in the expected directions between various illness perception dimensions and psychological outcomes in cancer (Dempster et al., 2012; Millar et al., 2005; Rozema, Vollink, & Lechner, 2009; Scharloo et al., 2010; Traeger et al., 2009). For example, studies have shown that the more symptoms an individual attributes to their illness (Millar et al., 2005; Scharloo et al., 2010) and the poorer their understanding of their condition (Dempster et al., 2012; Gould, Brown, & Bramwell, 2010; Traeger et al., 2009), the more likely they are to be distressed. In addition, the stronger the perception that cancer will have negative consequences (Dempster et al., 2012; Gould et al., 2010; Traeger et al., 2009), last a long time or be cyclical in nature (Gould et al., 2010; Rabin, Leventhal, & Goodin, 2004) and the weaker the belief in its controllability (Dempster et al., 2012; Gould et al., 2010; Rozema et al., 2009; Traeger et al., 2009), the greater the emotional distress. In addition, two of these studies found evidence to support the view that illness perceptions are stronger correlates of adaptive outcomes than are coping styles (Dempster et al., 2012; Rozema et al., 2009). However, as such studies only provide limited information about concurrent relationships, they are of little use in establishing whether illness perceptions play a causal role in activating or maintaining emotional distress after cancer.

Only three studies have been able to show prospective relationships between illness perceptions and emotional distress in cancer, and these differ in the dimensions that were found to predict subsequent distress. A study of newly diagnosed head and neck cancer patients (Llewellyn, McGurk, & Weinman, 2007) found that a stronger perception that cancer would last a long time, and a stronger sense of self-blame, predicted 26% and 21% (respectively) of the variance in depression six-eight months later. In contrast, a study in breast cancer (Millar et al., 2005), found no association between patients’ post-operative
perceptions of the duration or cause of their cancer and emotional distress 12-months later. However, they did find a positive association between post-operative illness identity and later emotional distress. Finally, a recent study among survivors of oesophageal cancer (Dempster et al., 2012) looked at the relationship between change in variables, rather than point prevalence, and found a relationship between change in illness perceptions over a one year period and changes in emotional distress. Specifically, a reduction in the perception of personal and/or treatment control over this period was associated with a corresponding increase in distress after controlling for medical, demographic and coping variables. However, it is important to note from this study that although both illness perceptions and coping added significantly to the variance in change on distress, the total amount of variance explained by the model as a whole was relatively small (7% and 10% for anxiety and depression respectively). Therefore, it is apparent that a lot of the variance in change in distress still remains unexplained after accounting for these variables.

The inconsistency in these results is perhaps not surprising. The CSM was developed to explain the relationship between an individual’s cognitions, coping behaviours and outcomes ‘at that time’, rather than how such cognitions might influence behaviour and outcomes in the future (Llewellyn et al., 2007). Therefore, while the CSM may be a useful approach for understanding concurrent distress, its utility for predicting persistent emotional distress from cognitions elicited around diagnosis is questionable. That is, a causal role for illness perceptions in maintaining distress has yet to be demonstrated.

3.3 Limitations of current theoretical approaches

Consequently, it can be seen that neither of the models described above (the cognitive model of adjustment to cancer and the CSM) have substantially advanced our understanding
of ‘why’ and ‘how’ distress is maintained after diagnosis of cancer. The fact that illness perceptions and negative illness-related thoughts are not clearly implicated is understandable, when one considers that most, if not all, individuals receiving a diagnosis of cancer will experience some thoughts related to negative perceptions of the illness, yet not everyone will experience persistent distress. Negative thoughts are generally fleeting, and an individual’s perceptions about their cancer in the early stages are likely to be unstable as they are assailed with new information and experiences. It has been suggested that negative thoughts only become a problem if the individual responds to them by engaging in excessive worry and/or rumination (Wells, 2009). On this basis, it may be argued that it is not the illness-perceptions per se, but the selection and use of worry in response to the negative thoughts that they trigger that leads to persistent emotional distress. Worry is prevalent in cancer and, while a certain level is considered normal and adaptive, individuals who experience high levels of generalised worry are more likely to develop a helpless/hopeless coping style in response to their concerns (Parle, Jones, & Maguire, 1996), and to develop more negative illness perceptions (Lehto & Cimprich, 2009). However, cognitive models such as the CSM and the cognitive model of emotional disorder do not attempt to explain the causes of such persistent worry and rumination.

3.4 A new theoretical approach - Metacognitive model of emotional disorder

The metacognitive model of emotional disorder was developed in response to the question of why some people are able to dismiss negative thoughts while others cannot and experience recurrent or prolonged distress: ‘Everyone has negative thoughts and everyone believes their negative thoughts sometimes. But not everyone develops sustained anxiety, depression, or emotional suffering’, page 1 (Wells, 2009).
In this model, as in traditional cognitive theories, dysfunctional beliefs have a central role in causing and maintaining distress. However, in contrast to those theories, in the metacognitive model it is the individual’s beliefs about their thinking (metacognitive beliefs) rather than the specific content of their thoughts, and their use of inflexible and recurrent thinking styles in response to negative thoughts, that underlies persistent emotional distress.

3.4.1 The S-REF Model

The basic theoretical underpinning of the metacognitive model of emotional disorder is the Self-Regulatory Executive Function model (S-REF: (Wells & Mathews, 1994)), see Figure 3.3 below.

Figure 3.3: The S-REF model of psychological disorder ((Wells, 2009), p.9)
The S-REF model was derived from prospective and experimental research into information processing models and is the first model to offer an account of the cognitive and metacognitive processes involved in the top-down control and maintenance of emotional disorder (Wells, 2009). It is based on multi-level cognitive architecture comprising three levels of interacting cognitive processing, including: a low-level of automatic and reflexive processing (i.e. negative automatic thoughts), a level of conscious processing of such thoughts and behaviours (cognitive style), and a level of stored metacognitive knowledge and/or beliefs (meta system) that guide the lower levels of ordinary cognitive processing towards a self-relevant goal. For most people, periods of emotional distress in response to an event are transitory, as the goal is reached and processing operations terminated. However, S-REF theory proposes that, for some people, activation of a particular toxic style of thinking called the cognitive attentional syndrome (CAS) occurs, and it is this that is central to the development and maintenance of emotional disorder. The CAS consists of cognitive processes such as persistent worry and rumination, focussing of attention on threat, and maladaptive coping strategies (e.g. avoidance or thought suppression). The model proposes that positive metacognitive beliefs about the benefits of, or need to engage in, such processes activate this style of responding, while negative metacognitive beliefs (i.e. about the danger or uncontrollability of worry and rumination) exacerbate and maintain it. This continuation of CAS processes ultimately ‘backfires’, by sustaining negative thinking and the sense of threat, rather than allowing such experiences to fade naturally.

3.4.2. Generic formulation for the metacognitive model of emotional disorder

Consequently, it can be seen that the S-REF, and hence metacognitive model of emotional disorder, looks beyond dysfunctional content of ordinary cognition (i.e. negative thoughts about cancer) to the generic processes that sustain it. Therefore, rather than being
disorder-specific, it allows for a universal case formulation of emotional disorder as shown in Figure 3.4 below:

![Universal case formulation diagram](image)

**Figure 3.4: Universal case formulation diagram** (Wells, 2009, p252)

Central to this formulation is the CAS, which represents a cognitive style of sustained and inflexible responding to thoughts, including processes such as: perseverative thinking (e.g. worry and rumination), focussing of attention on threat, and maladaptive coping strategies (e.g. avoidance or thought suppression). These CAS processes fail to modify dysfunctional beliefs, instead increasing the accessibility of negative information to support them (Wells, 2009), thus prolonging and intensifying distress. The cyclical relationship
between appraisal and the CAS signifies that negative thoughts or appraisals may trigger, be maintained by, or be the output of CAS processes but do not drive them.

The CAS is activated and driven by metacognitive beliefs. Two types of metacognitive belief are of particular importance: positive beliefs about the benefits of, and need to engage in aspects of the CAS (e.g. ‘if I worry about recurrence, I’ll detect early signs or symptoms’) that activate it, and negative beliefs about the danger or uncontrollability of CAS processes (e.g.: ‘worrying will make my cancer worse’; ‘I can’t stop worrying about recurrence’) that maintain or exacerbate it by causing worry about worry. The bidirectional arrow linking the CAS to metacognitive beliefs indicates that while these metacognitive beliefs activate the CAS, at the same time activity of the CAS strengthens or modifies these beliefs. In addition, negative metacognitive beliefs may also cause a direct emotional response, thereby exacerbating distress directly.

A further bidirectional arrow links the CAS to emotion in Figure 3.4, indicating that unpleasant emotions activate self-regulatory processing with the aim of reducing distress but, in the case of emotional disorder, activity of the CAS instead maintains or exacerbates this distress.

Finally, on the periphery of the formulation the self-world view is also linked by a bidirectional arrow. This represents other influences such as prior experiences or learning, which may shape the content of appraisals or be shaped by them, but do not drive the underlying mental processes that maintain distress.

3.4.3 Evidence for metacognitive model of emotional disorder.

The metacognitive model of emotional disorder was developed for use in mental health. Consequently, the vast majority of studies that have provided empirical support for the model have done so in mental health populations (see Wells, 2008, 2009 for a review) rather than
physical health or, more specifically, cancer. However, evidence of the model’s utility for understanding emotional distress in physical health is beginning to emerge. Metacognitive beliefs have been associated with heightened emotional distress in physical health populations including: Parkinson’s disease (Allott, Wells, Morrison, & Walker, 2005), chronic fatigue (Maher-Edwards, Fernie, Murphy, Nikcevic, & Spada, 2011), teenage and young adult (TYA) cancer survivors (Fisher, McNicol, Young, Smith, & Salmon, 2015; McNicol, Salmon, Young, & Fisher, 2013) and breast cancer (Thewes, Bell, & Butow, 2013).

3.5 Summary

In summary, it can be seen that the two predominant theoretical approaches currently applied to understanding emotional distress in cancer fail to explain why some people are vulnerable to persistent emotional distress after diagnosis and treatment, while others are not. In contrast, the metacognitive model of emotional disorder clearly indicates the psychological processes that underlie maintenance of emotional distress, and thereby offers several potential benefits over these more traditional models. Firstly, it allows patients vulnerable to emotional distress to be identified from the presence of modifiable causal factors, thus improving the potential effectiveness of intervention. Secondly, as intervention is focussed on modifying metacognitive beliefs and process, rather than the content of negative thoughts, it is easier to reconcile with the clinical reality of an often uncertain future and any objective physical changes, limitations and / or role changes imposed by the illness. Finally, because it doesn’t focus on the content of thoughts, it also offers potential for a trans-diagnostic intervention, which may be more appropriate to cancer patients who often present with mixed symptoms of anxiety, depression and trauma.
The remainder of this thesis is devoted to testing the utility of this model for understanding persistent emotional distress in cancer using a series of linked empirical studies. However, first it is necessary to establish that the available measurement tools are valid for assessing metacognitive beliefs and processes in cancer. Therefore, the first empirical chapter, Chapter 4, describes a study conducted to validate the Metacognitions Questionnaire (MCQ-30; Cartwright-Hatton & Wells, 1997) for use in a cancer population.
Chapter Four

Study one

Measuring metacognition in cancer: Validation of the Metacognitions Questionnaire 30 (MCQ-30).

(for Published article see Appendix B)
4.1. Introduction

The Metacognitions Questionnaire (MCQ) was developed by Cartwright-Hatton and Wells (Cartwright-Hatton & Wells, 1997) to explore the metacognitive dimensions that are central in the metacognitive model of emotional disorder. Factor analyses derived five subscales from the initial 65-item questionnaire (MCQ-65), three of which assess beliefs, including: ‘Positive beliefs about worry’; ‘Negative beliefs about the danger and uncontrollability of worry’; and negative beliefs about thoughts in general. The remaining two subscales assess the tendency to focus on cognitive events, ‘Cognitive self-consciousness’; and confidence in cognitive abilities, particularly memory and attention, ‘Cognitive confidence’. The MCQ-65 uses a four-point Likert response scale: 1 (do not agree); 2 (agree slightly); 3 (agree moderately); 4 (agree very much).

However, despite excellent psychometric properties (see Wells (Wells, 2009) for a review), the usefulness of the MCQ-65 was compromised by its length; consequently a shorter 30-item version was developed (Wells & Cartwright-Hatton, 2004). This MCQ-30 retained the factor structure and the response scale of the longer measure, with six items selected to represent each metacognitive dimension on the basis of highest factor loading and item clarity in previous studies.

Initial psychometric properties of the MCQ-30 were found, in a sample of 182 student and community participants, to be broadly similar to those of the longer measure (Wells & Cartwright-Hatton, 2004). Internal consistency of the subscales ranged from an adequate 0.72 to an excellent 0.93 with adequate test-retest reliability for four out of five subscales (ranging from r = 0.59 ‘Negative beliefs about worry’ to r = 0.87 ‘Cognitive self-consciousness’). Confirmatory and exploratory factor analysis confirmed an acceptable fit of the original five-factor model with most items loading on their predicted factors, except in the
case of ‘Need to control thoughts’ where only three out of six items loaded significantly. In addition, all five subscales were significantly and positively correlated with measures of worry (Penn State Worry Questionnaire, PSWQ (Meyer, Miller, Metzger, & Borkovec, 1990)) and Trait anxiety (State - Trait Anxiety Inventory, STAI (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983)) with the subscale ‘Negative beliefs about worry’ showing the strongest associations. Further studies have since assessed the psychometric properties of the MCQ-30 in mixed student and community samples in the UK (Spada, Mohiyeddini, & Wells, 2008) and Turkey (Yilmaz, Gencoz, & Wells, 2008). In both cases, the original five factor structure was replicated and positive correlations demonstrated with theoretically appropriate measures of worry (PSWQ), anxiety and depression.

Recently, interest has grown in applying the metacognitive model to understanding emotional distress in cancer (McNicol et al., 2013; Thewes, Bell, & Butow, 2013). Thewes et al (Thewes, Bell, & Butow, 2013) used the MCQ-30 to explore for the first time the association of metacognitive beliefs with Fear of Cancer Recurrence (FCR) among young women with early stage breast cancer. They found that the subscale ‘Negative beliefs about worry’ was the most highly correlated with FCR, and that the MCQ-30 total score accounted for 36% of the variance in this outcome, leading them to conclude that maladaptive metacognitions play an important role in FCR. However, caution is warranted in the interpretation of such findings, because without formal psychometric testing we do not yet know how the MCQ-30 operates in a cancer population. In order to have confidence in research conducted to test metacognitive theory and therapy in oncology settings (such as this), we first need measurement procedures that are valid for use in this population.

Consequently, the current study aims to explore, for the first time, the validity of the MCQ-30 in cancer. The primary aim is to explore whether the established five factor structure of the MCQ-30 is valid in this population, and to investigate the internal consistency
of its subscales. A second aim is to explore whether the theoretically expected associations between specific subscales of the MCQ-30 and anxiety and depression demonstrated in previous research (Wells & Cartwright-Hatton, 2004; Spada et al, 2008; Yilmaz et al, 2008) are replicated, thus providing evidence of concurrent validity in this population.

4.2. Methods

4.2.1 Ethics statement

This study was conducted within the context of a larger prospective cohort study exploring the association of metacognitive beliefs with emotional distress after cancer which was approved according to UK guidelines, by the NHS North West 5 Research Ethics Committee (reference: 09/H1010/70). There are no conflicts of interest to be declared.

4.2.2. Participants

A priori sample size calculations indicated a total sample size of 226 patients would provide 80% power to detect $R^2$ of maladaptive metacognition (MCQ-30) as low as .05, i.e. a small-medium effect size. The significance criterion was set at $p<.01$ to allow for multiple testing (3 outcomes).

Participants were recruited from patients at least 18 years old attending routine pre-treatment clinics at a National Health Service (NHS) teaching hospital, after receiving a diagnosis of primary non-metastatic breast or prostate cancer. Patients were excluded if they had recurrent or metastatic disease, or were considered by the clinical team or researcher to be too distressed or confused to give informed consent.
4.2.3. Measures

The Metacognitions Questionnaire 30 (MCQ-30) (Wells & Cartwright-Hatton, 2004) assesses metacognitive beliefs and processes. It comprises five subscales: ‘Positive beliefs about worry’; ‘Negative beliefs about worry’; ‘Cognitive confidence’; ‘Need to control thoughts’; and ‘Cognitive self-consciousness’. For each subscale, six items are scored 1-4, yielding total scores of 6 to 24. Participants are asked to indicate how much they generally agree with statements such as ‘Worrying helps me cope’ (Positive beliefs about worry); ‘My worrying is dangerous for me’ (Negative beliefs about worry); ‘I do not trust my memory’ (Cognitive confidence); ‘Not being able to control my thoughts is a sign of weakness’ (Need to control thoughts); and ‘I constantly examine my thoughts’ (Cognitive self-consciousness). High scores indicate, respectively, more positive and negative beliefs about worry, reduced confidence in memory, greater belief in the need to control thoughts and an increased tendency towards self-focused attention. The MCQ-30 has excellent internal consistency and good convergent and predictive validity in normal populations (Spada et al., 2008; Wells & Cartwright-Hatton, 2004; Yilmaz et al., 2008).

The Hospital Anxiety and Depression scale (HADS) (Zigmond & Snaith, 1983) was used to assess anxiety and depression. The HADS is a well-established measure of emotional distress specifically developed for use in physically ill populations. Fourteen items are scored on a four-point scale yielding two subscale scores of 0-21 with high scores indicating great anxiety or depression. A cut-off score of eight or more on each subscale indicates clinically significant levels of symptoms. The HADS has been extensively validated for use in cancer (Moorey et al., 1991; Vodermaier & Millman, 2011), and is one of the most widely employed measures of anxiety and depression symptoms in this population. In the current sample, both subscales had good internal consistency (Cronbach’s α: .84/.88 for T1/T2 depression; .88/.89 for T1/T2 anxiety).
The MOS social support survey (MOS; Sherbourne & Stewart, 1991) was used to check for differences between groups (completed T1 only vs. completed T1&T2) in perceived emotional support. This 19-item self-report measure was designed to assess four separate dimensions of perceived support among patients with chronic conditions. However, for this study, only the subscales concerning emotional support (‘emotional/informational support’, ‘positive social interaction’ and ‘affectionate support’) were used to produce a total score for ‘perceived emotional support’. As in a previous study in breast cancer (Hill et al., 2011), this score was dichotomised by designating the patients in the lowest third as having low emotional support.

4.2.4 Procedure

From February 2010 to May 2011, participants were consecutively recruited through two pre-treatment cancer clinics at a National Health Service (NHS) teaching hospital in North-West England. Suitable participants were identified by clinic staff, who gave them recruitment letters and information sheets for the study along with their appointment letters for routine pre-treatment consultations, and explained that participation in the research was entirely voluntary. When patients attended the clinic, those willing to see the researcher were given further information and asked for written consent. Participants were asked to complete study questionnaires in clinic (T1 – pre-treatment) and were given the choice of electronic (hand-held PC) or paper formats. Those unable to complete the questionnaires in clinic took a copy (paper version) home and returned them by post. Twelve months later, participants were mailed a second questionnaire pack (T2 – 12 months later), which they completed and returned by post.
4.2.5 Data analysis

The data were analysed using SPSS Version 20 and Mplus v6.12. Nonparametric statistics (Mann-Whitney or Kruskal-Wallis) were used for group comparisons between consenting patients who returned completed questionnaires at T1 and those that did not, and between participants who completed both assessments (completers) and those who completed T1 only (non-completers). In both cases, groups were compared on age group (divided above and below the median age), gender, and in the latter comparison also on educational level, perceived emotional social support, stage of disease, T1 HADS and IES.

To explore the validity of the MCQ-30 over time and under different circumstances, the data were analysed separately for both time points (pre-treatment & 12 months later).

Construct validity of the MCQ-30 was first assessed using Confirmatory Factor Analysis (CFA) to test the published five-factor measurement model. As the primary aim of this study was to assess validity, rather than achieve the best possible model fit, the decision was taken not to make minor modifications to the model based on the data (unless strongly supported by theory) as such modifications often just reflect idiosyncratic characteristics of the sample (MacCallum, Roznowski, & Necowitz, 1992). Instead, Exploratory Factor Analysis (EFA) was used to explore whether an alternative model would be more appropriate for this sample. Both sets of analyses (CFA and EFA), were performed in Mplus version 6.12 (L. K. Muthen & Muthen, 1998-2010), using the robust weighted least squares estimator (WLSMV(B. Muthen, 1984; B. Muthen, du Toit, & Spisic, 1997)) recommended for ordinal categorical data (Brown, 2006). The EFA tested models up to and including a five-factor structure without dictating where items should load. As previous studies identified MCQ-30 subscales as inter-correlated, an oblique rotation (Geomin) was used to establish the optimum pattern of item loadings. For both analyses (CFA & EFA), adequacy of model fit was
assessed based on two incremental fit indices: the Comparative Fit Index (CFI); and the Tucker-Lewis Fit Index (TLI), with values close to 0.95 indicating a well-fitting model (Hu & Bentler, 1999), and two absolute misfit indices: the Root mean Square Error of Approximation (RMSEA) with values <.05 indicating good fit and 0.5 - .08 adequate fit (Browne & Cudeck, 1993); and the Weighted Root Mean Square Residual (WRMR) with values less than .95 indicating good fit (Yu, 2002). For the EFA, the Standardised Root mean Square (SRMR) was used, instead of the WRMR, with values <.05 indicating good fit. Inter-correlations amongst the five latent factors of the published model were examined and the internal consistency of each subscale assessed using Cronbach’s alpha.

Concurrent validity of the MCQ-30 was then assessed (at each time point) by fitting the data to a structural model in which latent variables for anxiety and depression (each indicated by their seven constituent HADS items), were regressed onto the MCQ-30 factors. Adequacy of model fit was again assessed using the fit indices described above. As the MCQ-30 and HADS subscales were not normally distributed and the study sample relatively small, bootstrapping techniques were used to test the robustness of study findings.

4.3. Results

4.3.1 Sample

Of 370 patients who were invited to participate, 258 (70%) consented and 229 (62% of those approached, 89% of consenters) returned completed questionnaires at T1. There were no significant differences in age, gender and tumour stage between consenting patients who returned completed questionnaires and those that did not.

