BEHAVIOURAL SELF-BLAME IN CHRONIC ILLNESS:
A STUDY OF PREDICTORS AND CONSEQUENCES

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This thesis examined the relationship of behavioural self-blame and psychological adjustment to chronic illness and addressed some of the conceptual and methodological inconsistencies found in the existing literature on self-blame by studying different groups of chronically ill patients.

Study One comprised a questionnaire study and was designed to mainly examine 1) differences in the levels of self-blame in three different patient groups (i.e. diabetes, heart disease and breast cancer patients), 2) the relationship between perceptions of behavioural risk factors contributing to the patients' illness and level of self-blame and 3) the relationship between self-blame, self-efficacy and psychological adjustment. Findings showed that there are significant differences in the levels of self-blame across the three groups with breast cancer patients showing the least self-blame. These differences were explained in terms of the different levels of perceived lifestyle factors contributing to the cause of illness and its subsequent management in the three groups. Also, self-blame was higher when patients were asked to consider a specific negative event relevant to their illness than when they considered their illness in general. For all three groups, self-blame was correlated to the number of behavioural risk factors patients reported as having contributed to their illness. No relationship was found between self-blame and self-efficacy or psychological adjustment.

Study Two looked at the predictors of behavioural self-blame in heart disease patients by testing a theoretical model derived from evidence in the literature. The model included certain person (i.e. gender, age, characterological self-blame, and prior risk) and illness-related characteristics (i.e. type of diagnosis, time since diagnosis, perceived illness consequences, controllability of health behaviours) that had either direct paths to behavioural self-blame or indirect paths through their effect on behavioural causal attributions. The final model—showing gender and characterological self-blame as having both direct and indirect paths to self-blame, and prior risk, diagnosis and consequences as having only indirect paths—fit the data well. Also, behavioural attributions predicted
improved health behaviour after the illness. No relationship between behavioural self-blame or causal attributions and psychological adjustment was found.

Studies Three and Four addressed criticisms regarding the inconsistent conceptualisation and operation definitions of self-blame, which caution against unfounded generalisations such as the interchangeable use of the terms causality, responsibility and blame or the generalisation of results across different populations. This was done by 1) examining the degree to which self-blame is contingent upon the actual experience of illness, and 2) by comparing 14 negative events rated on dimensions relevant to blame and controllability. Specifically, in Study Three, non-patients were compared to patient counterparts from the previous study to look at differences in levels of self-blame for heart disease. Non-patients were found to have higher levels of self-blame than non-patients and showed no relationship between self-blame and behavioural risk suggesting a different understanding and utility of the concept. In Study Four, non-patients rated 14 negative events on the dimensions of blame, responsibility, control and avoidability. Two dimensional plots showed that while there were many similarities in the way the examined dimensions were applied to the 14 events, illnesses were represented separately from other negative events.

Overall, these studies suggest a strong cognitive component in self-blaming patients as opposed to the motivational elements suggested in the literature. Possible explanations and ways to theoretically link the contradictory findings are discussed in the last chapter of the thesis and include the consideration of self-regulation processes and of changes that the self is subject to throughout the course of an illness.
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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABSTRACT</td>
<td>ii</td>
</tr>
<tr>
<td>ACKNOWLEDGEMENTS</td>
<td>iv</td>
</tr>
<tr>
<td>TABLE OF CONTENTS</td>
<td>v</td>
</tr>
<tr>
<td>List of Figures</td>
<td>xl</td>
</tr>
<tr>
<td>List of Tables</td>
<td>xiii</td>
</tr>
<tr>
<td>Chapter One Overview of the Thesis</td>
<td>1</td>
</tr>
<tr>
<td>Chapter Two SETTING THE THEORETICAL BACKGROUND FOR THE STUDY OF SELF-BLAME</td>
<td>6</td>
</tr>
<tr>
<td>2.1 INTRODUCTION</td>
<td>6</td>
</tr>
<tr>
<td>2.2 ILLNESS AS A CHRONIC CONDITION</td>
<td>7</td>
</tr>
<tr>
<td>2.2.1 Illness representations and Leventhal’s model of illness behaviour</td>
<td>9</td>
</tr>
<tr>
<td>2.3 THEORIES OF CAUSAL ATTRIBUTION</td>
<td>11</td>
</tr>
<tr>
<td>2.3.1 Categorisation and basic assumption in theories of attribution</td>
<td>13</td>
</tr>
<tr>
<td>2.3.2 Motivations behind causal attributions</td>
<td>15</td>
</tr>
<tr>
<td>2.3.3 Dimensions in causal attribution</td>
<td>16</td>
</tr>
<tr>
<td>2.3.4 Common confusions about the organisation of causes</td>
<td>19</td>
</tr>
<tr>
<td>2.4 CAUSAL ATTRIBUTIONS IN PHYSICAL ILLNESS</td>
<td>21</td>
</tr>
<tr>
<td>2.4.1 Fact or artifact?</td>
<td>21</td>
</tr>
<tr>
<td>2.4.2 Attributions and Adjustment</td>
<td>23</td>
</tr>
<tr>
<td>2.4.3 Empirical evidence on illness attributions</td>
<td>24</td>
</tr>
<tr>
<td>2.5 A SYNTHESIS</td>
<td>36</td>
</tr>
</tbody>
</table>
Chapter Three  SELF-BLAME: THEORETICAL, METHODOLOGICAL AND EMPIRICAL CONSIDERATIONS

3.1  the theory of self-blame

3.1.1  The theoretical rationale for the functionality of self-blame

3.2  Empirical evidence on self-blame

3.2.1  Literature search and organization of results

3.2.2  General self-blame

3.2.3  Characterological self-blame

3.2.4  Behavioural self-blame

3.3  Limitations in the literature

3.3.1  Methodological explanations for the inconsistent results

3.3.2  Theoretical limitations in self-blame research

3.4  IDENTIFICATION OF RESEARCH QUESTIONS

Chapter Four  MEASURING SELF-BLAME IN CHRONICALLY ILL PATIENTS: A PILOT STUDY

4.1  INTRODUCTION

4.2  PHASE I: ITEM GENERATION AND PILOTING

4.2.1  Ethical Approval

4.2.2  Participants

4.2.3  Measures

4.2.4  Procedure

4.3  RESULTS FROM PILOT DATA

4.3.1  Item clarity and comprehensiveness

4.4  PHASE II: MAIN DATA COLLECTION

4.4.1  Participants and Recruitment Procedures
4.4.2 Measures ........................................................................................................ 81
4.4.3 Procedure ......................................................................................................... 83

4.5 RESULTS ........................................................................................................... 84
4.5.1 Sample ............................................................................................................. 84
4.5.2 Self-Blame Scale ............................................................................................. 86
4.5.3 Descriptive Analyses ....................................................................................... 89
4.5.4 Effects of specificity of reference and type of medical condition on self-blame ... 92
4.5.5 Behavioural risk factors and self-blame ........................................................ 93
4.5.6 Relationship between self-blame, self-efficacy and psychological adjustment. 95
4.5.7 Self-blame and time elapsed since diagnosis ................................................. 95
4.5.8 Self-blame and diet/exercise .......................................................................... 95

4.6 DISCUSSION ..................................................................................................... 96
4.6.1 Phase I: Item generation and piloting ............................................................ 96
4.6.2 Phase II: Main data collection ........................................................................ 97

Chapter Five PREDICTORS AND OUTCOMES OF BEHAVIOURAL SELF-BLAME

5.1 INTRODUCTION .................................................................................................. 104
5.1.1 Conceptual model of BSB ............................................................................. 107
5.1.2 Person characteristics .................................................................................... 108
5.1.3 Event characteristics ....................................................................................... 110
5.1.4 Outcomes of behavioural self-blame ........................................................... 111

5.2 METHOD ............................................................................................................ 113
5.2.1 Ethical Permission ........................................................................................ 113
5.2.2 Participants ..................................................................................................... 113
5.2.3 Measures ........................................................................................................ 114
5.2.4 Procedure ....................................................................................................... 117
5.2.5 Analyses .......................................................................................................... 117
5.3 RESULTS

<table>
<thead>
<tr>
<th>Subsection</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.3.1 Participants</td>
<td>119</td>
</tr>
<tr>
<td>5.3.2 Scales and reliabilities</td>
<td>119</td>
</tr>
<tr>
<td>5.3.3 Descriptives</td>
<td>121</td>
</tr>
<tr>
<td>5.3.4 Relationships among variables</td>
<td>123</td>
</tr>
<tr>
<td>5.3.5 Model testing</td>
<td>125</td>
</tr>
<tr>
<td>5.3.6 Outcome measures</td>
<td>129</td>
</tr>
</tbody>
</table>

5.4 DISCUSSION  

Chapter Six PERCEPTIONS OF BLAME IN NON-PATIENTS  

<table>
<thead>
<tr>
<th>Subsection</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.1 INTRODUCTION</td>
<td>136</td>
</tr>
<tr>
<td>6.2 METHOD</td>
<td>139</td>
</tr>
<tr>
<td>6.2.1 Participants and recruitment</td>
<td>139</td>
</tr>
<tr>
<td>6.2.2 Measures</td>
<td>140</td>
</tr>
<tr>
<td>6.2.3 Choice of Analyses</td>
<td>143</td>
</tr>
</tbody>
</table>

Chapter Seven INVESTIGATING SELF-BLAME IN NON-PATIENTS:

<table>
<thead>
<tr>
<th>Subsection</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.1 ANALYSES AND RESULTS</td>
<td>145</td>
</tr>
<tr>
<td>7.1.1 Participants</td>
<td>145</td>
</tr>
<tr>
<td>7.2 Differences between patients and non-patients</td>
<td>148</td>
</tr>
<tr>
<td>7.2.1 Relationships between self-blame, lifestyle risk and control: differences in patterns</td>
<td>150</td>
</tr>
<tr>
<td>7.3 DISCUSSION</td>
<td>152</td>
</tr>
</tbody>
</table>

Chapter Eight PERCEPTIONS OF BLAME, RESPONSIBILITY AND CONTROL FOR NEGATIVE LIFE EVENTS in non-patients  

<table>
<thead>
<tr>
<th>Subsection</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.1 ANALYSES AND RESULTS</td>
<td>157</td>
</tr>
</tbody>
</table>
Appendix VIII Questionnaire used in Study 2 (Chapter 5) 235

Appendix IX QUESTIONNAIRE used for Studies 3 and 4 (Chapters 6-8) 242

Appendix X AGGLOMERATION Tables for cluster solutions found in Study 4 (chapter 8) 248
LIST OF FIGURES

Figure 2.1: Leventhal’s self-regulation model ................................................................. 9
Figure 2.2: Basic structure of attribution conceptions as proposed by Kelley and Michela, (1980) ........... 14
Figure 2.3: Theoretical path models of the relationship between attributions and adjustment (distress) ........ 24
Figure 4.1: Frequency of risk factors perceived as relevant to illness (N=136) ...................................... 94
Figure 5.1: Conceptual diagram of the proposed relationships between BSB and its predictor and outcome variables. ............................................................................................................................. 112
Figure 5.2: Path diagram of the relationships among person characteristics, illness attributions and BSB. ........................................................................................................................................ 126
Figure 5.3: Path diagram of the relationships among event characteristics, illness attributions and BSB. ........................................................................................................................................ 127
Figure 5.4: Final path model of predictors of BSB .................................................................................. 128
Figure 8.1: Two-dimensional plot depicting the 14 negative events rated on the degree of blame assigned to the sufferer. .................................................................................................................. 161
Figure 8.2: Two-dimensional plot depicting the 14 negative events rated on the degree of responsibility assigned to the sufferer. ........................................................................................................... 162
Figure 8.3: Two-dimensional plot depicting the 14 negative events rated on the degree of avoidability the sufferer is perceived to have. ........................................................................................................... 163
Figure 8.4: Two-dimensional plot depicting the 14 negative events rated on the degree of control the sufferer is perceived to have over the occurrence of the event ........................................................................ 164
Figure 8.5: Dendrogram showing the three cluster solution yielded from the analysis of events rated on ‘blame’. ............................................................................................................................................... 166
Figure 8.6: Cluster solution superimposed on MDS solution for ‘blame’ rating of events. ......................... 167
Figure 8.7: Dendrogram showing the four cluster solution yielded from the analysis of events rated on ‘responsibility’. ........................................................................................................................................ 168
Figure 8.8: Cluster solution superimposed on MDS solution for ‘responsibility’ rating of events. ............. 169
Figure 8.9: Dendrogram showing the four cluster solution yielded from the analysis of events rated on 'avoidability'. 170

Figure 8.10: Cluster solution superimposed on MDS solution for 'avoidability' rating of events. 171

Figure 8.11: Dendrogram of the three cluster solutions of events rated on 'control'. 172

Figure 8.12: Cluster solution superimposed on MDS solution for 'control' rating of events. 173
LIST OF TABLES

Table 3.1 : Studies reporting the relationship between self-blame and adjustment in participants dealing with chronic illness. .............................................. 50

Table 4.1: Characteristics of the patient sample (N= 74). ........................................... 77

Table 4.2: Items included in the study following item generation procedures. ....................... 78

Table 4.3: Contribution of items to scale reliability (scale alpha = .73). ............................. 79

Table 4.4: Patient characteristics for each patient group .................................................. 85

Table 4.5: Item communalities for factor analysis obtained from reference to the general condition and the specific event. ...................................................... 87

Table 4.6: Pattern matrices showing the two-component solutions for general and specific self-blame. 88

Table 4.7: Mean values for all psychological variables. .................................................... 90

Table 4.8: Pearson’s correlations among main psychological and demographic variables (N in parentheses) ........................................................................... 91

Table 4.9: Frequencies of types of specific events mentioned by patients. .......................... 93

Table 5.1: Item communalities and structural coefficients for causal attribution items. ............... 120

Table 5.2: Item communalities and structural coefficients for perceived control items. .......... 121

Table 5.3: Means and standard deviations of main variables ................................................. 122

Table 5.4: Pearson’s correlations (r) among the variables in the model (155<N<160) ............... 124

Table 5.5: Hierarchical regressions of behaviour change and psychological adjustment variables on attributions and BSB. ..................................................... 130

Table 7.1: Matching characteristics of the samples in this study. Perfect matching is denoted by (✓) 147

Table 7.2: Demographic characteristics of patients and non-patients. .................................. 147

Table 7.3: Mean and standard deviation of the main variables in the study for each group. .......... 149

Table 7.4: Pearson’s correlations between self-blame, lifestyle risk and perceived control for patient and non-patient groups (82<N<85). .................................................. 151

Table 7.5: Hierarchical regressions of BSB on CSB, lifestyle risk and control items for both groups. 152
Table 8.1: Means and standard deviations of ratings of responsibility, avoidability, control and blame for each negative event (events with the lowest and highest mean are in bold). 

Table 8.2: Stress and $R^2$ values of the 2-dimensional solutions 

Table 8.3: T-tests for differences in mean scores between i) blame and responsibility and ii) control and avoidability for each negative event.
CHAPTER ONE

OVERVIEW OF THE THESIS

When people face unexpected negative events, they usually attempt to find an explanation for this event by examining possible causes such as themselves, others, the environment, and chance. Psychological research has examined the type of attributions people make for a negative event, as well as their potential impact on psychological adjustment. One particular relationship, that between self-blame for a misfortune and psychological adjustment, has been extensively studied in a variety of populations, but results have been far from conclusive. Conclusions become even more complicated when the two types of self-blame, behavioural self-blame (i.e. attributing a negative event to one’s own behaviour) and characterological self-blame (i.e. attributing a negative event to one’s own character) are considered. In particular, where a relationship to adjustment has been found, characterological self-blame has generally yielded a negative relationship. In contrast, behavioural self-blame has been found to have positive, negative or no relationship to measures of anxiety, depression or general distress. Studies on chronically ill patients mirror these inconsistencies and raise questions regarding the reasons for such a lack of consensus.

By focusing mainly on behavioural self-blame, the central aim of the present thesis is to identify the possible explanations for the inconsistent results and address some of them. Specifically, this thesis looks at the following:

1. Sample and event diversity.
2. Person and situation-related predictors of behavioural self-blame.
3. Time elapsed since diagnosis.

4. Operationalisation and measurement of self-blame.

5. Effects of behavioural self-blame on adjustment and health behaviour change.

Throughout the four studies presented in this thesis, an attempt is made to unpack the concept of behavioural self-blame and describe the context within which it is used as well as its functions.

Specifically, Chapters Two and Three review the literature on causal attributions and self-blame and portray the theoretical background of the thesis. Studies of causal attributions for illness are reviewed in Chapter Two and the evidence for their antecedents and consequences are considered. Chapter Three specifically identifies the inconsistencies in the findings regarding behavioural self-blame and the reasons behind them. Regarding its lack of conceptual clarity, evidence on the distinction between behavioural causal attributions and behavioural self-blame is presented and the implications of this evidence on the conceptual issues surrounding self-blame are discussed. Finally, Chapter Three concludes by identifying the main issues addressed by the studies in the thesis.

The study presented in Chapter Four deals with the issues of self-blame measurement and sample diversity. A self-blame scale is constructed and piloted, before it is administered in three different patient groups (i.e. heart disease, diabetes and breast cancer patients). Self-blame levels are compared across the three groups and the significant differences found are theoretically and empirically linked to differences in illness characteristics, mainly the number of behavioural risk factors associated with each illness. This chapter
also explores the relationship between self-blame and time since diagnosis, psychological adjustment, and adherence to healthy diet and exercise plans. All relationships are examined both for the whole sample and for each patient group separately. Findings demonstrate the plausibility of the idea that self-blame may be situation specific.

The possibility of situation-specificity in self-blame (and more specifically behavioural self-blame) raises the need to examine a number of predictors of behavioural self-blame in order to assess when self-blame is likely to be more evident. Chapter Five proposes a theoretical model of predictors and outcomes of behavioural self-blame and tests it on a sample of heart disease patients. The final model includes several person and situation-related variables (e.g. gender, characterological self-blame, prior risk, type of diagnosis) many of which affect behavioural self-blame indirectly through their effect on behavioural attributions. Behavioural attributions are also shown to predict changes in certain health behaviours such as diet and smoking. No relationship was found between self-blame and psychological adjustment.

Chapters Six, Seven and Eight deal with the issues of operationalisation of self-blame and the effects of event diversity on understanding blame and responsibility for illness. After introducing the rationale and methodology for the two studies in Chapter Six, Chapter Seven presents a comparison of self-blame between patients and non-patients. Patients were found to display lower levels of self-blame than non-patients suggesting that having actually experienced the threats of illness affects the cognitive and motivational components of illness explanations. Chapter Eight uses a quite different approach than the other studies in the thesis and attempts to offer a visual depiction of people’s
representations of negative events when issues of blame and control are considered. Using Cluster and Multidimensional Scaling analyses participants’ ratings of 14 negative events -including a variety of illnesses- on the dimensions of blame, responsibility, control and avoidability are examined. The resulting clusters and two-dimensional plots allow the examination of a) people’s overall representations of negative events along the four dimensions, thus informing on how these dimensions are understood and used, b) similarities and differences among the representations of these dimensions, c) perceived similarities and differences among the 14 negative events- all of which have been used in the literature and have yielded inconsistent results. Results show that while participants represented the 14 events very similarly along the four dimensions (and indeed no significant differences are found when these are tested statistically for each event) they represent illnesses separately from other negative events, suggesting that an illness may carry a different meaning than other misfortunes. The implications of these findings on the way self-blame is operationalised and relevant evidence generalised across situations are considered.

Finally, Chapter Nine brings together all the evidence presented in the thesis and reviews it in relation to the original research questions identified in Chapter Three. The theoretical implications of these findings are also discussed, and past and present evidence regarding behavioural self-blame are placed within the theoretical framework of self-regulation and the dynamics that develop through the course of adaptation to illness. It is also proposed that self-blame be studied through the prism of such a theoretical framework that allows the inclusion of many aspects of the illness experience. The chapter concludes with a
review of the limitations of the studies presented and a brief stipulation of the findings into a general conclusion.
SUMMARY

This chapter provides a comprehensive overview of the general theories and concepts that formed the background for the present thesis. The experience of chronic illness is briefly discussed. Leventhal's self-regulation model is considered as a framework for understanding the illness experience and the focus is directed specifically on causal attributions, a particular aspect within the Illness Perceptions component of the model. The theory and empirical evidence regarding causal attributions in illness is discussed. Finally, the theoretical and empirical issues discussed in the chapter are brought together in a discussion on how they can shape one's understanding of and inform research on self-blame.

2.1 INTRODUCTION

In order to understand the concept of self-blame, one needs to review a number of theoretical frameworks that have served as the background for self-blame research. This thesis is about self-blame in chronic illness. Put more simply, it is about people's explanation of the cause of their illness and the extent to which they see themselves as being responsible for causing their illness. This implies that when studying self-blame, one studies an illness cognition, a part of an illness representation, an attribution. It is therefore necessary to set the theoretical scene against which self-blame must be explored.
and understood. For this reason, the following sections consider the evidence on illness attributions placed within the scope of well-established models of illness cognitions.

2.2 ILLNESS AS A CHRONIC CONDITION

Both the medical and the psychosocial perspective on illness agree on certain categorizations of illness. One of the most frequently used is that of acute versus chronic illness. An acute illness makes an individual move from a healthy state to a temporarily unhealthy state and then back to healthy one. A ‘sick role’ is adopted while in the unhealthy state and is abandoned once the person recovers. In contrast, a chronic illness makes the individual move into an - often permanent - state where the boundaries between health and illness are not so clear and the effects of the illness must be incorporated in the everyday functioning of an otherwise healthy life (Bradley, 1994). Thus, the long-term management of the illness becomes a central part of a patient’s life and may involve a re-assessment of fundamental cognitions and behaviours in ways analogous to those seen in victimization (e.g. Collins, Taylor & Skokan, 1990).

It is these issues of management that make chronic illness particularly interesting from a social-psychological perspective. How do people cope with chronic illness? How do they understand and explain its occurrence and what role do these explanations play in modifying behaviour and maintaining a certain lifestyle? The answers to these questions have been sought repeatedly in psychological research but certain parameters have made the quest a complicated one. For example, there is a fair amount of uncertainty in medicine as to what the exact causes are for certain diseases. This has an impact on
people’s understanding of illness and the actions they take to avoid illness. For example, studies by Davison et al (1990) found that participants’ uncertainty regarding the relationship between lifestyle and heart disease contributed to their remarkable lack of concern about the effects that their diet could have on maintaining a healthy heart. Moreover, the public is the recipient of conflicting messages regarding the causes of disease. While health campaigns target certain risk behaviours and present certain behavioural profiles as risk factors for chronic conditions, progress in genetics stresses the role that hereditary predispositions can play in one’s health and implies a deterministic view of illness. And although to a health professional the two approaches are not mutually exclusive, it is left up to laypeople to make sense of this information and decide on the appropriate course of action (Mumma & McCorkle, 1982).

Apart from the issue of causal explanation, there are other features of chronic illness that make it a compelling topic for study. First, the time duration implied by the term chronic is an important factor (Bradley, 1994). Although patients may not actually experience symptoms every day of their chronic illness, the knowledge of a potential recurrence is permanent and the way the present is interpreted is defined by both past experiences and future possibilities (Bury, 1991). Finally, the variability in symptoms, prognosis, and significance across chronic illnesses (or sometimes within the same type of illness) is another characteristic of chronic illness. This feature implies that there is a large number of variables (e.g. stage of illness, type of symptoms, treatability, severity) that may be salient in people’s minds that must be considered when the experience of a chronic illness is explored. These salient characteristics of the experience are likely to influence how the
illness representations of the patient are shaped and may also affect how the patient copes with the illness.

2.2.1 Illness representations and Leventhal’s model of illness behaviour

The complicated process of understanding and adjusting to illness has been expressed in a model proposed by Leventhal and his colleagues (Leventhal and Nerenz, 1985). It is a self-regulation model that assumes that, given a problem, a person will be motivated to solve the problem and re-establish their state of health. Although not directly tested in the studies reported in this thesis, this model is used as a helpful framework for understanding the concepts involved. According to this model (Figure 2.1) there are three stages of self-regulation: interpretation (stage 1), coping (stage 2), and appraisal (stage 3).

Figure 2.1: Leventhal’s self-regulation model
Stage 1 involves the attempt to understand the problem and assign meaning to it using available information. This information comes from a variety of sources such as the characteristics of a problem (e.g. alarming symptoms), social messages (e.g. doctor’s advice), emotional responses to the threat (e.g. fear), and the individual’s representation of the threat (e.g. its cause, consequences).

Stage 2 involves the development of coping strategies broadly categorised into approach and avoidance strategies. Coping can refer to the individual’s attempt to a) come to terms with the diagnosis itself, b) deal with the crisis of an illness, or c) adjust to the illness through a process of cognitive adaptation.

Finally, Stage 3 involves appraisal, the evaluation of the strategies utilised so far and the decision of whether to proceed in the same way or use an alternative course of action. It is worth noting that there are bi-directional arrows going from each stage (or stage component) to the other stages. This implies a dynamic relationship between the various stages of the self-regulation process and expresses the possibility that at any point in the process information arising from a given stage can feed back to a previous stage and alter it.

Looking at the issues involved in being chronically ill discussed earlier, one can see how they could be explored with Leventhal’s model in mind. For example, at stage 1, the uncertainty of an illness cause can lead to unclear or contradictory illness representations which in turn can give rise to more negative emotions and affect the type and efficiency of the coping strategy used.
Leventhal’s model has been a very useful framework in describing illness cognitions. It has also proven to be a practical model because it is general enough to allow researchers considerable flexibility to ‘zoom in’ to its components and study them in more depth. For example, considerable research exists on the component of illness representations (Petrie & Weinman, 1996). Indeed, this component can be broken down into its own constituents which can easily be studied separately. The present thesis deals with a specific part of illness representations, causal attributions. Within attributions, it investigates the concept of self-blame as an attribution for one’s illness and attempts to explore its meaning and significance in the illness experience. Therefore, in the next sections, discussion will focus on theoretical and methodological issues regarding causal attributions in illness. Leventhal’s overall model will be considered again in the last chapter in an attempt to present all the findings in this thesis against a clear theoretical background.

2.3 THEORIES OF CAUSAL ATTRIBUTION

Causal thinking is a complicated set of processes which affects everyday thoughts, actions and emotions (Anderson, 1991). In turn, this set of processes is itself affected by beliefs and attitudes, creating a complex cognitive network of dynamically interacting parts which has been the focus of a considerable amount of research.

[…] attributional analyses may be propaedeutic to the application of other theories, for the simple reason that attributions arouse a diversity of other motives and mediate numerous other processes such as aggression, guilt, or motivational changes (Jones et al., 1972, pp xii).
To this day, the cornerstones of the psychological theories of causal attribution are the works of Heider (1958), Jones and Davis (1965), and Kelley (1973). Heider’s interest in ‘naive psychology’ led to an attempt to understand and formulate people’s common-sense rules for causal attributions (Hewstone, 1989).

According to Heider’s theory (1958), actor and act are seen as parts of a causal unit. Interpretations regarding this unit are affected by factors such as similarity and proximity: when two events are similar or proximate to each other, then the one is likely to be seen as the cause of the other. Also, there is a mutual influence between actor and act, and acts can become infused with characteristics of the person to whom they were attributed. Consequently, a ‘person’ attribution is more likely than a ‘situational’ one (fundamental attribution error) because the person is seen as the centre of a situation (‘prototype of origin’). Heider has also suggested that intentional actions are more readily attributed to personal dispositions than unintentional ones. Perceived intentionality is in turn based on the criteria of ‘equifinality (whether action is goal-directed rather than means-centred), local causality (whether people are seen as agents of an action, rather than passive recipients of environmental forces), and exertion (people are presumed to try harder to achieve intended effects or goals)’ (Hewstone, 1989, pp.14).

An important idea added to Heider’s theory is the concept of Correspondent Inference put forward by Jones and Davis (1965). This refers to one’s judgement that the actor’s behaviour (and intention behind it) is caused by, or corresponds to a particular trait that he or she possesses. For example, someone’s hostile behaviour would lead to the assumption
that the person is hostile by trait. In the process of inferring personal dispositions the perceiver is faced with two problems: the attribution of intention, and the attribution of dispositions. The attribution of intention involves deciding which effects of an observed action were intended by the actor. This suggests that the perceiver must believe that the actor is both capable of performing the action as well as aware of its consequences. Then, by considering the consequences of chosen actions, the observer makes a correspondent inference of dispositions when the chosen action has a few relatively unique consequences. Such inferences are stronger when the consequences of a chosen behaviour are socially undesirable.