Of the 229 participants who completed T1 questionnaires, 206 (90%) also completed the assessment 12 months later. No significant differences between those who completed
both time points (completers) and those that completed T1 only (non-completers) were apparent on T1 HADS, IES, age, gender, education, or tumour grade. However, non-completers were more likely than completers to report low levels of perceived emotional support at T1 (52% vs. 31% p=.034).

Sample characteristics for the participants at each time point are shown in Table 4:1.

### Table 4.1: Sample Demographic and Clinical Characteristics

<table>
<thead>
<tr>
<th>Total N</th>
<th>Pre-treatment sample</th>
<th>12 months follow-sample</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>229</td>
<td>206</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>61.3 (8.9)</td>
<td>61.5 (9.0)</td>
</tr>
<tr>
<td>Range</td>
<td>38 – 85</td>
<td>39-85</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>150 (66)</td>
<td>133</td>
</tr>
<tr>
<td>Male</td>
<td>79 (34)</td>
<td>73</td>
</tr>
<tr>
<td><strong>Marital status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married / co-habiting</td>
<td>151 (66)</td>
<td>139</td>
</tr>
<tr>
<td>Live alone</td>
<td>46 (20%)</td>
<td>37</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>88 (38)</td>
<td>76</td>
</tr>
<tr>
<td>School qualifications or higher</td>
<td>132 (58)</td>
<td>121</td>
</tr>
<tr>
<td><strong>Employment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Employed (full/part-time)</td>
<td>88 (38)</td>
<td>79</td>
</tr>
<tr>
<td>Retired</td>
<td>99 (43)</td>
<td>92</td>
</tr>
<tr>
<td>Retired (health)</td>
<td>16 (7)</td>
<td>14</td>
</tr>
<tr>
<td>Homemaker</td>
<td>13 (6)</td>
<td>9</td>
</tr>
<tr>
<td>Unemployed</td>
<td>10 (4)</td>
<td>9</td>
</tr>
<tr>
<td><strong>Cancer diagnosis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast</td>
<td>150 (66)</td>
<td>133</td>
</tr>
<tr>
<td>Prostate</td>
<td>79 (34)</td>
<td>73</td>
</tr>
<tr>
<td><strong>Tumour grade</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>56 (24)</td>
<td>54</td>
</tr>
<tr>
<td>Intermediate</td>
<td>107 (47)</td>
<td>97</td>
</tr>
<tr>
<td>High</td>
<td>62 (27)</td>
<td>52</td>
</tr>
<tr>
<td><strong>Distress outcomes</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety (HADS-A &gt;7)</td>
<td>117 (51)</td>
<td>70 (34)</td>
</tr>
<tr>
<td>Depression (HADS-D &gt;7)</td>
<td>28 (12)</td>
<td>44 (21)</td>
</tr>
<tr>
<td>PTSD symptoms (IES total ≥27)</td>
<td>136 (59)</td>
<td>77 (37)</td>
</tr>
</tbody>
</table>

_N.B. Missing data T1 (T2): Marital Status n=5(5); Live alone n=3(2); Education n=9 (9); Employment n=3(3); Tumour grade n=4(3)._
4.3.2. Factorial Structure

Confirmatory factor analysis of the MCQ-30 five-factor model showed overall a marginally adequate fit of the model to the data at the pre-treatment assessment: $\chi^2 (395) = 787.448$. p<.01, RMSEA = .066 (90% CI=.059-.073), CFI = .91, TLI = .90, WRMR = 1.218.

Exploratory Factor analysis which, unlike CFA, does not dictate where items should load, confirmed that a five-factor solution nevertheless provided the best model. Moreover, the fit indices ($\chi^2 (295) = 439.692$. P<.001, RMSEA = .046 (90% CI=.037-.055), CFI = .97, TLI = .95, SRMR = 0.046) together indicate a good fit of the model to this data. As shown in Table 4:2, all items loaded >0.4 on their expected factors (Wells & Cartwright-Hatton, 2004). However, as the items were allowed to load freely across any factors, minor discrepancies were observed between the EFA–derived solution and the published five factor model. Specifically, two items, MCQ3 and MCQ13, had their highest loadings on factors other than the expected ones. Item MCQ3 loaded higher on ‘Negative beliefs about worry’ (F1) than on its expected factor - ‘Cognitive self-consciousness’ (F4). Item MCQ13 had equivalent loadings on both its expected factors - ‘Need for control over thoughts’ (F5) - and ‘Cognitive self-consciousness’ (F4). Two further items (MCQ5 & MCQ29) also demonstrated significant (> .4) cross-loadings although for both the highest loading remained consistent with the published factor structure.

At the 12-month follow-up, CFA indicated an adequate fit of the data to the published five-factor model: $\chi^2 (395) = 684.184$. p<.01, RMSEA = .060 (90% CI=.053-.068, (p RMSEA<.05 )), CFI = .95, TLI = .95, WRMR = 1.048, therefore no Exploratory Factor Analysis was performed.
Table 4.2: Published scale structure and rotated (Geomin) factor loadings from EFA of the Metacognitions Questionnaire-30 at pre-treatment.

<table>
<thead>
<tr>
<th>Subscale: Positive beliefs about worry</th>
<th>MCQ-1</th>
<th>MCQ-7</th>
<th>MCQ-10</th>
<th>MCQ-19</th>
<th>MCQ-23</th>
<th>MCQ-28</th>
<th>EFA Factor Loadings</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Worrying helps me to avoid problems in the future</td>
<td>I need to worry in order to remain organized</td>
<td>Worrying helps me to get things sorted out in my mind</td>
<td>Worrying helps me cope</td>
<td>Worrying helps me to solve problems</td>
<td>I need to worry in order to work well</td>
<td>F1</td>
</tr>
<tr>
<td></td>
<td>0.08</td>
<td>0.09</td>
<td>0.05</td>
<td>-0.04</td>
<td>-0.10</td>
<td>-0.04</td>
<td>0.08</td>
</tr>
<tr>
<td>Subscale: Negative beliefs about worry</td>
<td>MCQ-2</td>
<td>MCQ-4</td>
<td>MCQ-9</td>
<td>MCQ-11</td>
<td>MCQ-15</td>
<td>MCQ-21</td>
<td>EFA Factor Loadings</td>
</tr>
<tr>
<td></td>
<td>My worrying is dangerous for me</td>
<td>I could make myself sick with worrying</td>
<td>My worrying thoughts persist, no matter how I try to stop them</td>
<td>I cannot ignore my worrying thoughts</td>
<td>My worrying could make me go mad</td>
<td>When I start worrying, I cannot stop</td>
<td>F1</td>
</tr>
<tr>
<td></td>
<td>0.58</td>
<td>0.65</td>
<td>0.70</td>
<td>0.69</td>
<td>0.62</td>
<td>0.76</td>
<td>-0.17</td>
</tr>
<tr>
<td>Subscale: Cognitive confidence</td>
<td>MCQ-8</td>
<td>MCQ-14</td>
<td>MCQ-17</td>
<td>MCQ-24</td>
<td>MCQ-26</td>
<td>MCQ-29</td>
<td>EFA Factor Loadings</td>
</tr>
<tr>
<td></td>
<td>I have little confidence in my memory for words and names</td>
<td>My memory can mislead me at times</td>
<td>I have a poor memory</td>
<td>I have little confidence in my memory for places</td>
<td>I do not trust my memory</td>
<td>I have little confidence in my memory for actions</td>
<td>F1</td>
</tr>
<tr>
<td></td>
<td>0.04</td>
<td>0.19</td>
<td>0.05</td>
<td>-0.10</td>
<td>-0.07</td>
<td>0.04</td>
<td>0.07</td>
</tr>
<tr>
<td>Subscale: Need for control over thoughts</td>
<td>MCQ-6</td>
<td>MCQ-13</td>
<td>MCQ-20</td>
<td>MCQ-22</td>
<td>MCQ-25</td>
<td>MCQ-27</td>
<td>EFA Factor Loadings</td>
</tr>
<tr>
<td></td>
<td>If I did not control a worrying thought, and then it happened, it would be my fault.</td>
<td>I should be in control of my thoughts all of the time</td>
<td>Not being able to control my thoughts is a sign of weakness</td>
<td>I will be punished for not controlling certain thoughts</td>
<td>It is bad to thinks certain thoughts</td>
<td>If I could not control my thoughts, I would not be able to function</td>
<td>F1</td>
</tr>
<tr>
<td></td>
<td>0.39</td>
<td>-0.04</td>
<td>0.32</td>
<td>0.15</td>
<td>0.13</td>
<td>-0.07</td>
<td>0.17</td>
</tr>
<tr>
<td></td>
<td>0.17</td>
<td>-0.11</td>
<td>0.06</td>
<td>0.15</td>
<td>-0.02</td>
<td>-0.00</td>
<td>-0.14</td>
</tr>
<tr>
<td></td>
<td>-0.14</td>
<td>-0.01</td>
<td>-0.06</td>
<td>0.21</td>
<td>0.11</td>
<td>-0.05</td>
<td>-0.10</td>
</tr>
<tr>
<td></td>
<td>0.06</td>
<td>0.03</td>
<td>0.07</td>
<td>0.27</td>
<td>0.18</td>
<td>0.27</td>
<td>0.52</td>
</tr>
<tr>
<td></td>
<td>0.72</td>
<td>0.03</td>
<td>0.50</td>
<td>0.64</td>
<td>0.64</td>
<td>0.50</td>
<td>0.64</td>
</tr>
</tbody>
</table>
### Subscale: Cognitive self-consciousness

<table>
<thead>
<tr>
<th>MCQ</th>
<th>Item</th>
<th>POS</th>
<th>NEG</th>
<th>CC</th>
<th>NC</th>
<th>CSC</th>
<th>Alpha</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>I think a lot about my thoughts</td>
<td>0.56</td>
<td>0.01</td>
<td>-0.01</td>
<td>0.43</td>
<td>-0.06</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>I am aware of the way my mind works when I am thinking through a problem</td>
<td>0.14</td>
<td>0.17</td>
<td>-0.07</td>
<td>0.49</td>
<td>-0.42</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>I monitor my thoughts</td>
<td>-0.03</td>
<td>0.08</td>
<td>-0.12</td>
<td>0.66</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>I am constantly aware of my thinking</td>
<td>0.27</td>
<td>0.02</td>
<td>0.05</td>
<td>0.66</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>I pay close attention to the way my mind works</td>
<td>-0.02</td>
<td>0.08</td>
<td>0.03</td>
<td>0.83</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>I constantly examine my thoughts</td>
<td>0.31</td>
<td>0.05</td>
<td>0.01</td>
<td>0.55</td>
<td>0.27</td>
<td></td>
</tr>
</tbody>
</table>

N.B. F1 ‘Negative beliefs about worry’ F2 ‘Positive beliefs about worry’ F3 ‘Cognitive confidence’ F4 ‘Need for control over thoughts’ F5 ‘Cognitive Self-consciousness’; Black= loading >.4; Underline = highest loading where item loads >.4 on more than one factor.

The mean and SDs of the five MCQ-30 subscales and the correlations amongst the five latent variables (CFA standardised solution) at both time points are presented in Table 4:3.

The internal consistency of the subscales was assessed using Cronbach’s alpha (Table 4:3) and ranged from 0.73 to 0.89 pre-treatment and from .79 to .91 at 12 month follow-up, indicating adequate to excellent internal consistency. At both time points the subscale with the lowest alpha coefficient was ‘Need for Control’.

**Table 4.3: Descriptive data, internal consistency and inter correlations among the five latent MCQ-30 factors (CFA standardised solution)**

<table>
<thead>
<tr>
<th></th>
<th>Pre-treatment</th>
<th>12 month follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (IQR)</td>
</tr>
<tr>
<td>POS</td>
<td>9.2 (3.95)</td>
<td>8 (8.7-9.7)</td>
</tr>
<tr>
<td>NEG</td>
<td>11.2 (4.17)</td>
<td>11 (10.6-11.7)</td>
</tr>
<tr>
<td>CC</td>
<td>10.0 (4.10)</td>
<td>9 (9.5-10.6)</td>
</tr>
<tr>
<td>NC</td>
<td>10.1 (3.68)</td>
<td>9.0 (9.6-10.6)</td>
</tr>
<tr>
<td>CSC</td>
<td>13.3 (4.39)</td>
<td>13 (12.7-13.9)</td>
</tr>
</tbody>
</table>

N.B. MCQ-30 subscales: ‘Positive beliefs about worry’ (POS); ‘Negative beliefs about worry’ (NEG); ‘Cognitive Confidence’ (CC); ‘Need for control over thoughts’ (NC); ‘Cognitive Self Consciousness’ (CSC)

*p<.05; **p<.001
4.3.3. Convergent validity

The hypothesised model of the relationship between metacognitive beliefs (using the MCQ-30’s published factor structure) and concurrent anxiety and depression is shown in Figure 4.1. Overall, the fit indices for this latent variable SEM (see Table 4:4) indicated an acceptable fit of the model. At both time points, ‘Negative beliefs about worry’ explained significant variance in both anxiety and depression and, as hypothesised, was the strongest of all the predictors. ‘Positive beliefs about worry’ also explained variance in anxiety at both time points but not depression. At the pre-treatment time-point, ‘Need for control over thoughts’ was associated with fewer symptoms of anxiety and this association fell just short of significant (p=.057) at the 12-month follow-up. There was no significant relationship between ‘Cognitive confidence’ or ‘Cognitive self-consciousness’ and anxiety or depression at either time-point.

<table>
<thead>
<tr>
<th>Fit Statistics</th>
<th>Pre-treatment</th>
<th>12-month follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chi Square Test of Model Fit</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Value</td>
<td>1354.58</td>
<td>1245.78</td>
</tr>
<tr>
<td>Degrees of Freedom</td>
<td>881</td>
<td>881</td>
</tr>
<tr>
<td>p-value</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>CFI/TLI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CFI</td>
<td>.93</td>
<td>.96</td>
</tr>
<tr>
<td>TLI</td>
<td>.93</td>
<td>.95</td>
</tr>
<tr>
<td>Root Mean Square Error of Approximation (RMSEA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Estimate (C.I)</td>
<td>.048 (.043-.053)</td>
<td>.045 (.039-.050)</td>
</tr>
<tr>
<td>Weighted Root Mean Square Residual (WRMR)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Value</td>
<td>1.147</td>
<td>1.009</td>
</tr>
</tbody>
</table>
Figure 4.1: Structural equation model of the relationship between latent factors for the dimensions of the MCQ-30 and HADS anxiety and HADS depression.

N.B. Rectangles indicate observed variables on MCQ-30 (MCQ) or HADS (H); ellipses indicate latent factors. Latent factors: Positive beliefs about worry (POS); Negative beliefs about worry (NEG); Cognitive Confidence (CC); Need to control thoughts (NC); Cognitive Self-consciousness (CSC); HADS Anxiety (HADS-A); HADS Depression (HADS-D). Figure shows standardized path coefficients and their significance. Solid line – significant at both time points; dotted line – non-significant; Brackets indicate coefficient at 12-month follow-up; Errors not shown; *** p<.001 ** p<.01 * p<.05
4.4. Discussion

The present study provides the first evidence to support the published five-factor structure of the MCQ-30 (Wells & Cartwright-Hatton, 2004) as valid and replicable in a cancer population. Although at the pre-treatment time point CFA showed only a marginal fit, subsequent EFA confirmed that a five-factor solution still provided the best solution. The improved fit observed for the EFA over the CFA was the result of items being allowed to load freely across any of the factors. However, all items still loaded on their expected factors with only minor discrepancies between the two models. At 12-month follow-up, fit was acceptable and comparable to that reported by the measure’s developers (Wells & Cartwright-Hatton, 2004). It is not clear why the fit should be slightly better at the 12-month follow-up. The mode of administration differed between the two time points with pre-treatment assessments largely being carried out on hand-held PCs while 12-month follow-ups were completed on paper. It is possible that this has some bearing on the improved fit observed at follow-up, as the latter assessment is closer to how the questionnaires have been administered during previous validation studies. Equally, the observed improvement in fit could be partly due to the timing of assessments in that the pre-treatment assessment was conducted relatively soon after diagnosis, during a period that is clinically busy and often emotionally turbulent. In contrast, the 12-month follow-up for most patients is likely to be a more settled time, at least clinically. However, taken together, these CFA and EFA results suggest that the established five-factor structure of the MCQ-30 is valid for use in a cancer population and that it remains valid across one year post-diagnosis and changing illness / treatment circumstances. In addition, the results indicate that the subscales possess good internal consistency comparable to those found in previous studies (Spada et al., 2008; Wells & Cartwright-Hatton, 2004; Yilmaz et al., 2008).
Two items (MCQ3 & MCQ13) also loaded on a factor different from that expected. However, only one of these loaded higher on that factor; Item MCQ3 (‘I think a lot about my thoughts’) had its highest loading on ‘Positive beliefs about worry’ rather than the expected factor ‘Cognitive self-consciousness’. Both of these items have also been found to cross-load on different factors previously (Yilmaz et al., 2008) although, in that study, item MCQ3 loaded >0.4 on ‘Negative beliefs about worry’ not on ‘Positive beliefs about worry’ as in the present study.

Preliminary evidence of the measure’s convergent validity is provided by the structural equation model of the relationship of the MCQ-30 latent factors with anxiety and depression. As hypothesised, and as shown previously in mental health, physical health, student and community populations, ‘Negative beliefs about worry’ was the strongest predictor of both anxiety (Allott et al., 2005; Spada et al., 2008; Wells & Cartwright-Hatton, 2004; Yilmaz et al., 2008) and depression (Spada et al., 2008; Yilmaz et al., 2008). In addition, ‘Positive beliefs about worry’ predicted anxiety at both time points. However, in contrast, ‘Need for control over thoughts’ was negatively related to anxiety at pre-treatment although this relationship was marginally non-significant at 12 month follow-up. This suggests that participants with lower conviction about the need to control their thinking experience greater anxiety. Such findings are unexpected, as previous studies in mental health, student and community samples have indicated that greater belief in the need to control thoughts predicts higher, rather than lower, levels of anxiety. This result may indicate a difference between this and previously studied mental health, student and community populations. However, further work would be required to establish whether this is a true population difference or just an artefact of the present data.

It is important to note that, by structural equation modelling standards, the study employed a small sample size, which may reduce the stability of the findings. Consequently,
further work is required to establish whether the apparent differences in item functioning and observed patterns of associations represent real differences in how the measure operates in mental health and cancer populations or are idiosyncratic to this data set. In addition, as only breast and prostate cancer patients were included in the study, it remains important to explore whether study findings can be replicated across different cancer diagnoses.

In summary, the current study provides initial evidence that the established five factor structure of the MCQ-30 is valid for use in a cancer population and that the subscales possess good internal consistency. Positive and negative beliefs about worry were associated with concurrent anxiety and depression as expected, although the negative relationship of anxiety with both ‘Need for control over thoughts’ is unexpected and therefore intriguing. Despite the limitations discussed above, we conclude from this study that the MCQ-30 is a sufficiently valid measure for assessing metacognitive beliefs and processes in breast or prostate cancer populations in the first year after diagnosis.
Chapter Five

Study two

The association of metacognitive beliefs with emotional distress after diagnosis of cancer.

(for Published article see Appendix C)
5.1. Introduction

Chapter 3 highlighted that the two predominant theoretical models (both based on the cognitive paradigm) used to understand emotional distress in cancer are unable to explain why emotional distress persists for some people, but not for others. An alternative model – the metacognitive model of emotional disorder – was suggested on the basis that it indicates psychological processes that underlie maintenance of emotional distress. All three theoretical approaches predict that content of negative thoughts (i.e. negative illness perceptions) will be associated with increased emotional distress. However, due to the putative causal role of metacognitive beliefs about worry in activating and exacerbating the CAS in response to such cognitions, the metacognitive model also makes two new predictions. Firstly, it predicts that metacognitive beliefs will be able to explain additional variance in emotional distress, over and above that explained by negative illness perceptions. Secondly, it predicts that the relationship between metacognitive beliefs and emotional distress will be mediated by CAS processes such as worry. Specifically, as described in Chapter 3, positive metacognitive beliefs will cause emotional distress by activating the CAS (i.e worry), while negative metacognitive beliefs will maintain emotional distress by causing both a direct emotional response and through exacerbating the CAS (i.e. causing worry about worrying (meta-worry)).

This study aims to test these predictions by examining, for the first time, the relative contribution of negative illness-perceptions and metacognitive beliefs to emotional distress after diagnosis of cancer, and by testing the mediational role of worry.
5.2. Method

A cross-sectional cohort design was used. Study participants were 299 patients recruited from routine pre-treatment clinics as part of the larger prospective cohort study. See Chapter 4 for a full account of the prospective study including; study inclusion criteria, recruitment and procedure.

5.2.1 Measures

Emotional distress was measured using the Hospital Anxiety and Depression Scale (HADS; (Zigmond & Snaith, 1983)) and the Impact of Events Scale (IES; (Horowitz, Wilner, & Alvarez, 1979)). The HADS has been described in detail in Chapter 4. The IES is a 15 item, self-report scale developed to assess the subjective impact of any specific event (e.g. diagnosis of cancer in this study). Individual items are scored on a four-point scale yielding a total score of 0-75, with high scores indicating more PTSD symptoms. In the current study this single factor model showed acceptable fit, supporting the validity of using the total score. No consensus exists on cut-off scores for clinically significant levels of PTSD symptoms. However, a total score of 27 or more provided an overall correct classification rate, for traumatic stress, of .80 in a large sample of motor vehicle accident survivors comprising both genders (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006), and has previously been used in cancer (Purnell et al., 2011). Internal consistency of the IES was excellent at both time points (pre-treatment & 12 months later) in the current sample (Cronbach’s α: .90/.94).

The Revised Illness Perception Questionnaire (IPQ-R; (Moss-Morris et al., 2002)) was used to assess negative illness perceptions. This comprises three parts, the first of which (‘Identity’) asks participants to indicate whether they have experienced any of 15 common symptoms (an additional item of particular relevance to prostate patients - ‘urinary problems’
- was added for this study) since diagnosis and, if so, whether they attribute them to cancer. Items endorsed as having been both experienced and attributed to cancer are counted, providing a total score of 0-15. As most patients with early stage prostate and breast cancer experience few symptoms, this scale was dichotomised (no symptoms vs. 1 or more symptoms). The second part of the IPQ-R comprises seven cognitive and emotional representation subscales. Items are scored 1-5, with high scores on the ‘Chronic timeline’, ‘Consequences’, and ‘Cyclical timeline’ subscales indicating a stronger belief that the illness will last a long time, have negative consequences and be cyclical in nature, respectively, and high scores on the ‘Personal control’, ‘Treatment control’ and ‘Illness coherence’ subscales indicating a stronger belief in the controllability of the illness and a greater personal understanding of it, respectively. As the IPQ-R was included to assess the relative importance of patients’ illness appraisal in predicting emotional response, the emotional representation subscale was disregarded. The final part, in which items are also scored 1-5, measures patients’ causal attributions about their illness. These items are typically not summed as a single scale, but may be analysed as separate items or as groups devised on the basis of theory ((Moss-Morris et al., 2002)). Previously, only psychological and/or behavioural attributions have contributed to the variance explained in quality of life (Scharloo et al., 2010) or emotional distress (Kulik & Kronfeld, 2005; Traeger et al., 2009) after diagnosis of cancer. Therefore, for this study, the seven items which reflect these attributions (i.e. ‘my own behaviour’, ‘my mental attitude’, ‘stress or worry’, ‘my emotional state’, and ‘my personality’ ‘family problems or worries’ and ‘overwork’) were used to generate a single causal subscale (‘Psychological cause’) and the rest discarded.

Metacognitive beliefs were measured using the Metacognitions Questionnaire 30- (MCQ-30; (Wells & Cartwright-Hatton, 2004)) which has been described previously in
Chapter 4. However, as the focus of this study was on testing specific predictions about the relationship of positive and negative metacognitive beliefs about worry with emotional distress only two (‘Positive beliefs about worry’ and ‘Negative beliefs about worry’) of the five subscales were included. These subscales ask participants to indicate how much they generally agree with statements such as ‘Worrying helps me cope’ (‘Positive beliefs about worry’); and ‘My worrying is dangerous for me’ (‘Negative beliefs about worry’). High scores indicate more positive and negative beliefs about worry, respectively.

Worry was measured using the Penn State Worry Questionnaire (PSWQ; (Meyer et al., 1990)). The PSWQ is a well-established measure developed to assess the level of worry independent of worry content. Participants are asked to rate to what extent statements, such as ‘When I am under pressure I worry a lot’, are ‘typical of me’. Sixteen items are scored 1-5, yielding a total score of 16 to 80, with higher scores indicating greater worry. However, a single factor model fitted the study data poorly. Some previous studies have indicated a two-factor model (Fresco, Heimberg, Mennin, & Turk, 2002; Yilmaz et al., 2008), with positively (PSWQ+ve) and negatively (PSWQ-ve), phrased items loading on separate factors. This model (with the exception of item 10 ‘I never worry about anything’, which loaded on both factors) provided the best fit to the study data and was therefore used in the present study, with Item 10 allowed to cross load.

The CAS-I (Wells, 2009) was included as an additional measure of the CAS. Developed primarily as a clinical tool, it is a state measure comprising two distinct parts. The first eight items, scored on a 0-8 scale, assess CAS processes and the extent to which individuals have been using maladaptive strategies to cope with negative thoughts or feelings. The remaining eight items assess metacognitive beliefs about the CAS and were redundant in this study due to the inclusion of the MCQ-30. Good internal consistency and significant
positive correlations with measures of depression, anxiety and stress have been reported for the CAS-I scale as a whole (Fergus, Bardeen, & Orcutt, 2012). For the present study, preliminary exploratory factor analysis of the first eight items indicated that a 3 factor model provided the best fit. Items 1 ‘How much time in the last week have you found yourself dwelling on or worrying about your problems?’ and 2 ‘How much time in the last week have you been focussing attention on the things you find threatening (e.g. symptoms, thoughts, danger)?’ loaded on the first factor and were summed to provide an alternative measure of the frequency of worry, the remaining items being disregarded.

5.2.2. Data Analysis

The data were analysed using SPSS Version 20, Stata 9 and Mplus v6.12. As fewer than 2% were missing at the scale level, and these data were confirmed to be missing completely at random, missing scales scores were imputed using the SPSS Expectation-Maximisation algorithm (Little & Rubin, 1987). As not all scales were normally distributed, this study used nonparametric statistics or bootstrapping techniques to ensure findings were robust.

Nonparametric statistics (Mann-Whitney or Kruskal-Wallis) were used to compare pre-treatment levels of emotional distress by age group (dichotomised at the median), gender, educational level, perceived emotional social support and stage of disease. Where significant differences were found these variables were entered as covariates in the subsequent analyses.

Preliminary regression analyses were used to identify the illness perceptions associated with each outcome (anxiety, depression and PTSD symptoms) after controlling for covariates.
To test the first prediction from the metacognitive model, separate hierarchical multiple regression analyses first tested the association of each outcome with metacognitive beliefs after controlling for identified covariates. Then these analyses were repeated, also controlling for the illness perceptions found in preliminary regression analysis to be associated with that outcome. To control for non-normality, final regression models were robustly assessed using bootstrapped sampling in Stata 9. To test the second prediction from the metacognitive model, the data were fitted to the hypothesised model (Figure 5.1) using structural equation modelling (SEM) in Mplus version 6.12 (L. K. Muthen & Muthen, 1998-2010).

Figure 5.1: Hypothesised path model of the relationship between metacognitive beliefs and emotional distress.

N.B: Solid lines predicted to be significant; Dotted lines not significant; + indicates positive direction of effect

Because visual inspection suggests there are similarities between some items on the PSWQ and the MCQ-30 subscale ‘Negative beliefs about worry’, a second model substituting the CAS-I for the PSWQ was included as an additional test to guard against bias due to common method variance. Fit was assessed using the robust weighted least squares estimator (WLSMV; (B. Muthen, 1984; B. Muthen et al., 1997)) recommended for ordinal categorical data (Brown, 2006). Analyses controlled for identified covariates and were conducted initially using the PSWQ, then repeated using the CAS-I. Adequacy of model fit
was assessed based on two incremental fit indices: the Comparative Fit Index (CFI) and the Tucker-Lewis Fit Index (TLI), with values close to .95 indicating a well-fitting model (Hu & Bentler, 1999), and two absolute misfit indices: the Root mean Square Error of Approximation (RMSEA) with values < .05 indicating good fit and 0.5 - .08 indicating adequate fit (Browne & Cudeck, 1993) and the Weighted Root Mean Square Residual (WRMR) with a cut-off value of .95 indicating good fit (Yu, 2002). For each model, we first confirmed the fit of the measurement component by simultaneously fitting the CFA measurement models for all the included latent variables, allowing them to correlate. The data were then fitted to the structural component of each model to assess the direct and indirect paths linking positive and negative metacognitive beliefs to emotional distress.

5.3. Results

T1 sample characteristics are summarised in Chapter 4, Table 4:1. A large proportion of the sample exceeded cut-off scores for clinically significant anxiety (51%) or PTSD symptoms (59%). Women with breast cancer were more anxious (U=3722, p<.001, r=-0.31), and reported more PTSD symptoms (U=4105.5, p<.001, r=-0.25) than men with prostate cancer. Younger patients also reported more anxiety (U=5117, p=.004, r=-0.19), depression (U=5370, p=.017, r=-0.16) and PTSD symptoms (U=5238, p=.009, r=-0.17). However, no outcome was related to education, perceived emotional support or tumour grade. Therefore, age and gender were the only covariates entered in subsequent analyses.

Results of the preliminary regression analyses are summarised in Table 5:1. For anxiety and depression, the final model accounted for 32% and 19% of the variance, respectively. After controlling for age and gender, illness perceptions - specifically higher
scores on ‘Identity’, ‘Chronic timeline’, ‘Consequences’ (for anxiety & depression) and ‘Psychological causes’ (for anxiety) - explained an additional 20% and 18% of the variance, respectively. In the analysis of PTSD symptoms, the final model accounted for 34% of the variance. Higher scores on the same four illness perception scales, together with higher scores on ‘Treatment control’ and lower scores on ‘Illness coherence’, explained an additional 22% of the variance in PTSD symptoms after controlling for age and gender. These findings were confirmed as robust using bootstrapped sampling.
Table 5:1: Final models of the variance in anxiety, depression and trauma explained by illness perceptions, after controlling for age & gender.

<table>
<thead>
<tr>
<th></th>
<th>Anxiety Model</th>
<th></th>
<th>Depression Model</th>
<th></th>
<th>PTSD symptoms Model</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R² change</td>
<td>Beta</td>
<td>t</td>
<td>Sig</td>
<td>R² change</td>
<td>Beta</td>
</tr>
<tr>
<td>Constant</td>
<td>-.56</td>
<td>.582</td>
<td>.582</td>
<td>-.79</td>
<td>.428</td>
<td>-.75</td>
</tr>
<tr>
<td>Gender</td>
<td>.12*</td>
<td>-.33</td>
<td>-5.49</td>
<td>&lt;.001</td>
<td>.01</td>
<td>-.15</td>
</tr>
<tr>
<td>Age</td>
<td>-.06</td>
<td>-.98</td>
<td>.326</td>
<td>.02</td>
<td>.28</td>
<td>.778</td>
</tr>
<tr>
<td>IPQ-R</td>
<td>.20*</td>
<td></td>
<td>.18*</td>
<td></td>
<td>.22*</td>
<td></td>
</tr>
<tr>
<td>Identity (0/1)</td>
<td>.14</td>
<td>2.34</td>
<td>.020</td>
<td>.14</td>
<td>2.10</td>
<td>.037</td>
</tr>
<tr>
<td>Chronic timeline</td>
<td>.17</td>
<td>2.20</td>
<td>.029</td>
<td>.18</td>
<td>2.11</td>
<td>.036</td>
</tr>
<tr>
<td>Cyclical timeline</td>
<td>.12</td>
<td>1.82</td>
<td>.070</td>
<td>.10</td>
<td>1.46</td>
<td>.15</td>
</tr>
<tr>
<td>Consequences</td>
<td>.14</td>
<td>2.05</td>
<td>.041</td>
<td>.15</td>
<td>2.00</td>
<td>.046</td>
</tr>
<tr>
<td>Personal Control</td>
<td>-.07</td>
<td>-1.25</td>
<td>.212</td>
<td>-.13</td>
<td>-1.96</td>
<td>.051</td>
</tr>
<tr>
<td>Treatment Control</td>
<td>.13</td>
<td>1.71</td>
<td>.088</td>
<td>.07</td>
<td>.82</td>
<td>.412</td>
</tr>
<tr>
<td>Illness coherence</td>
<td>-.00</td>
<td>-.06</td>
<td>.951</td>
<td>-.01</td>
<td>-.17</td>
<td>.865</td>
</tr>
<tr>
<td>Psychological cause</td>
<td>.22</td>
<td>3.45</td>
<td>.001</td>
<td>.10</td>
<td>1.42</td>
<td>.156</td>
</tr>
</tbody>
</table>

Model Summary

| R²       | .32      | .19      | .34      |
| Adj R²   | .28      | .15      | .31      |

*p<.001
5.3.1 The association of metacognitive beliefs and distress

Results of the regression analyses are shown in Table 5:2. After controlling for age and gender, metacognitive beliefs explained 34% of additional variance in anxiety and 19% in depression. Even after controlling also for illness perceptions, metacognitive beliefs added a further 23% and 9% in each outcome, respectively. The final model for anxiety accounted for 52% of the variance. Both ‘Positive beliefs about worry’ and ‘Negative beliefs about worry’ made significant individual contributions, with negative beliefs making the largest contribution of all the predictors entered. The final model for depression accounted for 25% of the variance, with negative beliefs making the largest contribution. Analysis of PTSD symptoms showed a similar pattern (Table 5:2). Metacognitive beliefs explained 29% of additional variance after controlling for age and gender, and 17% after controlling also for illness perceptions. The final model explained 51% of the variance, with ‘Negative beliefs about worry’ again making the biggest contribution.

These findings, confirmed as robust using bootstrapped sampling, support the first prediction from the metacognitive model that metacognitive beliefs add to the variance explained in distress and trauma after controlling for illness perceptions, with ‘Negative beliefs about worry’ making the biggest contribution to the variance in each outcome.
Table 5:2: Final models of the variance in anxiety, depression and PTSD symptoms explained by metacognitive beliefs after controlling for; age & gender (Model 1), and age, gender & illness perceptions (Model 2).

<table>
<thead>
<tr>
<th></th>
<th>Anxiety Model 1</th>
<th></th>
<th>Anxiety Model 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R² change</td>
<td>Beta</td>
<td>t</td>
<td>Sig</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>1.20</td>
<td>.233</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td>.12*</td>
<td>-.22</td>
<td>-.438</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>-.05</td>
<td>-1.01</td>
<td>.312</td>
</tr>
<tr>
<td>IPQ-R</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Identity (0/1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic timeline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consequences</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychological cause</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCQ-30</td>
<td></td>
<td>.34*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td></td>
<td>.15</td>
<td>2.70</td>
<td>.007</td>
</tr>
<tr>
<td>NEG</td>
<td></td>
<td>.52</td>
<td>9.13</td>
<td>.001</td>
</tr>
<tr>
<td><strong>Model Summary</strong></td>
<td>R²</td>
<td>.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adj R²</strong></td>
<td></td>
<td>.45</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Depression Model 1</th>
<th></th>
<th>Depression Model 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R² change</td>
<td>Beta</td>
<td>t</td>
<td>Sig</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>-.12</td>
<td>.903</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td>.02</td>
<td>-.05</td>
<td>-.81</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>.00</td>
<td>.02</td>
<td>.983</td>
</tr>
<tr>
<td>IPQ-R</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Identity (0/1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic timeline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consequences</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCQ-30</td>
<td></td>
<td>.14*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td></td>
<td>.06</td>
<td>.82</td>
<td>.411</td>
</tr>
<tr>
<td>NEG</td>
<td></td>
<td>.36</td>
<td>5.09</td>
<td>&lt;.001</td>
</tr>
<tr>
<td><strong>Model Summary</strong></td>
<td>R²</td>
<td>.16</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adj R²</strong></td>
<td></td>
<td>.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>PTSD symptoms Model 1</td>
<td></td>
<td>PTSD symptoms Model 2</td>
<td></td>
</tr>
<tr>
<td>--------------------------</td>
<td>-----------------------</td>
<td>--------------------------</td>
<td>-----------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td></td>
<td>R² change Beta t Sig</td>
<td>R² change Beta t Sig</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>3.07 .002</td>
<td>.33 .740</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.12* -.15 -2.90 .004</td>
<td>.12* -.20 -3.77 &lt;.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-.15 -2.77 .006</td>
<td>-.11 -2.05 .041</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IPQ-R</td>
<td>.22*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Identity (0/1)</td>
<td>.17 3.28 .001</td>
<td>.12 2.02 .045</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic timeline</td>
<td>.09 1.30 .194</td>
<td>.12 2.02 .045</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consequences</td>
<td>.12 2.02 .045</td>
<td>.10 1.56 .122</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treatment control</td>
<td>-.16 -2.95 .004</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Illness coherence</td>
<td>.05 .99 .322</td>
<td>.05 .99 .322</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychological cause</td>
<td>.29*</td>
<td>.17*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td>.12 2.09 .037</td>
<td>.09 1.58 .115</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEG</td>
<td>.49 8.25 &lt;.001</td>
<td>.41 7.14 &lt;.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Model Summary</strong></td>
<td><strong>R²</strong> .41</td>
<td><strong>R²</strong> .51</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adj R²</strong></td>
<td><strong>.40</strong></td>
<td><strong>.48</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N.B. MCQ-30 subscales: Positive beliefs about worry (POS); Negative beliefs about the danger and uncontrollability of worry (NEG)
*p<.001

5.3.2. SEM - relationship between metacognitive beliefs and emotional distress

CFA confirmed an excellent fit of the data to the measurement model. The data were then fitted to the full latent variable model, initially using the PSWQ to indicate the putative mediating variable. Age and gender were controlled for within the model (being correlated with the independent variable(s) by default, and having specified causal effects on the putative mediator(s) and final outcome(s)). The final path model for anxiety, depression, and PTSD symptoms is shown in Figure 5.2. The model was a good fit ($\chi^2$ (df=1617) =1922, p<.001, RMSEA = .029 (90% CI =.02-.03), CFI/TLI =.98/.98, WRMR = .89). As predicted, significant direct effects were apparent from ‘Negative beliefs about worry’ to anxiety (Beta=.50, p<.001) and PTSD symptoms (Beta=.70, p<.001) but not from ‘Positive beliefs...
about worry’. In addition, there was a significant indirect path from ‘Negative beliefs about worry’ to anxiety (Beta = .16, p = .025) mediated by PSWQ+ve, as predicted. However, there were no significant direct or indirect paths from ‘Negative beliefs about worry’ to depression and no indirect path mediated by worry to PTSD symptoms. In addition, the paths from ‘Positive beliefs about worry’ to both PSWQ+ve and PSWQ-ve were not significant.

The model testing was then repeated using the CAS-1 subscale as the mediating variable instead of the PSWQ. The final path model is shown in Figure 5.3. The model was a good fit ($\chi^2$ (df=919) = 1189, p < .001, RMSEA = .037 (90% CI = .03-.04), CFI/TLI = .98/.97, WRMR = .91). The pattern of significant direct paths seen above was replicated; there were significant direct effects of ‘Negative beliefs about worry’ on anxiety (Beta = .43, p < .001) and PTSD symptoms (Beta = .36, p < .001). In addition, there was also a significant indirect effect via the CAS-I on all three outcomes (Indirect Effects: anxiety Beta = .24, P < .001; depression Beta = .22, P = .017; PTSD symptoms Beta = .32 P < .001). There was no effect of ‘Positive beliefs about worry’ on either the CAS-1 or any of the outcomes.
Figure 5.2: Final path model of relationship of positive and negative metacognitive beliefs with anxiety, depression and PTSD symptoms, including mediation by worry (PSWQ)

N.B.: Solid lines p<.05 with standardised coefficients; Dotted lines not significant. Measurement model component of full SEM and pathways for covariates (Age & Gender) not shown but available on request from corresponding author. MCQ-30 subscales: Positive beliefs about worry (POS); Negative beliefs about worry (NEG). PSWQ subscales: Positively phrased items (PSWQ+ve); negatively phrased (PSWQ-ve).

Figure 5.3: Final path model of relationship between positive and negative metacognitive beliefs and anxiety, depression and trauma mediated by the CAS-I

N.B.: Solid lines p<.05 with standardised coefficients; Dotted lines not significant. Measurement model component of full SEM and pathways for covariates (Age & Gender) not shown but available on request from corresponding author. MCQ-30 subscales: Positive beliefs about worry (POS); Negative beliefs about worry (NEG).
5.4. Discussion

This is the first study to explore the utility of the metacognitive model of emotional disorder in an adult cancer population and, although only cross-sectional, findings are largely consistent with the theory that metacognitive beliefs and perseverative thinking (worry), rather than specific illness perceptions, cause and maintain emotional distress.

5.4.1 The relationship between metacognitive beliefs and distress

Negative illness perceptions were associated with distress after cancer diagnosis, consistent with both the CSM and S-REF models. However, after controlling for age and gender, metacognitive beliefs could explain more of the remaining variance than could illness perceptions for two of the three study outcomes (anxiety: 34 % versus 20%; PTSD symptoms: 29% versus 22%). In addition, after controlling for age, gender and illness perceptions, metacognitive beliefs added significantly to the variance in anxiety, depression and PTSD symptoms while, in each case, ‘Negative beliefs about worry’ made the biggest individual contribution to the variance out of all of the predictors. These latter findings are consistent with the metacognitive model, and with results of previous studies in mental health populations (see (Wells, 2009) for a review), the general population (Spada et al., 2008) and Parkinson’s disease patients (Allott et al., 2005) where ‘negative beliefs about worry’ was the predominant contributor to the variance in anxiety and depression.

The regression analysis also indicated that a second set of metacognitive beliefs, ‘Positive beliefs about worry’, made a unique contribution to the variance in anxiety. This finding is consistent with the metacognitive model of generalised anxiety disorder (Wells &
Mathews, 1994) in which positive metacognitive beliefs guide the selection of worry as an effective coping strategy which, in turn, increases emotional distress.

5.4.2 Mediation of the relationship between metacognitive beliefs and distress by the CAS

The metacognitive model proposes that the causal link between metacognitive beliefs and distress is the CAS and, in this respect, the findings partially support predictions from the model. Specifically, the relationship of anxiety with ‘Negative beliefs about worry’ was partially mediated, as predicted, by the PSWQ and the relationship of all three emotional distress outcomes with ‘Negative beliefs about worry’ was partially mediated by the CAS-1. That is, the findings are broadly consistent with the theory that negative metacognitive beliefs, (e.g. ‘worry is uncontrollable and dangerous’) cause a direct emotional response (anxiety and trauma symptoms) while also further increasing distress by exacerbating worry and activating meta-worry (e.g. ‘I worry too much about worrying’). The absence of any direct effect of ‘negative beliefs about worry’ on depression may reflect the wording of this measure which focuses specifically on beliefs about worry as opposed to other forms of persistent thinking (i.e. rumination) that are more closely associated with depression.

The hypothesis of full mediation between positive metacognitive beliefs and emotional distress - that is that ‘Positive beliefs about worry’, such as ‘worrying will help me notice if my cancer recurs’, causes emotional distress by driving worry about recurrence and self-focussed attention - was not supported. However, metacognitive theory would predict that, although positive metacognitive beliefs initially guide an individual towards the selection of CAS processes (i.e. worry) in response to negative thoughts or feelings, it is the negative metacognitive beliefs that ‘turbo charge’ distress by then exacerbating and maintaining these
processes. Thus it is possible that in a SEM which simultaneously tests all the pathways between metacognitive beliefs and emotional distress, the indirect pathway from ‘positive beliefs about worry’ to emotional distress via the CAS is masked by the inclusion of ‘negative beliefs about worry’.