Finally, Kelly (1967; cited in Hewstone, 1989) focused on the information used to arrive at a causal attribution. He outlined two different cases that depend on the amount of information available to the perceiver. In the first case, the perceiver has information from multiple sources and can perceive the covariation of an observed effect and its possible causes. In the second case, the perceiver is faced with a single observation and must take account of the configuration of factors that are plausible causes of the observed effect.

2.3.1 Categorisation and basic assumption in theories of attribution

The three theories outlined above are generally considered the major contributions in the field of causal attribution. They have been the centre of continuous criticism and have given rise to a considerable amount of research which in turn has lead to extensions or specifications of the main theories. Hence, what has come to be commonly known as Attribution Theory in psychology is really a collection of theories that explain how
common sense operates and how people provide themselves with explanations about events they observe.

Theories of attribution are often divided into two sub-fields: attribution theories and attributional theories (Kelley & Michela, 1980). As illustrated in Figure 2.2, attribution theories deal with the antecedents of attributions while attributional theories deal with the psychological consequences.

Figure 2.2: Basic structure of attribution conceptions as proposed by Kelley and Michela, (1980)

Forsteling (2001) reviews three basic assumptions behind theories of attribution. First, as with any cognitive approach to psychology, there is the assumption that cognitions mediate the relationship between stimulus and behaviour. Hence, theories of attribution focus on the nature of the cognitive processes that will result in an attribution being made as well as the variables that can affect those processes. A second assumption is that people typically attempt to understand the world around them by making attributions and do so in a methodical way similar to that of the scientific method. Individuals are believed to generate hypotheses about the occurrences of events and test them using available or
inferred information which will eventually either confirm the hypothesis or disconfirm it and lead to its reformulation. Finally, a third assumption is that it is functional to make attributions. It is believed that trying to understand the causes of observed events is inherently beneficial in that it enhances one’s sense of control and allows for future predictions to be made.

2.3.2 Motivations behind causal attributions

Possibly because of the last assumption discussed above, theories of causal attributions have mainly focused on how people reach conclusions about the causes of events and less on the reasons that such cognitive processes occur in the first place. The best attempt towards addressing the latter can be found in two reviews that emphasise the following points (Forsyth, 1980; Tetlock and Levi, 1982):

1. The control function. Causal explanations of events create a sense of control about one’s acquired experiences and anticipated future outcomes (Wortman, 1976). This function often provides an explanation about attributions that appear ‘unreasonable’. For example, blaming the victim for his/her misfortune or claiming responsibility for an outcome that is clearly beyond one’s control are both instances of counter-intuitive attributions that can be explained as ‘control-oriented’ responses to negative events (Jannoff-Bulman, 1983).

2. The self-esteem function. Quite commonly in attribution processes the actor/attributer makes an internal attribution for a positive outcome and an external attribution for a
negative outcome. Such self-serving biases are seen as enhancing self-esteem since they protect from embarrassment or humiliation.

3. **The self-presentation function.** Following from the logic behind the self-esteem function is the effort individuals make to control the way they are seen by those around them. By making external attributions for negative events and internal attributions for positive ones, people not only enhance their self-esteem but at the same time ensure a more positive public image.

**2.3.3 Dimensions in causal attribution**

Causal attributions can be subject to a number of classification criteria. These can refer to the type of relationship between the cause and the effect (*relational properties*) or to the type of cause (*qualitative properties*). The latter are of particular importance in this thesis. In contrast to the relational properties, which carry more significance for studies focusing on the perceptual and information processing aspects of causal thinking, the qualitative properties have attracted interest through studies focusing on attributional motivation and outcomes. The most popular theory on this is Weiner's (1986) attributional theory on motivation and emotion, which classifies causal attributions in three domains: locus, stability and controllability.

A. Relational Properties

**Proximal vs. distal relations:** Kelley (1983) stressed the role that the distance between the cause and the effect can have on attributions. He proposed that in a chain of causes which eventually led to the effect under scrutiny, it is the proximal causes (temporally closer to
the effect) rather than the distal ones (temporally distant) that will offer the best explanation.

Simple vs. complex relations: A simple causal cognition exists when only one cause is related to only one effect. Alternatively, a complex attribution exists when many causes or their interaction lead to one or more effects (Kelley, 1983)

B. Qualitative properties

Locus: It is very common for attributions to be conceptualised as either internal (with the causal factors lying within the person) or external (with causal factors lying outside the individual). Heider (1958) argued that such a distinction is of fundamental importance and many studies since then have used it as a framework. However, it has been criticised on several points (Miller et al, 1981). First, attributing more causality to internal factors does not entail attributing less causality on external ones. That is, internal and external factors are not necessarily on the same continuum nor are they mutually exclusive. Second, internality and externality may have a theoretical basis of interest but are so broad and ambiguous that they become practically meaningless. For example, attributing one's illness to an unhealthy diet may appear as an internal attribution but if that diet resulted from poor parental guidance or financial reasons then it may be closer to the externality dimension. In such cases, a theoretical debate might continue endlessly, but it is the actual meaning of the attribution (the behaviours and emotions it elicits to the patient) that carry the most interest. Third, and in line with the previous point, the various measures of external or internal attributions have shown lack of convergent validity indicating that actors and theorists do not conceptualise causality in the same way. Finally, the internal-
external distinction cannot account for instances where the individual acts intentionally on the external factors. Could a car accident be attributed to the icy road (external attribution) when the driver intentionally ignored the weather forecast warnings and did not take the necessary precautions?

In an attempt to clarify the internal-external dimension, White (1991) conducted two studies where participants were asked to judge whether different explanations of events were internal or external and whether they were reasons for or causes of the events. It was found that asking people to distinguish between internal or external causes is not enough since no reference is made to whether the behaviour was conscious, intentional and explained or not.

**Stability:** Weiner identified another dimension along which causes are perceived (Weiner et al., 1971). Depending on how changing or unchanging causes are believed to be over time, they are categorised as stable or unstable. For example, ability and intelligence are generally seen as stable factors, while luck or effort as unstable ones.

A popular approach in the literature is the combination of the locus and stability dimensions to create a taxonomy of causes. Based on this, a cause can be internal and stable (e.g. ability), internal and unstable (e.g. effort), external and stable (e.g. task difficulty) or external and unstable (e.g. chance) (Weiner et al. 1971).

**Controllability:** An important element missing from the above taxonomy is the extent to which a cause or factor leading to an outcome is controllable by the individual or not.
This was pointed out by Rosenbaum (1972; cited in Forsterling, 1988), who integrated this dimension in Weiner et al.’s (1971) scheme. However, Rosenbaum’s original dimension was that of ‘intentionality’ and not controllability implying that the two terms do not differ. This was later criticised by Weiner (1986), who differentiated intentional actions (e.g. intention to go on a diet) from controllable ones (e.g. uncontrollable eating behaviour).

Another dimension of cause categorisation later added to the above (Forsterling, 1988) is that of generality. Generality differentiates global causes (those having broad effects) from specific causes (those relating to a narrow field). Thus, for example, low intelligence would be categorised as global since its effects would probably appear in a wide spectrum of behaviours. In contrast, a special inability such as dyslexia would affect only a certain range of behaviours (i.e. language skills) and would be seen as specific.

2.3.4 Common confusions about the organisation of causes

Although research on causal attribution has been carried out for the last 30 years, there is still confusion about the organisation of causal factors (Anderson, 1991). First, there is difficulty in distinguishing phenomenal from scientific descriptions of causes. That is, it is not necessarily true that people’s way of thinking about the world matches the scientific approach. It may well be, for example, that people think about causes without realising that their reasoning follows the dimensions of locus, stability or generality. Finally, it may be that there is a perfect match between people’s categorisation of causes and the scientific relations between these variables. However, none of these instances indicate that
there is a necessary relation between people’s beliefs about the dimensions mentioned earlier and their actual effects.

Second, literature on causal attributions seems to assume that the way people can think is the same as the way people do think. Thus, study designs usually force people to think along the various attributional dimensions and eventually measure people’s ability to think that way. It is therefore important that this limitation be acknowledged when findings in the area are discussed.

As a result of the two previous points, a third confusion in attribution literature arises. It seems to be a common assumption that people think in terms of dimensions. This is probably an influence stemming from scientific reasoning where theories, laws, constructs and so on are generally framed in dimensional terms. However, it is possible that people think in categorical terms, or even, both categorical and dimensional terms. Anderson (1991) presented participants with 63 different causes and asked them to either rate them on given dimensions (i.e. internal vs. external), sort them in as many meaningful groups as they could think of (favouring both categorical and dimensional sorting), or sort them in exactly two groups (favouring dimensional sorting). He found that people typically engage in both categorical and dimensional thinking. It was suggested that categories are used very quickly in the beginning of the attribution process and indicate the implications for action, followed by dimensional thinking (mainly along the locus and controllability dimensions).
2.4 CAUSAL ATTRIBUTIONS IN PHYSICAL ILLNESS

The general principles on attribution theory presented above are based mostly on research within the area of social psychology. However, attributional approaches have been applied to many types of behaviour relevant to motivation, emotion and cognition. Regarding health, principles of attribution are increasingly used to investigate the role of such health cognitions in relation to the illness experience.

Illness is a negative, usually unexpected event that has profound physiological and psychological effects on the individual. It usually evokes a series of bodily responses that place an adjustment demand on the human organism making the need for psychological adaptation equally important. Since attributional search is seen as an attempt to understand, predict, and control events (including threats), it can be functional in the early stages of the adjustment process (Taylor, Lichtman & Wood, 1984). As with all other negative events, ill individuals may seek answers to questions such as ‘Why am I ill?’, ‘What caused my illness?’, ‘Could my illness have been prevented?’ ‘Did I cause the illness or is it due to an external cause? (Bishop, 1991)’.

2.4.1 Fact or artifact?

Benyamini, Leventhal & Leventhal (1997) approach attributions of health and illness from the perspective of Leventhal’s ‘self-regulation model’ (Leventhal, Meyer & Nerenz, 1980). As presented earlier in this chapter, the model proposes five cognitive dimensions (i.e. the illness representation component) to make sense of their health or illness: a) identity, i.e. the label given for a condition or symptom (e.g. cold, runny nose), b) cause, i.e. biological (e.g. virus), psychological (e.g. stress), c) time line, i.e. how long the
illness/symptom will last, d) consequences, i.e. physical or emotional effects of the condition on the person's life, and e) curability and controllability, i.e. the person's perception of whether the condition can be treated, and the extent to which it can be controlled. Based on these dimensions, it appears that illness cognitions mainly involve an illness/symptom representation, illness management procedures and outcome evaluation criteria. In the self-regulation model causal attributions are seen as part of the illness representation.

As part of this model however, attributions are seen as more likely to occur early in the disease progression and with serious or life-threatening conditions. However, not enough evidence exists on that yet. Benyamini, Baum, Newman et al (1997) claim that the methodology of attribution assessment has so far been flawed because probes were used extensively in order to assess the types of attributions. It should not be assumed that patients will spontaneously seek causal explanations for their condition. To illustrate this point they cite Lowery et al (1987) who found that chronically ill patients with arthritis, diabetes, hypertension, or past myocardial infarction reported they had never thought why this had happened to them. It may well be that these particular groups of patients see no use in causal explanations since re-occurrence is not as much an issue as is symptom management and quality of life. In any case, the way in which attributions are assessed may well affect yielded results and should therefore be considered during the interpretation of findings.

Aside from how salient or spontaneous causal attributions are, such attempts to explain disease appear in patients independently of their medical knowledge. Mabeck & Olesen
(1997) performed interviews to examine the attributions of patients in general practice. It was found that, regardless of their ability to understand scientific explanations about disease symptoms, patients used a number of metaphors to make sense of the problem indicating a mechanical understanding of the body (ethnomecanics). They thus provided some evidence towards a spontaneous attempt to offer a causal explanation.

2.4.2 **Attributions and Adjustment**

The main interest regarding attributions in illness (or any other negative event) is its potential effects on the individual's psychological well-being. It is believed that making an attribution helps one explain the event, find meaning in its occurrence and maintain a sense of an orderly world (Thomson, 1985; 1991; Thomson & Janigan, 1988, Witenberg, Blanchard, Suls et al, 1983). The following section includes a review of evidence for these effects. However as it will be discussed there, the majority of studies in the field are cross-sectional and can only talk about correlations and not causal relationships.

However, theoretical models regarding the causal relationship between adjustment and attributions have been proposed by Downey, Cohen Silver & Wortman (1990). According to them, there are three possible paths linking attributions and adjustment (Figure 2.3). One is the attribution driven model (model a), which holds that attributions are triggered by an event and, once formed, will determine the levels of distress experienced by the individual. Another model, the distress driven model (model b), claims that an event will elicit distress that in turn will lead to the formation of attributions. Finally, the correlational model (model c) holds that it is an undetermined factor that will be elicited by an event that will trigger both distress and attributions. In this model attributions and adjustment are not causally linked but are allowed to interact with each other. The studies
presented in the next section could support (or not) any of the three models through their cross-sectional design. As it will be demonstrated, longitudinal or experimental studies are needed to test and possibly enhance these models.

Figure 2.3: Theoretical path models of the relationship between attributions and adjustment (distress).

2.4.3 Empirical evidence on illness attributions

The studies on causal attribution for illness have focused on a variety of illnesses, however large proportion of those focus on cancer patients. Explaining the occurrence of cancer is a challenge for the patient: so far the aetiology of most types of cancer has not been established, the symptom onset is typically sudden and unexpected, and the course of the disease cannot be controlled or predicted by the patient (Gotay, 1985). From this point of view, explanations about cancer have been of special interest to attribution research. In a study of terminally ill male cancer patients (including pulmonary, colon, stomach, pancreas, bladder and primary unknown cancer patients), Linn, Linn & Stein (1982) found that compared to their healthy counterparts, cancer patients held weaker
convictions about the cause of the disease. These patients listed 'heredity' and 'God's
will' among the top four causes of cancer. Heredity was more strongly endorsed as a
reason among patients who have known about their disease for a longer time. It was
suggested that the lack of strong beliefs was 'a result of direct experience and a
consequent greater awareness of the complexities of causes that are almost always
involved in the development of cancer' (pp. 83 8). Similar results were yielded by Gotay
(1985) when she studied cancer patients' responses to the question 'Why me?'. Again, a
significant number of patients did not hold a strong explanation for their condition, and
God was the cause cited most frequently.

It has been indicated that people's causal attributions are much more specific when they
suffer from a disease with a better understood aetiology. For example, a study on lung
cancer patients' attributions about their medical condition revealed smoking to be the
most frequently mentioned factor (Faller, Schilling & Lang, 1995). Such a behavioural
attribution also suggested self-blame, thus triggering a cognitive dissonance process that
attempted to negate the involvement of smoking in the aetiology of cancer (e.g.
'Obviously smoking is usually the cause, but it is not really so in my particular case!' pp. 624). When the results of this study were compared to those of studies that have found
a profound lack of causal explanations among cancer patients, it was concluded that the 'I
don't know' response commonly expressed by patients denoted 'a subjective certainty
regarding the causal explanations rather than an absolute state of not knowing' (pp 624).
Therefore, while the frequency of 'I don’t know' answers have led researchers to conclude
that not everyone has an explanation for a negative event, it is likely that this lack of
explanation was due to subjective, biased reactions to the event rather than lack of knowledge.

Recently more attribution studies on specific symptoms or conditions have been reported. Fernandez & Sheffield (1996) studied people’s attributions about headache and found the most frequent explanations to be mental stress and alcohol. In another study, Clemet and Schoennesson (1998) examined the attributions of 57 self-defined gay men with HIV. Most participants had at least one explanation about their condition and the majority provided a combination of attributions (e.g. self- and other-blame) rather than a single explanation for contacting HIV. Finally, Deale, Chalder & Wessely (1998) studied the stability of attributions in patients with Chronic Fatigue Syndrome who participated in cognitive-behaviour therapy or relaxation training. It was found that patients’ causal attributions remained unchanged throughout treatment.

Other medical conditions have also attracted the interest of attribution researchers. For example, a study by Affleck, Tennen, Croog & Levine (1987) focused on interviews of heart attack victims seven weeks and eight years after their initial heart attack. They found that the majority of patients attributed their condition to stress and personal behaviours. Just over half of the sample reported gains and benefits from their experience and these patients were less likely to have a subsequent heart attack and exhibited less morbidity in the following years.
A. Antecedents of causal attributions in illness

The factors that lead to causal attributions for illness have not been studied as much as a) the nature of those attributions (e.g. internal vs. external, spontaneous vs. prompted etc.) and b) the consequences of attributional processes on the illness experience. Michela and Wood (1986) identified reality (i.e. factual information), prior knowledge, and sociodemographic factors as the three major categories of factors that, until then, had been found to affect the formation of illness attributions. However, since their chapter, the literature in the area has grown considerably and various other factors can be identified. For example, Furnham (1994) examined the structure and determinants of people’s explanations regarding a) their current state, b) ability to achieve better health, c) perceived probability of becoming ill, and d) perceived speed/likelihood of recovery. He found that for all four aspects studied, a common underlying structure emerged. Specifically, people explained their (expected) health outcomes in terms of behaviour, environment, medical treatment, life-style and fate/religion. In agreement with Michela and Wood (1986), Furnham also found that certain demographic variables (i.e. sex, education and marital status) consistently predicted health beliefs.

i) Factual information

Illness attributions can be affected by factual information (i.e. specific circumstances under which the illness occurred, information provided by respected ‘authorities’ such as physicians etc). For example, spinal cord injured victims in Bulman and Wortman’s (1977) study were more likely to attribute the accident to someone else when another person was indeed present (e.g. passenger, other driver) at the time of their injury. Similarly, Faller et al (1995) found that the most frequent illness attribution made by lung
cancer patients is smoking, an attribution that reflects medical research findings and widely held societal beliefs.

ii) Prior knowledge

One’s level of sophistication or prior knowledge about the illness may be an important factor. For example, Taylor (1983) found the more naïve the patients are, the more likely they are to employ basic attribution principles (e.g. attributing cancer to a fairly recent event and not taking into account that cancers may take even 15 years to grow).

According to Michela and Wood (1986) lay theories about causality may also be susceptible to folklore or conventional wisdom. The shift to a more patient-centred attitude to health that has marked the last decade may have changed the nature of the above relationships by giving people more knowledge about the factors that cause an illness and reducing the need to use conventional wisdom and folklore.

iii) Characteristics of the individual

*Gender:* The effects of sociodemographic factors on the formation of illness attributions have the focus of some studies. Consistent with experimental studies on achievement-failure attributions, significant differences have been found between men and women in the type and amount of attributions they make for an illness or health-related problem. Despite methodological weaknesses in differentiating between causal attributions, blame and attribution of responsibility (see Chapter 3), studies generally show that women tend to attribute their health problem more to themselves than their male counterparts. For example, Juvonen & Leskinen (1994) found that mothers accepted more responsibility and reported feelings of guilt for their child’s mental retardation more often than fathers.
did. Similarly, Vieyra, Tennen, Affleck, Allen & McCann (1990) and Abbey & Halman (1995) found that women were more likely to attribute their infertility to their behaviour than men were. Other studies supporting similar results have also focused on miscarriage and still birth (McGreal, Evans & Burrows, 1997), and children disabilities (Shapp, Thurman & DuCette, 1992). It must be noted, however, that gender differences are often omitted in studies of illness attributions mainly because the samples under study tend to be gender specific (e.g. breast cancer, heart disease etc.). Therefore, evidence on gender differences is by no means compelling.

Age: Age has also been found to play a role in attributional search for illness. Again, overlooking temporarily the lack of conceptual clarity among the different terms used to signify causal attributions, Heineman, Bulka & Smetak (1988) found that younger people tend to engage in more self-blame than older ones. Similar results were found by Manne & Sandler (1984) in their study of patients with genital herpes. They found that younger participants were more likely to blame their character for their illness than were older participants. In contrast to these studies, lack of a relationship between attributions and age have been reported in studies on parental attributions for a child’s death (Downey, Cohen, Silver & Wortman, 1990) and maternal attributions for severe perinatal complications (Affleck, McGrade, Allen & McQueeny, 1985).

Socio-economic status (SES): SES has also been found to affect illness attributions. Specifically, Affleck, Tennen, Croog & Levine (1987) found that MI patients with higher SES (and age) tended to make significantly more attributions to heredity than those of Lower SES. Lack of a relationship between income and causal attributions has been

*Culture:* The evidence linking culture differences to causal attributions is limited so no conclusive comments can be made. To illustrate the effect of culture on patients’ attributions, Sissons-Joshi (1995) carried out a cross-cultural study to compare beliefs about diabetes in England and India. Structured interviews revealed that more Indians than English reported diet as a cause for their condition and blamed themselves more than their English counterparts. In addition, it was found that more Indians than English did not see causality as central to their illness cognitions and displayed an inability or unwillingness to carry out causal reasoning. Similarly, Kohli and Dalal (1998) studied the attributions of Indian Hindu women diagnosed with cancer. It was found that the majority of attributions focused more on metaphysical beliefs such as fate, God’s will and Karma than to any other factors. In contrast, Hunt et al (1998) studied the illness concepts of Mexican American non-insulin dependent diabetes patients and found that most participants were aware of the biomedically accepted causes of their disease while at the same time tried to link their condition to their personal experience. Also, Downey et al (1990) found that white parents were more likely to make chance attributions about their child’s death than non-white parents. In contrast, Webb et al (1995) reported similar levels of self-blame between white and non-white American participants with traumatic brain injuries.

*Individual Differences:* It has also been suggested that personality affects people’s attributions, and that there is a ‘blaming personality style’ which is associated to poorer
adjustment regardless of who blame is directed towards (Wollert, Heinrich, Wood, Werner, 1983). It has also been proposed that coping (and related attributional processes) is personality in action under stress (Bolger, 1990). While these suggestions have received some support, work on this topic has been fairly limited (Mittelstaedt & Wollert, 1991; Wollert & Rowley, 1987; Anderson, Miller, Riger et al, 1994).

iv) Characteristics of the event

Severity: According to literature reviews on this issue (e.g. Michaela & Wood, 1986), the higher the severity of the event, the higher the tendency to make attributions. A study by Affleck et al (1987a) found that the higher the medically defined severity of participants’ heart attack, the higher the levels of blaming others endorsed by the patients. Also, Gotay (1985) found that patients with more advanced stages of cancer engaged in more self-blame than those in the earlier stages of the disease. Interestingly, Affleck et al (1987b) found that the severity of patients’ heart attack increased all types of attributions (self-blame, other-blame, chance, stress, heredity) supporting the assumption of Weiner’s attributional theory (Wong & Weiner, 1981). Similar findings have been reported when levels of perceived severity have been considered. Specifically, in mothers of children with insulin dependent diabetes, perceived severity of their child’s illness was positively associated with the number of attributions they made (Affleck et al, 1985). A somewhat contradictory finding has been reported by Tennen, Affleck & Gershman (1986). They found that medically defined but not perceived severity was associated with more attributions in mothers of children with severe perinatal complications. Thus, although a relationship between severity and attributions seems to be present, it is not clear enough for general conclusions to be drawn.
Controllability: The degree to which an event is seen as controllable has also been linked to people's attributions. Like severity, it is the individual's own perception of controllability that is important rather than some 'objective' measure of controllability. Event controllability has been considered as a motive for making attributions. Specifically, it has been proposed that certain attributions (e.g. behavioural attributions, self-blame) enhance feelings of future control and people will engage in those even if no culpability is evident to an outside observer (Janoff-Bulman, 1992). Research has looked at relationships among past controllability, attributions, future controllability and adjustment. Brickman, Rabinowitz, Karuza et al (1982) have also proposed a distinction between past and future control or, in other words, a distinction between taking responsibility for a problem versus taking responsibility for the future. The most well supported relationship is that between past controllability and self-blame. Studies have consistently shown that the higher the perceived past controllability of an illness, the higher the levels of behavioural self-blame (e.g. Timko and Janoff-Bulman, 1985; Dalal & Singh, 1992). In contrast, associations between attributions and future controllability have not been as consistent.

While some studies have reported a positive relationship between self-blame and future control of breast cancer (Timko and Bulman, 1985) and perinatal complications (Tennen, Affleck & Gershman, 1986), others did not find any support for such associations (Malcarne et al, 1995; Frazier, 1990; Frazier & Schauben, 1994). Similarly, expected associations between past and future controllability have not been supported by research. Apart from one study with cancer patients (Timko & Janoff-Bulman, 1985), studies with
accident victims (Dalal & Pande, 1988), rape victims (Frazier, 1990) and bereaved
women (Frazier & Schauben, 1994) have not found a correlation between past and future
controllability. Finally, associations between future control and adjustment are
inconsistent as well. The association between perceived future control and adjustment has
been found to be positive (Frazier, 1990; Gotay, 1985; Taylor, Lichtman & Wood, 1984),
negative (Frazier & Schauben, 1994), and non-existent (Malcarne et al., 1995; Timko &

v) Other factors

Sensky (1997) identified a series of biases that may affect the final outcome of the
attribution process. Specifically, more probability will be given to disease explanations
that come more readily to one’s mind. Emphasis should be placed in the role of the media
in rendering certain types of information as easily available to lay people. Furthermore,
the context within which attributions are made can be a potential influence. Clinical
settings for example would probably elicit more pathological attributions than a
laboratory setting. Finally, an already existing psychological state can influence patients’
attributions. Depressed mood has been repeatedly linked with more internal, stable and
global attributions that in turn can affect susceptibility to further illness or delay recovery.

B. Consequences of causal attributions in illness

i) Attributions and adjustment

Adjustment to a negative event has been the most widely studied consequence of
attributions (Harvey & Weary, 1984). The assumptions behind attribution theory hold that
people engage in attributions in order to make sense of the world around them. It is thus likely that once an attribution has been made, the individual is in a better position or state than before the attribution was made. Studies in search for evidence for this relationship have focused on conditions of attributions versus no attributions, on the number of attributions made, and on the type of attributions made (self, others, chance etc). In their review of studies on attributions and adjustment in serious illnesses, Tirnquist, Harvey, & Andersen, (1988) found that in general patients who report explicit attributions fare better than patients who make no attributions at all. While there is a number of studies supporting this conclusion (e.g. Witenberg et al, 1983; Thomson, 1981), several studies have also failed to find supporting evidence. For example, Taylor, Lichtman & Wood (1985) found no association between reporting an attribution and adjustment in breast cancer patients. Similar findings have been reported by Gotev (1985) and Sholomskas, Steil & Plummer (1990).

Regarding the number of attributions made, Taylor (1983) claimed that the greater the number of attributions made the better the individual’s adjustment because, even in the face of evidence that disconfirms one attribution, another attribution is available. However, two studies have reported evidence regarding this relationship and both failed to confirm its existence (Affleck et al, 1985; Tennen, Affleck & Gershman, 1986).

The majority of studies on attributions and adjustment focus on specific attributions. Self-blame as an internal attribution (or self-attribution of responsibility) is discussed in detail in the next chapter as it is the main focus of this thesis. Other attributions have included blaming others, chance or heredity, and environment. Blaming others was the focus of a
thorough literature review by Tennen & Affleck (1990). They found that blaming others was associated with poorer adjustment in 17 out of the 22 studies they reviewed. They also found that an individual’s appraisal of the situation (i.e. whether it is seen as a costly or beneficial event) and his or her own personality characteristics (e.g. attributional style, optimism) may determine the expression of other blame. However, there are some studies that do not support these findings (e.g. Van den Bout et al, 1988; Eiser, Havermans & Eiser, 1995; Hazzard, Weston & Gutteres, 1992; Reidy & Caplan, 1995). Attributions to chance are seen as external, unstable and uncontrollable attributions that have demonstrated no consistent relationship to adjustment for negative events (Michaela & Wood, 1986). Similarly, attributions to the environment have shown a positive (Affleck et al 1985), negative (Gotay, 1985) and non-existent (Bulman & Wortman, 1977; Sholomskas, Steil, Plummer, 1990) relationship to adjustment.