5.4.3 Study implications, limitations and conclusions

In summary, the findings support predictions from the metacognitive model that negative metacognitive beliefs cause and maintain distress by activating the CAS. However, because the study was cross-sectional, causality cannot be assumed; maladaptive metacognition may be a consequence of emotional distress, not a cause and, as these two opposing models would be mathematically equivalent, SEM would be unable to distinguish between them. Therefore a prospective test of the model is necessary, in order to establish the temporal precedence of maladaptive metacognition to persistent distress as more compelling evidence of causation. Furthermore, as the SEM was based on the assumption of no hidden confounders, the potential influence of unmeasured common causes cannot be eliminated. In particular, the information available from patients at the time of assessment did not include their history of anxiety, depression or PTSD symptoms. Consequently it is possible that, rather than maladaptive metacognitions causing elevated emotional distress, both are consequences of a pre-morbid psychiatric history. Another limitation is the sample. To balance the competing demands of maximising recruitment and generalizability, while minimising prognostic variability, sampling was restricted to the largest tumour groups in each gender - breast and prostate cancer; it cannot be assumed that findings would generalise to other cancers, particularly those with poorer prognosis. Although we controlled for gender (and therefore type of tumour) in the analyses, the study was insufficiently powered for
subgroup analyses. Further studies will be needed to test the stability of association of metacognitive beliefs with emotional distress across different tumour populations.

Despite these limitations, this study does provide first evidence of the applicability of the Metacognitive model to understanding emotional distress and trauma after diagnosis of cancer. Therefore, we suggest that there is potential to reduce vulnerability to emotional distress and trauma by modifying metacognitive beliefs and processes rather than using more traditional cognitive therapies. In a cancer context, an important potential advantage of this metacognitive approach to therapy is that it does not require engagement with the content of negative thoughts about cancer, which many individuals can find difficult or distressing (Baker et al., 2012). However, in order to explore this potential more fully, further study, both prospective and experimental, is warranted.
Chapter Six

Study three

A prospective study of the association of metacognitive beliefs and processes with persistent emotional distress after diagnosis of cancer.

(for Published article see Appendix D)
6.1 Introduction

The previous studies (Chapters 4 & 5) indicated that metacognitive beliefs (specifically ‘Positive beliefs about worry’ & ‘Negative beliefs about worry’) were associated with concurrent symptoms of anxiety, depression and trauma among patients recently diagnosed with breast or prostate cancer, and that they explained additional variance in these outcomes after controlling for age, gender and negative content of thoughts about cancer (i.e. negative illness perceptions). Structural equation modelling (Chapter 5) found evidence consistent with the central predictions of the metacognitive model that these beliefs cause and maintain distress directly, but also indirectly by driving worry. These findings provide the first evidence consistent with the theory that metacognitive beliefs underlie emotional distress experienced by cancer patients. However, in order to provide more compelling evidence of a causal role for metacognitive beliefs in maintaining emotional distress after cancer, prospective research is needed to demonstrate a temporal relationship. Consequently, the aim of this study is to explore whether metacognitive beliefs measured at the pre-treatment assessment (T1) predict symptoms of anxiety, depression and trauma twelve months later (T2), and to explore whether they add to the variance explained over and above previously implicated variables, including T1 distress and negative content of thoughts about cancer (i.e. T1 illness perceptions). Specifically it is hypothesised that:

(1) Metacognitive beliefs assessed around the time of diagnosis will prospectively predict variance in anxiety, depression and trauma 12 months later.

(2) Metacognitive beliefs assessed around the time of diagnosis will add to the variance explained in T2 anxiety, depression and trauma symptoms over and above demographic variables, T1 symptoms and T1 illness perceptions.
6.2 Method

This study used a prospective cohort design with a pre-treatment baseline (T1) and twelve month follow-up. See Chapter 4 for a full account of the prospective study including; study inclusion criteria, recruitment and procedure.

6.2.1 Measures

Anxiety and depression were measured using the Hospital Anxiety and Depression scale (HADS, (Zigmond & Snaith, 1983)), and trauma symptoms using the Impact of Events Scale (IES; (Horowitz et al., 1979)). The revised Illness Perceptions Questionnaire (IPQ-R (Moss-Morris et al., 2002)) was used to assess the content of thoughts (i.e. illness perceptions) about cancer, while metacognitive beliefs were measured using the Metacognitions Questionnaire 30 (MCQ-30 (Wells & Cartwright-Hatton, 2004)). For full details of these measures see Chapter 5.

All measures were assessed both at T1 and T2.

6.2.2 Analysis

The data were analysed using SPSS Version 20.

Nonparametric statistics (Mann-Whitney or Kruskal-Wallis) were used to compare T2 levels of each emotional distress outcome by age group (dichotomised at the median), gender, educational level, perceived emotional social support and stage of disease. Where significant differences in T2 outcomes were found (p<.05), the relevant variables were entered as demographic covariates in the first step of subsequent regression analyses.
In order to control for negative content of thoughts about cancer at T1 (i.e. illness perceptions), it is first necessary to establish the T1 illness perceptions that contribute to variance in T2 distress after controlling for baseline emotional distress. Therefore, the IPQ-R and MCQ-30 were analysed in parallel to identify which subscales within each measure independently predicted each T2 outcome. For the IPQ-R, hierarchical multiple regression analyses were first used to identify the T1 subscales associated with each T2 outcome (anxiety, depression and trauma) after controlling for demographic variables (Analysis 1 for each outcome). These analyses were then repeated, using just the significant IPQ-R subscales from Analysis 1, and also controlling for T1 symptoms of anxiety, depression or trauma (Analysis 2 for each outcome). As we had no a priori theory about which subscales would independently predict T2 outcomes, the IPQ-R subscales were included in each analysis using stepwise rather than forced entry. The subscales identified as independent predictors in Analysis 2 for each outcome were then entered as control variables in Analysis 3 for that outcome (see below).

This sequence of analyses was also used for the MCQ-30, thereby testing hypothesis 1. We first identified the T1 MCQ-30 subscales that independently predicted T2 outcomes after controlling for demographic variables (Analysis 1 for each outcome), and then entered these in a further analysis also controlling for T1 symptoms of anxiety, depression or trauma (Analysis 2 for each outcome). As with the IPQ-R analyses, as we had no a priori theory about which subscales would independently predict T2 outcomes, MCQ-30 subscales were included in each analysis using stepwise rather than forced entry. The subscales identified as independent predictors in Analysis 2 for each outcome were then entered as variables in Analysis 3 for that outcome (see below), which tested hypothesis 2.

Final hierarchical multiple regression analyses (Analysis 3 for each outcome) assessed whether the T1 MCQ-30 subscales which had been identified as significant predictors in
Analysis 2 (see above) were able to predict variance in T2 outcomes over and above that explained by demographic variables, T1 symptoms and the negative content of thoughts about cancer at T1 (i.e. IPQ-R subscales identified as significant predictors in Analysis 2). This final analysis used forced entry and bootstrapped sampling to ensure findings were robust.

6.3 Results

The demographic and clinical characteristics of the final sample (N=206) are shown in Table 4.1 (Chapter 4). Women with breast cancer and younger patients were more anxious at T2 (U=-3269.5, p<.001, r=-0.27; U=-3721, p<.001, r=0.26), and reported more trauma symptoms (U=3636, p=.003, r=-0.21; U=3638, p<.001, r=0.27) than did men with prostate cancer or older patients. Women with breast cancer also reported more symptoms of depression at T2 than did men with prostate cancer (U= 3857.5, p=.014, r=0.17). No outcome was related to education, perceived emotional support or tumour grade. Therefore, just age and gender were used as demographic covariates in subsequent analyses. The levels of anxiety, depression and trauma symptoms at both time points are shown in Table 6.1. Both anxiety and trauma symptoms significantly declined over time, whereas depressive symptoms significantly increased.

Table 6.1: Distribution of anxiety, depression and trauma scores at both time-points

<table>
<thead>
<tr>
<th></th>
<th>Time 1</th>
<th>Time 2</th>
<th>T1-T2 Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Median (IQR)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>Anxiety</td>
<td>7.6 (4.4)</td>
<td>7.5 (4-11)</td>
<td>6.2 (4.5)</td>
</tr>
<tr>
<td>Depression</td>
<td>3.3 (3.3)</td>
<td>2 (1-5)</td>
<td>4.1 (3.9)</td>
</tr>
<tr>
<td>Trauma</td>
<td>29.4 (16.9)</td>
<td>31 (14-42.3)</td>
<td>21.2 (18.9)</td>
</tr>
</tbody>
</table>
6.3.1 Association of T1 illness perceptions with T2 anxiety, depression and trauma

Regression of emotional distress on the IPQ-R subscales (Table 6.2) indicated that illness perceptions predicted between 10% (trauma) and 12% (anxiety) of the variance in T2 outcomes after controlling for age and gender (Analysis 1) and between 2% (trauma) and 3% (anxiety and depression) after also controlling for T1 symptoms (Analysis 2). The final models from Analysis 2 indicated that perceived lack of personal control and negative perception of the consequences of cancer predicted T2 anxiety (1% and 2% respectively), while poor understanding of the illness (‘Illness coherence’) predicted T2 depression and trauma. These IPQ-R subscales were therefore used to control for content of thoughts about cancer in the final hierarchical multiple regression analyses.
Table 6.2 Final models of the variance in T2 anxiety, depression and trauma predicted by T1 illness perceptions after controlling for age and gender (Analysis 1) and age, gender & T1 levels of symptoms (Analysis 2). $R^2$ change shows increment in variance explained when each set of variables was entered sequentially; beta, T and p are from the final model containing variables from all steps.

<table>
<thead>
<tr>
<th>ANALYSIS 1</th>
<th>T2 Anxiety</th>
<th></th>
<th></th>
<th>T2 Depression</th>
<th></th>
<th></th>
<th>T2 Trauma</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>Beta</td>
<td>T</td>
<td>p</td>
<td>$R^2$</td>
<td>Beta</td>
<td>T</td>
<td>p</td>
</tr>
<tr>
<td>Constant</td>
<td>3.05</td>
<td>.003</td>
<td></td>
<td></td>
<td>3.13</td>
<td>.002</td>
<td></td>
<td>4.74</td>
</tr>
<tr>
<td>STEP 1 - Demographics</td>
<td>.13***</td>
<td></td>
<td></td>
<td></td>
<td>.05**</td>
<td></td>
<td></td>
<td>.14***</td>
</tr>
<tr>
<td>Gender</td>
<td>-.19</td>
<td>-3.04</td>
<td>.003</td>
<td></td>
<td>-.13</td>
<td>-1.94</td>
<td>.054</td>
<td>-.14</td>
</tr>
<tr>
<td>Age</td>
<td>-.21</td>
<td>-3.28</td>
<td>.001</td>
<td></td>
<td>-.11</td>
<td>-1.62</td>
<td>.106</td>
<td>-.27</td>
</tr>
<tr>
<td>STEP 2 - IPQ-R #</td>
<td>.12***</td>
<td></td>
<td></td>
<td></td>
<td>.11**</td>
<td></td>
<td></td>
<td>.10***</td>
</tr>
<tr>
<td>Identity</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Cyclical timeline</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Chronic timeline</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Consequences</td>
<td>.22</td>
<td>3.41</td>
<td>.001</td>
<td></td>
<td>.19</td>
<td>2.75</td>
<td>.006</td>
<td>.19</td>
</tr>
<tr>
<td>Illness coherence</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td></td>
<td>-.18</td>
<td>-2.73</td>
<td>.007</td>
<td>-.24</td>
</tr>
<tr>
<td>Psychological attributions</td>
<td>.19</td>
<td>2.97</td>
<td>.003</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Personal control</td>
<td>-.14</td>
<td>-2.18</td>
<td>.030</td>
<td></td>
<td>-.13</td>
<td>-2.02</td>
<td>.045</td>
<td>ns</td>
</tr>
<tr>
<td>Treatment control</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

$R^2$ .25

Adj $R^2$ .23

107
### ANALYSIS 2

<table>
<thead>
<tr>
<th></th>
<th>T2 Anxiety</th>
<th></th>
<th>T2 Depression</th>
<th></th>
<th>T2 Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>Beta</td>
<td>$T$</td>
<td>$p$</td>
<td>$R^2$</td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td></td>
<td>2.79</td>
<td>.006</td>
<td></td>
<td>4.61</td>
</tr>
<tr>
<td><strong>STEP 1 - Demographics</strong></td>
<td></td>
<td>.13***</td>
<td></td>
<td></td>
<td>.05**</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td>-.03</td>
<td>-0.42</td>
<td>.675</td>
<td>-.08</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>-.17</td>
<td>-2.99</td>
<td>.003</td>
<td>-.15</td>
</tr>
<tr>
<td><strong>STEP 2 – T1 Symptoms</strong></td>
<td></td>
<td>.25***</td>
<td></td>
<td></td>
<td>.21***</td>
</tr>
<tr>
<td>T1 Anxiety</td>
<td></td>
<td>.49</td>
<td>8.03</td>
<td>.000</td>
<td></td>
</tr>
<tr>
<td>T1 Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.44</td>
</tr>
<tr>
<td>T1 Trauma</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>STEP 2 - IPQ-R</strong></td>
<td></td>
<td>.03*</td>
<td></td>
<td></td>
<td>.03**</td>
</tr>
<tr>
<td>Consequences</td>
<td></td>
<td>.13</td>
<td>2.17</td>
<td>.032</td>
<td>ns</td>
</tr>
<tr>
<td>Illness coherence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-.16</td>
</tr>
<tr>
<td>Psychological attributions</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Personal Control</td>
<td></td>
<td>-.11</td>
<td>-2.08</td>
<td>.039</td>
<td>ns</td>
</tr>
</tbody>
</table>

| $R^2$                | .41        |                       | .29           |                       | .39       |                       |               |                       |           |                       |
| Adj $R^2$            | .39        |                       | .27           |                       | .37       |                       |               |                       |           |                       |

N.B. IPQ-R subscales entered using stepwise method. * All eight IPQ-R subscales were included but only those found to be significant predictors for one or more outcome are shown. ** $p<.05$ *** $p<.01$, **** $p<.001$, ns - non significant, data not available using stepwise methods. ** Only subscales found to be significant predictors in Analysis 1 were entered. Shaded cells indicate that variable was not included.
6.3.2. Association of T1 metacognitive beliefs with T2 anxiety, depression and trauma

The results of the hierarchical multiple regression analyses to test hypothesis 1 are shown in Table 6.3. After controlling for age and gender (Analysis 1), metacognitive beliefs explained an additional 19% of the variance in T2 anxiety, 15% of the variance in T2 depression and 14% of the variance in T2 trauma. In all cases ‘Negative beliefs about worry’ made the largest individual contribution of all the predictors, with ‘Cognitive confidence’ also making a significant individual contribution. For anxiety, ‘Positive beliefs about worry’ was a further significant individual predictor of T2 symptoms. After controlling for T1 symptoms as well as demographic variables (Analysis 2), metacognitive beliefs continued to predict a small but significant proportion of variance in each outcome. It added a significant 2% to the variance in T2 anxiety, 5% to the variance in T2 depression and 1% to the variance in T2 trauma. In each case, ‘Cognitive confidence’ was the only MCQ-30 subscale that continued to make a significant individual contribution to the variance explained, and consequently this variable was the only metacognitive variable entered into the final set of analyses (Analysis 3).
Table 6.3 Final models of the variance in T2 anxiety, depression and trauma predicted by T1 metacognitive beliefs after controlling for age and gender (Analysis 1) and age, gender & T1 levels of symptoms (Analysis 2). $R^2$ change shows increment in variance explained when each set of variables was entered sequentially; beta, T and p are from the final model containing variables from all steps.

<table>
<thead>
<tr>
<th>ANALYSIS 1</th>
<th>T2 Anxiety</th>
<th>T2 Depression</th>
<th>T2 Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$ change</td>
<td>Beta</td>
<td>T</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>2.98</td>
<td>.003</td>
</tr>
<tr>
<td><strong>STEP 1 - Demographics</strong></td>
<td>.13***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>-.16</td>
<td>-2.67</td>
<td>.008</td>
</tr>
<tr>
<td>Age</td>
<td>-.20</td>
<td>-3.39</td>
<td>.001</td>
</tr>
<tr>
<td><strong>STEP 2 - MCQ-30</strong></td>
<td>.19**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td>.17</td>
<td>2.58</td>
<td>.011</td>
</tr>
<tr>
<td>NEG</td>
<td>.28</td>
<td>3.90</td>
<td>.000</td>
</tr>
<tr>
<td>CC</td>
<td>.12</td>
<td>2.00</td>
<td>.047</td>
</tr>
<tr>
<td>NC</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>CSC</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

$R^2$ .32
Adj $R^2$ .30
### Analysis 2

<table>
<thead>
<tr>
<th></th>
<th>T2 Anxiety</th>
<th></th>
<th></th>
<th>T2 Depression</th>
<th></th>
<th></th>
<th>T2 Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>Beta</td>
<td>T</td>
<td>$p$</td>
<td>$R^2$</td>
<td>Beta</td>
<td>T</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>3.70</td>
<td>0.00</td>
<td></td>
<td>2.34</td>
<td>0.020</td>
<td></td>
</tr>
<tr>
<td><strong>STEP 1 - Demographics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>.13***</td>
<td>-.03</td>
<td>-0.54</td>
<td>.590</td>
<td>-.09</td>
<td>-1.42</td>
<td>.158</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>-.20</td>
<td>-3.62</td>
<td>0.000</td>
<td>-.13</td>
<td>-2.18</td>
<td>.030</td>
</tr>
<tr>
<td><strong>STEP 2 – T1 Symptoms</strong></td>
<td></td>
<td>.25***</td>
<td></td>
<td></td>
<td>.21***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 Anxiety</td>
<td></td>
<td>.50</td>
<td>8.22</td>
<td>0.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 Depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T1 Trauma</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>STEP 3 - MCQ-30</strong></td>
<td>.02*</td>
<td></td>
<td></td>
<td></td>
<td>.05***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEG</td>
<td></td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CC</td>
<td></td>
<td>.14</td>
<td>2.53</td>
<td>.012</td>
<td>.23</td>
<td>3.85</td>
<td>.000</td>
</tr>
</tbody>
</table>

$R^2 = .40$  
$Adj R^2 = .39$

$R^2 = .31$  
$Adj R^2 = .30$

$R^2 = .38$  
$Adj R^2 = .36$

N.B. MCQ-30 subscales entered using stepwise method. MCQ-30 subscales: Positive beliefs about worry (POS); Negative beliefs about the danger and uncontrollability of worry (NEG); Cognitive confidence (CC); Need for control (NC); Cognitive self-consciousness (CSC). *$p<.05$  **$p<.01$, ***$p<.001$, ns - non significant, data not available using stepwise methods. ** Only subscales found to be significant predictors in Analysis 1 were entered. Shaded – not included.
6.3.3. Predictive ability of T1 metacognitive beliefs over and above demographic variables, T1 symptoms and content of thoughts about cancer

The results of the hierarchical multiple regression analyses to test the second hypothesis (Analysis 3) are shown in Table 6.4. For anxiety and depression, ‘Cognitive confidence’ added a significant 2% and 4% respectively to the variance in T2 symptoms over and above demographic variables, T1 symptoms and content of thoughts about cancer (i.e. relevant T1 illness perceptions). For anxiety, younger age, baseline symptoms, perceived lack of personal control and low cognitive confidence each made a significant individual contribution to the final model, which accounted for 42% of the variance in T2 symptoms. For depression, just younger age, baseline symptoms and low cognitive confidence made significant independent contributions to the final model, which accounted for 33% of the variance in T2 symptoms.

In the case of trauma, ‘Cognitive confidence’ did not make any significant contribution to the variance explained in T2 symptoms after controlling for demographic variables, T1 symptoms and T1 illness perceptions (‘Illness coherence’). In fact, younger age and T1 symptoms were the only variables to make a significant individual contribution to the final model, which accounted for 39% of the variance.
Table 6.4 Final models of the variance in T2 anxiety, depression and trauma predicted by T1 metacognitive beliefs after controlling for age, gender, T1 level of symptoms and T1 illness perceptions (Analysis 3). $R^2$ change shows increment in variance explained by each step; beta, T and p are from the final model containing variables from all steps.

**ANALYSIS 3**

<table>
<thead>
<tr>
<th></th>
<th>T2 Anxiety</th>
<th></th>
<th>T2 Depression</th>
<th></th>
<th>T2 Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$R^2$</td>
<td>Beta</td>
<td>T</td>
<td>p</td>
<td>$R^2$</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>2.50</td>
<td>.013</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>STEP 1 - Demographics</strong></td>
<td>.13***</td>
<td></td>
<td></td>
<td></td>
<td><strong>STEP 2 – T1 Symptoms</strong></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td>-.04</td>
<td>-.63</td>
<td>.527</td>
<td>T1 Anxiety</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td>-.18</td>
<td>-.06</td>
<td>.003</td>
<td>T1 Depression</td>
</tr>
<tr>
<td><strong>STEP 3 – IPQ-R</strong></td>
<td>.03**</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consequences</td>
<td></td>
<td>.12</td>
<td>1.94</td>
<td>.054</td>
<td>T1 Trauma</td>
</tr>
<tr>
<td>Illness coherence</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Personal control</td>
<td></td>
<td>-.11</td>
<td>-.20</td>
<td>.038</td>
<td></td>
</tr>
<tr>
<td><strong>Model Summary</strong></td>
<td>$R^2$ .42</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Adj $R^2$ .41</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

N.B. All variables entered using forced entry method. MCQ-30 subscales: Cognitive confidence (CC). ** Only subscales found to be significant predictors in Analysis 2 were entered. Shaded – not included. * $p<.05$ ** $p<.01$, *** $p<.001$
6.4. Discussion

This is the first study to explore whether metacognitive beliefs soon after cancer diagnosis, and before active treatment (T1), predict emotional distress 12 months later (T2). T1 metacognitive beliefs predicted T2 anxiety, depression, and trauma after controlling for age, gender and T1 symptoms, thus supporting hypothesis 1. This finding builds on previous research in non-clinical populations in which metacognitive beliefs prospectively predicted levels of anxiety and depression two (Weber & Exner, 2013), three (Hjemdal, Stiles, & Wells, 2013) and six months (Yilmaz, Gencoz, & Wells, 2011) later, after controlling for age, gender and T1 levels of symptoms.