C. Limitations in the literature

A number of methodological and theoretical limitations can be identified in the literature that may account for inconsistent findings among studies on attributions for negative events. These limitations range from small sample sizes and lack of power to lack of agreement on an operational definition for each attribution. A detailed discussion of these is presented in the next chapter, where the concept of self-blame is considered and studies on it are shown to be particularly inconsistent.
2.5 A SYNTHESIS

Based on the above presentation of the relevant theoretical and empirical work, a useful framework for understanding self-blame has emerged. Self-blame in chronic illness can be seen as a type of attribution or, to be more precise, a response that includes attributional elements. Using Leventhal’s model (Leventhal and Nerenz, 1985), self-blame can be placed within the Illness Representation component of a broader network of self-regulatory behaviours, emotions or cognitions that aim towards adjustment in a stressful situation such as chronic illness. As a type of attribution, it can be expected to range along several dimensions (internal-external, stable-unstable, global-specific), result from spontaneous or probed attributional processes, and be affected by the wide range of personal or situational characteristics. The next chapter will look specifically at studies on self-blame in chronic illness and present a comprehensive review of the findings. More importantly, it will show that, although the theoretical approaches described above could have lent themselves as theoretical guides towards clearer conceptualisations in the area of self-blame research, there are a considerable number of theoretical and methodological limitations in the literature, some of which the present thesis will address empirically.
CHAPTER THREE

SELF-BLAME: THEORETICAL, METHODOLOGICAL AND
EMPIRICAL CONSIDERATIONS

SUMMARY

This chapter aims to review the literature on self-blame and provide the background that led to the studies presented in this thesis. It begins by presenting the theory of self-blame (Janoff-Bulman, 1979). It then reviews the relevant literature and identifies those studies directly relevant to the subject matter of the thesis. Furthermore, it demonstrates a number of inconsistencies in research findings and explores the reasons behind these inconsistencies. It identifies methodological and theoretical limitations in the literature, many of which have not been previously considered. Finally, the resolution of these limitations is discussed and the emerging research questions addressed in the thesis are identified.

3.1 THE THEORY OF SELF-BLAME

In 1977, a study by Bulman and Wortman on attributions of blame in accident victims was the beginning of a series of studies on victimization and self-blame. In that study, the researchers interviewed 29 individuals paralyzed in serious accidents and found that blaming others and feelings of avoidability were positively correlated with poor coping ratings by the participants' rehabilitation staff. In contrast, self-blame was a predictor of good coping ratings.
Janoff-Bulman's (1979) theory had significant bearing on the well-founded theories of depression and learned helplessness. Specifically, according to Beck's cognitive model of depression (1967), depressed individuals are characterized by a readiness to interpret events in their life as personal failures and assume responsibility and blame for negative outcomes. In contrast, Seligman's (1975) learned helplessness theory claimed that depression is the result of the erroneous learning that outcomes such as rewards and punishments are uncontrollable. Thus, it appeared that the two theories contradict each other on the issues of responsibility (Abramson & Sackeim, 1977; Peterson, 1979). Do depressed individuals assume responsibility for events they feel they cannot control or not?

Janoff-Bulman's (1979) theory provided a resolution to this paradox by defining two types of self-blame: behavioural self-blame -referring to the *actions* that can lead to an event- and characterological self-blame -referring to the *personality characteristics* that can lead to an event. Behavioural self-blame (BSB) is control-related and focuses on those aspects of behaviour (or omissions of it) that are perceived to have contributed to the outcome. On the other hand, characterological self-blame (CSB) is esteem-related and focuses on the person's personality characteristics or permanent traits.

BSB suggests that, since an event is attributed to one's actions, the future is controllable: a similar future event can be avoided by choosing the right course of action. So, the victim does not feel helpless or hopeless, and adjustment and recovery become easier. For example, it may be very comforting for cancer patients to attribute their misfortune to past eating behaviour or for rape victims to attribute their misfortune to provocative appearance. The chances of the events happening again can be reduced considerably by changing
behaviour and the person feels less vulnerable. This type of self-blame has been linked to better adjustment and increased sense of control in patients and victims of violence. On the other hand, CSB suggests that the factors that led to the unfortunate outcome are permanent and usually unchangeable. In contrast to behavioural self-blamers who use past tense in their statements (e.g. ‘should (not) have done), characterological self-blamers use present tense (e.g. ‘I am...’, ‘I do...’). This type of blame is strongly linked to depression and is regarded as a maladaptive response to a negative event.

Recognising the distinction between the two types of self-blame leads to a resolution of the depression paradox. Characterological self-blamers will attribute failures or unfortunate events to their perceived inadequacies. It is these inadequacies that they view as generally stable and un-modifiable and consequently feel helpless. Thus, the conjunction of self-blame and helplessness need no longer be seen as paradoxical.

3.1.1 The theoretical rationale for the functionality of self-blame

Theorists endorsing the functionality of self-blame follow the motivational explanations of causal attributions (see Chapter 2) and view it as serving three basic needs (Miller & Porter, 1983). The first is the need for control over one’s life. Self-blame serves this need by implying that any harm done was caused by the individual and can be avoided in the future by a change in behaviour. In that way, victims feel in control of their life and their future. The second need served by self-blame is the belief in a fair, just world. According to Lerner (1980) people prefer to accept responsibility for their actions, even when there are no grounds for doing so, than to admit that the world is unfair and thus, unpredictable. ‘A Just World is one in which people ‘get what they deserve.’ The judgement of ‘deserving’ is
based on the outcome that someone is entitled to receive’ (Lerner, 1980, pp.11).

Specifically, the preconditions for deserving (or not) a certain outcome are determined by society. This is based on people’s behaviour and attributes: good behaviour (preparation, precautions, quantity and quality of production in their life) and good attributes (kindness, friendliness, generosity, conscientiousness, intelligence) deserve positive, desirable fates. In contrast, bad behaviour (carelessness, laziness, lack of productivity) and bad attributes (cruelty, ugliness, stupidity, unfriendliness) deserve some degree of punishment or bad fate.

Finally, self-blame serves the need to find meaning in significant events. Being raped or dying of cancer would be harder to tolerate if they were incomprehensible. It seems that being able to answer questions such as ‘why?’ or ‘why me?’ is crucial to coping and recovering from negative events. Self-blame provides an answer to these questions.

In her work on the psychology of trauma, Janoff-Bulman (1992) refined the above theoretical framework and used it as a way to explain most psychological reactions following a traumatic event. She proposed that at the core of one’s assumptive world are abstract beliefs about oneself, the external world, and the relationship between the two.

Specifically, the three fundamental assumptions are that a) the world is benevolent (i.e. the world is a good and safe place), b) the world is meaningful (i.e. there is a relationship between people and the events that happen to them, and the ‘Why?’ questions can be answered), and c) the self is worthy (i.e. people perceive themselves as good, capable and moral individuals). Although not held by everyone, these assumptions are accepted even indirectly by most people and form the bases of more complex philosophical and psychological positions. Like most schemas, the three assumptions are resistant to change, and in the face of disconfirming evidence, can guide behaviour to discount the
contradictory information. According to Janoff-Bulman, a traumatic experience can shatter a victim's fundamental assumptions, forcing them to question the belief in a good and meaningful world, and leave him or her disillusioned trying to preserve or restore as much of their assumptive world as possible. Following this line of thinking self-blame is the victim's attempt to minimise the threatening, meaningless nature of the event.

3.2 **EMPIRICAL EVIDENCE ON SELF-BLAME**

The impact that the theory had on explaining depression as well as its novel view on the adaptiveness of victims' self-blame intrigued many researchers who tested the theory on a variety of populations. However, to date, the studies have yielded inconclusive results even in attempts to replicate the original findings. The following paragraphs provide a comprehensive review of the empirical studies on self-blame.

3.2.1 *Literature search and organization of results*

The main criteria for paper selection during the literature search were fairly relaxed and simple. For electronic searches, the keyword *self-blame* had to appear in the title or abstract of a reference and would only be considered if the work was a paper, chapter, or book, written in English, and focused on adult populations. To identify those references relevant to the issue of self-blame but not bearing the term explicitly (e.g. research on issues of responsibility), follow-up searches for synonymous keywords under the same criteria were performed both electronically and manually.

From the identified references, a relatively small proportion focuses specifically on self-blame, discusses Janoff-Bulman's findings and directly informs on the theoretical and
empirical aspects of relevant research. More frequent is a rather general approach to self-blame, where the main focus is on coping styles of specific populations. Such studies tend to use standardized measures of coping which include self-blame (or acceptance of responsibility) in their structure. The most widely used coping measure that includes a scale for self-blame is the Ways of Coping by Folkman and Lazarus (1980). Used in many of the studies mentioned in this section, this measure includes confrontive coping (i.e. aggressive, hostile or risk-taking efforts to alter the situation), distancing coping (cognitive efforts to detach oneself from and minimize a situation), self-controlling coping (efforts to regulate one's feelings and actions), seeking social support (seeking informational, tangible and emotional support), escape-avoidance coping (wishful thinking and behavioral efforts to escape or avoid the problem), planful problem solving (problem-focused, analytic efforts to alter the situation), and positive reappraisal (creating positive meaning by focusing on personal growth). Self-blame is included in the coping style of accepting responsibility which involves acknowledgement of one's own role in the problem with a concomitant theme of trying to put things right.

Although coping studies provide some information about correlates of self-blame coping, they include little on no discussion of the operational definition or theoretical rational behind the term. Also, they occasionally imply a trait view of coping (i.e. a rather stable pattern of dealing with a problem) which, when applied to self-blame could contradict Janoff-Bulman's classification of behavioural and characterological self-blame.

For the purposes of the present review, studies have been grouped according to the way they define self-blame. Studies not adopting a distinction between behavioural and
characterological self-blame are seen as studying general self-blame (GSB). These include studies on coping. Studies specifically addressing Janoff-Bulman's theory and definitions are presented according to their findings on behavioural and characterological self-blame (BSB and CSB respectively). Table 3.1, presented in section 3.3 for illustration purposes relevant to that section, may be referred to here as a guide for the discussion that follows.

3.2.2 General self-blame

General self-blame has been studied in a variety of populations. In physical illness, it has been associated with younger age (Felton & Revenson, 1987; Blanchard-Fields & Robinson, 1987), problematic psychological adjustment to rheumatoid arthritis (Parker et al, 1988), psychological distress in infertile couples (Morrow, Thoreson & Penney, 1995), reactions to acute coronary syndromes (Pignalberi et al, 1998; Karanci, 1988)), adjustment to irritable bowel syndrome (Ali, Toner, Stuckles et al, 1998) and abortion (Stirtzinger et al, 1999). Also, associations have been reported between self-blame and lower quality of life in patients with inflammatory bowel disease (Moskovitz et al, 2000), greater number of physical symptoms in the elderly (Smith, Patterson and Grant, 1990), and higher anxiety and depression in patients with orthopedic or brain injuries (Curran et al, 2000).

Mentally ill populations have attracted research on the subject as well. The use of self-blame in emotion-focused coping styles has often been associated with depressed or emotionally distressed individuals (e.g. Kleinke, 1984; Weinberg, 1995; Kinderman, 1997; Grossi, 1999; Banazak, 2000) and has even been a central aspect of depression measures such as the Depression Coping Questionnaire (Kleinke, 1988). Also, along with self-isolation and wishful thinking, self-blame has been found to be a common coping strategy
in repatriated prisoners of war with post-traumatic stress disorder (Fairbank, Hansen and Fitterling, 1991) and other clinical samples (Wolfradt & Engelmann, 1999). Finally, a study by McCullough et al (1994) raises an interesting issue regarding potentially different approaches (practical or theoretical) to self-blame one could adopt. The study measured both coping styles and attributions and found that, compared to non depressed controls, dysthymia patients tended to make more external causal attributions but mainly employ wishful thinking and self-blame coping styles. These contradictory findings suggest a need to look more carefully into the theoretical aspects of self-blame and uncover the psychological processes it involves.

3.2.3 Characterological self-blame

The study by Bulman and Wortman (1977) gave rise to a series of studies looking mainly at the relationships between BSB, CSB and adjustment to negative events. However, for a number of methodological and conceptual reasons, discussed later, results verify Janoff-Bulman’s theory in some cases and fail to do so in others.

Of the two types of self-blame, CSB has yielded more consistent findings and has been negatively linked to adjustment. In physical illness, for example, Timko and Bulman (1985) found that characterological attributions were negatively associated with adjustment and perceptions of invulnerability to future recurrence. Similarly, Frazier (1990) and Frazier & Schauben (1994) found that CSB was significantly associated with increased post-rape depression. Also, Malcarne et al (1995) found a negative relationship between CSB and adjustment in cancer patients at four months post diagnosis, suggesting an effect of timing on the aforementioned relationship.
A small number of studies have failed to find any relationship between CSB and adjustment. For example, Gotay (1985) and Houldin et al (1996) found that both CSB and BSB were unrelated to adjustment in cancer patients. Also, Sholomskas, Steil & Plummer (1990) arrived at similar conclusions in their study of spinal-cord injured people. However, Gotay's (1985) and Sholomskas, Steil & Plummer (1990) studies had small sample sizes (n= 73 and n=31 respectively). Given the inconsistent findings regarding the association between CSB and negative outcomes, it may be more appropriate to conclude that there is no positive relationship between CSB and adjustment.

3.2.4 Behavioural self-blame

Behavioural self-blame has been a particularly problematic concept, and studies have yielded contradictory results. In agreement with Bulman and Wortman’s (1977) findings, Timko and Janoff-Bulman, (1985) showed that behavioural attributions of blame were positively associated with adjustment in breast cancer patients. This study was also the first to look at the mediating effects of control and vulnerability on the relationship between self-blame and adjustment. Similarly, Tennen, Affleck and Gershman (1986), in a study of mothers of infants with severe perinatal complications, presented further support for a positive relationship between behavioural self-blame and adjustment. Only partial support for the functionality of self-blame was provided by Taylor, Lichtman & Wood, (1984) who found that self-blame correlated with adjustment between 17 and 36 months after surgery in post-mastectomy patients but not earlier or later than that period.
In non-patient populations, Peterson, Schwartz & Seligman (1981) found that, while general self-blame was systematically related to depressive symptoms in undergraduate women, there are two attributional styles incompatible with depression: a style of attributing bad events externally or a style of attributing them to one's behavior. This indirectly provided support to Janoff-Bulman's theory. Also, Meyer and Taylor (1986) studied victim attributions and post-rape trauma, basing their hypotheses on Janoff-Bulman's theory. They found that although rape victims engaged in self-blame, they did not distinguish between behavioral and characterological self-blame.

However, many studies have found evidence contradicting that presented above. As already mentioned, Gotay (1985) studied 42 early-stage and 31 advanced-stage cancer patients and found that neither characterological nor behavioural self-blame were related to adjustment. In a later study on burn-injured patients, Kiecolt-Glaser and Williams (1987) concluded that behavioural self-blame (displayed by virtually all self-blaming participants in this study to some degree) is maladaptive and is associated with poorer compliance, more pain behaviour and greater depression. In 1988, the theory of adaptiveness of self-blame was challenged once again by Nielson and MacDonald's study of spinal-cord-injured patients. Results showed a strong association between self-blame and poor post-traumatic adjustment. Finally, Houldin, Jacobsen and Lowery (1996) studied the relationship between self-blame in a sample of 234 women diagnosed with stage I or stage II breast cancer. Although results failed to provide evidence for the different effects of characterological self-blame and adjustment, it was shown that more self-blame (of both kinds) was associated with poorer adjustment.
In a non-patient population, Sholomskas, Steil and Plummer (1990) managed to select a sample comparable to that of Bulman and Wortman’s (1977) in all relevant dimensions (i.e. size, diagnosis, time since diagnosis, mean age). Yet, the study failed to replicate the original findings, although it did provide evidence that blaming another for a negative event was associated with poor coping. Similar results were obtained by Frazier (1990), who found that, as with CSB, behavioral self-blame was positively associated with post-rape depression. This study also indicated that participants’ distinction between characterological and behavioral self-blame was not as clear cut as previously assumed.

Of all the studies reviewed above, two studies were identified for attempting to address the limitations of previous research by employing a prospective design. Malcarne et al (1995) studied self-blame and perceived control over disease progression and recurrence as predictors of distress in cancer patients. Patients were assessed near the time of diagnosis and 4 months later. This allowed for stronger conclusions regarding whether self-blame affects or is affected by psychological distress. Results supported the notion that characterological self-blame has negative effects on adjustment but found no evidence of beneficial effects of behavioral self-blame. Building on the work of Malcarne et al, Glinder & Compas (1999) examined the associations between self-blame and distress both cross-sectionally and prospectively, and were the first to study the effects of self-blame on cancer patients from diagnosis to a full year’s follow-up. Results showed that characterological self-blame was a predictor of distress over time (i.e. predicted distress at three months), while behavioural self-blame was a better predictor of distress cross-sectionally (i.e. correlated only to concurrent affective symptoms).
Finally, an interesting development in the self-blame literature was marked by Christensen et al (1999). Rather than focusing once more on the relationship between self-blame and adjustment, their study investigated the effects of behavioural self-blame on behaviour change in head and neck cancer patients. They found that behavioural self-blame was a good predictor of behaviour change (i.e. reduced smoking and alcohol consumption) only among those patients with high perceived control over future health.

3.3 LIMITATIONS IN THE LITERATURE

The previous section showed that results in the studies of self-blame have not been consistent. It has also shown that BSB is the concept that has yielded the most contradictory findings. To illustrate these inconsistencies, Table 3.1 shows a breakdown of the main studies on patient populations according to a) the type and size of sample they used, b) the design used, c) the types of blame investigated, d) the time elapsed since the negative event, e) the measures used to assess adjustment, and f) the results regarding the relationship between self-blame and adjustment (with ‘-’ signifying a negative relationship and ‘+’ signifying a positive relationship).

As can be seen in Table 3.1, only three studies (i.e. Schultz & Decker, 1985, Moulton et al, 1987, and Houldin et al, 1996) have sample sizes larger than 100 participants. All other studies on the table have considerably smaller sample sizes suggesting problems with power. Also, there is great variability in the time that had elapsed since the negative event in these studies. Time variables range from the day of diagnosis/event (Malcarne et al, 1995; Glinder & Compas, 1999) to 21 years later (Schultz & Decker, 1985). Similar variability
can be seen in the selection of adjustment variables and their measures presented in these studies. Specifically, studies define adjustment as combinations of the following: levels of anxiety and depression, mood state, levels of general adjustment to illness, general well-being, marital adjustment, hopelessness levels. The potential effects of such diversities on the validity of the results are discussed in detail in the next section.
Table 3.1: Studies reporting the relationship between self-blame and adjustment in participants dealing with chronic illness.

<table>
<thead>
<tr>
<th>Investigator(s)</th>
<th>Year</th>
<th>Design</th>
<th>Type of sample</th>
<th>Sample size</th>
<th>Mean time since event</th>
<th>Adjustment measures</th>
<th>Types of blame investigated</th>
<th>Relationship to adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Taylor, Lichtman &amp; Wood</td>
<td>1984</td>
<td>Cross-sectional</td>
<td>Breast cancer patients</td>
<td>78</td>
<td>1-60 months</td>
<td>Profile of Mood states, Index of Well-being, Scale of Marital Adjustment, Global Adjustment to Illness, Rosenberg Self-esteem Scale</td>
<td>General self-blame</td>
<td>None</td>
</tr>
<tr>
<td>Timko &amp; Janoff-Bulman</td>
<td>1985</td>
<td>Cross-sectional</td>
<td>Cancer patients</td>
<td>42</td>
<td>10 months</td>
<td>BDI</td>
<td>BSB, CSB</td>
<td>None direct, mediated by perceived invulnerability</td>
</tr>
<tr>
<td>Schultz &amp; Decker</td>
<td>1985</td>
<td>Cross-sectional</td>
<td>Spinal cord injuries</td>
<td>100</td>
<td>21 years</td>
<td>Index of Psychological Well-being, Life Satisfaction Index, Centre for Epidemiological Studies Depression Scale</td>
<td>General blame</td>
<td>Moderate and inconsistent across the three measures</td>
</tr>
<tr>
<td>Gotay</td>
<td>1985</td>
<td>Cross-sectional</td>
<td>Cancer patients</td>
<td>73</td>
<td>1-10 years</td>
<td>Bradburn Affect Balance Scale, Impact of Events Scale, Weissmen Social Adjustment scale</td>
<td>BSB, CSB</td>
<td>None</td>
</tr>
<tr>
<td>Tennen et al.</td>
<td>1986</td>
<td>Cross-sectional</td>
<td>Parents facing peri-natal complications</td>
<td>42</td>
<td>8.5 weeks since discharge</td>
<td>Profile of Mood States</td>
<td>BSB</td>
<td>Positive but indirect</td>
</tr>
<tr>
<td>Moulton et al.</td>
<td>1987</td>
<td>Cross-sectional</td>
<td>AIDS patients</td>
<td>103</td>
<td>Not reported</td>
<td>Profile of Mood states, Taylor Manifest Anxiety Scale, Beck Hopelessness Scale</td>
<td>General self-blame</td>
<td>Negative</td>
</tr>
</tbody>
</table>

Note: CSB= characterological self-blame, BSB=behavioural self-blame
<table>
<thead>
<tr>
<th>Investigator(s)</th>
<th>Year</th>
<th>Design</th>
<th>Type of sample</th>
<th>Sample size</th>
<th>Mean time since event</th>
<th>Adjustment measures</th>
<th>Types of blame investigated</th>
<th>Relationship to adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kiescolt-Glaser &amp; Williams</td>
<td>1987</td>
<td>Cross-sectional</td>
<td>Burn patients</td>
<td>49</td>
<td>1-30 days after admission</td>
<td>Brief Symptom Inventory</td>
<td>BSB/CSB</td>
<td>Positive with BSB</td>
</tr>
<tr>
<td>Nielson &amp; McDonald</td>
<td>1988</td>
<td>Cross-sectional</td>
<td>Spinal-Cord Injury patients</td>
<td>58</td>
<td>-</td>
<td>Millon Multiple Affect Scale, Beck Depression Inventory, Social Support Measures</td>
<td>General self-blame</td>
<td>negative</td>
</tr>
<tr>
<td>Sholomskas, Steil &amp; Plummer</td>
<td>1990</td>
<td>Cross-sectional</td>
<td>Spinal-Cord Injury patients</td>
<td>31</td>
<td>-</td>
<td>Hospital staff assessment of patients</td>
<td>BSB, CSB</td>
<td>None</td>
</tr>
<tr>
<td>Frazier &amp; Schauben</td>
<td>1994</td>
<td>Cross-sectional</td>
<td>Victims of rape, bereavement and relationship loss</td>
<td>59 (rape) 57 (bereaved) 55 (rel.loss)</td>
<td>7 ys (rape) 6 ys (bereav.) 4 ys (rel.loss)</td>
<td>Brief Symptom Inventory, McPhear Belief Scale</td>
<td>BSB, CSB</td>
<td>negative with both BSB and CSB</td>
</tr>
<tr>
<td>Malcarne et al</td>
<td>1995</td>
<td>Prospective</td>
<td>Cancer patients</td>
<td>72</td>
<td>At diagnosis and 4 mths later</td>
<td>Brief Symptom Inventory</td>
<td>BSB, CSB</td>
<td>at diagnosis: none at 4 months: negative with CSB</td>
</tr>
<tr>
<td>Holdin et al</td>
<td>1996</td>
<td>Cross-sectional</td>
<td>Breast cancer patients</td>
<td>234</td>
<td>-</td>
<td>Psychosocial Adjustment to Illness and Global adjustment to Illness Scales</td>
<td>BSB, CSB</td>
<td>None</td>
</tr>
<tr>
<td>Glinder &amp; Compass</td>
<td>1999</td>
<td>Prospective</td>
<td>Breast cancer patients</td>
<td>76</td>
<td>At diagnosis and then 3 mths, 6 mths &amp; 1y later</td>
<td>Symptom Checklist - 90</td>
<td>BSB, CSB</td>
<td>Negative with both BSB and CSB</td>
</tr>
<tr>
<td>Christensen et al</td>
<td>1999</td>
<td>Cross-sectional</td>
<td>Head/neck cancer patients</td>
<td>66</td>
<td>6.8 mths</td>
<td>Behaviour change (smoking, drinking)</td>
<td>BSB</td>
<td>BSB x control predicted behaviour change</td>
</tr>
</tbody>
</table>

Note: CSB= characterological self-blame, BSB=behavioural self-blame
3.3.1 Methodological explanations for the inconsistent results.

Inconsistencies regarding the adaptiveness of patient self-blame can be attributed to the following methodological problems.

a) Operational definitions and measurement.

The construct of self-blame has been operationalised differently by different studies. For example, while Tennen, Affleck and Gershman (1986) aim to study self-blame among parents of infants with perinatal complications (and indeed give this title to their paper), they ask their participants: ‘Today, do you think that something you (or someone else) did or did not do might have been, at least in part, responsible for your infant’s medical problems or immaturity?’ (pp. 692). They then asked the mothers to rate on 10-point scales, ranging from \( I = \text{not at all responsible} \) to \( 10 = \text{totally responsible} \), the extent to which they believed the problems were due to themselves, others or chance. In contrast, Frazier (1990) assessed the rape victims’ characterological and behavioural self-blame by asking directly, ‘How much do you blame things you did before the rape?’ or ‘How much do you blame things about your personality that you feel you can’t change?’ (pp. 300). As in these examples, in many studies on self-blame it is not clear from the beginning whether self-blame refers to ‘self-attribution of causality’ or ‘self-attribution of responsibility’ or a different term altogether.

Differentiating between causality, responsibility and blame in research and application has been viewed as an important issue since it may affect the construct validity of ‘self-blame’ (Shaver & Drown, 1986). Using the terms interchangeably reduces clarity and, thus, contributes to a vague theory that lacks consistency and predictive value. These distinctions
are discussed in Shaver’s (1985) model of blame attribution. Based on the philosophy of science and moral philosophy that discuss causation and responsibility respectively, she concludes that judgements of responsibility and blame tend to follow the occurrence of negative rather than positive events. In this line of reason, the cause of an event is that behaviour or situation that is sufficient for the occurrence of the event. Causality is dichotomous since a cause either exists or not. In contrast, responsibility characterises or even labels a process, can have degrees (high/low, minimum/maximum), and is thus variable. Responsibility usually connotes the perceiver’s judgement about a person in question and may be direct or indirect (i.e. vicarious responsibility). Finally, blame is the attribution made by the perceiver when the offender’s justification for an action is unacceptable.

While Shaver’s model refers mainly to situations where the perceiver is different from the actor, it can also be applied when the same person is both the actor and the perceiver. For example, when a medical problem is considered from the point of view of the patient the distinction among the three terms may apply: the past actions or omissions of a patient can cause the medical problem, and the degree of the patient’s negligence can be the ground of accepting responsibility. According to Shaver & Drown (1986), ‘a victim cannot be objectively blameworthy for the occurrence of a crime or an illness, unless the victim intentionally behaved in a manner to produce the suffering’ (p.704). In other words, the authors object to the use of the term ‘(self-) blame’, unless intention to suffer is involved.

Although a literary analysis of the term may support the author’s views, it is also important to examine the participant’s understanding of the term. Being able to differentiate between
responsibility, blame and cause does not imply that when presented with each term *alone* participants have its literal meaning in mind. For example, asking patients with a history of an unhealthy lifestyle whether they *blame* themselves for their current illness may not be inappropriate if, in retrospect, ‘not doing the right thing’ seems to them just as culpable as an intentionally harmful act.

Moreover, Shaver & Drown (1986) do not offer a comprehensive literature review. Indeed, there is contradictory evidence that participants in fact cannot differentiate among the three terms of responsibility, causality and blame (e.g. Wortman, 1983; Sholomskas, Steil & Plummer, 1990; Drown, 1985). Perhaps a more promising view was incorporated in Shaver & Drown’s comment that ‘the manner in which blame is inaccurately applied by victims may prove valuable in understanding their emotional adjustment. [Self-blame] is the outcome of an intensely personal dispute’ and ‘may carry the affective connotations that can lead to depression’ (p.701).

To follow the above point, Brewin, Robson & Shapiro (1983) demonstrated that it is possible to make causal attributions to self or others without blame. In a study of male victims of industrial accidents, participants’ causal attributions (including self-attributions) were measured separately from blame assessments. Measures of blame and culpability focused on the moral evaluations of participants and included items relating to negligence and just cause. In contrast, causal attributions focused only on the cause–effect relationship between actors and events. Results showed that men who felt more culpable for their accidents had faster recoveries while men who only made self-attributions did not have
consistent recovery rates. The moral component of self-blame has also been supported by studies on non-patient populations (e.g. Kelly, 1998).

In any case, a clear and realistic operational definition of self-blame and, consequently, a systematic investigation of participants understanding of it is necessary for a study to be well-founded, complete and comparable to others.