Before controlling for T1 symptoms, metacognitive beliefs explained a greater proportion of variance in T2 anxiety, depression and trauma than did illness perceptions. Furthermore the illness perception subscales that were predictive (‘Consequences’, ‘Personal control’, ‘Psychological attributions’, ‘Illness coherence’) could be considered to be markers for worry or rumination in that they may be the outcome of these processes. Of the five MCQ-30 subscales included in Analysis 1, two (‘Negative beliefs about worry’ and ‘Cognitive confidence’) independently predicted T2 anxiety, depression and trauma, with a third (‘Positive beliefs about worry’) also significantly contributing to the variance in anxiety. In all three cases, ‘Negative beliefs about worry’ made the largest individual contribution, as would be predicted by the metacognitive model of emotional disorder (Wells, 2009). These findings are also consistent with those of Yilmaz et al (Yilmaz et al., 2011) who reported that, in their non-clinical sample, ‘Negative beliefs about worry’ predicted levels of anxiety and depression six months later. However, in the current study when T1 levels of distress were controlled, the relationship of ‘Negative beliefs about worry’ with anxiety and depression was no longer significant. Instead, ‘Cognitive confidence’ was the only metacognitive variable to contribute to variance. The reasons for this are not clear.
One possibility is that this finding is due to the limitations of using hierarchical regression in a prospective study design, where baseline emotional distress inevitably predominates in predicting future distress. In Chapter 5 a strong cross-sectional association of T1 symptoms of anxiety, depression and trauma with metacognitive beliefs and processes was demonstrated. Consequently, as the metacognitive beliefs and processes measured to predict T2 distress also (according to theory) cause T1 distress, there is likely to be considerable overlap in the variance in T2 distress explained by T1 symptoms and metacognitive beliefs, leading to underestimation of the importance of the putative causal variables. That is, by controlling for baseline symptoms we may be masking the effect of the beliefs and processes that underlie its maintenance. To resolve this dilemma, approaches to analysis are required that can distinguish putatively causal effects arising from metacognitive beliefs and processes (causing symptoms of distress to be maintained) from the confounding effect resulting from symptom maintenance. Such differentiation is not feasible using standard hierarchical regression, but may be possible using structural equation modelling techniques to model the effect of change in metacognitive beliefs on change in emotional distress. This is addressed in Chapter 7.

As well as being able to explain more of the variance in T2 distress than did illness perceptions in Analysis 1, metacognitive beliefs (‘Cognitive confidence’) were also able to explain additional variance in anxiety and depression over and above age, gender, T1 symptoms, and T1 illness perceptions (Analysis 3). This supports hypothesis 2 for these two outcomes. However, for trauma, metacognitive beliefs (‘Cognitive confidence’) no longer significantly predicted T2 symptoms after including T1 illness perceptions (‘illness coherence’) in the analysis (Analysis 3). However, it should be noted that the proportion of variance in T2 trauma explained by ‘Cognitive confidence’ is unchanged between Analysis 2 (controlling for T1 trauma) and Analysis 3 (controlling for T1 trauma and T1 ‘illness
coherence’). Furthermore, there is little difference in the variance explained by ‘Cognitive confidence’ in trauma (1%) and in anxiety (2%). Therefore, this apparent discrepancy (i.e. metacognitive beliefs explaining additional variance over and above illness perceptions for T2 anxiety and T2 depression but not T2 trauma) may be an artefact of the present data. To our knowledge this is the first study to explore the prospective relationship between metacognitive beliefs (as measured by the MCQ-30) and trauma symptoms, making it difficult to judge the reliability of this finding.

One limitation of the study is the restriction of the sample to breast and prostate cancer patients. These populations were selected because they represent the largest tumour groups in each gender and have a broadly similar prognosis. However, this means it is not possible, in this sample, to separate out any effects that may be due to tumour group or gender. Furthermore, we cannot assume that the predictive effects found in this study would generalise to other cancer populations. Further studies will be needed to test the stability of the observed predictive effect of metacognitive beliefs on persistent emotional distress across genders and different tumour and prognostic groups. In addition, despite the prospective design, it should be noted that causality can still not be assumed as the influence of unmeasured confounders cannot be ruled out. In order to provide more compelling evidence of a causal role for metacognitive beliefs, further studies are necessary that adopt different approaches to design, such as experimental manipulation.

In summary, the findings of the current study provide promising first evidence that metacognitive beliefs can help to predict anxiety, depression and trauma one year after diagnosis of breast and prostate cancer. Furthermore, they support the hypothesis that metacognitive beliefs add to the variance explained in persistent anxiety and depression over and above that explained by negative content of thoughts about cancer. Consequently, therapeutic approaches targeting metacognitive beliefs and processes - rather than the content
of negative thoughts about cancer - may prove beneficial for preventing persistent emotional distress in these populations.
Chapter Seven

Study four

Identifying causal predictors of emotional distress 12 months after cancer diagnosis: A Latent Growth Curve Analysis
7.1. Introduction

In the previous chapter (Chapter 6) metacognitive beliefs around the time of diagnosis (T1) of breast or prostate cancer were shown to prospectively predict variation in the symptoms of anxiety, depression and trauma 12 months later (T2). However, the amount of additional variance explained after controlling for the level of symptoms at T1 was small. It was suggested that this finding may be partly because prospective regression analysis is limited in its ability to advance our understanding of the underlying causal processes and that alternative approaches are needed that can disentangle effects arising from causal processes from the confounding effects of enduring symptoms.

One such approach is structural equation modelling (SEM). For theory testing, SEM has several advantages over regression analysis (Musil, Jones, & Warner, 1998). Firstly, rather than just testing individual relationships between predictor variables and a single outcome, as in multiple regression analysis, SEM permits simultaneous representation of multiple associations and causal paths by a series of structural equations determined by theory, which is then tested to determine how well a hypothesised model fits the data (Byrne, 2012; Musil et al., 1998). Thus it becomes possible to test the model in its entirety. In addition, whereas regression analysis uses observed variables and is unable to assess or correct for error, SEM uses latent variables with multiple indicators and provides explicit estimates of the associated error variance parameters (Byrne, 2012). In cross-sectional analyses, SEM has provided preliminary evidence supporting a key predictions from the metacognitive model (Chapter 5). That is, that negative metacognitive beliefs (i.e. belief in the danger and uncontrollability of worry) cause both a direct emotional response, and also exacerbate distress indirectly by activating worry about worry (meta-worry). Prospective SEM enables us to build on this work because, as well as making comparisons between subjects at one point in time, it also makes comparisons within subjects (over time) providing
a clearer assessment of temporal relations between metacognitive beliefs, cognitive processes and emotional distress. Furthermore, by using latent growth curve (LGC) modelling it is possible to separate out the enduring component of T1 emotional distress (i.e. that portion of T1 distress that is maintained across time points) from the change components (i.e. change in symptoms over time), that is: to test whether change in metacognitive beliefs affects change in worry which in turn affects change in distress.

The current study explores the clinical utility of the metacognitive model in cancer by conducting the first prospective test of its key theoretical predictions using LGC modelling. Specific predictions are that:

(1) Increase in metacognitive beliefs (‘Negative beliefs about worry’, ‘Positive beliefs about worry’, ‘Cognitive confidence’) over 12 months will be associated with an increase in emotional distress (anxiety; depression; trauma).

(2) The relationship between increase in positive metacognitive beliefs (i.e. ‘Positive beliefs about worry’) and increase in emotional distress will be fully mediated by increase in CAS processes (i.e. worry and threat-focused attention).

(3) The relationship between increase in negative metacognitive beliefs (i.e. ‘Negative beliefs about worry’, ‘Cognitive confidence’) and increase in emotional distress will be partially mediated by increase in CAS processes (i.e. worry and threat-focused attention).
7.2 Method

7.2.1. Design

This study used data from the prospective cohort which has been described previously (Chapters 4 & 6).

7.2.2 Measures

All measures were administered at both time points.

Emotional distress was assessed using the Hospital Anxiety and Depression Scale (HADS, (Zigmond & Snaith, 1983)) to measure symptoms of anxiety and depression and the Impact of Events Scale (IES; (Horowitz et al., 1979)) to measure symptoms of trauma. Metacognitive beliefs were measured using the Metacognitions Questionnaire 30 (MCQ-30, (Wells & Cartwright-Hatton, 2004)). As previous prospective regression analyses (Chapter 7) indicated that only three components of metacognitive belief (‘Positive beliefs about worry’, ‘Negative beliefs about worry’ and ‘Cognitive confidence’) independently predict variance in emotional distress, these were the only MCQ-30 subscales used in the present study. The CAS was assessed using two different measures. Firstly, the Penn State Worry Questionnaire (PSWQ, (Meyer et al., 1990)) was used to assess worry; a key component of the CAS. Secondly, in a separate set of analyses, the Cognitive Attentional Syndrome Scale (CAS-I, (Wells, 2009)) was used to (a) guard against possible bias in the conclusions drawn, due to common method variance between the ‘Negative beliefs about worry subscale’ of the MCQ-30 and the PSWQ (as described previously in Chapter 5); and (b) provide a broader assessment of the key CAS processes (i.e. worry and threat focussed attention). For full details of these measures see Chapter 5.
7.2.3 Analysis

The data were analysed using Mplus v6.12. As not all scales were normally distributed, bootstrapping techniques were used to ensure findings were robust to non-normality (Efron & Tibshirani, 1993).

Latent Growth Curve Model (LGC) modelling tested predictions that change in metacognitive beliefs is associated with change in emotional distress and that this relationship is mediated by change in key CAS processes (i.e. worry & threat focussed attention). LGC models generally comprise two parts (Byrne, 2012; MacKinnon, 2008), each represented by latent factors: the ‘intercept’ – which represents the starting point for each variable (T1) and an enduring component that is maintained over time; and the ‘slope’ which represent the growth in each variable over time. This allows the relationship between change in variables over time (i.e. the slope latent variables) to be assessed separately, but alongside the relationship between the baseline levels of each variable (i.e. the intercept latent variables).

The data were fitted to the hypothesised LGC model (Figure 7.1) using the robust weighted least squares estimator (WLSMV, (B. Muthen, 1984; B. Muthen et al., 1997)) recommended for ordinal categorical data (Brown, 2006). Data were fitted, initially using the PSWQ (i.e. worry) as mediator and then repeated using the CAS-1 (i.e. worry and threat-focused attention). Analyses controlled for covariates (age and gender) identified in previous analysis (Chapter 6). Adequacy of model fit was assessed by two incremental fit indices: the Comparative Fit Index (CFI) and the Tucker-Lewis Fit Index (TLI), with values close to .95 indicating a well-fitting model (Hu & Bentler, 1999), and two absolute misfit indices: the Root mean Square Error of Approximation (RMSEA) with values <.05 indicating good fit and 0.5 - .08 indicating adequate fit (Browne & Cudeck, 1993), and the Weighted Root Mean Square Residual (WRMR) with a cut-off value of .95 indicating good fit (Yu, 2002). In each case the hypothesised model (Figure 7.1) was initially tested for each outcome,
including all direct and indirect paths with latent factors for the MCQ-30 subscales, the PSWQ/CAS-1 and emotional distress (i.e. anxiety, depression, trauma). Relationships modelled between intercept latent variables and slope latent variables were constrained to be equal (i.e. it was assumed that they were reflections of the same processes). In addition, to achieve the most parsimonious model a backward elimination approach was used whereby the least significant path was deleted from the initial model and the analyses rerun until all remaining paths significantly contributed to a final model. Final models were then re-run to obtain bootstrapped standard errors and 95% confidence intervals to establish that findings were robust to non-normality.

**Figure 7.1 Hypothesised LGC model of the relationship between change in metacognitive beliefs and change in emotional distress, mediated by change in worry**

**N.B.:** Time 1 (t1) & Time 2 (t2) latent variables: Metacognitive beliefs (M); CAS (C); Emotional distress outcomes (E); Metacognitive Beliefs (MCB):
7.3 Results

Demographic and clinical characteristics of the sample (N=206) are reported in Chapter 5 (Figure 4.1).

The final LGC models for anxiety, depression, and trauma, first using the PSWQ (i.e. worry) as indicator of the CAS and then using the CAS-1 (worry and threat focussed attention), are shown in Figures 7.2-7.4. All paths found to be significant in each final model are shown. Paths that were not robust to non-normality (as indicated by bootstrapped analysis) are shown as dashed lines.

The results testing hypotheses 1-3 are presented separately below for each emotional distress outcome in turn.

7.3.1 Anxiety

The final LGC model for anxiety using the PSWQ as mediator (Figure 7.2a) was a good fit to the data ($\chi^2$ (df=2734) =3264, p<.001, CFI/TLI =.97/.97, RMSEA = .031 (90% CI =.03-.04), WRMR = .95). In this model, change in ‘Negative beliefs about worry’ and ‘Positive beliefs about worry’ were either directly (‘Negative beliefs about worry’) and/or indirectly (‘Negative beliefs about worry’; Positive beliefs about worry’) associated with a change in anxiety. There was no significant association between change in ‘Cognitive confidence’ and change in anxiety. Therefore, hypothesis 1 was supported for 2/3 metacognitive beliefs.

After bootstrapping, robust effects were found indicating that change in ‘Negative beliefs about worry’ directly affected change in ‘Worry’ (b = 1.28, bootstrapped SE (95% C.I) = 0.33 (0.66-2.00), p<.0001) and change in anxiety (b = 0.95, bootstrapped SE (95% C.I.) =0.45 (0.32-2.24), p<.035)’. However, previous paths indicating that change in ‘Positive beliefs about worry’ affected change in ‘Worry’ (b = 0.17; bootstrapped SE (95%
CI) = 0.14 (-0.10 – 0.37); p=.213), and that change in ‘Worry’ affected change in anxiety (b = 0.25; bootstrapped SE (95% CI) = 0.20 (-0.18 – 0.64); p=.216) were not robust.

Thus, although both hypotheses 2 and 3 were supported in the final model, neither finding was robust to non-normality.

The final LGC model for anxiety using the CAS-1 as the mediating variable (Figure 7.2b) demonstrated a good fit $\chi^2$(df=1441) =1828, p<.001, CFI/TLI =.97/.96, RMSEA = .036 (90% CI =.03-.04), WRMR = .95) to the data. In this model, change in ‘Negative beliefs about worry’ was both directly and indirectly associated, and change in ‘Positive beliefs about worry’ indirectly associated, with change in anxiety, supporting hypothesis 1. However, once again there was no significant association between change in ‘Cognitive confidence’ and change in anxiety.

Bootstrapped analysis indicated that the path between change in ‘Positive beliefs about worry’ and change in ‘CAS’ was not robust (b = 0.27; bootstrapped SE (95% CI) = 0.16 (-.12 – 0.49). However, robust effects were found indicating that change in ‘Negative beliefs about worry’ directly affected growth in anxiety (b = 0.78; bootstrapped SE (95% CI) = 0.41 (0.36 – 0.55); p=.058), and affected change in the ‘CAS’ (b = 1.12; bootstrapped SE (95% CI) = 0.36 (0.73 – 1.98); p=.002), which in turn affected change in anxiety ((b = 0.36; bootstrapped SE (95% CI) = 0.16 (0.13 – 0.59); p=.001).

Thus, while hypothesis 2 was supported in the final model, this finding was again not robust. However, hypothesis 3 was robustly supported for the subscale ‘Negative beliefs about worry’.
Figure 7.2a: Final LGC path model of relationship of change in metacognitive beliefs with change in anxiety symptoms, mediated by change in worry (PSWQ) over 12 months
Figure 7.2b: Final LGC path model of relationship of growth in metacognitive beliefs with growth in anxiety symptoms, mediated by growth in worry (CAS-I) over 12 months

N.B Solid lines with standardised coefficients shown for paths between metacognitive beliefs, worry and anxiety; Measurement model component for T1 and T2 latent variables and for covariates (Age & Gender) not shown. T1 & T2 latent variables: Positive beliefs about worry (P); Negative beliefs about worry (N); CAS (C); Worry (W); Anxiety (A). Growth Curve latent variables: intercept (i); slope (s). MCQ-30 subscales: Positive beliefs about worry (POS); Negative beliefs about worry (NEG)
Figure 7.3a: Final LGC path model of relationship of growth in metacognitive beliefs with growth in depression symptoms, mediated by growth in worry (PSWQ) over 12 months.
Figure 7.3b: Final LGC path model of relationship of growth in metacognitive beliefs with growth in depression symptoms, mediated by growth in worry (CAS-I) over 12 months

N.B Solid lines with standardised coefficients shown for paths between metacognitive beliefs, worry and depression; Measurement model component for T1 and T2 latent variables and for covariates (Age & Gender) not shown. T1 & T2 latent variables: Positive beliefs about worry (P); Negative beliefs about worry (N); Cognitive confidence (C); Worry (W); CAS (C); Anxiety (A). Growth Curve latent variables: intercept (i); slope (s). MCQ-30 subscales: Positive beliefs about worry (POS); Negative beliefs about worry (NEG) Cognitive Confidence (CC).
Figure 7.4a: Final trimmed LGC path model of relationship of growth in metacognitive beliefs with growth in trauma symptoms, mediated by growth in worry (PSWQ) over 12 months.
Figure 7.4b: Final trimmed LGC path model of relationship of growth in metacognitive beliefs with growth in trauma symptoms, mediated by growth in worry (CAS-I) over 12 months

N.B. Solid lines p<.05 with standardised coefficients shown for paths between metacognitive beliefs, worry and trauma; Measurement model component for T1 and T2 latent variables and for covariates (Age & Gender) not shown. T1 & T2 latent variables: Positive beliefs about worry (P); Negative beliefs about worry (N); Worry (W); CAS (C); Trauma (T). Growth Curve latent variables: intercept (i); slope (s). MCQ-30 subscales: Positive beliefs about worry (POS); Negative beliefs about worry (NEG)
7.3.2 Depression

The final LGC model for depression using the PSWQ (figure 7.3a) was a good fit to the data ($\chi^2$ (df=2730) =3248.35 p<.001, CFI/TLI =.97/.97, RMSEA = .030(90% CI =.03-.03), WRMR = .97). In this model, change in ‘Negative beliefs about worry’ and ‘Cognitive confidence’ were both directly associated with change in depression. In addition, changes in ‘Negative beliefs about worry’ and ‘Positive beliefs about worry’ were associated with change in worry. However, as change in worry was not associated with a change in depression, there were no significant indirect paths between metacognitive beliefs and depression. Consequently, hypothesis 1 was supported for ‘Negative beliefs about worry’ and ‘Cognitive confidence’ only.

Bootstrapped analysis indicated that both the path between change in ‘Negative beliefs about worry’ and change in ‘worry’ (b=1.21; SE (95% CI) = 0.35 (0.53-1.89) p=.001); and the path between change in ‘Negative beliefs about worry’ and change in depression (b=0.74; SE (95% CI) = 0.34 (.07-1.40) p=.029) were robust. However, the path between change in ‘Cognitive confidence’ and change in depression was not robust. Thus, neither hypothesis 2 nor hypothesis 3 were supported.

The final LGC model for depression using the CAS-1 as mediator (Figure 7.3b) was a good fit to the data ($\chi^2$ (df=1444) =1827.2, p<.001, CFI/TLI =.96/.96, RMSEA = .036 (90% CI =.03-.04), WRMR = .99). In this model, all three metacognitive belief subscales were either directly (‘Cognitive confidence’) or indirectly (‘Negative beliefs about worry’, ‘Positive beliefs about worry’) associated with change in depression, supporting hypothesis 1.

Bootstrapped analysis indicated a robust indirect effect of ‘Negative beliefs about worry’ on depression. Change in ‘Negative beliefs about worry’ affected a change in the ‘CAS’ (b=1.19; SE (95% CI) = .36 (0.80 – 1.96) p=.001), which in turn affected change in
depression (b=.41; SE (95% CI) =0.16 (0.14-0.79) p=.009). No other paths in the final model were robust to non-normality.

Thus, while hypothesis 2 was supported in the final model, this finding was not robust. Hypothesis 3 was not supported.

7.3.3 Trauma

The LGC model for trauma using the PSWQ (figure 7.4a) was a good fit to the data ($\chi^2$ (df=4063) =4586.50, p<.001, CFI/TLI =.97/.97, RMSEA = .025 (90% CI =.02-.03), WRMR = .98). However, in this final model although change in both ‘Negative beliefs about worry’ and ‘Positive beliefs about worry’ affected change in worry, only ‘Negative beliefs about worry’ affected growth in trauma. Consequently, the predicted indirect paths between metacognitive beliefs and trauma were not supported, and hypothesis 1 was supported for ‘Negative beliefs about worry’ only.

Bootstrapped analysis indicated that the direct path between change in ‘Negative beliefs about worry’ and change in trauma was robust (b=1.14; SE (95% CI) = 0.17 (0.80-1.48) p<.0001), as was the path between change in ‘Negative beliefs about worry’ and change in worry (b=1.30; SE (95% CI) = 0.23 (0.85-1.75) p<.0001). The path between change in ‘Positive beliefs about worry’ and change in worry was not robust. Thus, neither hypothesis 2 nor hypothesis 3 was supported.

The fit of the final model for trauma using the CAS-1 as mediator (Figure 7.4b) was also good ($\chi^2$ (df=2444) =2793.63, p<.001, CFI/TLI =.98/.97, RMSEA = .026 (90% CI =.02-.03), WRMR = .92). Changes in all three metacognitive beliefs subscales were either directly (‘Cognitive confidence’) or indirectly associated (‘Negative beliefs about worry’; ‘Positive beliefs about worry’) with change trauma. Thus, hypothesis 1 was broadly supported.
However, there was no direct path between change in ‘Negative beliefs about worry’ and change in trauma in this model, which is surprising.

Bootstrapped analysis indicated that there was a robust indirect effect of change in ‘Negative beliefs about worry’ on change in trauma, with change in ‘Negative beliefs about worry’ predicting change in ‘CAS’ (b=1.25; SE (95% CI) = .39 (0.69 – 2.38) p=.002), which in turn predicted change in trauma (b=.61; SE (95% CI) =0.08 (0.42-0.74) p=.014). However, the path between growth in ‘Positive beliefs about worry’ and growth in ‘CAS’ was not robust to non-normality, and the direct effect of growth in ‘Cognitive confidence’ on growth in trauma was just borderline (b=0.13; SE (95% CI) = .05 (-.02 – 0.21), p=.014).

Consequently, while hypothesis 2 was supported in the final model this finding was not robust. Hypothesis 3 was not supported.

7.4 Discussion

The aim of this study was to expand on evidence supporting the value of the metacognitive model of emotional disorder for understanding the causal process underlying persistent emotional distress. In Chapter 6 it was suggested that controlling for baseline distress might mask the effects of underlying causal variables. Consequently the current study used an LGC modelling approach to test causal effects arising from a change in metacognitive beliefs and processes, while controlling for the confounding effects resulting from maintenance of symptoms.

7.4.1 Hypothesis 1: Change in metacognitive beliefs over 12 months will be associated with change in emotional distress

After controlling for the relationship between the starting levels / enduring component (i.e. intercept) of metacognitive beliefs and distress, change in metacognitive beliefs over
time was associated with change in emotional distress (i.e. anxiety, depression, trauma) in most cases, thus broadly supporting hypothesis 1. Specifically, change in ‘Negative beliefs about worry’ was directly and/or indirectly associated with a change in emotional distress for all three outcomes (anxiety, depression, trauma), although the exact relationship differed according to the mediating variable used. Change in ‘Positive beliefs about worry’ was also associated with change in anxiety, depression and trauma when the CAS-1 was used as the mediating variable, although these findings were not robust. Finally, change in ‘Cognitive confidence’ was directly associated with change in depression, and also with change in trauma (when the CAS-1 was used). However, this variable had no association with anxiety suggesting that ‘Cognitive confidence’ does not play a role in predicting future anxiety when other metacognitive beliefs are accounted for. These findings, taken together, give a different picture of the relative importance of these three metacognitive subscales in predicting emotional distress than that implied in Chapter 6. However, they are more in line with theoretical predictions from the metacognitive model of emotional disorder - suggesting that while all three types of metacognitive belief are associated with persistent emotional distress, it is ‘Negative beliefs about worry’ that has the strongest causal relationship. This is also consistent with previous prospective research which found that ‘Negative beliefs about worry’ was the strongest prospective predictor of both anxiety and depression in a non-clinical sample (Yilmaz et al, 2011).

7.4.2 Hypothesis 2: The association between change in positive metacognitive beliefs and change in emotional distress is fully mediated by change in the CAS

The metacognitive model of emotional disorder predicts that positive metacognitive beliefs maintain distress insofar as they guide individuals towards use of CAS processes such as worry, rumination and threat-focussed attention, in response to negative thoughts and
feelings. This hypothesis of full mediation is supported in the final models for anxiety (2a & 2b) and in the models using the CAS-1 as mediator for depression (3b) and trauma (4b). However, in all of the models the paths between change in ‘Positive beliefs about worry’ and change in ‘Worry’ or the ‘CAS’ were not robust to non-normality. Thus, while a change in ‘Positive belief about worry’ does appear to cause a corresponding change in CAS processes as would be predicted, this effect is small when other factors (i.e. ‘Negative beliefs about worry’) are included in the model and hence not robust to non-normality. The finding that indirect and direct paths between ‘Positive beliefs about worry’ and emotional distress are non-significant when ‘Negative beliefs about worry’ are included in the model is consistent with previous cross-sectional mediation analysis (Chapter 5), and also fits with metacognitive theory which suggests that, while positive metacognitive beliefs are important for initial activation of the CAS, it is the negative metacognitive beliefs that are most influential in maintaining the CAS, and thus exacerbating distress.

7.4.3 Hypothesis 3: The association between change in negative metacognitive beliefs and change in emotional distress is partially mediated by change in the CAS.

Hypothesis 3, predicting that the relationship between change in ‘Negative beliefs about worry’ and change in emotional distress would be partially mediated by CAS processes, was supported in both of the final models for anxiety (2a & 2b). However, the indirect path was robust only for the model which used the CAS-1 as indicator (Figure 2b). For depression and trauma, this hypothesis was not supported. For both of these outcomes, change in ‘Negative beliefs about worry’ had a direct effect on change in outcomes when the PSWQ was used as the mediator variable (Figure 3a & 4A), but an indirect effect when the CAS-1 was used (Figure 3b & 4b). The lack of indirect effect observed in models 3a & 4a is clearly due to the lack of association between change in ‘Worry’ (as measured by the PSWQ)
and change depression and trauma, as for both models change in ‘Negative beliefs about worry’ was strongly predictive of change in ‘Worry’. The reason behind the lack of a direct effect in models 3b & 4b however is less obvious. It may be due to the wording of the ‘Negative beliefs about worry’ subscale, which focuses entirely on worry as opposed to other aspects of repetitive thinking (i.e. rumination & threat focused attention), which are of more relevance to depression and trauma and are included in the CAS-1. These findings are consistent with the previous cross-sectional analyses (Chapter 5), in that partial mediation of the relationship between this subscale and emotional distress is supported for anxiety, but not for depression. However, it differed in the findings related to trauma. In this case, the hypothesis of partial mediation was not supported in the current study, but was in cross-sectional analysis when the CAS-1 was used as the mediator variable. It is not immediately clear why such a discrepancy should arise. It may be due to the inclusion of ‘Cognitive confidence’ in the prospective model, which was not included in the cross-sectional analysis.

Change in ‘Cognitive confidence’ did not have an effect on change in CAS processes as measured by either the PSWQ or the CAS-1. Hence the hypothesis of partial mediation was not supported for this subscale in any of the models tested. This is perhaps not surprising as both of the measures used to indicate the CAS are predominantly focussed on worry (i.e. a form of future orientated repetitive thinking) and on future threat, whereas the subscale ‘Cognitive confidence’ assesses lack of confidence in memory. These kinds of metacognitive beliefs are more likely to activate rumination (i.e past orientated repetitive thinking) in an attempt at gap filling or a search for meaning.

7.4.4 Study implications, limitations and conclusion

In summary, this study provides further evidence supporting predictions from the metacognitive model that change in metacognitive beliefs is associated with change in
emotional distress. The evidence supporting predictions about mediation of this relationship by change in the CAS is, however, less clear-cut. Results partially support theoretical predictions that change in positive metacognitive beliefs cause a change in emotional distress by activating change in CAS processes. The relatively small (and non-robust) effect found is consistent with the metacognitive model’s assertion that it is negative metacognitive beliefs rather than positive metacognitive beliefs that are the key to understanding persistent emotional distress. The prediction of partial mediation of the relationship between change in negative metacognitive beliefs and change in emotional distress was not supported for ‘Cognitive confidence’ and was only supported for ‘Negative beliefs about worry’ with respect to predicting change in anxiety.

It should be noted, as alluded to above, that the findings of this study are limited by the measures used, a view which is supported by inconsistent findings between LGC models using different measures of the CAS (PSWQ vs CAS-1). In particular, the MCQ-30 and the PSWQ are predominantly or entirely focussed on worry, and as such are more relevant to exploring the relationship of metacognitive beliefs and processes with anxiety than with depression or trauma. This view is supported by the lack of association between change in worry as measured by the PSWQ and change in depression and trauma. In contrast, the CAS-1 items used in this study provide a broader measure of the CAS, asking about current use of worry or rumination (Item 1) and threat focussed attention (Item 2). Consequently, this measure is more likely to be of relevance to, and therefore associated with, all three outcomes. Another explanation, which may also contribute to the lack of association between change in worry (as measured by the PSWQ) and change in distress, is the issue of common method variance. It has previously been noted that there is a degree of item overlap between the ‘Negative beliefs about worry’ subscale of the MCQ-30 and the PSWQ (Chapter 6). Indeed, this was the main reason for including an additional measure of the CAS in the
current study. Consequently, it is possible that when both scales are included in the model change in ‘Negative beliefs about worry’ is the stronger predictor of distress, therefore reducing the effect of change in the PSWQ on change in distress.

In addition to problems associated with the measures used, it is also important to note that the study employed, by structural equation modelling standards, a relatively small sample size. This may be a further factor contributing to the null and/or non-robust findings. Finally, while LGC modelling has the potential to test theoretical predictions about causal processes, it may be argued that with just two waves of data, it is impossible to demonstrate that prior change in the independent variable is related to change in the mediator or the outcome (MacKinnon, 2008). Consequently, as this study provides little more than correlation between change in variables over time, it falls short of providing conclusive evidence of causation.

Despite these limitations it can be concluded that the current study does provide additional evidence supporting the clinical utility of the metacognitive model for persistent emotional distress in cancer. It is clear that further studies are required to provide stronger evidence of causality, and that these need to adopt different approaches to design such as prospective studies with three or more waves of data, and experimental studies of interventions designed to manipulate key metacognitive and/or CAS variables. However, the consistent finding that change in metacognitive beliefs affects a change in the CAS (as measured by the CAS-1), which in turn affects a change in emotional distress, is encouraging. It lends further weight to the view that intervention aimed at modifying metacognitive beliefs (in particular ‘Negative beliefs about worry’) and interrupting CAS processes (incl. worry and threat focussed attention) has the potential to reduce vulnerability in persistent emotional distress in this population, and suggests that further research is warranted.
Chapter Eight

Study five

Exploring the utility of Attention Training Technique (ATT) to reduce emotional distress in cancer patients: A case series
8.1. Introduction

Cognitive behavior therapy (CBT) is the most common psychological approach used to treat emotional distress in cancer patients. However, CBT has its limitations as described previously (Chapter 1), and its suitability and acceptability in cancer, where negative thoughts often reflect clinical reality, is questionable. An alternative theory on which to base psychological intervention is the Self-Regulatory Executive Function model (S-REF, Wells & Mathews, 1994), which underpins the metacognitive model of emotional disorder as described in Chapter 3. The intervention based on this model, metacognitive therapy (MCT), does not focus on modifying negative content of thoughts, but on modifying the metacognitive beliefs and processes that maintain them. It assumes that emotional disorder develops when flexible control of attention is lost because it is bound up with perseverative, worry-based processing and monitoring for threat – the cognitive attentional syndrome (CAS). The aim of MCT is to enable the person not to engage in worry, rumination, or other coping strategies when negative thoughts and feelings occur. Chapters 4-7 provided evidence supporting the theory that underlies this view. In particular, Chapter 5 reports that around the time of diagnosis, the association between metacognitive beliefs about the danger and uncontrollability of worry and emotional distress was partially mediated by worry and threat-focused attention. Furthermore, in Chapter 7, change in worry and threat-focused attention mediated the relationship of change in both positive and negative metacognitive beliefs about worry with change in anxiety and depression. Such findings support the view that an intervention targeting the CAS has the potential to reduce emotional distress.

Attention Training Technique (ATT), a component of metacognitive therapy, was developed specifically to modify the CAS and increase metacognitive awareness. It is an auditory attention task. Although not originally intended for use as a treatment in its own right, preliminary evidence from mental health research has shown large treatment effects.
(Wells, 2009). Studies using single case experimental designs have shown significant clinical benefits of ATT across different emotional disorders, including recurrent major depressive disorder (Papageorgiou & Wells, 2000), panic and social phobia (Wells, White, & Carter, 1997), and hypochondriasis (Papageorgiou & Wells, 1998). Furthermore, in hypochondriasis a randomized controlled trial of ATT versus no treatment (Cavanagh & Franklin, 2000) reported significant improvements in a range of health anxiety outcomes (including degree of health worry, disease conviction and of behavioural indices). These treatment gains were achieved after just six sessions, and maintained at 18-month follow-up.

ATT may have considerable clinical utility in cancer patients as it is does not require them to address their negative thoughts or feelings. In addition, as it has the potential to be delivered through guided self-practice, ATT may be particularly useful where engagement with face-to-face interventions is not practical (e.g. for patients who: are engaged in active treatment; have difficulty taking time out from usual commitments; or are geographically distant from treatment centres). In the current study, in order to explore this potential, ATT was modified to be delivered via a single face-to-face session with all remaining sessions conducted over the telephone. This represents a significant departure from how ATT has been used previously.

The aim of this study was to explore the utility of ATT for patients experiencing emotional distress after diagnosis and primary (i.e. surgical) treatment of cancer. Specifically, to:

(1) provide an initial test of efficacy of ATT in this population.

(2) explore participant experience of the intervention to assess acceptability and practicality, and to provide information to guide further development and modification of ATT for this population.
8.2. Method

8.2.1 Design

A non-concurrent multiple baseline case series (P. J. Watson & Workman, 1981) with three-month follow-up was used. Replication across patients begins to establish proof-of-principal for treatment efficacy across participants who may have differing emotional symptom presentations. This is particularly useful in cancer populations as emotional distress is often heterogeneous in presentation, comprising symptoms of anxiety, depression, trauma and/or fear of recurrence. In a multiple baseline design, the dependent variable is assessed across a no-treatment baseline for a minimum of three data-points (Smith, 2012). Patients in the current study were allocated to a four- or five-week baseline after which ATT began.

8.2.2 Participants

Five patients who attended a large NHS University Hospital for routine oncology outpatient appointments or for assessment by the psycho-oncology team were included in the study. Patients were eligible if they met the following criteria: (1) diagnosis of primary breast cancer, prostate cancer, or choroidal melanoma at least six months previously, (2) a clinically significant level of emotional distress as assessed using the Hospital Anxiety and Depression Scale (HADS total <14), (3) aged 18 years or above, (4) not in receipt of concurrent psychological treatment, (5) not in the palliative phase of care, (6) free from, or stable on, psychotropic medication, (7) no evidence of psychotic illness, current alcohol or substance abuse. These diagnostic groups were chosen for several reasons: (1) all three are similar in that they have a relatively good prognosis for survival, (2) breast and prostate cancer are the largest diagnostic groups in each gender, while choroidal melanoma (although
rare) can occur in either gender, and (3) links between clinicians and researchers within these clinical populations were already established.

**Participant 1** was a 57 year old woman who had been diagnosed with Grade 2 breast cancer one year before recruitment into the study. Initial treatment was by wide local excision (WLE) proceeding to mastectomy with auxiliary node clearance (ANC) two months later. This was followed by adjuvant hormonal therapy for five years. The participant reported a previous episode of anxiety (two and a half years earlier), and a series of difficult life events since, which meant that her diagnosis was experienced as just one more in a long line of stressors. She reported that she initially approached her diagnosis with a positive attitude, but that now she was worrying not just about the possibility of cancer recurrence, but also feeling generally vulnerable and anxious about ‘*what will happen next?*’

**Participant 2** was a 62 year old woman who had been diagnosed with Grade 1 breast cancer 16 months previously. Initial treatment was by mastectomy followed by five years adjuvant hormonal treatment. Three months before diagnosis, this participant suffered a stroke which left her with mild left side muscle weakness; she also had ongoing health problems including acute pancreatitis and chronic pain. Participant 2 reported a considerable family history of cancer, including: both parents, a sister who died of breast cancer and a niece who carries the BRCA gene. She reported a childhood onset of anxiety, for which she had previously received counselling and had been on antidepressant medication (diazepam) for the past three years. She also reported periodically experiencing PTSD symptoms (including: intrusive thoughts, avoidance, physical symptoms and feeling ‘on guard’ or ‘jumpy’) following a traumatic incident when she was twenty-five. At the time of inclusion in the study, Participant 2 described feeling anxious and depressed, very alone, frightened about possible recurrence and ashamed about her difficulty coping.
Participant 3 was a 56 year old woman who had been diagnosed with Grade 3 breast cancer three and a half years before inclusion in the study. Initial medical treatment was by WLE, followed by chemotherapy, radiotherapy and adjuvant hormonal treatment. One year previously, Participant 3 was seen by a Clinical Psychologist as part of the assessment for risk-reducing mastectomy. During this assessment she explained that she wanted surgery because she was experiencing severe anxiety about the possibility of cancer recurrence. However, she had not received this surgery by the time of inclusion in the study. Both her parents had been diagnosed with cancer; she had also in the last five years lost both her husband and sister-in-law to cancer. Currently, she reported that fear of cancer recurrence was severely disrupting her life. She reported thinking about it every minute of every day and constantly checking herself in response to the thought ‘it (the cancer) is still there’. She also reported that she found it impossible to look to, and plan for, the future; was resentful of other people’s relationships and felt socially isolated.

Participant 4 was a 61 year old woman who had been diagnosed with Grade 3 Ductal Carcinoma in Situ (DCIS) one year prior to inclusion in the study. Primary treatment was by mastectomy followed by chemotherapy, radiotherapy and five years adjuvant hormonal therapy. This participant reported a prior history of depression and anxiety from age 23 years although she had received no intervention until three months before cancer diagnosis when she experienced low mood following a leg fracture. Participant 4 reported no family history of cancer. At the time of inclusion in the study, she reported feeling very low and spending a lot of time worrying, including worrying about the possibility of cancer recurrence and how she would cope. She also reported that she lacked motivation, was still experiencing considerable pain and felt physically exhausted.

Participant 5 was a 57 year old woman who had been diagnosed with choroidal melanoma eight months previously. Primary treatment was by plaque radiotherapy followed
by six-monthly liver screening arranged by her GP. Patients diagnosed with choroidal melanoma are usually offered biopsy for prognostic testing. However, in Participant 5’s case this was not possible due to the size and position of the tumour. Aside from cancer, this participant also reported physical comorbidities and a prior history of depression and anxiety related to multiple traumatic events experienced during and since childhood. She had previously seen a psychiatrist during one of her episodes of depression but had always been reluctant to take medication. In addition to her own problems, Participant 5 described supporting other family members with both mental health and profound disability issues. Currently she described feeling irritable, tearful and generally exhausted. Because of her low mood, she said that she often felt as though she wanted to lock herself away, which in turn made her feel guilty and selfish because of responsibilities in caring for family.

8.2.3 Outcome measures

8.2.3.1 Weekly

Participants completed two self-report measures at weekly intervals during the baseline and intervention phases. The total score of the Hospital Anxiety and Depression Scale (HADS, (Zigmond & Snaith, 1983)) was used to monitor levels of distress and individual scores from two items from the Cognitive Attentional Syndrome Scale (CAS-1, (Wells, 2009)) were used to monitor use of worry (Item 1: ‘How much time in the last week have you found yourself dwelling on or worrying about your problems?’) and threat-focused attention (Item 2: ‘How much time in the last week have you been focussing attention on the things you find threatening (e.g. symptoms, thoughts, danger?).’ These measures were also used at the subsequent post-treatment and follow-up assessment. For full details of the HADS and the CAS-1 see Chapter’s 4 & 5 respectively.
8.2.3.2 Pre-treatment, Post-treatment and Follow-up

Two additional self-report measures were used for pre-treatment, post treatment and follow-up assessments. The Metacognitions Questionnaire (MCQ-30,(Wells & Cartwright-Hatton, 2004)) was used to assess changes in metacognitive beliefs (see Chapter 4 for full details), while one subscale from the Fear of Cancer Recurrence Inventory (FCRI, (Simard & Savard, 2009)) was used as a measure of cancer-specific distress (i.e. fear of cancer recurrence - FCR). The FCRI comprises 42 items across 7 subscales including: ‘FCR triggers’, ‘FCR severity’, ‘psychological distresses’, ‘functional impairment’, ‘reassurance and coping strategies’. Respondents indicate the frequency of symptoms within each subscale on a 5-point Likert-scale with higher scores indicating higher FCR. The subscale ‘FCR severity’ specifically assesses presence and severity of thoughts and images related to FCR, and as such has been recommended as a screening tool for a clinically significant FCR (cut-off score 13; (Simard & Savard, 2008)). The FCR severity subscale has previously been used for this purpose in a study of breast cancer survivors (Thewes, Bell, & Butow, 2013; Thewes, Bell, Butow, et al., 2013; Thewes et al., 2012).

The Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-I/NP, (First, Spitzer, Gibbon, & Williams, 2002) relating to Generalised Anxiety Disorder (GAD), Major Depressive Disorder (MDD) and Post Traumatic Stress Disorder (PTSD) was used to provide an independent clinician-rated assessment of current and past history of anxiety and mood disorders.

8.2.4 Intervention

ATT is introduced to patients as a way to reduce worry, rumination and self-focused attention. It comprises an externally focused auditory attention task which is given to the participant on CD. The task has three components: (1) selective attention; (2) rapid attention
switching; and (3) divided attention. Participants are asked to practice allocating their attention as directed, while treating any negative thoughts or feelings that arise as background noise, without any attempt to suppress, analyse or remove them from the mind. Each component is practiced sequentially during a 12 minute period. In the selective attention task, practiced for approximately five minutes, the patient is asked to focus attention on specific sounds among a competing array on the CD and at different spatial locations within the environment. They are asked to focus on each sound as guided, while not being distracted by others. The rapid attention switching task follows, and is practiced for five minutes, during which the patient is asked to shift attention rapidly between different sounds and spatial locations. Initially, about ten seconds is devoted to each sound, with this gradually reducing to one every five seconds. Finally, in the divided attention task, practiced for approximately two minutes, the patient is asked to try and listen to all of the sounds simultaneously.

8.2.5 Procedure

Patients who consented to the study were sent a pre-treatment questionnaire pack and contacted by telephone to participate in a SCID interview. Following this, they were allocated to a no-treatment baseline ranging from 3-5 weeks. No therapeutic input was provided during this time, although participants were offered short weekly phone calls (approx. ten minutes) to monitor symptoms. No participants took up this offer.

On completion of baseline, each participant was invited to participate in a single face-to-face session with the therapist either in the patient’s home or at the hospital. The rationale for ATT was explained, the patient practised ATT, and any difficulties or concerns relating to how to use it and how to fit it into daily life were discussed.
At the start of the session, each participant was asked to describe their current emotional problems and an idiosyncratic case formulation was developed and presented. This helped the participant become socialised to the aims of ATT. ATT was then practiced in session. Before practicing ATT, each participant was asked to rate their balance of attention on a 7-point scale ranging from ‘-3’ (entirely externally focused on the environment around me) to ‘+3’ (entirely internally focused on my thoughts, feelings or body). This was then repeated immediately after ATT practice. After practicing ATT for the first time a two-point reduction in self-focused attention is typical (Wells, 2009). If this did not occur, the therapist explored with the participant possible reasons for the lack of positive change and corrected any misunderstandings regarding how to practice ATT.