A similar lack of coherence in the supposed effects of self-blame is evident in the measurement of outcome variables in self-blame studies. Measures of adjustment have included measures of coping (e.g. Tennen et al, 1984), mood state (e.g. Tennen et al, 1986), distress (e.g. Malcarne et al, 1995), depression (Timko & Janoff-Bulman, 1985), general measures of adjustment to illness (e.g. Houldin, Jacobsen & Lowery, 1996), or a combination of the above (e.g. Moulton, Sweet, Temoshok & Mandel, 1987). Finally, behaviour change was investigated as an outcome measure in patients with head and neck cancer (Christensen et al, 1999). This 'polyphony' of constructs has been coupled by a similar polyphony in measures within each construct, covering a wide range of methodologies from standardized and unstandardised questionnaires, surveys and observer ratings (e.g. most of studies mentioned above) to qualitative data (e.g. Anderson, 1999). In addition, some studies base their findings on qualitative data. As a result, comparison and integration of the findings, let alone a meta-analysis of them, becomes a difficult task.
b) Statistical power

As mentioned in the previous section, a large proportion of studies on self-blame (as well as on other specific attributions) employ small samples for their analyses. This has an impact on the statistical power of a study (Robins, 1988). Power is the probability of rejecting a false null hypothesis. It is a function of the significance criterion (typically $p<.005$), effect size and sample size (Cohen, 1988). Power calculations should ideally be carried out prior to data collection. As suggested by Hall (2000), to have 80% power with $p<.005$ significance criterion when a medium effect size is expected, a sample size of 64 is needed for a correlation analysis and 102 for a t-test. Effect sizes in psychology tend to be medium to small (Cooper & Findley, 1982) and effects of self-blame on adjustment or behaviour change should not be an exception. Power and effect sizes are not discussed in self-blame studies and there is a possibility that even significant results are a result of Type 1 error.

c) Timing

Another aspect relevant to the inconsistencies in the self-blame literature is the timing of the measurement. As with the study of other attributions (e.g. Agrawal & Dalal, 1993; Major, Mueller & Hildebrandt, 1985; Dirksen, 1995), the time interval between participants' experience and self-blame measurements has varied considerably among the different studies. For example, ranging from a few days after the diagnosis (e.g Moulton et al, 1987) to several years (e.g. Timko & Janoff-Bulman, 1985), the effect of elapsed time on perceptions of responsibility or blame has not been accounted for. When a negative experience is recent and 'vivid' in one's memory, it is likely to be charged with powerful emotions that affect cognition. Reacting to statements about the event will only reflect the individual's present state which in many cases may not be permanent. On the other hand, if
too much time has passed since the negative event, the individual may have completely overcome the experience, large parts of which may have even been forgotten. Comparing data obtained at such potentially different circumstances can be a misleading task and should be avoided.

Timing issues are also involved in the design various studies have used. The majority of them are cross-sectional (see Table 3.1), making data susceptible to time of measurement effects. For example, being in the hospital (whether as an in-patient or an out-patient), waiting to see the doctor or having just come out of a possibly stressful follow-up session may affect the sample’s responses. Conducting time series studies to control for time of measurement effects is one way around the problem. However, even more preferable are prospective studies, which have lately been utilised with very promising results (e.g. Glinder & Compas, 1999; Richards, Elliot, Shewchuck & Fine, 1997). The latter research design, not only allows for the control of timing effects, it also resolves problems of potential reciprocal relations. Indeed, by employing a prospective design in their study, Malcarne et al (1995) found that ‘initial distress predicts increases in later self-blame, just as initial self-blame predicts increases in later distress’ (pp.414). Unfortunately, only a small number of studies on self-blame have used a prospective design possibly due to time and cost considerations.

d) Sample diversity

By focusing on Janoff-Bulman’s initial observations of better adjusted self-blaming victims (namely severe accident and rape victims), a chain-effect was initiated whereby subsequent studies set out to explore that same observation hoping to provide critical evidence for or
against it. However, the theory was unquestionably expanded to other populations without ensuring the applicability of the construct and the logic behind it. Participants such as cancer patients (e.g. Malcane et al, 1995), AIDS patients (e.g. Moulton et al, 1987) or heart disease patients (Affleck, Tennen, Croog & Levine, 1987), possibly have different experiences when compared to each other as well as to rape or severe accident victims. As Forsythe and Compas (1987) have shown, the effectiveness of different coping strategies or attributions varies from one event to another and often depends on the controllability of the events. Frazier and Schauben (1994) have provided some evidence towards the generalisability of the relationship between self-blame and recovery from negative events. They performed their study on college students who had suffered one of three negative events (i.e. rape, bereavement, and relationship loss). Results showed that bereavement was seen as less controllable than relationship loss and involved less behavioural and characterological self-blame. Also, in all three groups, both types of self-blame were related to poor adjustment. Although this study acknowledges the need to systematically test for generalisability across different populations, it leaves room for some criticism. First, comparisons for differences in responses were only performed between two of the three groups (i.e. bereavement and relationship loss). There was no comparison of these groups with that of rape victims. Consequently, one cannot safely say whether rape –the event originally involved in the formulation of the theory- is in any way similar to bereavement or relationship loss. Finally, it is important that the similarities and or differences of negative events be studied systematically on dimensions that are relevant not only theoretically but also empirically based on lay people's own representations of negative events.
3.3.2 Theoretical limitations in self-blame research

Apart from the methodological weaknesses of the research on self-blame a number of theoretical limitations also exist. These limitations can be divided into two categories: a) those regarding the purpose or reasons for self-blame and b) those regarding its relation to adjustment. This section reviews these limitations beginning in both instances with those recently reported in the literature.

a) Lack of theoretical scrutiny

A large number of the studies mentioned earlier focus mainly on coping strategies as a collection of spontaneous behaviours employed in order to achieve psychological adjustment. Consequently a general theoretical perspective is adopted (e.g. Lazarus’ model of problem vs. emotion focus coping), through which self-blame is seen as one of many different reactions to a stressful situation, and discussion typically revolves around more general ideas of coping motivation and effectiveness. However, Janoff-Bulman (1992) had a slightly different approach when describing self-blame. The term was placed in the context of trauma reinterpretation and seen as a cognitive strategy that ultimately contributes to the process of emotional recovery. Using the coping concepts of primary and secondary appraisal (and not coping strategies) proposed by Lazarus (1966), Janoff-Bulman (1992) claims that “a third type of appraisal process becomes very evident in the case of traumatic events. These are not appraisals that occur during the initial confrontation with the traumatic situation, but rather interpretations and redefinitions of the event that occur over the course of coping and adjustment” (pp. 116). Thus, it is obvious that the conceptualization of self-blame depends largely on the level of theoretical scrutiny that researchers apply when formulating their research questions. It is possible that if Janoff-Bulman’s findings had not
been surprising enough to spur a series of studies and eventually create the theoretical and methodological challenges that they have, self-blame would have remained a concept tied to the ideas of coping strategies and seen as more of an emotional reaction (similar to guilt) and less of a cognitive process related to attributions.

\[ b) \text{Alternative reasons for self-blame} \]

One factor not previously considered in the formulation and application of the self-blame theory is the level of consistency between the actual risk factors involved in the patient's condition and his or her causal attributions for the event. Self-blame may be the result of identifying those behavioural or personality factors in the patient's life that are believed to be related to a given chronic condition and attributing the condition to them. For example, heart disease patients who believe their heart attack is directly related to their smoking or dietary behaviour or their stress-prone personality may blame themselves for their illness. Similarly, a breast cancer patient may blame herself for the seriousness of her condition because of her neglect to have regular breast cancer screening. In other words, it is possible that in some studies self-blame reflects the participants' causal attributions and not a need to regain control.

To take the above point further, self-blame (or lack of it) may be affected by the social environment within which physical illness is experienced. With the links between disease and lifestyle being emphasised by the media and health campaigns targeting unhealthy behaviours, there is an increasing awareness of the role of the individual in maintaining health. Psychological literature has repeatedly addressed the impact of this awareness on
people's health cognitions (e.g. Blaxter, 1993; Lupton, 1994). As Bradley (1994) has written:

The perceived failure of medicine to remove [the chronic] diseases, and the consequent demand placed upon health services to care for the chronically sick, provide the backdrop to the call for the individual to be responsible for improving the health of the nation [...] Control of conditions like cancer and heart disease depends upon modification of the individual's behaviour and habit of living...(pp.198).

It is, thus, possible that patient self-blame is a reflection of a more widely held notion of individual responsibility for health. To the best of our knowledge, this parameter has not been empirically investigated.

c) Self-blame and adjustment.

MacLeod (1999) revisited the assumptions behind Janoff-Bulman's model and presented an alternative interpretation 'of why attributions appear to mediate psychological adjustment' (ppl9). The first assumption underlying Janoff-Bulman's model is that victims of misfortune are likely to engage in an attributional search in order to make sense of the event. However, this is not always the case. As Gotev (1985) found in her study of cancer patients, a large proportion of the sample (24%) had not looked for a reason for their situation. Moreover, Taylor, Lichtman & Wood (1984) found little difference in recovery between breast cancer patients who engage in causal thinking and those who don't. While such contradictions may be due to differences in timing considerations (i.e. patients may be preoccupied with different aspects of a disease at different points in time), it is also possible that attributional thinking (and behavioural self-blame in particular) is unrelated to psychological adjustment (MacLeod, 1999).
A second aspect of the self-blame theory criticised by MacLeod is the role of perceived control as a mediator between self-blame and adjustment. He argues that past evidence for this relationship is considerably weak and, despite some useful suggestions for a distinction between control over outcomes and control over recurrence (Tennen, Affleck, & Gerslman, 1986; Malcarne, Compas, Epping-Jordan and Howell, 1995), any attempts for a re-appraisal remain inadequate.

Finally, McLeod suggested that the possible inconsistencies in the literature may be due to unidentified yet better moderators or predictors of the relationship between perceived control and adjustment. Specifically, it is stressed that results may be affected by how controllable the incident really is (i.e. the objective controllability of the incident rather than its comparative controllability). For example, while a relationship breakdown seems more controllable than bereavement, it can nevertheless be an event of very low control. Moreover, in low control events, it may be more important to consider the perceived likelihood of recurrence rather than perceived control per se.

Inconsistent results regarding the adjustive function of behavioural self-blame may also be due to a possible interaction between behavioural and characterological self-blame. The importance of this relationship has been stressed by Janoff-Bulman (1992) herself. Specifically, she cautioned that ‘the effects of characterological self-blame should be partialled out of correlations between behavioral self-blame and coping outcomes. This has not been done in research studies, and thus the adaptive value of behavioral self-blame is apt to be missed or seriously underestimated’ (pp. 200). Unfortunately, this clarification
came somewhat late in the debate and few studies have tested and/or controlled for this interaction (e.g. Malcarne et al, 1995).

Finally, inconsistent findings may also be due to a reciprocal relation between self-blame and outcome variables. Specifically, it is important to examine whether self-blame and adjustment are part of a perpetuating emotional cycle rather than two separate states in a cause and effect relationship. If, for example, self-blame (whether behavioural or characterological) is a result of a causal search that took place during a period of significant experienced anxiety or depression (e.g. soon after a negative event), it is likely that it may contribute to more anxiety or depression which may, in turn, strengthen self-blame and so on, explaining the studies showing a negative correlation between adjustment and self-blame. Similarly, a positive relationship might be explained by successful coping already in use at the time of the attributional search. In this case, the mere availability of an explanation (even as self-implicating as self-blame) may promote emotional well-being. As already noted, timing considerations might be of importance here as well. The amount of time elapsed since the event might determine the presence or absence of successful coping and thus, the positive or negative nature of the reciprocal relationship.

3.4 IDENTIFICATION OF RESEARCH QUESTIONS

Research on self-blame needs to address the weaknesses of past studies and work towards revising the theory to account for specific populations. The present thesis attempts to
address some of the issues mentioned above and provide evidence for an alternative conceptualization of self-blame.

\textit{a) Sample diversity}

The issue of sample diversity is addressed in two ways in this thesis. First, a comparative approach is used and the degree to which levels of self-blame differ across populations is investigated. Levels of self-blame and its relationship to adjustment are compared among heart disease, breast cancer and diabetes patients. The reasons for detected differences are explored. This issue is also addressed by looking at the illness representations of non-patients and exploring the place that various illnesses—compared to other negative events—hold in the lay representations of negative events. Furthermore, a comparison of levels of self-blame between patient and non-patient samples matched on all relevant variables is performed to explore the role that the actual experience can have on the levels of self-blame.

\textit{b) Reasons for self-blame}

The role of self-blame and the reasons for its occurrence are addressed in two ways in the thesis. Specifically, the relationship between self-blame and causal attributions is investigated and the possibility that self-blame is a result of ‘reality based’ attributions is examined. This issue is addressed rather indirectly at first by relating the number of behavioural attributions patients made to their levels of self-blame. Later in the thesis, a model looking at predictors of self-blame is proposed and tested.
c) Timing

Ideally, the effect of the time elapsed since diagnosis should be investigated longitudinally. However, the studies presented in this thesis adopted cross-sectional designs for both methodological reasons relating to other variables and practical considerations. A good cross-section of data on time elapsed since diagnosis was available in all studies involving patient samples. Timing information was repeatedly examined in relation to self-blame and adjustment in all relevant studies.

d) Self-blame and adjustment

The relationship between self-blame and adjustment was investigated in both an exploratory and a model testing approach in separate samples. Adjustment was assessed through measures of depression and anxiety. To incorporate the recent findings by Christensen et al (1999) a measure of health behaviour change was also incorporated in the third study reported in the thesis.

e) Operationalization and measurement of self-blame

Shaver and Drown's (1986) proposal for a distinction between responsibility, causality and blame is addressed in the last study where participants' responses to a series of items differing in the use of these three terms are compared. Also, the need for a multi-item measure is briefly addressed in the first study. The first steps towards the design of a scale sensitive to the different types of self-blame that takes into account the potential differences between causality, responsibility and blame is presented.
To summarise, the following general research questions are investigated in this thesis:

a) Does self-blame differ across different patient populations? If yes, in what way?

b) What are the personal and situational characteristics, if any, that predict behavioural self-blame?

c) Does the time elapsed since diagnosis affect patient self-blame?

d) Is there a relationship between self-blame and adjustment or behaviour change?

e) How do people understand the concept of blame in illness? Do they differentiate between similar terms such as blame and responsibility or control and avoidability?
CHAPTER FOUR

MEASURING SELF-BLAME IN CHRONICALLY ILL PATIENTS: A PILOT STUDY

SUMMARY

This study aimed to explore self-blame in chronic illness by comparing responses in three groups of chronically ill patients (i.e. heart disease, breast cancer and diabetes patients). In the first part of the study, the preliminary work before the main data collection is presented. Specifically, the generation of items towards the construction of a self-blame measure is presented, which was deemed necessary after identifying the predominantly single-item measures used in past research in the area. Following item generation, the measure was piloted within a patient population to identify potential problems with item clarity and comprehensiveness. The final set of items was then identified and used in the next phase of the study where its psychometric properties were assessed on a larger sample before entered in further analyses. In the second phase, relationships between self-blame and other illness attributions, self-efficacy, psychological adjustment (i.e. anxiety and depression), and diet and exercise health behaviours were examined. Furthermore, the effects of the level of event specificity (i.e. general condition vs. specific negative event relevant to illness) was examined. Results showed significant differences across illness groups and between general and specific self-blame. Also, the number of behavioural risk factors patients associated with their illness correlated highly with the levels self-blame. Diet and exercise levels were negatively correlated with self-blame in the diabetes group. No relationship was found between self-blame and self-efficacy or psychological adjustment. The strengths and limitations of the study are discussed and suggestions for further research are made.
4.1 INTRODUCTION

Self-blame has been the focus of attribution research for the last 25 years. Janoff-Bulman and Wortman's (1977) study on self-blame and coping in severe accident victims is considered the cornerstone for this line of research. In that study, it was found that, contrary to other findings, self-blame was a predictor of good coping, suggesting the functionality of self-blame. Various possible explanations for this relationship were proposed including control over one's life (Wortman, 1976), preservation of the essential belief in a 'just world' (Lerner, 1980), and need to find meaning in significant events (Silver & Wortman, 1980). Whatever the explanation, self-blame was seen as arising from a need to minimise the uncertainty created after a negative event.

The majority of studies on self-blame have focused on victims of rape and severe accidents having led to spinal cord injuries (e.g. Nielson & MacDonald, 1988; Sholomskas, Steel & Plummer, 1990; Schultz & Decker, 1985; Meyer & Taylor, 1986; Janoff-Bulman, 1982). In physical illness, interest on self-blame is due mainly to its possible implication on psychological adjustment and recovery from physical illness. However, studies have been inconclusive on the existence and nature of the above relationship. With findings suggesting that the association between self-blame is positive (e.g. Bulman & Wortman, 1977), negative (e.g. Malcarne et al, 1995) or non-existent (e.g. Taylor et al, 1984), it has been difficult to come to a conclusion about the effects of self-blaming behaviour on adjustment or recovery.

The diversity in findings has been attributed to a number of reasons. First, the samples used have differed in a number of dimensions such as type of disease or injury, mean age,
and time elapsed since the negative event (Gotay, 1985; Kiecolt-Glaser & Williams, 1987; Schultz & Decker, 1985). Second, the means of measuring psychological adjustment varied considerably, ranging from unstandardised observer ratings (e.g. nurses) to self-ratings of coping, anxiety or depression (Bulman & Wortman, 1977; Sholomskas, 1986). Finally, there has been great variety in the operationalisation and measurement of self-blame (Shaver & Down, 1986).

Regarding the latter issue, the most commonly used method of assessing self-blame has been by directly asking participants to rate their attributions of general, BSB and CSB on Likert scales. The single items used in the majority of research have been variations on the questions of a structured interview originally used by Janoff-Bulman (1979). Specifically, in Janoff-Bulman’s original research, general self-blame was assessed by asking participants to respond to the question ‘How much do you blame yourself for what happened?’ on a 7-point Likert scale. Responses were then converted into percentages and were considered in conjunction with percentage of blame assigned to four factors: self, other people, environment, and chance. CSB and BSB and were assessed by asking ‘If you assigned blame to yourself, how much would it be to the type of person you are, and how much to choosing the wrong thing in the situation?’. Responses were given on a 10-point scale with 1 = ‘I am the type of person who has bad things happen to them’ and 10 = ‘I chose the wrong thing to do in this particular situation’. In addition to these aspects of self-blame, perceived avoidability was assessed by asking ‘To what extent do you believe you could have avoided what happened?’ rated on a 7-point scale with 1 = not at all and 7 = completely.
A number of limitations can be identified in the above measures. First, the four factors on which blame is assigned are limiting and not necessarily mutually exclusive. For example, people may view environmental factors as affected by chance, or others as part of their environment. Assigning percentages to these factors is not informative enough, unless used in association with a qualitative framework such as interviews where there is the possibility of probing. Second, BSB and CSB are assessed by the same item and are treated as a single dimension. Hence, a value on the relevant 10-point scale can only show a tendency towards one type of self-blame rather than the other and limits the type of analyses that could possibly be performed if the two were measured as separate dimensions (e.g. correlations between BSB and CSB). Third, these items were constructed by the investigator instead of consulting the target population to discover the ways they described self-blame.

Subsequent research has been substantially influenced by Janoff-Bulman’s methodology and has used these items or modified versions. For example, Frazier (1990) used modified items on BSB and CSB asking participants ‘How much do you blame things you did before the rape (e.g. walking alone at night)?’ and ‘How much do you blame things in your personality (e.g. being too trusting) that you feel you can’t change?’. Both items were scored on a 5-point scale. While these items measure BSB and CSB as separate dimensions, they are only single item measures of these constructs.

Apart from the methodological problems with self-blame measures, several conceptual issues discussed in the previous chapter have been raised by a number of researchers (e.g. Shaver & Drown, 1986). These emphasise the richness of the construct and the need for a multi-item measure that will allow a consistent and systematic approach.
Sholomskas, Steil & Plummer (1990) considered all the above inconsistencies and designed a study specifically to replicate Bulman & Wortman's 'original self-blame, other-blame and coping relationship on a comparable sample and event, using both the original instruments and ones which could address the issues reviewed' (pp.552). They found that other-blame to be a better predictor of poor coping than self-blame. Relevant to these issues is a recent systematic literature review on the relationship between attributions and adjustment to serious illness by Hall (2000). She found most attributions to be unrelated to psychological outcomes, attributing the confusion regarding this relationship to small, under-powered studies. It must be noted, however, that causal attributions lack the evaluative component present in self-blame (Brewin 1988) and therefore should not be considered a typical example of self-blame. Also, the above study focused only on the relationships between outcome, types of attributions, and sample size. It did not take into account the measurement of attributions and outcomes nor did it differentiate among the various types of illness (acute, chronic, accidents etc.).

A possible explanation for the above inconsistencies is that the nature of self-blame may vary from one situation to the other. It may be the case that the underlying psychological processes that can lead to self-blame are different for victims depending on the nature of the adverse event (e.g. rape versus accident versus chronic illness). Similarly, the same person may engage in self-blame for some aspects of the negative experience and not others, thus finding it easier to assess their role in more specific situations (e.g. high cholesterol indices in a blood test) occurring after and within the scope of a diagnosis. Although to the best knowledge of the present researcher comparative studies on self-
blame have not been reported, indications of differences in self-blame across situations can be found when comparing relevant research findings. For instance, levels of self-blame in cancer patients appear to be lower than in victims of violence. To illustrate, in Malcarne et al (1995), mean self-blame of cancer patients was 1.90 (SD=1.01) for behavioural (BSB) and 1.56 (SD=0.98) for characterological self-blame (CSB) measured on a 5-point Likert scale (where 5= high self-blame). On the other hand, Frazier (1990) found mean self-blame of rape victims to be 2.64 (SD=10.45) for BSB and 2.62 (SD=1.38) for CSB on the same 5-point scale. Although these differences may be due to different study designs, they indicate that the situation or condition may be a determinant of self-blame. Hence, further research is needed to evaluate this possibility. As an implication of this, it is worth exploring the effect that specificity of reference may have on levels of self-blame. Apart from looking at the overall situation and making comparisons across conditions, it is important to examine whether referring to an illness as a whole would yield different levels of self-blame than referring to a specific event within the illness (e.g. bad test results or a specific episode of health deterioration).

The discrepant results in prior studies on self-blame have also been attributed to the role of attributions and perceived control over the cause of the illness (Tennen et al, 1986). Specifically, it has been suggested that the strength and direction of the relationship between adjustment and self-blame is mediated by perceived control over the perceived causes of an event. Various forms of perceived control have been studied including locus of control (Taylor, Lichtman & Wood, 1984) and perceived avoidability (Schulz & Decker, 1985; Davis, Lehman, Silver et al, 1996). Another concept closely related to issues of control is self-efficacy. In an experimental study of the relationship between
self-blame, coping self-efficacy and abortion, Mueller and Major (1989) found that self-efficacy was moderately related to self-blame and predicted adjustment 3 weeks post-abortion. However, this study employed an experimental design using counselling interventions. The degree to which these results generalise to different medical conditions needs further exploration.

Finally, it has been suggested that variables other than psychological adjustment may be affected by self-blame. Specifically, Christenssen et al (1999) stress the importance of investigating the relationship between self-blame and health behaviours. Although their study provides some evidence of such a relationship (i.e. more behavioural self-blame was associated with less smoking or drinking), it is based on a small sample (N=66) and focuses only on one patient group (head/neck cancer patients) and their related health behaviours (i.e. smoking and drinking).

The present study investigated self-blame across different chronic conditions. To address the shortcomings of previous measures, a multi-item measure of self-blame was developed in consultation with members of the public and was administered to a sample of patients with diabetes or heart disease. The first step towards this goal included the generation of the appropriate items.

Once a multi-item self-blame scale was designed, levels of self-blame were compared among diabetes, heart disease and breast cancer patients. These particular groups of patients were chosen for two reasons. First, cancer, diabetes and heart disease are three of the most common chronic illnesses today. According to the national statistics provided by
the Department of Health (DOH, 2000) out of 971 male deaths per 100,000 registered in the year 2000, 378 were due to a circulatory condition with 227 specifically due to ischaemic heart disease. Cancer was the second most common cause of death accounting for 260 deaths. Female deaths showed a similar pattern, with 397 out of the 1043 deaths per 100,000 due to circulatory conditions and 236 due to cancer. Detailed tables of these statistics are given in Appendix I. Although diabetes does not appear to claim as many lives as cancer and heart disease, it is one of the most common medical conditions in the UK with over 1.4 million people already diagnosed and an estimated 1 million remaining unidentified (Diabetes UK, 2000). It can also lead to mortality through circulatory problems which have already been established as a leading cause of death above. All three conditions are therefore serious enough to warrant investigation. Another reason for choosing these conditions is that they carry different degrees of lifestyle involvement and are thus ideal for exploring the relationship between the behavioural attributions and self-blame. Specifically, both heart disease and diabetes have been linked to a number of behavioural risk factors such as diet and exercise. In contrast, breast cancer has been more certainly linked to uncontrollable factors such as heredity and age and less so to health behaviours such as diet, exercise and smoking.

Thus, looking at these serious and comparatively diverse medical conditions, the present study explored the relationship between self-blame and measures of psychological adjustment (anxiety and depression), self-efficacy, behavioural attributions and self-care activities (diet and exercise) through comparisons among the patient groups.
In order to investigate the effect that specificity of reference can have on reported levels of self-blame, self-blame was measured twice in this study: once with reference to the participants' condition in general, and then regarding a specific recent negative event relevant to their condition (e.g. alarming symptoms).

The research questions addressed were the following:

a) Are there differences in levels of self-blame across different medical conditions?

b) Does the specificity of reference (i.e. referring to specific rather than general events) affect the levels of self-blame reported by participants?

c) What is the relationship between self-blame and psychological adjustment in the patient groups under study? What is the role of generalised self-efficacy in that relationship?

d) What is the relationship between self-blame and current health behaviours? (i.e. diet and exercise)?

Specific research hypotheses were tentative due to the novelty of the research questions and the markedly inconsistent findings in past research regarding self-blame and adjustment. Nevertheless, it was expected that self-blame would be significantly different across patient groups and references of different specificity. Regarding the relationship between self-blame and psychological adjustment, it was expected that, if a relationship were found, it would be moderated by general self-efficacy. Specifically, self-blame would lead to low adjustment only when generalised self-efficacy was low. Finally, it was expected that there would be a positive correlation between health care activities and self-blame for the medical condition.
4.2 PHASE I: ITEM GENERATION AND PILOTING

4.2.1 Ethical Approval

Access to the patient sample for this phase of the study was allowed following an application to the South West Surrey Local Research Ethics Committee accompanied by the consent of the relevant consultants. Approval was granted within approximately two months (Committee approval letter in Appendix II).

4.2.2 Participants

Non-patients: An opportunity sample of 15 members of the public (6 males and 9 females) was recruited in order to obtain the items for the self-blame scale. Participants' mean age was 35 years.

Patients: A total of 74 patients were recruited from the Royal Surrey County Hospital outpatient clinics. Patients were diagnosed with either heart disease or diabetes. Their mean age was 50.5 years (SD=12.8) and their mean time elapsed since diagnosis was 6 years (SD=5.4). A breakdown of the patients' characteristics is presented in Table 4.1.
Table 4.1: Characteristics of the patient sample (N= 74).

<table>
<thead>
<tr>
<th></th>
<th>Diabetes</th>
<th></th>
<th>Heart Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>Gender</td>
<td>20</td>
<td>10</td>
<td>25</td>
</tr>
<tr>
<td>Mean age in years</td>
<td>42.8</td>
<td>46.6</td>
<td>57.2</td>
</tr>
<tr>
<td>(SD)</td>
<td>(13.8)</td>
<td>(11.9)</td>
<td>(8.1)</td>
</tr>
</tbody>
</table>

4.2.3 Measures

Item generation: The self-blame items were generated both by reviewing existing literature and through informal conversation with members of the public. The latter involved asking people to state possible explanations for falling ill. When necessary, probing was used to bring the focus on issues of blame and responsibility. The items finally included in the study were selected a) on the basis of how often they appeared during the item generation process (items appearing less than twice were omitted) and b) on how relevant they were to issues of responsibility and blame.

A total of 20-items were finally selected from both past literature and the interview material (Table 4.2; also see Appendix III for actual questionnaire). Occasionally, items with exactly the opposite meaning to those already in the scale were included to control for response biases. All items were scored on 7-point Likert scales ranging from 1 ('strongly disagree') to 7 ('strongly agree'). An 'I don't know' option was also available at the end of each response scale which corresponded to a '0' score. The
comprehensibility of the scale was tested on the patient sample of heart disease and diabetes patients.

Table 4.2: Items included in the study following item generation procedures.

| 1. Don't know what caused my illness | 11. My illness is God's way of punishing me |
| 2. I could have prevented getting ill | 12. My illness is just a matter of bad luck |
| 3. I am responsible for my own health | 13. I had nothing to do with getting ill |
| 4. I feel guilty when I see my doctor | 14. I am not the only one responsible for my illness |
| 5. I should have looked after myself better | 15. My illness could have happened to anyone |
| 6. People blame me for my illness | 16. Other factors apart from me led to my illness |
| 7. I deserve to be in this situation because of who I am | 17. I don't feel responsible for getting my illness |
| 8. I am to blame for my illness | 18. Getting my illness was God's will |
| 9. I don't deserve to be looked after | 19. I should have taken better care of myself and I would have never fallen ill |
| 10. I should have lived more moderately | 20. I couldn't help getting sick |

4.2.4 Procedure

Participants were approached while waiting for their outpatient appointments at the Royal Surrey County Hospital. They were asked to complete the questionnaire in the presence of the researcher and were encouraged to express any thoughts regarding the phrasing and content of the items. Once the questionnaire was completed, the researcher discussed the questionnaire with participants and noted any comments they had regarding the clarity of the items. Time spent with each participant ranged from 5-10 minutes.
4.3 RESULTS FROM PILOT DATA

4.3.1 Item clarity and comprehensiveness

Certain items were characterised as problematic by the patients. Specifically, items so identified by 30% or more of participants were items 1, 4, 9, 11, 15 and 19. These were characterized as 'odd', 'unclear', 'hard to answer' or 'irrelevant to the other items'. Some concern was also expressed regarding items 11 and 18 that included attributions to God.

To investigate the extent to which the concerns expressed by the patients were reflected in the psychometric properties of the items the contribution of each item to scale reliability was consulted (Table 4.3). It must be noted, however, that reliability analysis was not an aim of the study at this stage, and issues around scale construction and reliability (e.g. factor analysis, item total correlations etc.) were addressed in the next phase of the study.

Table 4.3: Contribution of items to scale reliability (scale alpha = .73).

<table>
<thead>
<tr>
<th>Item</th>
<th>Reliability if Item Deleted</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Don't know what caused my illness</td>
<td>.7482</td>
</tr>
<tr>
<td>2. I could have prevented getting ill</td>
<td>.7001</td>
</tr>
<tr>
<td>3. I am responsible for my own health</td>
<td>.7372</td>
</tr>
<tr>
<td>4. I feel guilty when I see my doctor</td>
<td>.7393</td>
</tr>
<tr>
<td>5. I should have looked after myself better.</td>
<td>.6926</td>
</tr>
<tr>
<td>6. People blame me for my illness</td>
<td>.7084</td>
</tr>
<tr>
<td>7. I deserve to be in this situation because of who I am</td>
<td>.7358</td>
</tr>
<tr>
<td>8. I am to blame for my illness</td>
<td>.7050</td>
</tr>
<tr>
<td>9. I don't deserve to be looked after</td>
<td>.7345</td>
</tr>
<tr>
<td>10. I should have lived more moderately</td>
<td>.7202</td>
</tr>
<tr>
<td>11. My illness is God's way of punishing me</td>
<td>.7333</td>
</tr>
<tr>
<td>12. My illness is just a matter of bad luck</td>
<td>.6997</td>
</tr>
<tr>
<td>13. I had nothing to do with getting ill</td>
<td>.7185</td>
</tr>
<tr>
<td>14. I am not the only one responsible for my illness</td>
<td>.7026</td>
</tr>
</tbody>
</table>
As seen in Table 4.3, some of the items already identified as problematic by the participants appeared to affect the scale’s reliability as well (i.e. items 1, 4, 9 and 15). Thus, they were removed from the scale. Items 11, 18 and 19 were excluded on the basis of participants’ comments (the content of item 19 was essentially maintained through item 5 which has similar but clearer meaning). All other changes involved minor improvements in the wording of the items. The final form of the scale is shown Part Two of the questionnaire in Appendix IV.

4.4 PHASE II: MAIN DATA COLLECTION

4.4.1 Participants and Recruitment Procedures.

In order to gain access to participants for the second phase of this study, the consultants of three clinics (i.e. diabetic, cardiac, and breast cancer) at the Royal Surrey County Hospital (Guildford, Surrey) were contacted. Each consultant was presented with the study’s aims, rationale and protocol as well as the researcher’s Curriculum Vitae, and was asked for his consent to approach patients during clinic hours. All three consultants allowed access to their patients without any objections to the study’s methodology.
Once the consultants’ approval was granted, an application was sent to the South West Surrey Local Research Ethics Committee. Following a brief correspondence with the committee during which some necessary clarifications were made regarding the study’s methodology, ethical approval to conduct the study was granted. A copy of the approval letter is provided in Appendix V.

4.4.2 Measures

A copy of the measures used in this study is given in Appendix IV.

Demographic Characteristics

Patients were asked to report their age, gender, and occupation. The latter was then coded in four categories based on a simplified version of the Standard Occupational Classification (SOC, 2000). All professional and associate professional occupations (i.e. managers and senior officials, professionals, associate professional and technical occupations) were grouped in category 1. Category 2 included intermediate occupations (i.e. Administration and secretarial skilled trades, personal service, sales and customer care). Category 3 included all elementary occupations (i.e. process plan and machine operatives and elementary trade, plant, storage and administration and service occupations). Finally category 4 included all retired participants and housewives who did not report any prior occupation.

In this section patients were also asked to report their diagnosis or reason for appointment and the time of initial diagnosis if known.
Self-Blame measures

The 14 items obtained during the first phase of this pilot work were used here to assess patients' self-blame about their illness. Participants responded on 7-point Likert scales, where 1= 'strongly disagree' and 7= 'strongly agree'. The scale was completed twice, first with respect to their condition in general, and second with respect to a specific self-selected negative event relevant to their condition (e.g. bad test results, alarming symptoms etc.) that occurred within the last three months.

Risk Factors

A risk factor checklist containing 22 possible factors known to contribute to various chronic conditions was presented. Patients were asked to tick those factors that according to their subjective opinion contributed to their illness. Any number of factors could be ticked, and an 'other' option was available at the end of the list to allow factors not on the list to be added. In order to avoid bias in categorising a risk factor as behavioural, two independent judges categorised each risk factor on the list as either behavioural or non-behavioural in nature. Agreement was 99%. Specifically, risk factors of diet, stress, alcohol, being overweight, smoking, and sexual behaviour were seen as behavioural. All other factors were considered non-behavioural.

Self-efficacy

The Generalized Self-efficacy Scale (Schwarzer & Jerusalem, 1993) was used to assess patients' perceptions of their ability to successfully deal with problems. The Generalized Self-Efficacy Scale is a 10-item measure that is designed to assess optimistic self-beliefs to cope with a variety of difficult demands in life. It explicitly refers to personal agency,
i.e. the belief that one's actions are responsible for successful outcomes. Internal validity has been reported to range from .82 to .93.

Measures of Psychological Adjustment

Psychological adjustment was assessed through the measurement of anxiety and depression using the Hospital Anxiety and Depression Scale (HADS, Zigmond & Snaith, 1983). The HADS is designed to measure anxiety and depression in general medical outpatient populations. It consists of seven depression and seven anxiety items selected to distinguish the psychological effect of physical illness. The HADS has good validity and reliability coefficients (as high as .77 and .76 respectively), is brief and simple, and is acceptable by patients (Bowling, 1995).

Diet and Exercise

Finally, a set of 7 items developed by Toobert & Gasgow (1994) assessing the patients' adherence to dietary and fitness guidelines was included in the questionnaire. Of those, 5 assessed participants' dietary habits in terms of calorie control and consumption of fibre, fat, and sweets, and 2 assessed participants' activity and exercise levels. Questions were rated on 5-point scales, with points on the scales representing either percentages or frequencies.

4.4.3 Procedure

Participants were recruited through three hospital outpatient clinics, Cardiac, Breast, and Diabetes, at the Royal Surrey County Hospital in Guilford (UK). Participants had to be less than 70 years old and have a specific diagnosis for the condition rather than a general
or suspicious complaint. Patients waiting for their follow-up appointment were approached by the clinic's nurse and were told about the researcher's presence. Those interested in knowing more about the study were introduced to the researcher, who informed them about the study and invited them to participate. All participants were informed of their right to withdraw from the study at any time and were required to provide written consent. Participants were provided with a 9-page self-administered questionnaire with an estimated completion time of 30 minutes. There were no limitations in the amount of time available for completion and all participants were encouraged to ask questions if necessary. Upon receipt of the questionnaire, the researcher initiated an informal conversation with each participant to ensure that patients were not made uncomfortable by reading the questionnaire.

4.5 RESULTS

4.5.1 Sample

A total of 146 patients were approached by the clinic's nurses and agreed to hear more about the study. Of those, 137 adults (47% male, 53% female; mean age 51.6 years, SD=12.03) agreed to participate. Thus, the response rate for this study was 94%. For the remaining 6%, the reasons for decline were illiteracy (N=3), visual impairment (N=2) and questionnaire length (N=4). Participants were diagnosed with one of the following conditions: diabetes (N=43, 67% male, 33% female; mean age 46.3 years, SD=14.3), breast cancer (N=55, 100% female; mean age 51.8 years, SD=9.6) and heart disease (N=39, 89% male, 11% female; mean age 51.8 years, SD=8.4). In all three patient groups
the majority of participants reported an intermediate occupation or better. Table 4.4 shows a breakdown of patient characteristics in the three illness groups.

**Table 4.4: Patient characteristics for each patient group**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Sex</th>
<th>Occupation (N)*</th>
<th>Mean Age (years)</th>
<th>Mean time since diagnosis (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>29 (male)</td>
<td>11 11 8 8</td>
<td>46.3 (SD: 14.3)</td>
<td>12.8 (SD: 12.6)</td>
</tr>
<tr>
<td></td>
<td>14 (female)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast cancer</td>
<td>55 (female)</td>
<td>16 17 5 16</td>
<td>51.8 (SD: 9.5)</td>
<td>2.8 (SD: 2.3)</td>
</tr>
<tr>
<td>Heart disease</td>
<td>35 (male)</td>
<td>12 4 7 14</td>
<td>51.8 (SD: 8.4)</td>
<td>4.2 (SD: 6.7)</td>
</tr>
<tr>
<td></td>
<td>4 (female)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: 1: Professional Occupations 2: Intermediate Occupations 3: Elementary Occupations 4: Retired or Housewives

The three patient groups differed significantly in age ($F(2, 136)=11.7, p<.001$) and Scheffe's post hoc multiple comparisons indicated that heart disease patients had a significantly higher mean age from both diabetes and breast cancer patients. Similarly, the groups differed in the time elapsed since diagnosis ($F(2, 132)=20.2, p<.001$) with diabetes patients having a higher mean time since diagnosis from heart disease and breast cancer patients. Therefore these two variables were subsequently included in all relevant analyses.
4.5.2 Self-Blame Scale

Two sets of factor analyses were run (Principal Components with oblique rotation), one for the general condition and one for the specific negative event. Initial solutions (i.e. eigenvalues >1) resulted in four components for general self-blame and three components for specific self-blame (see Appendix VI for relevant output). These solutions were not retained for the following reasons. First, examination of the scree plots (Appendix VI) in both cases indicated that a two factor solution would be more appropriate. This was reflected in the very small amount of variance explained by the last and smallest components (i.e. 7.3% in general self-blame and 8.6% in specific self-blame). Second, in both cases the last component consisted of only two loadings and could not be interpreted in any meaningful way to support retaining them. Therefore, different solutions were sought for varying the number of extracted components. Prior to extracting two factor solutions for both cases of self-blame, a three factor solution was extracted for general self-blame (see Appendix IV) to ensure that a potentially meaningful solution was not being overlooked. This solution suffered the same problems as previously, with the smallest component accounting for little percentage of the variance (i.e. 8.4%), including only two loadings and being hard to interpret.

The most meaningful solution consisted of two components in both cases of self-blame. The item communalities (i.e. the proportion of variance that each item has in common with other items) are shown in Table 4.5. Particularly low communalities were characteristic of items 7, 13 and 14.
Table 4.5: Item communalities for factor analysis obtained from reference to the general condition and the specific event.

<table>
<thead>
<tr>
<th>Item</th>
<th>General</th>
<th>Specific</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I could have prevented my condition</td>
<td>.68</td>
<td>.79</td>
</tr>
<tr>
<td>2. I am responsible for getting my condition</td>
<td>.63</td>
<td>.87</td>
</tr>
<tr>
<td>3. I deserve to be in this situation because of choices I made</td>
<td>.82</td>
<td>.75</td>
</tr>
<tr>
<td>4. People blame me for my condition</td>
<td>.49</td>
<td>.59</td>
</tr>
<tr>
<td>5. I deserve to be in this situation because of who I am</td>
<td>.42</td>
<td>.56</td>
</tr>
<tr>
<td>6. I am to blame for my condition</td>
<td>.73</td>
<td>.77</td>
</tr>
<tr>
<td>7. My condition was God’s way of punishing me</td>
<td>.17</td>
<td>.01</td>
</tr>
<tr>
<td>8. I am not responsible for my condition</td>
<td>.34</td>
<td>.34</td>
</tr>
<tr>
<td>9. I should have taken better care of myself</td>
<td>.67</td>
<td>.54</td>
</tr>
<tr>
<td>10. I couldn’t prevent my condition</td>
<td>.76</td>
<td>.53</td>
</tr>
<tr>
<td>11. I should have behaved more moderately</td>
<td>.41</td>
<td>.33</td>
</tr>
<tr>
<td>12. I had nothing to do with getting my condition</td>
<td>.77</td>
<td>.78</td>
</tr>
<tr>
<td>13. My condition could have happened to anybody</td>
<td>.001</td>
<td>.47</td>
</tr>
<tr>
<td>14. My condition was just a matter of bad luck</td>
<td>.17</td>
<td>.30</td>
</tr>
</tbody>
</table>

The structural coefficients (or factor loadings) of the items are shown in

Table 4.6. They have been re-arranged in descending order for General- Component I. Regarding self-blame for the condition in general, component I accounted for 40.5% of the variance and component II accounted for 10.3% of the variance (Cronbach’s alpha=
In the case of the specific negative event, component I accounted for 43% of the variance and component II for only 12% of the variance (Cronbach’s alpha = .89 and .74). Also, in both the general and the specific conditions, component II expressed exactly the opposite meaning of other items in the scale and its items had been included to avoid response bias (see section 4.2.3). The correlation of the components was also examined and was found to be negative (r = -.40, N = 137, p < .001 for the general condition and r = -.47, N = 88, p < .01 for the specific event) verifying that the two components have captured the opposite meaning expressed by their items. Therefore the items loading on component II (i.e. items 12, 13, 14 in Table 4.6) were excluded from further analyses.

Table 4.6: Pattern matrices showing the two-component solutions for general and specific self-blame.

<table>
<thead>
<tr>
<th>Item</th>
<th>General Component</th>
<th>Specific Component</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>II</td>
</tr>
<tr>
<td>1. I deserve to be in this situation because of choices I made</td>
<td>.89</td>
<td>-.05</td>
</tr>
<tr>
<td>2. I am to blame for my condition</td>
<td>.83</td>
<td>-.08</td>
</tr>
<tr>
<td>3. I should have taken better care of myself</td>
<td>.80</td>
<td>-.05</td>
</tr>
<tr>
<td>4. I am responsible for getting my condition</td>
<td>.79</td>
<td>-.03</td>
</tr>
<tr>
<td>5. I could have prevented my condition</td>
<td>.71</td>
<td>-.11</td>
</tr>
<tr>
<td>6. People blame me for my condition</td>
<td>.69</td>
<td>-.01</td>
</tr>
<tr>
<td>7. I deserve to be in this situation because of who I am</td>
<td>.66</td>
<td>.07</td>
</tr>
<tr>
<td>8. I should have behaved more moderately</td>
<td>.63</td>
<td>-.01</td>
</tr>
<tr>
<td>9. My condition was God’s way of punishing me</td>
<td>.43</td>
<td>.17</td>
</tr>
<tr>
<td>10. My condition was just a matter of bad luck</td>
<td>-.35</td>
<td>.13</td>
</tr>
<tr>
<td>11. My condition could have happened to anybody</td>
<td>-.27</td>
<td>-.06</td>
</tr>
<tr>
<td>12. I couldn’t prevent my condition</td>
<td>-.05</td>
<td>.85</td>
</tr>
<tr>
<td>13. I had nothing to do with getting my condition</td>
<td>-.27</td>
<td>.76</td>
</tr>
<tr>
<td>14. I am not responsible for my condition</td>
<td>.06</td>
<td>.60</td>
</tr>
</tbody>
</table>
Although the retained component was very similar in both the general and the specific conditions, it was not identical. Specifically items 10 and 11 loaded on opposite factors for the general and the specific conditions. These items also had very low communality indices when used for the general condition (items 13 and 14 in Table 4.5): Therefore, these two items were deleted to make the components equivalent. Therefore, the resulting scale consisted of 9 items (i.e. items 1, 2, 3, 4, 5, 6, 7, 8, 9) according to Table 4.6). and was perceived as measuring general self-blame (symbolized as GSBg when referring to self-blame for the overall medical condition and GSBsp when referring to the self-blame for the specific negative event). Internal reliability for this component was satisfactory for both conditions (Cronbach’s alpha= .89 for both GSBg and GSBsp). Composite scores for GSBg and GSBsp were calculated and entered in all relevant subsequent analyses.

4.5.3 Descriptive Analyses

Means and standard deviations for all measures used in this study are presented in Table 4.7. In general, participants showed relatively low levels of self-blame for either their condition in general and the relevant negative event. Although low observed means raise concerns about floor effects, there were sufficient numbers of patients who rated their self-blame high enough to provide adequate range for conducting inferential statistical tests (Glinder & Compas, 1999). Specifically, 17-23% of patients scored higher than 3 on a 7-point scale on the self-blame scale.
Table 4.7: Mean values for all psychological variables.

<table>
<thead>
<tr>
<th>Item Scale</th>
<th>Potential Score Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>GSbg</td>
<td>1-7 (7=max GSB)</td>
</tr>
<tr>
<td>GSBsp</td>
<td>1-7 (7=max GSB)</td>
</tr>
<tr>
<td>HADS (anxiety)</td>
<td>0-21 (8-10 borderline)</td>
</tr>
<tr>
<td>HADS (depression)</td>
<td>0-21 (8-10 borderline)</td>
</tr>
<tr>
<td>Self-efficacy</td>
<td>10-40 (40=max self-efficacy)</td>
</tr>
<tr>
<td>Diet</td>
<td>1-5 (5=healthy diet)</td>
</tr>
<tr>
<td>Exercise</td>
<td>1-5 (5=max exercise)</td>
</tr>
<tr>
<td>Number of behavioral risk factors</td>
<td>0-6</td>
</tr>
</tbody>
</table>

Anxiety and Depression levels were relatively low, self-efficacy levels were relatively high, and diet and exercise levels were average indicating moderate compliance to medical and healthy living guidelines. The correlations between all variables in the study are shown in Table 4.8. Since the three patient groups differed in age and time since diagnosis these variables were also included in the analyses.
Table 4.8: Pearson’s correlations among main psychological and demographic variables (N in parentheses)

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8a</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1)</td>
<td>GSBg</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2)</td>
<td>GSBsp</td>
<td>.47** (88)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3)</td>
<td>HADS (anx)</td>
<td>-.09 (135)</td>
<td>.07 (88)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4)</td>
<td>HADS (depr)</td>
<td>-.05 (135)</td>
<td>.03 (88)</td>
<td>.66** (135)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5)</td>
<td>S</td>
<td>elf-efficacy</td>
<td>-.03 (135)</td>
<td>.11 (88)</td>
<td>-.30** (134)</td>
<td>-.29** (134)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6)</td>
<td>Diet</td>
<td>-.05 (134)</td>
<td>-.18 (87)</td>
<td>-.34** (133)</td>
<td>-.22* (133)</td>
<td>.19* (133)</td>
<td>-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7)</td>
<td>Exercise</td>
<td>-.01 (135)</td>
<td>.16 (88)</td>
<td>-.25** (134)</td>
<td>-.16 (134)</td>
<td>.12 (134)</td>
<td>.28** (134)</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8)</td>
<td>No. of behavioral risk factors a</td>
<td>.65** (137)</td>
<td>.18 (88)</td>
<td>-.09 (135)</td>
<td>-.13 (135)</td>
<td>.08 (135)</td>
<td>.04 (135)</td>
<td>-.04 (135)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>9)</td>
<td>Age</td>
<td>.09 (137)</td>
<td>-.12 (88)</td>
<td>-.18* (135)</td>
<td>-.06 (135)</td>
<td>.05 (135)</td>
<td>.15 (135)</td>
<td>.01 (135)</td>
<td>.03 (137)</td>
<td>-</td>
</tr>
<tr>
<td>10)</td>
<td>Time since diagnosis</td>
<td>-.13 (133)</td>
<td>.20 (84)</td>
<td>.03 (131)</td>
<td>-.05 (131)</td>
<td>-.01 (131)</td>
<td>.02 (131)</td>
<td>.04 (131)</td>
<td>-.12 (133)</td>
<td>-.05 (133)</td>
</tr>
</tbody>
</table>

Note: * p<.05, **p<.001,
a: Spearman’s correlations

Significant correlations were found between GSBg and GSBsp (r=.47, N=88, p<.01)
indicating that the higher the self-blame for one’s medical condition the higher his/her self-blame for the negative event related to the condition. Also the Number of behavioural factors correlated significantly with GSBg (r=.65, N=137, p<.01) meaning that the greater the number of behaviour factors people identified as having contributed to their illness, the higher their levels of self-blame for their general medical condition.
Participants’ age showed a low but significant negative correlation with anxiety ($r=−.18$, $p<.05$) suggesting that the lower the age the higher the anxiety. No other significant correlations were found between age and the remaining psychological variables. Similarly, time since diagnosis did not correlate significantly with any of the other variables in the analysis.

4.5.4 Effects of specificity of reference and type of medical condition on self-blame

Participants showed relatively low levels of self-blame for the medical condition in general. Specifically, when referring to their medical condition in general, participants’ mean self-blame score was $1.99$ (SD = .20). Looking at the frequencies of participants’ responses, approximately 10% of the sample scored on the high end of the scale (indicating at least moderate levels of GSBg).

Regarding the specific negative event, 87 out of 137 participants (64%) responded to the relevant section. Of those, 55 (40%) clearly stated the specific event that their answer referred to, and 32 (24%) provided answers to the relevant section without specifying a negative event. The most common negative events reported were bad test results (e.g. high cholesterol or glucose levels), alarming symptoms (e.g. angina), and illness recurrence (e.g. metastasis of cancer). Table 4.9 shows the frequencies of the reported specific negative events. Here, mean self-blame was $2.26$ (SD=1.44). Approximately 17% of the participants scored on the high end of the scale.
Table 4.9: Frequencies of types of specific events mentioned by patients.

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bad test results</td>
<td>11</td>
<td>8.0</td>
</tr>
<tr>
<td>Alarming symptoms</td>
<td>22</td>
<td>16.1</td>
</tr>
<tr>
<td>Recurrence of illness</td>
<td>11</td>
<td>8.0</td>
</tr>
<tr>
<td>Infection</td>
<td>3</td>
<td>2.2</td>
</tr>
<tr>
<td>Other</td>
<td>8</td>
<td>5.8</td>
</tr>
<tr>
<td>Not specified</td>
<td>32</td>
<td>23.4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>87</strong></td>
<td><strong>63.5</strong></td>
</tr>
</tbody>
</table>

To test for differences in self-blame across medical conditions and types of reference (i.e. general vs. specific), a mixed-factors ANOVA was carried out with medical condition as the between-subjects factor and specificity of reference as the within-subjects factor. Results showed significant main effects of both the type of medical condition ($F(2, 85)=11.8, p<.001$), and the specificity of reference ($F(1, 85)=12.7, p<.01$). Post hoc analysis (Scheffe test) of the between-subjects factor revealed that the breast cancer group showed significantly lower levels of self-blame from the heart disease and diabetes groups. All groups showed higher levels of self-blame for the specific negative event. No significant interactions were revealed.

4.5.5 Behavioural risk factors and self-blame

Both the number and type of risk factors identified by patients were considered. Out of 137 participants, 122 (90%) identified at least one risk factor. The mean number of risk factors was 2.4 (min = 0, max=11, SD = 1.6), with 52% identifying 2 to 4 risk factors. Figure 4.1 shows the frequencies of risk factors selected by the participants. The most frequent were stress, followed by heredity, age, and being overweight.
The number of behavioural risk factors identified differed across the three medical conditions (Kruskal-Wallis $x^2=15.9$, df=2, $p<.001$) with heart disease patients identifying more behavioural risk factors (mean=.56, sd=.50) than diabetes patients (mean=.42, sd=.50) and breast cancer patients (mean=.07, sd=.26).

**Figure 4.1**: Frequency of risk factors perceived as relevant to illness (N=136)

When self-blame scores between patients who identified behavioural risk factors and patients who did not were compared, it was found that the former had higher GSBg ($t=-7.77$, df=135, $p<.001$) as well as GSBsp ($t=-.2.63$, df=86, $p<.05$).
4.5.6 Relationship between self-blame, self-efficacy and psychological adjustment.

Correlations between self-blame (general and specific) and the psychological adjustment variables of depression and anxiety indicated that adjustment is generally unrelated to self-blame. Self-efficacy, was also unrelated to self-blame, and consequently did not appear the have any effect on the relationship between self-blame and adjustment.

When the above relationships were examined separately for each patient group relationships between the above variables remained statistically non-significant.

4.5.7 Self-blame and time elapsed since diagnosis

As already mentioned, Pearson’s correlations between self-blame in the whole sample and time elapsed since diagnosis yielded no significant results. When the same correlations were computed separately for each patient group results showed that GSBg correlated significantly with time since diagnosis in the diabetes group (r=.34, N=42, p<.05). There is therefore some indication of a link between time since diagnosis and self-blame in the diabetes group suggesting that as time passes diabetes patients show increased acceptance of responsibility for their condition.

4.5.8 Self-blame and diet/exercise

Regarding patient’s reported levels of diet and exercise, no significant correlations with self-blame were found when the whole sample was considered. Looking at each patient group separately however, a significant correlation was found between diet and GSBg (r=-.42, N=43, p<.01) and GSBsp (r=-.49, N=30, p<.01) in the diabetes group suggesting
that the higher the acceptance of blame for their illness or a relevant specific event, the less healthy the diet adopted by patients. For the same group, a significant correlation was found between GSBg and exercise levels ($r = -0.32$, $N=43$, $p<.05$). No significant relationships between self-blame and diet/exercise levels were found in the other patient groups.

4.6 DISCUSSION

4.6.1 Phase I: Item generation and piloting

One of the criticisms in studies of self-blame has been the use of single item measures. This study aimed at generating additional items for the measure of self-blame in patients with chronic illness and piloting those items on patient populations to improve their clarity and comprehensiveness. Out of the 20 items yielded from informal interviews with members of the public, 7 items had to be deleted following feedback from a patient sample. The remaining 13 items underwent minor changes in wording and grammar. One item was added to include an explicit measure of behavioural self-blame, leading to a 14-item measure of self-blame.

Two interesting observations arise when looking at the content and phrasing of the items. First, both the words ‘blame’ and ‘responsibility’ appear in the list of items, indicating that they may be equally available in people’s vocabulary about illness avoidability. While there are several studies in the literature indicate that this is the case (e.g. Sholomskas, Steil & Plummer, 1990), none of the studies aimed specifically at exploring this possibility. Since Shaver and Drown (1986) have cautioned about the potential
implications that differences in meaning may have on the conceptualisation and measurement of self-blame, a study is presented later in the thesis to explore people’s understanding and application of the above terms on different negative events including various illnesses.

A second observation is that many of the items include evaluative words such as ‘I should have...’ and ‘I could have...’. Such phrases show not only a focus on the self, but also a critical approach towards issues of personal agency. There is thus some evidence of the existence of the attributional and evaluative components within the concept of self-blame discussed previously in the thesis (Brewin, 1985).