At the end of the session, participants were asked to take the CD home and practice ATT twice a day, every day, for eight weeks. A plan for when and how they might achieve this and any potential barriers were discussed and agreed. Finally, each participant was provided with a diary containing a guide to using ATT / Frequently Asked Questions (FAQ), a ‘record of practice’ sheet, eight weekly study questionnaires (HADS/CAS-1) and reply-paid envelopes for their return.

Weekly pre-arranged telephone calls were used to monitor and support each participant’s guided practice of ATT, including discussion and resolution of any problems, and a reminder to complete and return study questionnaires. At the end of the eight week intervention period, participants were advised that they were no longer required to practice ATT. Within one week of completing the intervention phase they were sent a post-treatment questionnaire pack and asked to participate in an exit interview (which included a repeat administration of the SCID) conducted by telephone. Exit interviews were semi-structured according to an interview guide (see Appendix A). Participants were prompted to talk about their understanding, and experiences of using ATT and asked to rate on a scale of 1-10 their
satisfaction with ATT in general, how it was introduced, the practice, the weekly phone calls and whether they thought it would remain useful to them in the future (see Appendix A for full details).

Participants who completed the intervention were followed up by post after three months and asked to complete the follow-up questionnaires (HADS/CAS-1/MCQ-30/FCRI). They were also asked to report any continued use of the ATT CD.

8.2.6 Analysis plan

The most commonly used method for determining an intervention effect in single case-experimental design research is graphical representation and visual inspection (Smith, 2012). Weekly scores on the HADS and level of worry/rumination and threat-focussed attention on the CAS-1 (items 1 & 2) were therefore plotted. In addition, pre-intervention, post intervention and three-month follow-up scores were plotted for the MCQ-30 subscales and the FCRI severity scale.

Clinical significance of treatment effects on emotional distress (HADS total) were assessed as they have been previously in an open trial of metacognitive therapy in adolescent and young adult survivors of cancer (Fisher et al., 2015). This used the Jacobson method “criterion c” (Jacobson, Roberts, Berns, & McGlinchey, 1999) to establish whether participants could be considered recovered as a result of treatment. This method allocates each patient to one of four possible outcomes: (1) reliable deterioration; (2) no change; (3) reliable improvement; and (4) recovered. The first three outcomes are derived solely from a reliable change index (RCI), which compares treatment outcomes to normative data from non-clinical samples to determine whether the magnitude of change is statistically significant. However, to be classified as ‘recovered’, patients need to demonstrate both a reliable change and a HADS score that falls below the established clinical cut-off (criterion c). The RCI used
previously (Fisher et al., 2015) and hence in this study was an 8-point change from baseline (mean) to end of treatment, with normative data having been drawn from a large non-clinical sample (Crawford, Henry, Crombie, & Taylor, 2001). The cut-off point used was 13.

A review of the notes taken during monitoring calls and exit interviews was used to explore the participants’ perspective on the ATT intervention. Specifically, this review focussed on acceptability and feasibility including identifying any problems experienced and potential barriers to use.

8.3 Results

Only two of the five participants enrolled in the study completed the intervention as directed. In addition, two completed the intervention phase, but had one (Participant 5) or more gaps (Participant 2) between sessions. In the remainder of this chapter these four participants are collectively referred to as ‘treatment completers’ to distinguish them from the one participant (Participant 4) who dropped out completely. Participant 4 could not be contacted from week 4. After several unsuccessful attempts to contact and re-engage her by both telephone and post, she was considered lost to follow-up. Of the four treatment completers, Participant 2 failed to return her post-treatment questionnaires and Participant 3 failed to return her three-month follow-up questionnaires despite several reminder calls and letters.

8.3.1. Session by session scores on outcome measures

Figure 8.1 shows the HADS-distress, worry and threat-focussed attention scores across the baseline and intervention phases and at follow-up. Baseline scores across all three measures were relatively stable for each participant. After ATT was introduced, a reduction in HADS score was seen across the intervention phase for all four treatment completers.
Corresponding reductions were seen in worry and threat-focussed attention with the pattern of scores for both outcomes mirroring that seen for the HADS. Three participants (Participants 1, 3 & 5) encountered one or more distressing situations during the course of the study. In each case, the HADS score either stabilised or increased in line with the changes in worry and threat-focussed attention, although it did not return to pre-treatment levels.

At three-month follow-up, Participants 1 and 2 showed further improvement across all three outcomes, albeit very minor improvements for Participant 2. In contrast, Participant 5’s scores returned to baseline levels. Participant 3 did not return her follow-up questionnaire.

**Figure 8.1: Emotional distress, worry and threat-focused attention scores across baseline, treatment and follow-up phases**

**Participant 1**

**Participant 2**
N.B. Distress – HADS total score; Worry - CAS-1 Item 1; Attention – CAS-1 Item 2; B- baseline assessment; Trt – treatment assessment; Post – post-treatment assessment; 3 month – 3-month follow-up assessment
Figure 8.2 shows each participant’s pre-treatment, post-treatment and follow-up scores on the MCQ-30 subscales and the FCRI-severity scale.

Participant 1 had high pre-treatment scores on three of the five MCQ-30 subscales: ‘Negative beliefs about worry’, ‘Positive beliefs about worry’ and ‘Cognitive self-consciousness’. Small reductions were seen across all three of these subscales at post-treatment with further reductions at follow-up for two: ‘Positive beliefs about worry’ and ‘Cognitive self-consciousness’. The reduction in ‘Negative beliefs about worry’ from pre-treatment to follow-up was negligible. Fear of cancer recurrence (as indicated by the FCR-severity scale) initially worsened between pre and post-treatment. However, at the follow-up assessment it had reduced substantially (-15 points from pre-treatment) to below the level considered clinically significant.

Participant 2 had extremely high pre-treatment scores across all five of the MCQ-30 subscales. This participant did not return her post-treatment questionnaire but did return her follow-up. There was little change in her scores from pre-treatment to follow-up. Participant 2 also had an extremely high FCR severity score at pre-treatment. Unfortunately, as she failed to complete this subscale at either post-treatment or follow-up, it is not known whether ATT was effective in reducing this.

Participant 3 also had high pre-treatment scores on three out of five MCQ-30 subscales including: ‘Negative beliefs about worry’, ‘Need to control thoughts’ and ‘Cognitive self-consciousness’. Substantial reductions (8-12 points) were seen across all three of these subscales between the pre and post-treatment assessments. However, since this participant did not return her follow-up questionnaire it is not known whether these improvements were maintained at three-month follow-up. Participant 3 also had an extremely high pre-treatment FCR-severity score. This was almost halved by post-treatment.
Figure 8.2: MCQ-30 subscales & FCRI-Severity scale at pre-treatment, post-treatment and follow-up

Participant 1

Participant 2

Participant 3
Participant 4

Participant 5

N.B. POS – Positive beliefs about worry; NEG - Negative beliefs about worry; CC – Cognitive confidence; NC – Need for control over thoughts; CSC – Cognitive Self-Consciousness; FCR-Severity – Fear of Cancer Recurrence Inventory – Severity scale
Participant 5 had high pre-treatment scores on ‘Negative beliefs about worry’ and ‘Cognitive self-consciousness’. Her scores on the ‘Negative beliefs about worry’ subscale reduced substantially between pre- and post-treatment but returned to the pre-treatment level at follow-up indicating that treatment gains in this area were not maintained. Similarly, ‘Cognitive self-consciousness’ improved between pre- and post-treatment but, deteriorated again by follow-up. In contrast, the FCR-Severity score improved between pre and post-treatment and between post-treatment and follow-up. At the three-month follow-up the FCR-severity score was less than half the pre-treatment score and below the level indicating clinically significant FCR.

8.3.2. Clinically significant change

At post-treatment, two participants had made reliable improvement (Participant 1 & 5), and one met criteria for recovery (Participant 3). The remaining participant (Participant 2) did not return her post-treatment questionnaire but had showed ‘no change’ by the final week of the intervention.

At three-month follow-up, another participant (Participant 1) also met criteria for recovery. However, Participant 3, who was recovered at post-treatment, did not return her follow-up questionnaire so it is not known whether her status as ‘recovered’ was maintained. The remaining two participants (Participant 2 and 5) were unchanged at follow-up relative to baseline.

8.3.3. Acceptability and feasibility of the intervention

During exit interviews, the four treatment completers reported that they were glad that they had taken part in the study, and that they had benefitted from participation. Although Participant 2’s scores on the outcome measures suggested no improvement she reported less
worry. Satisfaction ratings for the intervention and confidence in its longer term
effectiveness are shown in Table 8.1. All treatment completers rated the overall intervention
extremely highly, stating that it had significantly improved their mood: e.g. ‘been useful as
it’s made me feel clear (…) my mood is generally better than 9 weeks ago, don’t feel as
anxious, not as worried’ (Participant 1). One participant also commented that her improved
mood had been noticed by those around her: ‘friends noticed I am more relaxed, I am not
saying stupid things like ‘I’m not going to be here’ (Participant 3). In addition to this general
improvement in mood, several treatment completers also reported that they felt better able to
cope with new or ongoing stressors that they encountered during the course of the
intervention: e.g. ‘even though difficult situations have continued, I have managed to not lie
awake all night thinking about it. Things have been really bad but I’ve not engaged with the
worries’ (Participant 5). Furthermore, all treatment completers felt confident that the
intervention would be effective for them in the longer term: e.g. ‘It will be a useful tool to
have in life for the future and I will use it’ (Participant 1).

Table 8.1: Participants’ exit interview ratings (0-10) of satisfaction with, and
confidence in the intervention

<table>
<thead>
<tr>
<th>Question</th>
<th>Participant</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Satisfaction with ATT (overall)</td>
<td>9</td>
</tr>
<tr>
<td>Satisfaction with first session</td>
<td>10</td>
</tr>
<tr>
<td>Satisfaction with home practice</td>
<td>9</td>
</tr>
<tr>
<td>Satisfaction with phone calls</td>
<td>10</td>
</tr>
<tr>
<td>Confidence in ATT working in the future</td>
<td>9</td>
</tr>
</tbody>
</table>

By the end of the intervention phase, all four treatment completers stated clearly that
they had recognised that the goal was not to eradicate thoughts but to respond to them

2 (...) indicates a pause in participants’ talk
differently: e.g. ‘I still get thoughts (...) but they are diluted a little (...) I feel a difference (...) I’ve changed in the way I deal with them (negative thoughts)’ (Participant 1). However, it is also interesting to note that the patients who were reliably improved as a result of the intervention all said that they had doubted it could work at the outset: e.g. ‘it wasn’t what I expected (...) I thought how is this going to help me, how can this work’ (Participant 3), and that it took time to understand the approach: e.g. ‘As time goes on you start to realise it’s not about getting rid of worries (...) taken me a while to realise it’s about how I respond to them’ (Participant 5). Although it was apparent that none of the participants understood the goal at the outset, Participant 5 suggested that this might not be due to inadequacy of the initial introduction but because ‘No matter what you say at the outset, initially you do think they (negative thoughts) are going to go away because you can’t imagine anything else, can’t imagine a case where you still have them, but aren’t worrying about them’ (Participant 5). In addition to this lack of understanding of the overall goal, initial misconceptions of how to do the task included that it was important to empty the mind or stop negative thoughts, and that it was important to be able to hear all the sounds as directed. Frustration at not being able to ‘do the task right’ discouraged several participants from practicing, and for two almost caused them to drop out completely: ‘I couldn’t focus, the more I was trying (...) the more I couldn’t do it’ (Participant 2); ‘I just couldn’t do it ...was getting angry and frustrated, close to giving up, the whole left, right, behind you thing nearly stopped me from doing it’ (Participant 5). For these reasons, all four treatment completers agreed that the phone calls were a vital part of the intervention: ‘I wouldn’t have got through without (therapist). Being able to ask stupid questions was vital (...) if I had just been sent away with the CD (...) I wouldn’t have been able to do that’ (Participant 5).

Despite the initial difficulties, treatment completers were in agreement that they liked the combination of home practice and weekly phone calls and preferred it to the idea of
attending for weekly face-to-face sessions. Furthermore, it was acknowledged by all that 'practice was key', although they all found it problematic to maintain practicing twice a day every day. Several participants (Participant 1, 3 & 5) suggested that this level of practice could be viewed as too time-consuming by some people. However, rather than suggesting it should be reduced, Participant 1 suggested that it was merely important to stress that 'you need to be committed to it and have to practice... to take the time to do it' (Participant 1). Finally, participants were unanimous in saying that they would recommend ATT to others, and that they thought it should be further developed for use in cancer. Given the importance of practice to the success of ATT, they were also in agreement that it would have been difficult, if not impossible, to engage while still on active treatment. For this reason, all suggested the most appropriate time to be offered ATT would be at the end of treatment:

'I would say 'yeah (...) go for it!' (...)It is difficult to understand, feels so arty farty, not concrete enough. Can’t put it into words but it is not a solid enough explanation of what you can achieve ....it can change your life, teaches you a different way of thinking about thinking’ (Participant 5).

8.4. Discussion

8.4.1. Is ATT an effective intervention for reducing emotional distress after cancer?

The primary aim of this study was to provide an initial test of the efficacy of ATT for reducing emotional distress in a cancer population. These results suggest that ATT has the potential to reduce emotional distress. Three of the four treatment completers were categorised as either ‘recovered’ or ‘reliably improved’ after treatment. In addition, levels of worry and self-focussed attention decreased between pre- and post-treatment for these participants and these outcomes co-varied with distress for all participants across baseline, intervention and follow-up.
In addition, pre- to post-treatment reductions were observed for these three participants on three of the five MCQ-30 subscales (‘Negative beliefs about worry’, ‘Need for control over thoughts’ and ‘Cognitive self-consciousness’). This pattern of reductions is consistent with a previous study in recurrent major depression (Papageorgiou & Wells, 2000) and consistent with the expectation that ATT will reduce internally-focused attention, while also reducing beliefs about the uncontrollability of worry and the need to engage with thoughts.

However, it is important to note that not all of those who completed treatment improved in these outcomes (i.e. in distress, worry, threat-focused attention, and metacognitive beliefs) after ATT, or maintained post-treatment improvements at three-month follow-up. Therefore, unlike previous case series studies testing the efficacy of ATT for reducing emotional disorder in mental health populations (Cavanagh & Franklin, 2000; Papageorgiou & Wells, 1998, 2000; Wells et al., 1997), it is clear that ATT in the current study was not effective for all. The most likely explanation for this comparatively poor success rate is differences in how ATT was implemented between studies. All the previous mental health studies (Cavanagh & Franklin, 2000; Papageorgiou & Wells, 1998, 2000; Wells et al., 1997) involved weekly face-to-face sessions, including in-session practice of ATT. In contrast, the current study is the first to adopt a guided self-practice approach to delivering ATT. It is apparent from the gaps in the data and feedback from patients that there was considerable difficulty in getting patients to implement ATT correctly using this approach. All of the treatment completers in the current study reported: having had severe doubts at the outset that ATT could work, initial difficulty in understanding how to practice ATT, and that without the monitoring phone calls they would have likely given up. Indeed, the one
patient who dropped out consistently reported difficulty in understanding how to practice ATT prior to leaving the study.

It may be that ATT alone (delivered in this format) is insufficient to produce lasting change. It is encouraging to note, however, that regardless of whether or not a clinically significant improvement in HADS score was achieved, all treatment completers did verbally report a change in the way they responded to negative thoughts about cancer that was broadly consistent with the expected effects of ATT.

In contrast to the HADS distress score, FCR-severity scores improved for all three treatment completers who provided data at post-treatment, and improvements were maintained at follow-up for the two who provided complete data, irrespective of changes in the other outcome measures. It is not clear why the results for this outcome measure should be different. It may be that it is simply an effect of social desirability (i.e. participants enrolled in the study with the expectation of reducing cancer-related distress and wished to please the researcher). However, the observed reductions in FCR are consistent with previous studies which found an effect of ATT on reducing dysfunctional illness related beliefs (Cavanagh & Franklin, 2000; Papageorgiou & Wells, 1998, 2000; Wells et al., 1997); health worry, disease conviction (Cavanagh & Franklin, 2000), and negative illness-related behaviour (Papageorgiou & Wells, 1998). It is also consistent with the expectation that ATT alters participants’ balance of attention, so that that they become less internally and more externally focussed. Metacognitive theory would suggest that, as a result of this altered balance of attention, hypervigilance to internal cues (i.e. negative illness related thoughts, physical sensations) previously perceived as threatening is likely to be reduced (Sharpe et al., 2010). In addition, because of increased attentional flexibility, individuals become able to disrupt persistent worry-based processing, thus freeing-up cognitive resources for
more adaptive information processing (Wells & Mathews, 1994; Wells et al., 1997) of negative illness related thoughts or feelings.

8.4.2. Is ATT a practical and acceptable intervention?

The second aim of the study was to explore the participants’ perspective on acceptability and practicality of ATT to guide future development of the intervention. The intervention was well-received by the four treatment completers. Only one participant dropped out and from the information obtained during the monitoring calls it seems likely this was due to difficulty in understanding how to do the task. Other participants reported similar problems; in particular Participant 2 found ATT so difficult and frustrating that she almost left the study, and Participant 5 reported that she would have left had it not been for the further explanation provided by the therapist phone calls. However, all of the treatment completers reported a clear understanding of the goals of ATT by the end of the treatment as well as a perceived benefit of taking part and confidence that it would continue to work for them in the future. In general, a self-guided format was welcomed although it was suggested that future developments should seek to increase therapist contact by having a face-to-face session at mid treatment in order to consolidate understanding of the task and increase compliance with practice. These findings suggest that ATT is an acceptable intervention and that the therapist support component was vital to its success.

As well as the importance of therapist contact, all participants stated that by the end of treatment they had recognised the importance of practice, but that this should be given greater emphasis at the outset. This poses somewhat of a dilemma in how to ‘sell’ ATT to future participants, as it was also mentioned that the time commitment involved in practising ATT
(i.e. twice a day for 8 weeks) could be seen as onerous by some. Indeed, several potential participants had cited this as the reason for not wanting to participate, and all four treatment completers admitted to reducing the frequency of practice as time progressed. In addition, it became apparent that follow-up calls were insufficiently formalised leading to missed appointments on both sides, which undermined the professionalism of the intervention as well as increasing the likelihood of participants practising ATT incorrectly. Consequently, it is clear that if ATT is to be successfully delivered in a guided self-practice format there is a need to formalise the way it is provided to ensure that participants complete the intervention as directed. It is suggested that a formal schedule of practice and therapist appointments should be agreed with each participant during the initial session. This should then be reviewed as part of each subsequent follow-up. In addition, the importance of keeping telephone appointments should be stressed to ensure participants are fully aware that this as a key component of the treatment and not merely an optional extra.

8.4.3. Limitations and conclusions

Despite some positive findings, it is clear that caution is warranted in reaching a conclusion about the effectiveness of ATT in cancer as this is a small, uncontrolled study. Significant practical problems were encountered both in keeping telephone appointments and in obtaining participants self-report data, which are likely to have undermined intervention effectiveness. Furthermore, the study is also limited by the characteristics of the sample in that all participants were female and all but one had been diagnosed with breast cancer.

However, despite these issues, as a proof of principle test this study represents a promising start. Patients who completed treatment were extremely positive. Verbally they all reported a change in the way they were responding to negative thoughts about cancer and a relationship between change in worry, threat-focussed attention and emotional distress was
shown in the patient reported outcome data. However, ATT delivered in the current guided self-help format clearly did not work for all and as such there is a need for substantial further development. Based on the interview feedback this may include increasing therapist contact, developing a more structured program of practice and phone calls, including additional components of MCT (i.e. detached mindfulness) and including signposting for patients who require further intervention (i.e. full MCT).
Chapter Nine

General Discussion & Conclusions
9.1. Re-orientation to thesis aims

This thesis explored the utility of the metacognitive model of emotional disorder for understanding persistent emotional distress after cancer. Because the metacognitive model was developed for use in mental health, its application in physical health research has previously been limited. Several studies have explored the contribution of metacognitive beliefs to emotional distress in various physical health conditions and in cancer (e.g. Allott et al, 2005; Thewes, Bell & Butow, 2013), but none has formally tested the theory. Therefore, this thesis represents the first attempt to formally test theoretical predictions from the metacognitive model in cancer, using the findings of several linked empirical studies - one cross-sectional (Study 2), two prospective (Studies 3 and 4) and a case series study (Study 5):

9.2. Validity of the MCQ-30

The starting point for this work was to test the validity of the Metacognitions Questionnaire (MCQ-30) for use in cancer. Although this measure has been used in cancer before (Butow et al., 2013; Fisher et al., 2015; Thewes, Bell, & Butow, 2013), there has been no formal assessment of its psychometric properties in this population. Therefore, before beginning to test predictions from the metacognitive model, it was necessary to establish the MCQ-30’s validity for use in cancer. To this end, Study 1 used confirmatory and exploratory factor analysis techniques to confirm the validity of the published five-factor structure, and establish that no alternative pattern of loadings was a better fit to the data. In addition, concurrent validity was assessed using structural equation modelling to test the expected relationship between the five MCQ-30 subscales and anxiety and depression. The results of this study suggest that the MCQ-30 is valid for use with breast and prostate cancer within the first year after diagnosis. However, it is acknowledged that from just one study it is premature to claim that the MCQ-30 is valid for use in other cancer diagnoses, or indeed
other time frames within the cancer journey.

9.3. Testing theoretical predictions from the metacognitive model of emotional disorder

Having established the validity of the MCQ-30, it was then used to test predictions from the metacognitive model, in a series of linked cross-sectional, prospective and experimental studies. Three overarching predictions were tested:

(1) Metacognitive beliefs will be associated with both current and future emotional distress, and negative metacognitive beliefs will be the largest predictor

(2) Metacognitive beliefs will predict additional variance in current and future emotional distress over and above previously implicated factors including: ‘content of cognition’ (i.e. illness perceptions) & baseline symptoms of distress

(3) CAS processes such as worry and threat–focused attention will mediate the relationship between metacognitive beliefs around diagnosis and current and future emotional distress

The specific findings from each study have been discussed in the preceding chapters. In this final chapter the findings from each study are brought together and discussed under the heading of the particular prediction (1-3 above) addressed.