4.6.2 Phase II: Main data collection

The nature of self-blame in patients with different chronic illnesses and potential differences among these patient groups was explored. Specifically, using a multi-item measure of self-blame, this study investigated self-blame in diabetes, breast cancer and heart disease patients, and looked at differences in participants’ responses when self-blame is regarded for the medical condition in general and a specific negative event related to the medical condition. This study also looked at the relationship between self-blame and psychological adjustment and potential differences in these associations in the three patient groups. Additional measures of health related self-care (i.e. diet and exercise) were also explored as possible correlates of self-blame.

Results showed that levels of self-blame for developing the medical condition varied significantly across the three patient groups. Specifically, breast cancer patients showed
lower levels of self-blame than patients in the other two groups. The question that results from this observation is why such a difference exists. Traditionally, self-blame is seen as an attempt to increase feelings of control over uncontrollable events (Janoff-Bulman, 1979). In this study, although measures of perceived control over the condition were not included, one would expect cancer to be the least controllable of the three conditions in terms of onset and recurrence and, thus, to involve a greater need for a sense of control leading to greater levels of self-blame. This, however, is not the case making the present findings beg for an alternative explanation.

One dimension along which breast cancer inherently differs from diabetes or heart disease is the degree of lifestyle involvement. Although some behavioural risk factors such as lack of exercise and being overweight have been associated with increased risk of breast cancer, they are not considered as strong predictors as age or heredity (Vogel, 2001). Furthermore, the need for lifestyle changes following diagnosis is unlikely to be of the same degree as for diabetes or heart disease patients. In the two latter cases, patients are expected to monitor or change habits such as diet and activity levels, since these factors are considered important predictors of disease onset and recurrence. Therefore, it is possible that self-blame levels in this study reflect differences in the degree of lifestyle involvement associated with each condition.

A similar rationale may explain the results regarding the differences in self-blame when referring to the general condition or a more specific negative event. This study showed that, overall, levels of self-blame are higher when considering a specific event than the medical condition in general. It is possible that a post-onset complication (e.g. high
glucose levels) is seen as part of the management of the illness or its consequences, is perceived as the patient’s own responsibility, and thus leads to higher levels of self-blame that the medical condition in general.

Another indication that patient self-blame may reflect the association between a given illness and lifestyle factors is given in this study by an exploration of the risk factors patients associate with their illness. Results showed that patients who identified at least one behavioural risk factor showed more self-blame than patients who identified no behavioural risk factors. It should be noted however that risk factors were assessed by using a checklist and therefore analyses has little quantitative value. Knowing the degree to which patients considered these factors as relevant to their medical condition might give better insight into the relationship between self-blame and risk factor involvement.

Regarding the relationship between self-blame and adjustment, results showed no relationship between the relevant variables in either the overall sample or the specific patient groups. As discussed in earlier chapters, previous research has consistently found a relationship between characterological self-blame and depression in chronic patients but similar findings for behavioural self-blame have not been consistent. The self-blame measure in this study was rather generic and did not differentiate between behavioural and characterological self-blame. Hence, claims can only refer to feelings of general self-blame, which appeared to be unrelated to anxiety or depression. Inconsistent findings in this area have been attributed to timing considerations (Michela & Wood, 1986). In this study, the mean time since diagnosis was approximately 6 years, and evidence for a correlation between time since diagnosis and measures of general self-blame were relatively weak and group specific. It is possible that, a stronger association exists at the
very early stages of the illness experience (i.e. at diagnosis) not captured by the design of this study. After some time, although self-blame may still be present in patients’ illness cognitions, it becomes generally unrelated to psychological adjustment.

Examination of the relationship between the time since diagnosis and self-blame revealed that as time passes diabetes patients tend to accept more blame for their illness. These findings make sense when the nature of the illness is considered. Diabetes is a disease that primarily requires lifestyle management in order to be kept in control. Patients with this condition have to constantly adhere to certain health behaviour guidelines in order to keep their glucose levels within limits and avoid hypo- or hyper- glycemic episodes. Therefore, it may be that the self-blame that diabetes patients show with time stems from an increasing acceptance of the responsibilities related to their condition and the accompanied awareness of their role in disease onset and management.

Finally, an examination of the relationship between self-blame and health behaviour yielded some interesting findings. In diabetes patients, a negative relationship was found between acceptance of blame and healthy eating. These results are especially interesting when considering the fact that diet is of central importance to this particular group of patients. A possible explanation for this finding may be that the direction of the relationship runs from dietary habits towards the acceptance of blame. In other words, diabetes patients who are failing to stick to the recommended dietary guidelines may be more ready to accept responsibility for their illness (or a specific negative event related to it) than patients who are more successful (intentionally or not) in sticking to a healthy
diet. A similar explanation may apply in the case of exercise and its negative relation to self-blame in the same patient group.

By evaluating the results discussed in this section, several of the study’s strengths and limitations become apparent. On the positive side, this study utilised a new approach to the study of self-blame. Unlike the majority of the studies available in the literature, where a single group of patients is involved and the focus is on the self-blame-adjustment relationship, this study looked at self-blame comparatively across three medical conditions and investigated their similarities and differences regarding self-blame behaviour. Consequently, it has revealed the importance of the illness (or event) characteristics as possible explanations of self-blame and has shown the need for further investigation in this area.

On the other hand, a number of limitations are evident in this study and should be acknowledged. First, the sample used poses some restrictions in the analysis and interpretation of results. Specifically, while the overall sample size is acceptable for an exploratory study, the patient groups are rather small. Also, the medical conditions chosen did not have an even distribution of male and female participants and thus did not allow gender effects to be studied. Although it can be argued that the samples in this study reflected the sex distributions in the actual population, statistical restrictions for the study of gender differences remain. Second, the measure of self-blame used in the study was too generic to allow for the examination of some of the issues raised in previous chapters. Specifically, there was no opportunity to look for relationships involving behavioural and characterological self-blame. Although each type of self-blame was represented by a
specific item in the scale, the creation of a behavioural and a characterological self-blame sub-scale did not result during the item generation phase or the factor analysis. Therefore, interpretation had to rely on a more generic measure of self-blame. Finally, the low self-blame scores should be considered. While there was enough variability for the appropriate analyses to be run, the findings should be interpreted with caution. Furthermore, the observed floor effects may be an indication of low sensitivity of the measure used. While low mean self-blame scores are common in the literature (e.g. Malcarne et al, 1995; Nielson & McDonald, 1988), they have been surprisingly overlooked. It is possible that low levels of self-blame—especially in the presence of self-attributions—may suggest a reluctance to endorse negative evaluative comments about oneself even though personal agency is acknowledged. Clearly, more attention should be paid to the levels of self-blame reported and the possible reasons for them.

The findings of this study give rise to a number of research questions that should be further investigated. Specifically, it is important to understand the origins of patient self-blame and determine the factors that affect its occurrence and intensity. Since self-blame differs among medical conditions, there may be variables related to the experience of the given illness (e.g. prior health behaviour, knowledge of medical facts) that determine the levels of self-blame. Similarly, individual characteristics such as sex may affect self-blame and should be explored. Finally, rather than focusing on measures of psychological adjustment, the potential relationship between self-blame and health behaviours should be further explored.
In conclusion, the present study was designed to approach self-blame in chronic patients in a comparative manner and explore potential differences among different patient groups. Results revealed significant differences in self-blame among diabetes, breast cancer and heart disease patients, with cancer patients showing less self-blame than the remaining two patient groups. Also, self-blame tended to be higher when a specific negative event was considered than when patients referred to their illness in general. Overall, no substantial evidence of a relationship between self-blame and psychological adjustment was found. In spite of the study’s limitations, it has been shown that more research on the conditions that determine the presence and level of self-blame in patients needs to take place, and refinement of the existing theory must be considered.
SUMMARY

This chapter aims to construct and test a model of the predictors and outcomes of behavioural self-blame. Drawing mainly from the evidence of attribution research, a model is proposed that describes the paths leading from personal and situational characteristics to behavioural causal attributions and behavioural self-blame. Similarly, the paths from behavioural self-blame to psychological adjustment and behaviour change are also described. The model is then tested on a sample of patients with heart disease using path and regression analysis as applicable. Results show that a number of situational and personal variables affect self-blame mainly through their link with behavioural causal attributions. Evidence for the relationship between behavioural self-blame and adjustment or health behaviours is also presented but interpreted with more caution due to methodological restrictions.

5.1 INTRODUCTION

The significance of self-blaming behaviour lies in its potential relationship with psychological adjustment. To date, research on self-blame has focused almost entirely on clarifying this relationship, and attempting to replicate Janoff-Bulman’s (e.g. 1979; 1982) findings of a positive relationship between behavioural self-blame (BSB) and adjustment,
and a negative relationship between characterological self-blame (CSB) and adjustment. However, research results have been inconsistent—especially regarding BSB—and attempts to replicate Janoff-Bulman’s original work have not been successful (Sholomskas, Steil & Plummer, 1990).

As mentioned in previous chapters, a number of reasons for these inconsistencies have been put forward including differences in sample characteristics, diversity of measures used, variations in time elapsed since the onset of the event, and differences in the operational definitions employed by different researchers. Two implications for research can be identified in the above explanations. First, there is a possibility that BSB is problem specific which would explain the variability in results across different population samples and timings. This possibility has been considered by Shaver & Drown (1986) after reviewing studies of self-blame in chronic illness. They argue that without close conceptual analysis of the variables involved, it is possible that self-blame may for example ‘have positive effects on coping with perinatal complications and immediate spinal cord injury, negative effects on adjustment to the disease of a spouse and no important influence on coping with breast cancer or long-term adjustment to permanent disability’ (pp. 699). Some evidence supporting the possibility of problem specificity regarding self-blame has also been demonstrated in the previous chapter of this thesis, where different levels of self-blame were expressed by different patient groups.

The second implication of the proposed reasons behind the inconsistencies in the self-blame literature relates to the lack of operational definitions and conceptual clarifications. Specifically, it has been shown that most studies fail to differentiate between causality,
responsibility and blameworthiness and use self-blame as synonymous with causal attributions or attributions of responsibility (Shaver & Drown, 1986). In the absence of empirical support for the similarity of these concepts, it is not possible to generalise from these findings.

A similar issue worth pursuing further is the relationship between causal attributions and self-blame. As already discussed earlier in the thesis, the distinction between causal attributions and self-blame (whether behavioural or characterological) has been proposed and empirically supported by Brewin (1988). Causal attributions involve locating the cause of an event. Self-blame presupposes a causal attribution to oneself and involves a moral evaluation of that attribution resulting in self-recrimination. In general, causal attributions can exist without the presence of self-blame, while self-blame requires a self-attribution of causality. The empirical evidence for this distinction includes only a few clinically relevant studies. For example, Brewin (1994) found that in victims of industrial accidents causal self-attributions were associated with less tension and anxiety while moral self-blame was associated with quicker return to work. However, the evidence is limited and there is a need for a more integrative approach that clarifies the relationships between the two variables.

It thus becomes important to shift some of the focus placed on the outcomes of BSB onto its antecedents or predictors in order to clarify the way self-blame forms and functions. If BSB is 'problem specific', what are the specific circumstances under which it occurs? Regarding illness, are there any illness or patient characteristics that can predict the
existence of self-blame? Also, what is the relationship between self-blame and causal attributions and how much do they have in common?

5.1.1 Conceptual model of BSB

The goal of the present study was to test an integrative model of BSB and attempt to give BSB a clear, comprehensive place in the attributional process. Using the limited and rather fragmented evidence available in the literature, a conceptual model was drawn that incorporated both predictor and outcome variables of BSB (Figure 5.1). Behavioural causal attributions are treated as one of the main predictors of behavioural self-blame that often act as a mediator between predictor variables and BSB. A presentation of the variables in the model as well as the rationale for their inclusion is presented below.

Regarding the antecedents of BSB very little is known. With most studies focusing on the outcomes of self-blame, evidence on its antecedents is only circumstantial. In contrast, evidence on the antecedents of causal attributions is more available. However, it is very rarely that antecedents of attributions and self-blame are considered in the same study which, combined with the already existing conceptual lack of clarity, limits the understanding of the area even further.

In general, the antecedents of either concept can be grouped into two main categories: person characteristics and event characteristics. Person characteristics include such variables as demographics (i.e. age, sex etc.) and individual differences variables (e.g. coping style, personality type etc.). Event characteristics include variables like severity, controllability/avoidability and time elapsed since the occurrence.
Demographic characteristics: In general, few sociodemographic factors have been shown to consistently predict attributions. Heinemann, Bulka and Smetak (1988) found that age was a significant predictor of responsibility attributions for spinal cord injuries. In a study of people with genital herpes, Manne and Sandler (1984) found that younger people were more likely to engage in self-blame than older ones. Moreover, Rodin (1986) suggested that older age may give rise to self-blame through the loss of control of certain health related activities. In contrast, Affleck et al (1987a) found that older men endorsed personal behaviour as a cause of their heart attack more strongly than younger men. Other studies have found no association between age and attributions (e.g. Downey, Silver and Wortman, 1985) but of those only one used a patient sample. Specifically, Koslowsky et al (1978) found that age, education, occupation, income, and religion did not predict attributions for myocardial infarction. Overall, the evidence seems to support a relationship between patients’ age and causal attributions even though the nature of the relationship is not very clear.

Sex has been investigated more extensively in attribution research with several studies aiming specifically to explore sex differences in causal attributions. For example, Vieyra et al (1990) found that women were more likely to attribute their infertility to themselves, reflecting findings in achievement literature in which women are more likely to attribute failure to themselves (Eccles et al, 1984). Similar findings were reported elsewhere (e.g. Abbey & Halman (1995), Juvonen & Leskinen, 1994; Hanninen & Aro, 1996).
In light of this evidence, the present model depicts age as an indirect predictor of BSB through its effect on causal attributions. In contrast, sex is expected to have both a direct and an indirect effect on self-blame since, apart from their tendency for self-attributions mentioned above, women are known to be more prone to emotion-oriented behaviours (e.g. Folkman & Lazarus, 1980)

**Individual differences:** The concept of CSB implies the involvement of more stable, unchangeable aspects of the self in the attributional process. In the present model, CSB is seen as both a direct and an indirect predictor of BSB since its esteem-related properties (Janoff- Bulman, 1992) would affect both the cognitive and emotional aspects of the attributional process.

**Prior behaviour:** Although it has not attracted much attention, a person’s behaviour prior to the negative event has been found to be a good predictor of self-blame. For example, Sholomskas, Steil & Plummer (1990) found that alcohol use was the best predictor of self-blame following a severe car accident. Similar results have been reported by Christensen et al (1999) regarding self-blame and prior alcohol and tobacco use in patients with head and neck cancer. However, neither of the studies differentiated between attributions and self-blame. On the contrary, they use causality and blame interchangeably in their work and discuss findings in terms of causal attributions. In the present model, prior behaviour is proposed to have an indirect effect on BSB through its effect on causal attributions.
5.1.3 Event characteristics

Severity/Consequences: Event severity (often measured through participants' perceptions of event consequences) has been found to affect causal attributions and self-blame. Although results come from cross-sectional studies and report mainly correlations, it has been shown that a positive correlation exists between event severity and causal attributions (e.g. Affleck et al, 1987; Tennen et al, 1996; Miller & Porter, 1983). Therefore, in this model illness severity is linked to behavioural attributions expecting that the more severe the event the greater the need for a comprehensive explanation.

Elapsed time: The few prospective studies in the area of illness attributions have found that self-attributions of responsibility change over time. For example, cancer patients' levels of CSB but not BSB were found to be significantly higher 4 months after initial diagnosis (Malcarne et al, 1995), while a the proportion of spinal cord injured patients attributing responsibility to themselves was significantly smaller one year after the accident (Richards et al, 1997). However, it is not clear whether time affects self-blame directly or indirectly through the adoption of alternative attributions that do not implicate the self. For this reason, both links are adopted in the model and their statistical significance will be explored.

Type of diagnosis: Relating back to the argument that self-blame may be problem specific, several studies have reported significant differences in the levels of self-blame between different populations (e.g. Weinberg 1994; Frazier & Schauben, 1994). However, exploring the reasons behind these findings was outside the scope of those studies. Here, following the argument in the previous chapter that differences in self-
blame are in essence due to different explanations available (e.g. lifestyle factors), it is proposed that the type of diagnosis (e.g. MI, hypertension etc) will affect behavioural attributions and indirectly self-blame.

Control and avoidability: Perceptions of control and avoidability have been proposed as potential determinants of self-blame. To illustrate, Davis et al (1996) found that perceived avoidability of spinal cord injuries contributed to self-blame even after controlling for participants’ causal attributions. A positive correlation between control and self-attributions has also been reported (Abbey & Halman, 1995). Therefore, in the present model, perceptions of control are seen as a direct predictor of BSB.

5.1.4 Outcomes of behavioural self-blame

Following the ample but quite unclear literature on the relationship between self-blame and psychological adjustment, the variables of anxiety and depression are treated as outcome variables in the present model. Furthermore, according to recent findings of a relationship between self-blame and behaviour change in patients with head and neck cancer (Christensen et al, 1999), the present model treats BSB as a predictor of changes in health behaviour. Specifically, it is proposed that, as a result of the evaluation of the instrumental role of the individual in engaging in the identified causes of the illness, BSB will affect the degree to which those behaviours have changed since the onset of the illness.
Figure 5.1: Conceptual diagram of the proposed relationships between BSB and its predictor and outcome variables.

To summarise, the present study tested the above model in order to clarify the conditions under which behavioural self-blame occurs and the possible implication it may have on other aspects of behaviour. The population sampled for the purposes of this study was patients diagnosed with some form of cardiovascular condition. The reason for this choice was that the particular group of conditions lends itself to the study of all the variables in the model. Apart from involving a relatively common category of medical problems, the general ‘umbrella’ of cardiovascular conditions’ covers closely related problems and syndromes (e.g. hypertension, angina, myocardial infarction) that may be due to behavioural (i.e. lifestyle) and/or non-behavioural factors, vary in severity, affect both men and women of a wide age range, be psychologically demanding and require lifestyle change. Therefore, it can be expected that all levels of the variables in the model will be represented in the particular population and thus tested properly.
5.2 **METHOD**

5.2.1 *Ethical Permission*

Two Consultant-Cardiologists from the Royal Surrey County Hospital supported the study by allowing patient recruitment at their outpatient clinics. Ethical permission for the study was granted by the South West Surrey Local Research Ethics Committee (see Appendix VII for the approval letter). Approval was granted approximately three months after initial application.

5.2.2 *Participants*

Participants were recruited at two cardiac outpatient clinics. The inclusion criteria were age, willingness to participate and knowledge of a specific diagnosis. Specifically, participants up to and including the age of 75 could participate in the study. There were two reasons for the choice of this particular cut-off point. First, the length of the questionnaire and the time required to complete it could be tiring for older people. Second, the older the participants, the higher the likelihood of their symptoms being associated with older age, in which case most parts of the questionnaire might seem irrelevant. Participants below the age limit who were willing to participate had to be aware of a specific diagnosis in order to fill in the questionnaire. Patients waiting for test results to confirm a diagnosis or seeing the doctors for the first time were excluded from the study.
A six-page questionnaire was administered to the patients (see Appendix VIII). Specifically the questionnaire assessed the following:

**Demographic and medical characteristics:** Patients' age, sex, occupation, diagnosis and type of treatment were recorded in the first section of the questionnaire. Ideally, this information should have been obtained from the patients' medical files. However, access to the files was not available. In order to assess the accuracy of the participants' responses to the diagnosis and treatment questions, the information obtained by 20 of the participants (chosen randomly during the data collection period) was verified by the clinic nurse. Accuracy was high (98%).

**Self-blame:** In order to assess BSB and CSB, the items found in the literature most consistently were used. Patients were asked how much they blamed their illness on things the did (their behaviour), and how much they blamed their illness on who they are (their character). Answers were given on a 5-point Likert scale with 1='not at all' and 5='completely' (see Part F of questionnaire in Appendix VIII). In contrast to the measure used in the previous study (chapter 4), these questions allowed for the differentiation between CSB and BSB and could potentially be used to make comparisons to findings from past research.

**Causal attributions:** A scale measuring participants’ specific causal attributions was generated specifically for the purposes of this study. Seven factors commonly associated with the onset of heart disease were identified by reviewing medical literature on risk.
factors for heart disease (American Heart Association, 2002; British Heart Foundation, 2002). These were: smoking, unhealthy diet, being overweight, little or no exercise, stressful lifestyle, and heredity. Chance was also included to offer an option for those participants who might feel none of the above risk factors could account for their illness. The latter two factors were not directly relevant to the proposed model. However, they were included in the scale in order to offer participants a reasonably broad and balanced range of choices that included both behavioural and non-behavioural attributions. Participants were asked to indicate the extent to which they felt their condition was caused by the above factors by rating them on a 5-point Likert scale where 1='not at all due to it' and 5='a lot due to it' (see Part B of questionnaire in Appendix VII). Issues regarding scale reliability are dealt with in the results section.

Avoidability of risk factors: Each of the risk factors mentioned above was rated on perceived avoidability (e.g. 'I believe an unhealthy diet is a habit I can avoid'). Additionally, participants rated the perceived avoidability of their behaviour and character (e.g. 'I believe I can change my behaviour if I want to', 'I believe I can change my character if I want to'). As with the causal attribution items above, the main focus here was on perceived avoidability of behavioural factors. Answers were given on 5-point Likert scales with 1='strongly disagree' and 5='strongly agree' (see Part C of questionnaire in Appendix VII).

Prior behavioural risk and risk change: In order to assess behavioural risk prior to illness onset, patients were asked to report the degree to which they engaged in certain risk behaviours associated with heart disease prior to their illness. The scale was a modified
version of the Toobert & Glasgow (1994) scale used in the previous chapter. Items referred to participants’ weight (e.g. Before the onset of my condition... I was overweight), diet (e.g. ...I was following a calorie controlled diet), exercise (e.g. ...I exercised regularly), smoking (e.g. ...I used to smoke), and stress (e.g. ...I led a stressful life). Answers were rated on a 5-point scale with 1= Not at all and 5= A lot (see Part A of questionnaire in Appendix VII). Using responses to these items, a risk index was created. Every response equal or higher than 3 was considered an indication of a risky behaviour and was given a point of 1. Responses equal or lower than 2 were considered an indication of little or no risk and were given a point of 0. Thus, a composite index score was obtained that ranged from 0 (little or no risk) to 9 (high risk).

In order to assess perceived behaviour change, the same health behaviour items were repeated later in the questionnaire (see Part E of questionnaire in Appendix VII), this time enquiring about behaviour since the onset of participants’ condition (e.g. ‘Since the onset of my condition I have been overweight’). Each response was subtracted from its earlier (baseline) equivalent giving an index of positive, negative or no behaviour change. A positive score indicated improved health behaviour, while a negative score indicated a decrease in the health behaviour. A score close or equal to zero indicated no change.

Consequences: Perceived consequences of the medical condition was assessed by including the ‘consequences’ subscale from Weinman et al’s (1996) Illness Perceptions Questionnaire (IPQ; see Part D of questionnaire in Appendix VII). The IPQ is a relatively well validated and reliable measure that has been used with patients with heart disease. Its sub-scales can be used individually. The ‘consequences’ sub-scale consists of seven items
referring to the perceived impact of illness on one’s life. Items are scored on a 5-point 
scale with 1 = strongly disagree and 5 = strongly agree. In the present sample, the sub-
scale yielded a satisfactory Chronbach’ a alpha of .73.

Anxiety/Depression: Participants’ anxiety and depression levels were measures with the 
Hospital Anxiety and Depression Scale (HADS) by Zigmond and Snaith (1983; see Part 
G of questionnaire in Appendix VII). The scale includes 7 items for anxiety and seven for 
depression each ranging from 0 = no anxiety/depression to 21 = high anxiety/depression. 
Internal reliability for the anxiety and depression scales was satisfactory (alpha=.86 and 
.76 respectively).

5.2.4 Procedure

Patient recruitment took place in the cardiology outpatient clinic at Royal Surrey County 
Hospital. The procedure followed for patient recruitment and questionnaire administration 
was identical to that followed in Study 1-Part II (see Chapter 4).

5.2.5 Analyses

Correlations and factor and reliability analyses were conducted where appropriate in order 
to prepare the data for further analysis. Path analysis of the proposed model was 
conducted using LISREL, a structural equation modelling program. The maximum 
likelihood (ML) technique was used for parameter estimation, and the goodness of fit of 
the model with the observed data was estimated using a variety of fit indices reported by 
LISREL. Specifically, the model $\chi^2$, the Root Mean Square Error of Approximation Index
(RMSEA), the Non-Norm Fit Index (NNFI), the Standardised Root Mean Square Residual (SRMSR), and the Goodness of Fit Index (GFI) were used. The RMSEA looks at the precision of fit and should ideally be smaller than 0.05. The NNFI is a relative fit index that compares the specified model with a baseline model and also penalizes for model complexity. NNFI should ideally be greater than 0.90. The GFI compares the existing model fit with a null model and represents the percent of covariances explained by the covariances implied by the model. It should be higher than 0.95. Finally, the SRMR is based on standardised residuals, looks at the average difference between predicted and observed variances or covariances and should be smaller than 0.05.

Overall, well fitting models should have small and non-significant $\chi^2$ and as many of the other fit indices above or below the suggested cut-off point as appropriate.

Sample size considerations: Obtaining the correct sample size is very important in LISREL. The $\chi^2$ statistics used can be affected by sample size with too large samples risking unfounded rejection and too small risking unfounded acceptance. There are several suggestions of preferred sample size in the literature and rules of thumb are often used. Stevens (1996) suggests at least 15 cases per measured variable, while 50+(8*n) is commonly found in the literature with n meaning the number of variables in the model. An alternative suggestion is to have five participants per estimated parameter in the model. Here, with 15 variables in the model and 14 parameters to estimate, a sample size of 120-170 participants is adequate.
5.3 RESULTS

5.3.1 Participants

A total of 160 patients participated in the study. Of those, 115 were men (72%) and 45 were women (28%). Their mean age was 61.3 years (SD: 7.4 years). The mean time elapsed since diagnosis was 6.1 years (SD=9.4). The majority of patients (62%) were following up on an incident of myocardial infarction (MI) or unstable angina. The remaining participants (38%) were diagnosed with less aggressive, often asymptomatic conditions such as atrial fibrillation and high blood pressure. At least half the sample (51%) was on medication for their condition and approximately 40% had had Percutaneous Transluminal Coronary Angioplasty (PTCA) or Coronary Artery Bypass Graft Surgery (CABG).

5.3.2 Scales and reliabilities

Causal attributions: Principle Component Analysis with oblique rotation, revealed two components.

Table 5.1 shows the item communalities and pattern matrix.

Component 1 accounted for 34% of the variance (before rotation) and included all 5 behavioural attributions (i.e. smoking, unhealthy diet, being overweight, little or no exercise and stressful lifestyle) central to the purposes of the study. Reliability (Cronbach’s alpha) for the additive scale including items loading more than .3 on this component was .70. Component 2 accounted for 18% of the variance (before rotation) and included the attributions to heredity and chance. The two items correlated at \( r = -.25 \). The
two components had a low correlation of $r=-.19$. As explained earlier, this component was not relevant to the proposed model and its items were only offered to counterbalance the questions on the behavioural attributions. Therefore, it was not included in subsequent analyses.

Table 5.1: Item communalities and structural coefficients for causal attribution items.

<table>
<thead>
<tr>
<th>Item</th>
<th>Communalities</th>
<th>Component I</th>
<th>Component II</th>
</tr>
</thead>
<tbody>
<tr>
<td>unhealthy diet</td>
<td>.67</td>
<td>.82</td>
<td>-0</td>
</tr>
<tr>
<td>overweight</td>
<td>.53</td>
<td>.70</td>
<td>-0</td>
</tr>
<tr>
<td>smoking</td>
<td>.37</td>
<td>.62</td>
<td>.11</td>
</tr>
<tr>
<td>little exercise</td>
<td>.44</td>
<td>.67</td>
<td>-0</td>
</tr>
<tr>
<td>stressful life</td>
<td>.28</td>
<td>.48</td>
<td>-.16</td>
</tr>
<tr>
<td>heredity</td>
<td>.70</td>
<td>-.10</td>
<td>-.85</td>
</tr>
<tr>
<td>chance</td>
<td>.55</td>
<td>-.12</td>
<td>.71</td>
</tr>
</tbody>
</table>

Perceived control of risk factors: Principle Component Analysis with oblique rotation of the behaviour control items revealed two components. Table 5.2 shows the item communalities and pattern matrix. Component 1 included all the behaviour items (i.e. diet, smoking, stressful lifestyle and behaviour) and accounted for 33% of the variance. Component 2 included the non behavioural items (i.e. character, heredity and chance) and accounted for 20% of the variance. Cronbach’s alpha for component 1 was relatively low (alpha=.68) but not far from the acceptable levels. The two components had a low correlation of $r=-.13$. As already explained, Component 2 was not relevant to the scope of
this study and was excluded from further analysis. A composite score indicating perceived behaviour control was obtained but its effects were interpreted with caution due to its marginally acceptable reliability coefficient.

Table 5.2: Item communalities and structural coefficients for perceived control items.

<table>
<thead>
<tr>
<th></th>
<th>Communalities</th>
<th>Pattern matrix</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>I</td>
</tr>
<tr>
<td>smoking</td>
<td>.54</td>
<td>.74</td>
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<tr>
<td>behaviour</td>
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<td>.73</td>
</tr>
<tr>
<td>diet</td>
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<tr>
<td>stress</td>
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<td>.59</td>
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<tr>
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<td>.28</td>
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<tr>
<td>chance</td>
<td>.61</td>
<td>-.13</td>
</tr>
<tr>
<td>heredity</td>
<td>.58</td>
<td>-.0</td>
</tr>
</tbody>
</table>

5.3.3 Descriptives

Means and standard deviations for all the main variables in the study are shown in Table 5.3. In general, participants showed relatively low levels of both types of self-blame. Prior risk behaviour was reported at average levels on the 0-9 scale and levels of anxiety and depression were relatively low. Regarding reported behaviour change since the onset of the condition, smoking showed the largest positive change, followed by stress. People tended to perceive their condition as rather long lasting, of average impact on their lives, and quite controllable.
Table 5.3: Means and standard deviations of main variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Stand. Deviation</th>
<th>Min-Max</th>
<th>Potential range</th>
</tr>
</thead>
<tbody>
<tr>
<td>BSB</td>
<td>2.25</td>
<td>1.20</td>
<td>1-5</td>
<td>1-5</td>
</tr>
<tr>
<td>CSB</td>
<td>1.99</td>
<td>1.12</td>
<td>1-5</td>
<td>1-5</td>
</tr>
<tr>
<td>Behavioural attributions</td>
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<td></td>
</tr>
<tr>
<td>Behaviour Control</td>
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</tr>
<tr>
<td>Prior risk behaviour</td>
<td></td>
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<tr>
<td>Consequences</td>
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</tr>
<tr>
<td>Anxiety</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Behaviour change</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calorie controlled diet</td>
<td>1.08</td>
<td>1.35</td>
<td>(-3) – (4)</td>
<td>(-4) - (+4)</td>
</tr>
<tr>
<td>Fat consumption</td>
<td>.80</td>
<td>1.01</td>
<td>(-2) – (4)</td>
<td>(-4) - (+4)</td>
</tr>
<tr>
<td>Weight</td>
<td>~0</td>
<td>1.08</td>
<td>(-4) – (3)</td>
<td>(-4) - (+4)</td>
</tr>
<tr>
<td>Smoking</td>
<td>1.10</td>
<td>1.49</td>
<td>0-4</td>
<td>(-4) - (+4)</td>
</tr>
<tr>
<td>Exercise</td>
<td>-.40</td>
<td>1.48</td>
<td>(-4) – (4)</td>
<td>(-4) - (+4)</td>
</tr>
<tr>
<td>Stress</td>
<td>0.74</td>
<td>1.17</td>
<td>(-2) – (4)</td>
<td>(-4) - (+4)</td>
</tr>
</tbody>
</table>

122
5.3.4 Relationships among variables

Before examining the hypothesized paths between self-blame and its predictor variables, the relationships among all the variables were considered. As shown in Table 5.4, BSB showed a high to moderate significant correlation with CSB (r=.59, p<.01), behavioural attributions (r=.69, p<.01), prior risk behaviour (r=.51, p<.01), and change in diet and smoking habits (r=.34, p<.01 and r=.25, p<.01 respectively). Behavioural attributions followed a similar pattern. Also, there was no relationship between BSB and psychological adjustment (i.e. anxiety and depression) and there was only a weak correlation between anxiety and CSB (r=.20, p<.05).

The correlation between CSB and anxiety (r=.20, p<.05) verified, though weakly, the negative relationship between CSB and adjustment. Regarding BSB and adjustment, Janoff-Bulman (1992) suggests that this can be properly explored only by calculating the partial correlation of BSB and adjustment measures, controlling for CSB. When this was done however, no significant correlations were found between BSB and anxiety (r=-.12, p=.13) or depression (r=-.06, p=.43).
Table 5.4: Pearson's correlations ($r$) among the variables in the model (155<$N<$160)

<table>
<thead>
<tr>
<th></th>
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<th>2</th>
<th>3</th>
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<th>13</th>
<th>14</th>
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<tbody>
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<td>1. BSB</td>
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<td>2. CSB</td>
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<tr>
<td>3. Behavioural attr.</td>
<td>.69*</td>
<td>.52**</td>
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<td>4. Behaviour control</td>
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<td>-.03</td>
<td>-.08</td>
<td></td>
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<td>5. Prior risk</td>
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<td>.35**</td>
<td>.53**</td>
<td>-.09</td>
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<td>6. Consequences</td>
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<td>.14</td>
<td>.19*</td>
<td>.003</td>
<td>.19*</td>
<td></td>
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<td>7. Anxiety</td>
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<td>.06</td>
<td>-.18*</td>
<td>.10</td>
<td>.43**</td>
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<td>8. Depression</td>
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<tr>
<td>9. High calorie diet</td>
<td>.24**</td>
<td>.16*</td>
<td>.35**</td>
<td>.002</td>
<td>.36**</td>
<td>.16*</td>
<td>.08</td>
<td>.04</td>
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<tr>
<td>10. Fat consumption</td>
<td>.16*</td>
<td>-.02</td>
<td>.17*</td>
<td>.000</td>
<td>.16*</td>
<td>.10</td>
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<td>.15</td>
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<tr>
<td>11. Weight</td>
<td>.11</td>
<td>.09</td>
<td>.17*</td>
<td>.12</td>
<td>.26**</td>
<td>-.13</td>
<td>-.24**</td>
<td>-.16*</td>
<td>.03</td>
<td>.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Smoking</td>
<td>.25**</td>
<td>.27**</td>
<td>.33**</td>
<td>-.11</td>
<td>.37**</td>
<td>.29**</td>
<td>.04</td>
<td>.01</td>
<td>.17*</td>
<td>.22**</td>
<td>-.05</td>
<td></td>
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</tr>
<tr>
<td>13. Exercise</td>
<td>.07</td>
<td>-.05</td>
<td>.13</td>
<td>.18*</td>
<td>.29**</td>
<td>-.12</td>
<td>-.25**</td>
<td>-.21**</td>
<td>.16*</td>
<td>.10</td>
<td>.22**</td>
<td>.04</td>
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<td></td>
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<td>14. Stress</td>
<td>.09</td>
<td>.20*</td>
<td>.16*</td>
<td>.03</td>
<td>.18*</td>
<td>.02</td>
<td>-.19*</td>
<td>-.15</td>
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<td>.19*</td>
<td>.16*</td>
<td>.21</td>
<td>.06</td>
<td></td>
</tr>
</tbody>
</table>
5.3.5 Model testing

To allow for a more clear examination of the paths involved, the model was tested in three phases. First, the predictors of self-blame were examined by dividing the variables into two conceptually distinct sub-models (i.e. person characteristics vs. event characteristics). Each sub-model was examined for adequate fit and potential modifications were considered. Then, the sub-model including the outcome measures was examined and finally the sub-models were combined in one overall model tested globally.

The first sub-model (Figure 5.2) fit the data with $\chi^2=0.15$ (df=2, p=.921), RMSEA=~0, GFI=1.00, NNFI=1.04, SRMR=0.0038. Looking at the standardised solution (weights have been standardised to allow for variability in the measurement scales of variables), this sub-model shows that the higher the level of behavioural attributions the higher the level of self-blame ($\beta=.52$). Sex, here a dummy variable, is positively associated with the level of behavioural attributions ($\gamma=.20$) but negatively associated with BSB ($\gamma=-.28$). CSB has significant effects on BSB both directly ($\gamma=.25$) and indirectly through its effect on attributions ($\gamma=.40$). Prior risk behaviour had a significant indirect effect on BSB through behavioural attributions ($\gamma=.48$). Finally, the path coefficient for age was non significant ($\gamma=-.05$).
Figure 5.2: Path diagram of the relationships among person characteristics, illness attributions and BSB.

The second sub-model included the event characteristics (Figure 5.3). There was adequate goodness of fit with $\chi^2=3.31$ (df=4, p=.51), RMSEA= ~0, GFI=0.99, NNFI=1.02, SRMR=0.03. The type of diagnosis affected self-blame indirectly through its effect on behavioural attributions ($\gamma=.44$). In turn, behavioural attributions influenced BSB ($\beta=.68$). Perceived consequences had an indirect effect on self-blame through its effect on behavioural attributions ($\gamma=.13$). Time since diagnosis and behaviour control yielded positive but non-significant path coefficients.
Finally, the third sub-model containing the outcome variables was tested (i.e. the paths extending to the right of BSM and behavioural attributions in Figure 5.1). However, analysis of the fitted covariance matrix (i.e. Σ matrix) was not positive definite. Attempts to explain and address the problem of the non positive definite covariance matrix included examination of the following: high multicollinearity of the variables, outliers causing too much variability in the data, and Haywood cases (i.e. negative estimated error terms for an indicator of a latent variable). However, none of the above explained the problem. The problem persisted even after attempting to change the combination of the exogenous variables by excluding variables that appeared to correlate the least with the others. Proceeding with LISREL, when a matrix has been found to be non-positive definite may result in an arbitrary solution, therefore this section was excluded from the model and was analysed separately adopting a more exploratory, data-driven approach using regression analyses.
The remaining two sub-models were combined in a final overall test. The overall model included those variables with significant paths in the sub-models (i.e. sex, CSB, prior risk, diagnosis, and consequences) as the exogenous variables, and BSB and behavioural attributions as the endogenous variables. The final model (Figure 5.4) fitted the data well with $\chi^2=2.03$ (df=3, $p=.57$), RMSEA= ~0, GFI=1.00, NNFI=1.01, SRMR=0.01. According to it, there were three direct and five indirect significant paths to BSB.

Behavioural attributions, sex and CSB had a direct effect on BSB ($\beta=-.53$, $\gamma=-.28$ and $\gamma=.24$ respectively). All other effects on BSB were mediated by behavioural attributions. Specifically, there was a positive effect of sex ($\gamma=.35$), CSB ($\gamma=.29$), prior risk ($\gamma=.55$) and type of diagnosis ($\gamma=.44$) on behavioural attributions. Finally, there was a positive effect of illness consequences on behavioural attributions though the path coefficient was not statistically significant.

**Figure 5.4:** Final path model of predictors of BSB
Following examination of the model and confirmation of a good fit, it is important to examine alternative theoretical models that would fit the data equally well. An alternative model that could potentially change the theoretical understanding of self-blame is one where an additional path would indicate an effect of BSB on behavioural attributions (i.e. creating a loop where BSB ‘causes’ behavioural attributions which in turn ‘cause’ BSB). When this model was tested the fit improved even further ($\chi^2 = .73$, $df=2$, $p=.69$) but the added path was not significant. Since the improved fit was apparently due to loss in degrees of freedom and model parsimony was compromised, the alternative model was not considered adequate.

5.3.6 Outcome measures

As mentioned earlier, the use of LISREL was not possible for this section of the model due to the covariance matrix being non positive definite. Since attempts to remedy the problem were not successful, it was decided to proceed with regression analysis. It must be noted however, that results in this section should be interpreted with caution since the above problem would theoretically stand for regression analysis as well.

Eight hierarchical regressions, one for each outcome measure in the study, were run. In order to examine the unique contribution of BSB to the outcome variables, behavioural attributions were entered first on a separate block. As shown in Table 5.5, significant regression equations were obtained when changes in diet, fat consumption and smoking were each regressed on behavioural attributions and BSB. For diet and fat consumption change behavioural attributions explained approximately 12% of the variance while for
smoking they explained 10% of the variance. No significant regression models were obtained for exercise and weight change, anxiety and depression.

Table 5.5: Hierarchical regressions of behaviour change and psychological adjustment variables on attributions and BSB.

<table>
<thead>
<tr>
<th>Model</th>
<th>F</th>
<th>Adj. R²</th>
<th>Beta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caloric diet change</td>
<td>10.66**</td>
<td>.12</td>
<td>.35**</td>
</tr>
<tr>
<td>Attribution</td>
<td></td>
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</tr>
<tr>
<td>BSB</td>
<td></td>
<td></td>
<td>-.002</td>
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<tr>
<td>Fat consumption</td>
<td>2.57</td>
<td>.02</td>
<td>.11</td>
</tr>
<tr>
<td>Attribution</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BSB</td>
<td></td>
<td></td>
<td>.09</td>
</tr>
<tr>
<td>Weight change</td>
<td>2.21</td>
<td>.03</td>
<td>.17</td>
</tr>
<tr>
<td>Attribution</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BSB</td>
<td></td>
<td></td>
<td>-.01</td>
</tr>
<tr>
<td>Smoking change</td>
<td>9.31**</td>
<td>.10</td>
<td>.29**</td>
</tr>
<tr>
<td>Attribution</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BSB</td>
<td></td>
<td></td>
<td>.05</td>
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<tr>
<td>Exercise change</td>
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<td>.01</td>
<td>.15</td>
</tr>
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<td>Attribution</td>
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<td></td>
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<td>BSB</td>
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<td></td>
<td>-.04</td>
</tr>
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<td>Anxiety</td>
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<td>.01</td>
<td>.08</td>
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<td></td>
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<tr>
<td>BSB</td>
<td></td>
<td></td>
<td>-.03</td>
</tr>
<tr>
<td>Depression</td>
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<td>.01</td>
<td>-.06</td>
</tr>
<tr>
<td>Attribution</td>
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<td></td>
</tr>
<tr>
<td>BSB</td>
<td></td>
<td></td>
<td>.06</td>
</tr>
</tbody>
</table>

5.4 DISCUSSION

The present study proposed a theoretical model of behavioural self-blame and attempted to clarify its position in the attributional process for illness. It was proposed that BSB is mainly a result of causal attributions for one’s illness which in turn can be influenced by a
number of patient and event characteristics. It was also proposed that some of these characteristics can influence BSB directly without the mediating role of attributions.

The proposed model fit the data well and revealed that as expected, three variables directly affect BSB: the patient's sex, CSB, and the level of behavioural attributions themselves. More specifically, while being male was positively associated with behavioural attributions, it was negatively associated with BSB. This indicates that while men tend to acknowledge behaviour attributions more strongly, they do not feel as blameworthy as women. This finding could be a reflection of the inaccurate but popular preconception that heart disease predominantly affects men. Men find it easier to accept the illness and the behaviours that may have contributed to it without feeling blameworthy about it. Regarding CSB, the model showed it can have a direct positive effect on self-blame but also an indirect effect through behavioural attributions. This is consistent with the definition of CSB as a more stable internal assignment of blame that is often accompanied by depression and can thus affect both the attribution of causality and the evaluation of one's role in bringing about those causes. Finally, verifying the claims of Brewin (1985), BSB is directly affected by behavioural attributions. The model depicts and verifies the notion that after one identifies one's action as the cause of an event, one can then engage in an evaluation of that action and decide how instrumental he or she really was in causing the event.

Regarding the other predictor variables in the model, the type of diagnosis and the amount of risky health behaviour prior to the illness appear to have an indirect effect on BSB through their effect on attributions. It is plausible that suffering from a condition that is
associated with a number of risk behaviours or having actually adopted those behaviours in the past will make a series of behavioural causal attributions available to the patient during the search for explanation and meaning.

The perceived consequences of the illness did not prevent the model's fit but also did not yield statistically significant paths. Illness consequences or perceived severity have been previously linked with causal attributions (e.g. Tennen, Affleck & Gershman, 1986) so it was theoretically unfounded to delete the paths. However, more research is needed to establish their true contribution to the model.

Finally, age, time since diagnosis and perceived control had to be excluded from the model. In the present sample, these variables did not correlate with the remaining variables of the model and prevented further analysis. Since some evidence exists that justified their inclusion in the model, more research is needed to clarify their role.

Regarding time since diagnosis in particular, it has failed to correlate with self-blame in all samples examined so far in the thesis. It therefore seems possible that the only timing that may affect self-blame (and its relationship to adjustment) is right after the actual event or diagnosis. At that time, the search for an explanation is peaking, the situation makes the individual particularly vulnerable to anxiety and depression and self-blame may be found to affect adjustment levels. Soon after that, and once an explanation has been adopted, the effects of timing possibly diminish.

After examining a slightly different model, one that shows a bi-directional relationship between BSB and attributions, the relationship between attributions and self-blame was
further clarified. It was found that while statistically the model fit the data even better, its parsimony was affected by adding a non-significant path. Therefore, to the extent that one can rely on the data and accept the non-significance of the added path, the original view of BSB as an evaluation step that follows the causal search is supported and is consistent with evidence in the literature (e.g. Martin & Lee, 1992).

Regarding the outcome variables originally included in the model, LISREL path analysis was not possible due to calculations resulting in a non-positive definite covariance matrix. This meant that the values in the calculated matrix were such that prevented certain algebraic calculations (e.g. inversion of matrix may be impossible due to division by zero values). Thus no conclusion could be reached through this type of analysis. However, following a data driven approach, it was found that the recently proposed relationship between BSB and behaviour change (Christensen et al., 1999) can be explained by the role of behavioural attributions in the behaviour modification process. According to the regression analyses changes in diet and smoking are predicted by one's behavioural attributions rather than one's levels of BSB. This suggests that behaviour change is not necessarily a result of an evaluation of one's causal behaviour. Acknowledging the risk one was in before the illness and subsequently changing his or her behaviour may be a result of pressure or compliance. Also, it must be noted that since no objective measures of behaviour change were obtained the above relationship should be interpreted with caution. It may be the everyday intention to change rather than actual change that is reflected in these measures. Clearly, more work is required to ascertain the role of attributions and self-blame in behaviour change, especially when considering the potential benefits it may have on clinical application (Michela & Wood, 1995).
In evaluating the methodology and findings of the present study, a number of strengths and limitations can be identified. One of the study’s strengths is the fact that, to the best knowledge of the researcher, it is the first study to explore the predictors of BSB and propose a comprehensive theoretical model of illness attributions and self-blame. With the majority of studies focusing on the relationship between self-blame and adjustment and the inconsistencies regarding the methodology and the findings, the need for a broader approach to self-blame was necessary. This study clarifies not only the role of BSB in relation to causal attributions, but also the conditions under which they both occur in the context of the illness experience. Also, it follows up on the idea of a relationship between self-blame and behaviour change and offers an alternative explanation. Finally, it attempts a shift from a purely data-driven approach (though such an approach was in one instance inevitable) to understanding self-blame towards a theory-driven one that may prove more promising in resolving some of the inconsistencies.

Looking at the limitations of the study, it must be noted that the model tested in this study is only one of a potentially infinite number of models that could fit the data. Accepting a model in LISREL means that the model is plausible, not true. According to Maruyama (1998) ‘data never can confirm a model; they can only fail to disconfirm it’ (pp. 272). Further validation of the model is required, preferably with larger samples and alternative models ruled out before the present model can be irrevocably accepted. Finally, the present model tested a large number of parameters with few degrees of freedom. Its parsimony was therefore compromised and goodness of fit was easier to achieve. A more
parsimonious model would have been more desirable and theoretically more significant, but the evidence available was not clear enough to support a more economical model.

Another limitation of the present study is the measures of behaviour change. Both the baseline and the ‘since the onset’ measures of health behaviour are self-reports and were not validated by more objective indices (e.g. actual weight entries). Therefore, they are subject to biases and may not reflect the true change in participants’ health behaviours. If a link between self-blame, attributions and behaviour change is to be explored further, it is important that it be accompanied by objective measures of the behaviour in question so that effects can be assessed more precisely.

To sum up, this study has examined the predictors of self-blame and has provided evidence of a unidirectional relationship between attributions and BSB. It has also examined the effects of attributions and self-blame on perceived behaviour change and has offered both conceptual and empirical clarifications. In the following chapters, a comparison between patient and non-patient populations on issues of self-blame are examined and the role of the illness experience is juxtaposed with the illness representations of the general public to investigate the potential role of preconceived notions of responsibility in illness and misfortune in the attribution process.
SUMMARY
This chapter introduces two studies investigating self-blame in non-patient populations. Specifically, it presents the common background and method of the studies given in Chapters Eight and Nine as well as the research questions investigated. The main issues raised and explored in this and the following chapter are a) whether self-blame is a response observed only in people dealing with a traumatic event (possibly due to their need to enhance feelings of control), b) whether there is grounds for generalising the original findings on self-blame in victims of different misfortunes, and c) whether there is indeed reason for concern regarding the interchangeable use of the terms of ‘blame’, ‘responsibility’, control, and ‘avoidability’ as it has been suggested in the literature.

6.1 INTRODUCTION
So far, the evidence presented in this thesis points towards a situational conceptualisation of behavioural self-blame. In other words, it has been shown that the intensity of self-blame depends largely on factors such as the patient’s lifestyle prior to illness, the relevance of that lifestyle with the condition suffered and so on. Thus, it could be said that in patient populations self-blame is a result of a ‘rational’ or ‘pragmatic’ exploration of the potential factors that may have lead to the illness and a recognition of the link (or lack of) between a given illness and lifestyle. In this chapter, the focus shifts from patient to non-patient
populations and the plausibility of the above approach is examined by looking at the illness representations of people free of illness.

Therefore, the next phase of the present research explored four main issues. The first is whether the relationship between lifestyle risk factors and behavioural self-blame found in patients is true for non-patient populations. If it is, then an indication will exist that self-blame is a result of a more generalised way of thinking common both to patients and non-patients.

Second, in line with the previous issue, an exploration is presented of whether the tendency for self-blame is evident only in patients when compared to their non-patient counterparts. If the explanation of behavioural self-blame as one's way of increasing one's sense of control is correct (Janoff-Bulman, 1992) then it is expected that behavioural self-blame for a particular event or illness will be more evident in people whose sense of control has been challenged (i.e. patients). Despite lack of relevant evidence in the previous studies of a relationship between controllability and self-blame, perceived controllability is included in this study in order to investigate comparatively (i.e. between patients and non-patients) its potential role in illness explanations.

The third issue examined is that of perceptions of self-blame regarding different negative events. Specifically, researchers appear to assume that Janoff-Bulman's theory, which originated from work on paralysed victims of severe car accidents and rape victims, is applicable to other situations such as chronic illness patients and seek to replicate her results without ensuring that situations are indeed comparable. It is thus important to investigate
how similarly or differently various negative events are perceived, and to evaluate the extent
to which treating different misfortunes as comparable negative events indeed reflects the
representations of the general public.

Finally, from a more methodological perspective, the study will investigate the similarities
and differences between the concepts of blame and responsibility, and control and
avoidability. As reviewed earlier in the thesis, Shaver & Drown (1986) claim that
differentiating among causality, responsibility and blame in research and application may
affect the construct validity of ‘self-blame’. Using the terms interchangeably may reduce
clarity and thus contribute to a theoretical vagueness resulting in lack of consistency and
predictive value. Although a literary analysis of the term may support the authors’ views, it
is also important to examine the participants’ understanding of the term. Being able to
differentiate between responsibility, blame and cause does not imply that when presented
with each term alone participants have its literal meaning in mind. For example, asking
patients with a history of an unhealthy lifestyle whether they blame themselves for their
current illness may not be inappropriate if, in retrospect, ‘not doing the right thing’ seems to
them just as culpable as an intentionally harmful act. Moreover, Shaver & Drown (1986) do
not provide a comprehensive review of the literature and consequently offer only
suggestions on how the relevant concepts should be approached, rather than evidence on
research findings. Indeed, there is contradicting evidence that participants actually
differentiate among the three terms of responsibility, causality and blame (e.g. Wortman,
To conclude, the study presented in the following two chapters aimed at investigating the following research questions:

1. When matched on all relevant characteristics, do patients and non-patients show a similar tendency for self-blame for a given illness?

2. What are people's representations of negative events along the dimensions of responsibility, blame, control and avoidability? Are the resulting patterns for each dimension similar to each other?

3. Are there any differences in people's perceptions of different types of events (e.g. illnesses, accidents etc), or are there any other underlying characteristics of events (e.g. severity) that may influence their representations?

4. Do people distinguish among the concepts of responsibility and blame, and control and avoidability? How likely is it that these concepts are used interchangeably?

6.2 METHOD

6.2.1 Participants and recruitment

A random sample of 1000 members of the public residing in the Surrey and London area was contacted by post. The sample's information had been previously purchased by the Department of Psychology, University of Surrey from a market research support company.

In a cover letter accompanying the questionnaire, recipients were given information about the aims of the study and how their address was obtained. They were informed about issues of anonymity and confidentiality, and were encouraged to fill in the questionnaire survey
and return it within two weeks. Due to financial constraints, no reward was offered for participation and no reminder letters were sent.

For the purposes of the matched-pairs design of the study, the patient sample presented in Chapter Six was used. Patients and non patients were matched on gender, occupation and lifestyle risk factors. Gender and lifestyle risk factors were considered because they were found to have a significant effect on self-blame in the studies reported earlier in this thesis. Occupation was considered due to suggestions (but no conclusive evidence) in the literature that socio-economic factors may affect people’s perceptions of control and responsibility, with people of lower socio-economic status (SES) feeling less responsible or in control of unhealthy behaviours (Blaxter, 1993). Because data in previous studies in the thesis was not detailed enough on this to allow for an examination of the relationship between self-blame and socio-economic status, participants in this study were matched on this variable to account for potential effects.

6.2.2 Measures

A six-page questionnaire was used that consisted of two parts. Part I included the measures that were used in the patient-non-patient comparisons (reported in Chapter 7). Part II included the measures used to assess non-patients’ illness representations on the dimensions of responsibility, blame, control and avoidability (reported in Chapter 8). The questionnaire is included in Appendix VII.

Self-blame: In order to answer the first two research questions of this study, measures of self-blame, lifestyle risk factors and perceptions of control were assessed. However, since
this group had not experienced any serious illnesses for which self-blame could be assessed, a hypothetical scenario was presented. Specifically they were presented with the following instructions:

Imagine yourself in the following situation: You have recently been experiencing disturbing chest pains. After being admitted to the hospital and undergoing the appropriate examinations, you are diagnosed as suffering from heart disease. Keeping in mind that in all other respects your life is exactly the same as in reality, please read the following statements carefully and respond by ticking (✓) the box that best describes how you feel.

Following these instructions, participants were asked to provide ratings of behavioural and characterological self-blame (using the same items as Chapter Five).

**Behavioural risk factors:** In order to assess participants’ degree of risky lifestyle, they were asked to indicate the degree to which they engaged in certain risk behaviours associated with heart disease (i.e. being overweight, following a calorie controlled diet, eating foods high in fat/fibre/sugar, exercising regularly, smoking cigarettes, leading a stressful life, and leading a physically active life). The scale used was identical to the scale used for the study reported in Chapter 5, with responses ranging from 1=“not at all” to 5=‘a lot’. As in Chapter 5, a 9-point risk index was calculated following re-coding of values where appropriate. Every response equal or higher than 3 was considered an indication of a risky behaviour and was given a point of 1. Responses equal or lower than 2 were considered an indication of little or no risk and were given a point of 0. Thus, a composite index score was obtained that ranged from 0 (little or no risk) to 9 (high risk).
Perceived controllability of health behaviours: Participants perceived controllability over the above health behaviours as well as their general behaviour and character were assessed. Each controllability item was scored on a 5-point scale ranging from ‘strongly disagree’ (1) to ‘strongly agree’ (5) as in Chapter Five.

Illness representations and perceptions of blame, responsibility, control and avoidability: In order to investigate research questions 3-4, participants were asked to rate a list of negative events on a number of scales regarding issues of responsibility and control. Specifically, a total of fourteen different negative events identified from the literature on self-blame were used as the stimuli in this study. These events included illnesses (e.g. AIDS, heart disease), violent events (e.g. rape) and other potentially life threatening situations (e.g. severe burn injuries; see APPENDIX VIII). In order to increase the measure’s sensitivity to participants’ representations of the events, slightly different aspects of the same event were included on the list. For example, rather than including ‘cancer’ as a single, general event, two types of cancer varying in the degree of associated personal involvement were included (i.e. breast cancer and lung cancer).