9.3.1 Metacognitive beliefs will be associated with both current and future distress and negative beliefs will be the largest predictor

The predicted association between metacognitive beliefs and emotional distress was initially tested cross-sectionally, around the time of diagnosis, in Study 1 & 2 (Chapter 4 & 5) then again prospectively (at 12-month follow-up) in Study 3 & 4 (Chapter 6 & 7). Across all of these studies the subscale ‘Negative beliefs about worry’ was consistently associated
with current and future anxiety and depression (Studies 1-4) and future trauma (Studies 2-4).

In addition, as predicted, it was the largest contributor to variance in emotional distress in each case. In contrast, ‘Positive beliefs about worry’ was associated with current and future anxiety only, although again this was a consistent finding across studies. The results regarding the remaining three MCQ-30 subscales were not consistent across the studies. Around the time of diagnosis, less belief in a ‘Need to control thoughts’ was associated with greater concurrent anxiety but not depression, whereas ‘Cognitive confidence’ and ‘Cognitive self-consciousness’ were not associated with either outcome (Study1). In contrast, in the prospective studies multiple regression analysis indicated that ‘Cognitive confidence’ around diagnosis was associated with future anxiety, depression and trauma (Study 3), although not all of these associations were confirmed when tested using structural equation modelling (Study 4). Neither ‘Need to control thoughts’ nor ‘Cognitive self-consciousness’ were prospectively associated with emotional distress.

The findings related to positive and negative beliefs about worry are consistent with the metacognitive model which suggests that, while positive metacognitive beliefs activate the CAS, it is negative metacognitive beliefs that are primarily responsible for maintaining and exacerbating it. The generic metacognitive model tested in this thesis refers to two broad categories of belief; positive and negative metacognitive beliefs, and the MCQ-30 subscales ‘Negative beliefs about worry’ and ‘Positive beliefs about worry respectively’ are clearly defined in these terms. The lack of consistency regarding the remaining three subscales (‘Cognitive confidence’, ‘Need to control thoughts’ and ‘Cognitive self-consciousness’) is perhaps not surprising given that they refer to how one’s memory works, the need to control thoughts and the tendency to engage in monitoring one’s mind, domains of metacognition that are not considered to be universally relevant across all emotional disorders (Wells, 2009). There is some evidence in in Study 3 and 4 that ‘Cognitive
confidence’ is associated with future emotional distress in cancer. It is unclear why this subscale in particular should be associated with distress in this population, although the model suggests that low confidence in cognitive abilities may lead to a sense of uncertainty, of worry about how the mind is working (i.e. whether it has been affected by cancer or treatment) and/or activate rumination in an attempt at gap filling or searching for meaning. In addition, previous studies have indicated an association between low confidence in cognitive abilities and obsessional thoughts and checking (Cartwright-Hatton & Wells, 1997; Hermans, Martens, De Cort, Pieters, & Eelen, 2003), which fits with the type of behaviour often seen in patients with high fear of recurrence.

9.3.2 Metacognitive beliefs will predict additional variance in current and future emotional distress over and above previously implicated factors including; ‘content of cognition’ (i.e. Illness perceptions.) & baseline symptoms of distress

In Study 2, a competitive test using hierarchical multiple regression assessed the relative contribution of illness perceptions and metacognitive beliefs around the time of diagnosis to concurrent emotional distress. Positive and negative beliefs about worry were able to explain more of the variance in concurrent anxiety and trauma than illness perceptions, when controlling for just age and gender, but this was not the case for depression. However, metacognitive beliefs were able to add significantly to the variance over and above inclusion of illness perceptions for all three emotional distress outcomes. In prospective analyses, a similar picture was apparent with metacognitive beliefs able to explain more of the variance in future anxiety, depression and trauma than illness perceptions after controlling for just age and gender. In addition, they were able to add a small but significant amount to the variance in future anxiety and depression, but not for trauma, after controlling for baseline symptoms of distress and illness perceptions. These findings are
broadly consistent with the theoretical prediction that, due to their causal role in driving engagement with negative thoughts and feelings, metacognitive beliefs will contribute over and above content of cognition (i.e. illness perceptions).

It should be noted, however, that after controlling for baseline distress, the amount of additional variance explained by either set of predictors (illness perceptions or metacognitive beliefs) is small. This suggests that as predictors per se, they offer little advantage over screening for distress at baseline. However, as discussed previously (Chapter 2), while baseline distress is a consistent predictor of future distress, in reality this merely tells us that in most cases distress is maintained from baseline. We still need to identify the factors that underlie this maintenance before we can reduce vulnerability to persistent distress. Consequently, further exploration of the data to test whether an interaction between baseline distress and metacognitive beliefs offers a more clinically useful prediction would be a valuable next step.

9.3.3. CAS processes such as worry and threat–focussed attention will mediate the relationship between metacognitive beliefs around diagnosis and current and future emotional distress

Two studies within this thesis explicitly tested whether CAS processes mediated the relationship between metacognitive beliefs and current and/or future emotional distress. In both cases the relationship between ‘Negative beliefs about worry’ and all three emotional distress outcomes was mediated by worry and threat focussed attention (as assessed using the CAS-1), although the hypothesis of partial rather than full mediation was only supported for concurrent anxiety and depression and change in anxiety over time. In contrast, the prediction of full mediation of the relationship between ‘Positive beliefs about worry’ and emotional distress was not supported for any of the concurrent outcomes (Study 2) nor was it
robustly supported for the relationship between change in metacognitive beliefs and change in emotional distress over time (Study 4). However, as discussed previously (Chapter 5 & 7), these findings are consistent with the metacognitive model’s assertion that while positive metacognitive beliefs activate the CAS it is negative metacognitive beliefs that are the key to understanding persistent emotional distress and hence these will appear to be the stronger predictor of emotional outcomes when both are assessed simultaneously.

Aside from these explicit tests of mediation, Study 5 tested whether an intervention designed to disrupt the CAS was able to reduce emotional distress among patients who were more than six months from diagnosis. Although success of the intervention was variable between participants, in each case it was apparent that scores for worry and threat-focused attention co-varied with those for HADS-distress, reducing in parallel after introduction of Attention Training Technique (ATT) for those who were either reliably improved or recovered by the end of treatment. While this is consistent with the model’s assertion that activation of the CAS underlies maintenance of distress, covariance cannot be considered strong evidence of support for the model as it does not indicate a direction of effect. Furthermore, findings from such a small uncontrolled study must always be interpreted with caution.

In summary, the findings from the main body of this thesis indicate that the above theoretical predictions are broadly supported, but only with confidence in respect to ‘Negative beliefs about worry’. In addition, the null findings relating to ‘Positive beliefs about worry’ can be understood in the context of the metacognitive model where they are purported to activate the CAS but not be of primary importance in its maintenance. However, this thesis finds no substantial evidence to support any of the remaining three domains of metacognitive belief assessed by the MCQ-30, as being reliably associated with either current or future distress in this population once ‘Negative beliefs about worry’ is
accounted for. These findings are generally consistent with previous research in mental health (see (Wells, 2009) for a review), general population samples (Spada et al., 2008) and Parkinson’s disease patients (Allott et al., 2005). In these studies, the subscale ‘Negative beliefs about worry’ has consistently been identified as the predominant contributor to variance in both anxiety and depression, whereas the importance of the other domains of metacognitive belief measured by the MCQ-30 has varied across the different populations studied.

9.3.4 Problems with testing theoretical models

Although these findings provide promising first evidence to support the utility of the metacognitive model in cancer, there are several methodical limitations that deserve further attention.

The first issue to note is that while this study controlled for baseline emotional distress (the predominant predictor identified in Chapter 2), it did not control for neuroticism, which was also found to be a consistent predictor of emotional distress. The finding in one study (Aarstad et al., 2005), that neuroticism reduced to non-significant the effect of depression at diagnosis on depression at six years, suggested it is an important vulnerability factor underlying persistent emotional distress, although the mechanism of action is unclear. Cross-sectional studies have begun to find evidence that negative metacognitive beliefs mediate the relationship between neuroticism and anxiety in non-clinical (Dragan, Dragan, Kononowicz, & Wells, 2012) and student populations (Dragan & Dragan, 2014; van der Heiden et al., 2010), and between neuroticism and health anxiety in a student population (Bailey & Wells, 2013). These findings suggest that while controlling for neuroticism in the current study may have impacted on the observed association between baseline and follow-up emotional distress, it would have been unlikely to have affected the relationship between
baseline metacognitive beliefs and persistent emotional distress. Further prospective and developmental research is required to explore the relationship between such temperament traits and the development of metacognitive beliefs.

A significant limitation to Chapter 5 is the cross-sectional nature of the study. Cross-sectional research can never be used to provide evidence of causality as the models and variables presented, although based upon theory, are only correlational in nature. The data presented can just as equally be used as evidence that emotional distress gives rise to metacognitive beliefs and processes as vice versa, or that metacognitive beliefs, worry and self-focussed attention are simply associated with emotional distress. Chapters 6 and 7 attempted to address this issue by using a prospective design. In addition, there is a problem inherent in using prospective multiple regressions (Chapter 6) to isolate the effects of underlying causal factors from baseline symptoms of the emotional outcome it is predicted to maintain. That is, that generally, in prospective regression studies it is considered good practice to control for baseline levels of the variable you are trying to predict. However, as discussed in Chapter 6, it is apparent that by doing this we may actually be masking the effect of underlying causal variables (i.e. metacognitive beliefs) due to the overlap in variance explained by these variables and symptoms of distress at baseline. This methodological issue may explain the predominant finding in the literature that baseline distress is the largest or only significant predictor of persistent emotional distress after cancer (Chapter 2). In Study 4 (Chapter 7), latent growth curve analysis was used to partial out variance attributable to enduring symptoms of emotional distress by looking at the association between changes in variables in parallel to the association between variables at baseline. While this is an improvement over the standard hierarchical regression approach, it is still limited in the context of this thesis as with only two waves of data it is impossible to demonstrate temporal precedence of change in metacognitive beliefs over change in emotional distress. Thus it is
essentially still cross-sectional and subject to the same limitations on interpretation as Study 2. Consequently, in order to provide more compelling evidence of a causal role for metacognitive beliefs and processes further prospective research with a minimum of three waves of data is needed. Another approach would be to conduct prospective research looking at the predictive utility of metacognitive beliefs in groups divided according to baseline emotional status. However, in order for such a study to be sufficiently powered a substantially larger sample would be needed than was achieved for this thesis.

A linked issue to the problem of disentangling causal factors from the emotional outcomes they are predicted to maintain is related to how these various constructs are operationalised. For example, distress outcomes are frequently defined by the cognitive processes that the metacognitive model suggests cause and maintain them; i.e. persistent worry is a key defining component of anxiety disorder and also a key component of the CAS. Furthermore as noted in Chapter 5, visual inspection of items on each measure indicated similarities between some of the items on the PSWQ, used to assess the CAS, and the ‘Negative beliefs about worry’ subscale. Consequently, it could be argued that is it not surprising that associations between emotional distress, the CAS and ‘Negative beliefs about worry’ were found for anxiety, but not for depression and trauma when the PSWQ was used. The inclusion of the CAS-1 scale as an alternative to the PSWQ goes some way to guarding against bias due to the common method variance resulting from this overlap in measurement. However, as the CAS-1 is intended as a clinical tool and therefore has not been formally psychometrically tested, it is clear that there is a need for improvement in measurement tools available for measuring the CAS. Unfortunately the issue of overlap between hypothesised causal factors of the CAS and symptoms of emotional distress is difficult to resolve, although future research should take steps to minimise overlap in how these constructs are operationalised where possible. In addition, as the measures of metacognitive beliefs and
CAS processes used in the current study were designed for use in mental health populations, and most specifically in relation to generalised anxiety disorder, it is likely that they lack sensitivity in a cancer population where heterogenous presentation of anxiety, depression and trauma symptoms are common. This is likely to have led to an enderestimation of the role of metacognitive beliefs in mediating the relationship between baseline and follow-up depression and trauma. Before we can establish a more accurate picture of the association between metacognition and persistent emotional distress in cancer, further studies are required to develop more appropriate measures of metacognitive beliefs and CAS processes for use in this population.

A final measurement issue pertains to whether the self-report measures used in this thesis are accessing the relevant cognitions or causing them (Ogden, 2003). In her analysis of social cognition models, Ogden highlights that the standard practice used in theory testing, that is asking participants to complete self-report questionnaires about their cognitions, assumes that questionnaire items are able to access pre-existing cognitions, whereas an alternative explanation is that such cognitions are created by completing the questionnaire. This issue clearly applies equally to all self-report measures used in psychological research. However, it may be particularly pertinent to this thesis as it could be argued that by asking patients to respond to questions about their ‘thinking about cancer’, their feelings, and their ‘thinking about thinking’, we are in effect increasing patients' focus on their internal thoughts and feelings which is precisely the process predicted to cause and maintain distress. While this issue is not one that can be easily resolved in this type of cohort study, it is important to bear in mind when reflecting on the findings. Furthermore, it is particularly important to consider this issue in clinical work, where completing self-report measures may initially increase rather than reduce self-focused attention as was seen for some of the ATT participants in Study 5.
A final methodical issue is related to the question’ to what extent is it possible to test a theoretical model such as the metacognitive model of emotional disorder?’ A good clinical prediction model consists of linked constructs that are sufficiently specific to generate testable hypotheses (Ogden, 2003). That is, in order to provide strong statistical evidence of a causal relationship, arrows linking constructs should clearly indicate a direction of effect. However, in contrast, clinically useful models tend to be more fluid and flexible so that they can be used to create a narrative which the clinician can then use to help the patient understand their difficulties. In reality, most psychological models fall somewhere between the two and in this respect the metacognitive model is no different. While it has been possible to generate some directional, and therefore testable, hypotheses, it also recognised that the model contains feedback loops which are inherent to its clinical utility. In this sense it may be argued that while key predictions can and have been tested in the current thesis, the model itself can never be tested in its entirety. Consequently, support for the model can only come from combining evidence from this and further studies in the future that use a variety of observational and experimental approaches to test different predictions and components of the model.

9.4 A final test - can an intervention designed to disrupt the CAS reduce emotional in cancer?

As has been stated above, evidence presented in this thesis supports the hypothesis that metacognitive beliefs add to the variance explained in persistent anxiety and depression over and above that explained by negative content of thoughts, and that the relationship between metacognitive beliefs and emotional distress is mediated by the CAS. Consequently, therapeutic approaches targeting metacognitive beliefs and CAS processes – rather than the
content of negative thoughts about cancer – should have the potential to reduce emotional
distress in this population. Study 5 (presented in Chapter 8) is an initial proof-of-principle
test of whether an intervention aimed at disrupting the CAS might be of use in a cancer
population. The findings of this study were inconclusive regarding the overall effectiveness
of ATT for reducing distress in cancer. For participants who fully engaged with the
intervention and understood the goal of treatment, ATT delivered in this format appeared to
be effective. However, for those who did not engage fully, it clearly was not. Therefore the
main issue appears to be the difficulty of getting participants to engage with and do the ATT
task correctly. The intervention was well received by those who completed the study, all of
whom verbally reported having changed the way in which they responded to negative
thoughts about the cancer. However, it seems clear that using the ATT CD as a stand-alone
intervention is unlikely to be an effective form of treatment. There is a clear need to ensure
participants are well-socialised to the model, engaged in meta-level dialogue and committed
to practising tasks as directed and this is likely to require more regular therapist contact.
While the result of this case-series study must be interpreted with caution, given the scale of
the study as well as the problems encountered, when taken together with the positive findings
of previous studies of full metacognitive therapy (MCT) in adult breast cancer (Butow et al.,
2013) and adolescent and young adult survivors of cancer (Fisher et al., 2015; McNicol et al.,
2013) they do suggest that there is promise in pursuing further development and testing of
interventions based on MCT in this population.

9.5 Recommendations for future research.

It can be seen that while the current thesis presents valuable first evidence in support of the
utility of the metacognitive model for understanding emotional distress after cancer,
considerable further research is warranted. Firstly, it is evident that there is a need for refinement or development of new instruments for assessing metacognitive beliefs and processes in cancer populations. Measures are needed that have items which go beyond asking about worry in order to reflect the diversity of symptom presentation that is common in cancer patients. Only then will study measures have sufficient specificity to provide an accurate picture of the relationship between maladaptive metacognition and emotional distress after cancer. Secondly, it is evident that further prospective research is required. The current study pertains only to breast and prostate cancer patients in the first year after their primary treatment. Consequently, there is a need to replicate the current study in a more heterogenous sample in order to establish the generalisability of current findings and control for potential gender and cancer diagnosis effects. Prospective studies with longer follow-up, and at least three waves of data, are necessary to test the hypothesis of a temporal relationship between metacognitive beliefs and processes and emotional distress. It will also be useful to explore differences between groups that exhibit different trajectories of distress (i.e. never distressed, recovered, maintained), while controlling for the occurrence of any additional stressors or cancer recurrence in the intervening period. Aside from such observational studies, experimental treatment studies are required that allow metacognitive beliefs and processes to be manipulated to test the hypothesis of a corresponding change in emotional symptomology. In conclusion, it is noted that evidence of causality is never derived from a single study but through triangulation of evidence from a variety of studies that use different methodologies to address the same question.
References


Baker, P., Beesley, H., Dinwoodie, R., Fletcher, I., Ablett, J., Holcombe, C., & Salmon, P. (2012). 'You're putting thoughts into my head': a qualitative study of the readiness of patients with breast, lung or prostate cancer to address emotional needs through the first 18 months after diagnosis. Psychooncology. doi: 10.1002/pon.3156


List of Appendices

A: Prospective Study: Study Questionnaire

B: ATT Case Series Study: Exit Interview Guide

C: Cook S.A; Salmon, P; Dunn, G; Fisher, P. Measuring metacognitions in cancer:
   Validation of the Metacognitions Questionnaire 30 (MCQ-30). PlosOne

D: Cook S.A; Salmon, P; Dunn, G; Holcombe, C; Cornford, P; Fisher, P. The association of metacognitive beliefs with emotional distress after diagnosis of cancer. (2015). Health Psychology 34 (3); 207-215


F: Prospective Cohort Study: Participant Feedback Leaflet
Appendix A

Case Series Study: Exit Interview Guide
EXIT INTERVIEW

Questionnaires:

- MCQ-30
- FCRI by post
- HADS/CAS-1

Interview: by telephone or in-person (LMC)

1) SCID - current GAD, MDD, PTSD

2) Feedback on ATT Intervention (unstructured)

What was your understanding of the goal of the intervention?
Was this achieved?

When you have negative thoughts about [………..] how do you respond to them?
Is this different to how you would have responded to them previously? [if yes, how?]

On a scale of 1-10 how satisfied would you say you are with the intervention?

- In general
- How it was introduced at the first session
- Practising ATT at home
- Weekly phone calls

[Explore with reference to good/bad point / suggestions for improvements]

On a 1-10 scale how much confidence do you have that this technique will work for you in the longer term…….[explore response]

What would you say to a patient considering whether or not to try ATT?

Do you think it should be developed for use for people who have had cancer?

If yes, when would an intervention like this be most useful?
Appendix B


Appendix C


http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4321533/pdf/hea_34_3_207.pdf
Appendix D


Appendix E

Prospective Cohort Study: Participant Feedback Leaflet
What happens next?

Soon we will be inviting people to meet in a small group of other patients to discuss how we can use these findings to improve the care offered to cancer patients.

If you are interested in being involved in this, we would love to hear from you. You can contact us at the address below.

Contact

SHARON COOK
MRC Population Health Scientist Fellow
Division of Clinical Psychology, Institute of Psychology, Health and Society

THE UNIVERSITY OF LIVERPOOL
The Whelan Building, Quadrangle
Brownlow Hill
Liverpool
L69 3GB

Direct Tel: +44 (0)151 795 0347
sacook@liv.ac.uk

Acknowledgments:

We gratefully thank the staff and patients of the Royal Liverpool & Broadgreen University Hospitals NHS Trust for supporting this study.

The study was approved by:
NHS North West 5 Research Ethics Committee
(reference: 09/H1010/70)

The study was funded by:
This research was conducted as part of a Population Health Scientist Fellowship funded by the Medical Research Council.
http://www.mrc.ac.uk/index.htm

Feedback for Study Participants
**Why was the study being done?**

For many people, receiving a diagnosis of cancer is a traumatic experience. So feeling frightened, anxious, or emotionally overwhelmed is an understandable and common response.

With time, most people find their emotions settle down.

But this is not the case for everyone. Studies have shown that one in five people continue to have problems with anxiety or depression, even some time after treatment has ended.

The aim of this study was: **to explore why some people find it difficult to recover emotionally, while others do not?**

**What our theory says is happening.**

Negative thoughts, such as ‘this isn’t fair’, or ‘I’m not strong enough to get through this’ are common, especially when diagnosed with something like cancer. Many psychologists believe that it is these sorts of thoughts that make some people distressed.

However, a new theory suggests that *it is not negative thoughts that cause distress, but the way in which we respond to them.* That is, worrying about them or trying to stop them often makes us feel worse!

*We respond in this way because of assumptions we hold about our thinking,* such as:

- ‘Worrying about a problem will help me find a solution’
- ‘It is bad to have negative thoughts’

**The study & what we found.**

You were asked to complete questionnaires twice. The first time was at your pre-treatment appointment. The second was twelve months later. The questionnaires asked about your mood, your thoughts about your cancer and your beliefs about worry and the need to control thoughts.

*We found that….***

- **People who believe they have no control over worry, or that it could harm them, worry even more, often “worrying about worry” as well as any negative thoughts and feelings they have about their cancer.**
  - The stronger these beliefs, the greater the level of worry, and anxiety or depression.

*We also found that….***

- Over the twelve month period, if the strength of these beliefs about worry changed so did the level of worry, and anxiety or depression.

**What this means?**

These findings can be used to help people diagnosed with cancer who are anxious or depressed.

Specifically, they suggest that **if we can change these beliefs, and teach people to respond differently to negative thoughts and feelings they will worry less.**

*This will reduce their anxiety or depression.*

We are currently testing this in a small group of patients.

We thank you for your valuable help with this study.