The 14 events were rated in response to 4 different questions. These were the following:

1) How much do you think the sufferer is responsible for causing the event?

2) How much do you think the sufferer is to blame for the event?

3) How much do you think the sufferer could have avoided the event?

4) How much do you think the sufferer has control over the cause of the event?
Answers to these questions were given on a five-point scale with 1='not at all' and 5='completely'. Participants' responses were then analysed separately for each of the four questions using Cluster analysis and multi-dimensional scaling.

Finally, participants were asked to report whether they had ever experienced one or more of the negative events on the list.

6.2.3 Choice of Analyses

Pearson’s correlations, regression analysis and t-tests were used in order to investigate research questions 1 and 2. A path analysis testing the model proposed in the previous chapter was also considered but was dismissed due to the relatively small response rate and consequently sample size of the survey (see relevant Results section).

Multidimensional Scaling (MDS) was used to investigate participants’ representations of the 14 negative events. Interpretation of each plot was assisted by cluster analysis as suggested by Shephard, Romney and Nerlove (1972). These methods of analysis were chosen because of their ability to provide measures of similarity and/or proximity in a clear way. In MDS, data are represented in a readily interpretable and communicable visual form (i.e. plots of points in multidimensional space) and few assumptions about the data are made. The aim of MDS is to identify distinct regions in the output plot that can reveal meaningful partitions of the space. Cluster analysis was used to discover natural groupings based upon the original data and ensure an objective way of interpreting and labelling the
results. Also since cluster analysis is a non-dimensional method (Shephard et al, 1972) it can provide solutions that are not captured in two- or three- dimensional space.

Finally, in order to test for significant differences between perceptions of a) blame and responsibility and b) avoidability and control for the 14 negative events a series of t-tests was performed.

For purposes of clarity and organization the findings of the present study are presented in two separate chapters. Chapter Seven presents the findings and discussion regarding research questions 1 and 2. Chapter Eight presents the findings and discussion of research questions 3 and 4.
CHAPTER SEVEN

INVESTIGATING SELF-BLAME IN NON-PATIENTS:
COMPARISONS WITH PATIENTS

SUMMARY

This chapter presents the results of the first part of a study using a non-patient sample to study self-blame. Specifically, a comparison of self-blame levels is presented between patients and non-patients matched on all relevant characteristics. A total of 85 pairs were studied and results showed higher levels of self-blame (both BSB and CSB) in the non-patient group as well as different regression models with behavioural risk factors predicting BSB only in the patient group. Possible explanations of these findings are discussed and the limitations of the study are considered.

7.1 ANALYSES AND RESULTS

7.1.1 Participants

One hundred and forty-four (N=144) non-patients responded to the questionnaire giving a response rate of approximately 14%. This was not surprising, considering the limitation in resources that did not allow for follow-up reminders or calls to maximize response rates. Of the 144, five reported having experienced one of the negative events listed in the grid and were excluded from the analysis leaving a sample of 139. Of these, 47 were men and 92 were women. Mean age for the overall sample was 58.6 (SD: 12.8) years.
For the analyses reported in the present chapter, this sample was juxtaposed to the patient sample reported in Chapter Five. Patients and non-patients were matched on personal characteristics found in the previous study to be predictors of self-blame. Specifically, of the five factors seen as predictors of BSB in the final model of Chapter Six (i.e. sex, CSB, risk behaviour, diagnosis and consequences), participants were matched on sex and behavioural risk factors. CSB was treated here as a dependent variable for exploratory purposes so matching was not appropriate. Another variable that was considered for matching was participants’ occupation as an indicator of socio-economic status (SES) using the major groups of the Standard Occupational Classification (SOC 2000).

Although analyses in both samples (as well as data presented in previous chapters) did not reveal any relationships between SES and self-blame or lifestyle risk factors, SES was taken into account to ensure similarity in the general lifestyle of participants and the extent to which that can affect their perceptions of and knowledge about illness. Hence, although SES was not allowed to limit the matching only to pairs with corresponding SES, it was allowed to guide the matching procedure as much as possible. To illustrate the way matching criteria were applied, a woman credit controller with a lifestyle index of 6 would be allowed to match with a woman medical secretary with a lifestyle index of 6 since both occupations fall under the general group of ‘administrative and secretarial occupations’.

A total of 85 matched pairs were found in the manner described above. As shown Table 7.1, 27 pairs matched perfectly on sex, behavioural risk and occupation, 18 pairs matched on sex, behavioural risk and occupation class, and 40 pairs matched only on sex and behavioural risk.
Table 7.1: Matching characteristics of the samples in this study. Perfect matching is denoted by (\checkmark).

<table>
<thead>
<tr>
<th>N pairs (patient-non-patient)</th>
<th>Sex</th>
<th>Behavioural Risk</th>
<th>Exact occupation</th>
<th>Occupation class</th>
</tr>
</thead>
<tbody>
<tr>
<td>27</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>√</td>
<td>√</td>
<td></td>
<td>√</td>
</tr>
<tr>
<td>40</td>
<td>√</td>
<td>√</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Once all possible matched pairs had been obtained, the matched sample’s characteristics were obtained. As a whole, the sample included participants with a mean age of 58.1 (SD: 12). The most frequent occupation category reported was professionals. Table 7.2 shows a breakdown of the main characteristics for the patient and non-patient groups.

Table 7.2: Demographic characteristics of patients and non-patients.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients</th>
<th>Non-patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>85</td>
<td>85</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>39</td>
<td>39</td>
</tr>
<tr>
<td>Female</td>
<td>46</td>
<td>46</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>61.0</td>
<td>55.2</td>
</tr>
<tr>
<td>SD</td>
<td>7.3</td>
<td>16.7</td>
</tr>
</tbody>
</table>
Table 7.2 (Cont.): Demographic characteristics of patients and non-patients.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients</th>
<th>Non-patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Occupational Classification</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Managers &amp; Senior Officials</td>
<td>15</td>
<td>24</td>
</tr>
<tr>
<td>Professional</td>
<td>7</td>
<td>14</td>
</tr>
<tr>
<td>Associate Professional</td>
<td>6</td>
<td>12</td>
</tr>
<tr>
<td>Protective Service</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Administrative &amp; Secretarial</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Skilled Trades</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Personal Service</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Process, Plan &amp; Machine Operatives</td>
<td>-</td>
<td>3</td>
</tr>
<tr>
<td>Elementary Occupations</td>
<td>11</td>
<td>6</td>
</tr>
</tbody>
</table>

7.2 DIFFERENCES BETWEEN PATIENTS AND NON-PATIENTS

The means and standard deviations of the main variables in the study are presented in Table 7.3. It can be seen that non-patients have generally scored higher on all variables except perceived controllability of stress and character.

In order to check for significant differences in self-blame between patients and non-patients, matched-pairs t-tests were used. Significant differences were found for both BSB ($t=2.98$, df=166, $p<.01$) and CSB ($t=2.23$, df=166, $p<.05$), with patients scoring lower than non-patients. However, due to the large positive correlation between BSB and CSB, the extent to which the above t-tests were examining two different types of self-blame or group differences on essentially one variable could be seen as debatable.

Therefore, a multivariate analysis of variance (MANOVA) was appropriate. Lack of a
priori power analysis for the particular test, and in light of the small numbers of participants in each group, the test was to be interpreted with caution. The analysis was carried out with BSB and CSB as the dependent variables and the type of population (i.e. patients, non-patients) as the independent variable. Results showed that there were no significant main effects for either BSB (F(1, 91)=2.87, ns) or CSB (F(1,91)=.88, ns). Since the results of the MANOVA could be due to lack of statistical power, it was concluded that they would be used as indications for caution when interpreting the results of the matched-pairs t-tests.

Regarding differences in perceived controllability of risk factors, independent sample t-tests revealed that no significant differences between the two groups.

**Table 7.3:** Mean and standard deviation of the main variables in the study for each group.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patients</td>
<td>Non-Patients</td>
</tr>
<tr>
<td>BSB</td>
<td>2.01 (1.15)</td>
<td>2.51 (1.01)</td>
</tr>
<tr>
<td>CSB</td>
<td>1.85 (1.02)</td>
<td>2.21 (1.12)</td>
</tr>
<tr>
<td>Controlability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet</td>
<td>4.17 (.64)</td>
<td>4.24 (.77)</td>
</tr>
<tr>
<td>Smoking</td>
<td>4.42 (.81)</td>
<td>4.62 (.79)</td>
</tr>
<tr>
<td>Stress</td>
<td>3.12 (1.03)</td>
<td>3.07 (1.05)</td>
</tr>
<tr>
<td>Behaviour</td>
<td>3.63 (.89)</td>
<td>3.85 (.85)</td>
</tr>
<tr>
<td>Character</td>
<td>2.51 (.97)</td>
<td>2.42 (1.13)</td>
</tr>
</tbody>
</table>

Note: *p<.05, **p<.01
7.2.1 Relationships between self-blame, lifestyle risk and control: differences in patterns

When looking at the relationships between self-blame (both BSB and CSB) and lifestyle risk, some interesting differences were detected. As shown in Table 7.4, BSB is positively correlated with lifestyle risk for both patients and non-patients ($r=.25$, $p<.05$ and $r=.23$, $p<.05$ respectively). CSB correlated significantly with lifestyle risk only for the non-patient group. Because of the strong correlation between BSB and CSB, partial correlations were calculated between BSB and lifestyle risk in non-patients, controlling for CSB. It was found that the relationship between BSB and lifestyle risk was no longer significant ($r=.12$, $N=81$, $p=ns$).

Furthermore, a significant negative correlation was found between BSB and stress avoideability in the patient group ($r=-.31$, $N=84$, $p<.01$) but not in the non-patient group. This means that patients who view a stressful lifestyle as avoidable tend not to blame themselves for their illness, a pattern not evident in the non-patient sample. However, when CSB (which also shows a negative correlation with perceived stress avoidability) is controlled for, the relationship between BSB and stress avoidability becomes non-significant ($r=-.14$, $df=81$, $p=ns$). It was found, thus, that it is CSB that is responsible for this relationship yielding a negative correlation of $r=-.23$, $N=81$, $p<.05$ when BSB is controlled for.
Table 7.4: Pearson’s correlations between self-blame, lifestyle risk and perceived control for patient and non-patient groups (82<N<85).

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<tr>
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<th>1</th>
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<td>.45**</td>
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<tr>
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<td>.24*</td>
<td>.23*</td>
<td>.27*</td>
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<td></td>
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<td>.01</td>
<td>-.11</td>
<td>.27*</td>
<td>.07</td>
<td>.40**</td>
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<td><strong>8. CHARACTER</strong></td>
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<td>.02</td>
<td>.40**</td>
<td>.41**</td>
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<td>-.05</td>
<td>.25*</td>
<td>.24*</td>
<td></td>
</tr>
</tbody>
</table>

Note: * p<.05, **p<.01

Variables were entered in two separate hierarchical multiple regressions -one for each group- in order to compare the strength that the independent variables had in predicting BSB in each group. In each regression, CSB was entered as the first step (block), lifestyle risk as the second, and all five control items as the third. Results (Table 7.5) showed that for both groups, CSB was a significant predictor of BSB (beta=.58, p<.001 for patients and beta=.57, p<.001 for non-patients). Lifestyle risk was a significant predictor of BSB
only for the patient group (beta=.28, p<.01), and control items did not predict BSB for either group.

Table 7.5: Hierarchical regressions of BSB on CSB, lifestyle risk and control items for both groups.

<table>
<thead>
<tr>
<th></th>
<th>Patients</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>Non-patients</th>
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<tbody>
<tr>
<td></td>
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<td>t</td>
<td>Adj. R^2</td>
<td>F</td>
<td>Beta</td>
<td>t</td>
<td>Adj. R^2</td>
</tr>
<tr>
<td>CSB</td>
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<td>6.24**</td>
<td>.40</td>
<td>8.74**</td>
<td>.57</td>
<td>6.07**</td>
<td>.35</td>
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<td>Risk</td>
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<td>3.14**</td>
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</tr>
<tr>
<td>Diet</td>
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<td>Behaviour</td>
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<td>-.66</td>
<td></td>
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<td>.01</td>
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</tbody>
</table>

Note: * p<.05, **p<.01

7.3 DISCUSSION

The first part of the present study looked at similarities and differences in self-blame and its correlates between patients and non-patients. Using a matched-pairs design, it examined whether the groups differ a) in the amount of self-blame they are ready to accept regarding an illness (i.e. heart disease) and b) the predictors of behavioural self-blame.
Regarding differences in self-blame, there was some evidence that non-patients were ready to blame themselves for a potential heart attack more than their patient counterparts. This was true for both behavioural and characterological self-blame. However, as mentioned in the results section, these findings were subject to certain statistical limitations. Therefore the discussion that follows should be seen as an opportunity to address potentially relevant theoretical issues rather than a particular interpretation of evidence.

The explanation of the differences between the matched groups may lie in the actual illness experience. Specifically, non-patients responded to the hypothetical scenario using an illness representation formed through ways other than the actual illness experience. They do not have a personal experience to draw from and thus form their illness perceptions indirectly (e.g. through the experiences of others) resulting in more crude conclusions about blame and responsibility issues. In contrast, patients, having gone through the illness experience, have been exposed to the actual situation, and are more aware of the factors it involves. The above point could be illustrated using the example of acquisition of information about a chronic illness. Non-patients are likely to receive information on a disease quite passively, as targets of health campaigns or by exposure to media coverage. The information they receive is likely to be simplified in order to be generalisable to the public, and often out of context. In contrast, patients diagnosed with a specific condition, possibly having undergone diagnostic tests yielding a specific patient profile, are likely to receive more accurate, detailed and specific information about their condition and what caused it. They may also be more likely to have a personal interest in fine-tuning their knowledge since it can directly affect the progression of their illness.
Therefore, it is possible that the difference detected in this study is due to different knowledge about illness and perhaps different illness representations.

The differences in the regression models found in the two groups provide some support for the above explanation. In non-patients, lifestyle risk did not predict BSB indicating that, though scoring higher on self-blame, non-patients did not take into account their own lifestyle and possible risk behaviours when responding to the self-blame items. In contrast, patients replied on a more pragmatic basis, and their lifestyle risk score predicted their BSB scores.

The present findings, however, can be interpreted through the scope of other theoretical perspectives as well. The Just World Hypothesis (Lerner, 1980) and other theories stressing the importance of control over future events (e.g. Wortman, 1976) could propose that non-patients scored higher on self-blame driven by the need to attribute a common and potentially fatal disease to a controllable agent (i.e. themselves) regardless of whether their lifestyle justified making such a connection. In contrast, patients may have had these assumptions challenged and made more accurate attributions using actual knowledge drawn from their experience.

Conversely, the different levels of self-blame between patients and non-patients may be seen as patients' tendency to underestimate the degree to which their health behaviour and lifestyle contributed to their illness, compared to their non-patient counterparts. Consistent with coping literature but in contrast to the control-enhancing theoretical explanations of self-blame in victims of misfortune (Janoff-Bulman, 1992), it may be that
patients engage in a denial-like pattern of causal thinking and downplay the role of personal health behaviour in their illness in order to avoid guilt or feelings of responsibility. Although this explanation is possible, it does not seem very likely when the results of previous studies in this thesis and timing issues in the patient group are considered. Specifically, the evidence presented so far in the thesis suggests a relationship between lifestyle involvement and self-blame (Chapters Four and Five) with higher lifestyle involvement implied by an illness relating to higher levels of self-blame. If coping or defence mechanisms were in play, one would expect similar levels of self-blame in different patient groups, unless there were reasons to believe that these mechanisms were used more by some patient groups and less by others. Moreover, coping and defence mechanisms are typically used relatively early in stressful situations and tend to diminish once adjustment has been achieved (Lazarus, 1966; Folkman & Lazarus, 1980). With the mean time since diagnosis for the patient group being approximately five years and psychological adjustment scores at acceptable levels, there would be no reason to expect any coping or defence mechanisms in operation at the time of data collection.

Clearly, more work is needed to explore the possible underlying mechanisms behind these intriguing differences.

There are certain limitations in this study that should be noted. First, the low response rate of the non-patient population limits the generalisability of results. However, the sample size is large enough to provide adequate statistical power of the results and warrant further investigation. Second, the variables on which the patients were matched with non-patients are sufficient only to the extent that the studies presented earlier in the thesis are reliable and valid. Since, to the best knowledge of the author, there are no other studies available
that investigate the determinants of self-blame, pair-matching in this study had to rely on the results of the path analysis in Chapter Six. A potentially better way (but perhaps not as feasible methodologically) of examining those differences would be to use a within-subjects repeated measures design and look at people's tendency for self-blame regarding an illness before and after a diagnosis.
CHAPTER EIGHT

PERCEPTIONS OF BLAME, RESPONSIBILITY AND
CONTROL FOR NEGATIVE LIFE EVENTS IN NON-PATIENTS

SUMMARY

This chapter explores people's understanding of negative events and investigates the way
the represent them in terms of blame, responsibility, avoidability and control dimensions. A
list of 14 negative events including accidents and illnesses was presented to the same
participants described in the previous chapter, who were asked to rate them on the above
dimensions. Data was analysed using Cluster Analysis and Multidimensional Scaling.
Results showed a clear differentiation between illnesses and other negative events in
people's representations. Also, negative events were grouped along the dimensions
according to the levels of responsibility in a manner that verified earlier findings in the
thesis about the relationship between lifestyle involvement and perceptions of blame.
Results are discussed in reference to theoretical and methodological criticisms presented
earlier in the thesis.

8.1 ANALYSES AND RESULTS

For this phase of the study, the entire sample of non-patients (N=139) presented at the
beginning of Chapter Eight was used. As mentioned, the sample consisted of 47 men and 92
women. The mean age for the overall sample was 58.6 (SD: 12.8) years.

Looking at the mean ratings of the 14 events on responsibility, avoidability, control and
blame (Table 8.1), it can be seen that AIDS had the highest means on all four dimensions
followed by lung cancer. Childhood diabetes had the lowest scores followed by breast cancer and then rape.

**Table 8.1:** Means and standard deviations of ratings of responsibility, avoidability, control and blame for each negative event (events with the lowest and highest mean are in bold).

<table>
<thead>
<tr>
<th>Event</th>
<th>Blame mean</th>
<th>Responsibility mean</th>
<th>Avoidability mean</th>
<th>Control mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Disease</td>
<td>2.67</td>
<td>2.94</td>
<td>3.03</td>
<td>2.87</td>
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<tr>
<td>SD</td>
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<td>.97</td>
<td>.96</td>
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<td>High Blood Pressure</td>
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<td>2.45</td>
<td>2.67</td>
<td>2.55</td>
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<tr>
<td>SD</td>
<td>1.01</td>
<td>.98</td>
<td>1.04</td>
<td>.99</td>
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<tr>
<td>Adult diabetes</td>
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<td>1.99</td>
<td>1.99</td>
<td>2.18</td>
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<tr>
<td>SD</td>
<td>1.24</td>
<td>1.99</td>
<td>1.08</td>
<td>1.16</td>
</tr>
<tr>
<td>Childhood Diabetes</td>
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<td>1.16</td>
<td>1.23</td>
<td>1.16</td>
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<tr>
<td>SD</td>
<td>.78</td>
<td>.44</td>
<td>.62</td>
<td>.46</td>
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<tr>
<td>Lung cancer</td>
<td>3.38</td>
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<td>SD</td>
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<td>.76</td>
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<td>.84</td>
<td>.65</td>
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<td>Stroke</td>
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<td>2.21</td>
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<tr>
<td>SD</td>
<td>1.04</td>
<td>.98</td>
<td>.98</td>
<td>.97</td>
</tr>
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<td>AIDS</td>
<td>3.61</td>
<td>3.89</td>
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<td>3.82</td>
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<td>1.08</td>
<td>1.05</td>
<td>1.07</td>
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<td>Rape</td>
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<td>SD</td>
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<td>.94</td>
<td>.94</td>
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<td>1.01</td>
<td>1.16</td>
<td>1.00</td>
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<td>Paralysis (car accident)</td>
<td>1.72</td>
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<td>1.72</td>
<td>1.73</td>
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<td>SD</td>
<td>.90</td>
<td>.81</td>
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<td>.90</td>
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<tr>
<td>Paralysis (sports accident)</td>
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<td>SD</td>
<td>1.19</td>
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<td>1.36</td>
<td>1.22</td>
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<tr>
<td>Burn injuries (work)</td>
<td>2.43</td>
<td>2.55</td>
<td>2.81</td>
<td>2.69</td>
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<td>SD</td>
<td>1.03</td>
<td>1.16</td>
<td>1.21</td>
<td>1.13</td>
</tr>
<tr>
<td>Burn injuries (home)</td>
<td>3.06</td>
<td>3.29</td>
<td>3.51</td>
<td>3.34</td>
</tr>
<tr>
<td>SD</td>
<td>1.15</td>
<td>1.23</td>
<td>1.22</td>
<td>1.22</td>
</tr>
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</table>
Multidimensional Scaling (MDS): MDS analysis was run in order to depict the 14 negative events in multidimensional space and investigate any underlying dimensions in participants’ representations of the events. The choice of the number of dimensions suitable for a given data set must offer a balance between statistical and substantive criteria (Everitt & Dunn, 1991). Statistical criteria focus on goodness of fit measures while more substantive criteria focus on the interpretability of the solutions. Regarding goodness of fit measures, the common guidelines for acceptable stress and $R^2$ measures were followed (explained below). Regarding interpretability, practical criteria of clarity and simplicity were employed.

Four classical metric MDS solutions were obtained, one for each of the four questions, using the SPSS ALSCAL procedure and employing the Euclidean distance. The goodness of fit measure for MDS analyses is expressed by the solution’s stress and $R^2$ values. According to Kruskal (1964, cited in Everitt & Dunn, 1991) stress levels lower than 0.15 are excellent with values close to zero being ideal. Similarly, $R^2$ values approaching 1 are perfect. In all four cases, unidimensional solutions gave the lowest stress values. However, since participants did not make explicit judgements of similarities or differences on the stimuli, but rather rated the events on four 5-point scales, they were, in a way, forced to think along a specific dimension at a time (e.g. blame for the sufferer). Hence, it was not surprising that unidimensional solutions would yield the best fit values. It was decided that accepting these solutions would be less informative than 2 or 3-dimensional ones. For that reason, 2-dimensional solutions were preferred when stress
values allowed it. Table 8.2 shows the S-stress (SPSS ALSCAL's variant of a stress measure) and $R^2$ values obtained for each 2-dimensional solution.

**Table 8.2:** Stress and $R^2$ values of the 2-dimensional solutions

<table>
<thead>
<tr>
<th></th>
<th>S-stress</th>
<th>$R^2$</th>
</tr>
</thead>
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<tr>
<td>Responsibility</td>
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<td>.98</td>
</tr>
<tr>
<td>Control</td>
<td>.04</td>
<td>.99</td>
</tr>
<tr>
<td>Avoidability</td>
<td>.08</td>
<td>.97</td>
</tr>
</tbody>
</table>

Figure 8.1 shows the 2-dimensional MDS plot for all 14 events rated on the sufferer’s blame for causing the event. It is important to note that MDS plots are simple configurations in space and therefore could be rotated in anyway provided that the relative distances among the points in the plot are maintained. Events placed on the far left of the plot (such as childhood diabetes, breast cancer or rape) were perceived as different from events on the far right of the plot (such as AIDS). Looking at the mean ratings for each event (shown both in Table 8.1 and on the plot of Figure 8.2) it becomes obvious that the horizontal dimension is the amount of blame participants see as attributable to the sufferer. For example, a rape victim or a patient with breast-cancer is seen as less blameworthy than a patient with lung-cancer or AIDS. Thus, the space could be partitioned in three main areas showing the groupings according to the degree of blame assigned by participants.

Furthermore, there appears to be an alternative partitioning of space when looking along the vertical dimension of the plot. All illnesses can clearly be separated form all other events as
demonstrated by the dashed horizontal line on the plot. With events like ‘adult diabetes’ or ‘heart disease’ at opposite ends from ‘paralysis from a sports accident’ or ‘burn injuries in the workplace’, it is plausible that the vertical dimension depicts participants’ categorization of the events into a specific context of actual illnesses versus other health-affecting hazards. Therefore diseases are grouped together at the top of the plot while at the bottom one finds events that, regardless of how blameworthy the sufferer is, occurred as accidents during everyday activities.

**Figure 8.1:** Two-dimensional plot depicting the 14 negative events rated on the degree of blame assigned to the sufferer.

When responses to the ‘responsibility’ question were analysed (Figure 8.2), the solution was very similar. The spread of the events along the horizontal dimension followed the same pattern as the previous plot with those events perceived as low on responsibility appearing...
on the left side of the plot (e.g. breast cancer), those perceived as high on responsibility on
the right side of the plot (e.g. AIDS) and those perceived as average on responsibility
appearing in the middle. Regarding the vertical dimension, again, illnesses were clearly
separated from other accidents and hazards, and were depicted at the top of the plot.

Figure 8.2: Two-dimensional plot depicting the 14 negative events rated on the degree of
responsibility assigned to the sufferer.

Overall, considering issues of blame and responsibility for the 14 negative events yielded
very similar representations, revealing a common underlying way of understanding the two
dimensions and making judgements along them.
Looking at the representations of avoidability and control of the 14 negative events a similar picture is presented. As shown by their mean ratings, avoidability of events (Figure 8.3) is depicted when moving horizontally along the plot with rape and childhood diabetes seen as less preventable than AIDS, lung cancer, or burn injuries happened at home. Once more, the vertical dimension differentiated between illnesses and other events, with illness depicted at the top of the plot.

**Figure 8.3:** Two-dimensional plot depicting the 14 negative events rated on the degree of *avoidability* the sufferer is perceived to have.

Finally, Figure 8.4 shows the representation of the events rated on the control the sufferer is typically perceived to have over the occurrence of the event. Although the events on this plot are slightly more dispersed than in the previous plots -perhaps suggesting a more
complicated way of categorising events- the main principles are the same. Horizontally, one moves from the less controllable occurrences on the left of the plot, to the more controllable one at the right. Illnesses are once again separated from the other events and appear towards the top of the plot.

**Figure 8.4:** Two-dimensional plot depicting the 14 negative events rated on the degree of control the sufferer is perceived to have over the occurrence of the event.

Cluster analysis: Hierarchical agglomerative clustering of the 14 negative events (employing between-groups linkage and squared Euclidean distance) was run for each of the four questions (i.e. blame, responsibility, control and avoidability) in order to facilitate a more extensive and objective interpretation of the MDS plots. All variables were measured on the same scale so no transformations were required. In clustering techniques, it is occasionally difficult to decide on the appropriate number of clusters. Two strategies
were used to determine the number of clusters. First, the Euclidean distance schedule was examined for large increases in coefficients between agglomeration steps (agglomeration schedules are shown in Appendix IX). This can be also done more informally by examining the differences between fusion levels in the dendrogram produced by the statistical program (Everitt, 1993). Large changes are taken to indicate a particular number of clusters. Second, cluster profiles were examined for meaningful cluster differences and judgements were made on the basis of interpretation.

Inspection of the agglomeration coefficients and assessment of the face validity of the dendrograms suggested a three- or four- cluster solution for all four questions. In order to test the stability of the clusters across different algorithms, all four analyses were re-run using the within-group linkage clustering method. Stability was measured in terms of 'transfer of cluster membership' (i.e. minimal transfer of membership across different clustering methods). Clusters were highly stable with no change in cluster membership when comparing the between and within group linkage methods.

Regarding the ratings of blame, analysis yielded three clusters as shown in the dendrogram in Figure 8.5. The first cluster (Cluster I) included child diabetes, breast cancer, rape, paralysis after a car accident and domestic violence. All five events can be seen as relatively unforeseeable and possibly attributable to uncontrollable factors such as chance or heredity. In contrast to Clusters II and III there appears to be little or no lifestyle involvement regarding the occurrence of the events and thus sufferers are not responsible for them. The second cluster (Cluster II) included high blood pressure, stroke, adult diabetes, heart disease, paralysis due to a sports accident and both examples of severe
burn injuries (i.e. at home and at work). These events are less unexpected than those of Cluster I since some measures can be taken to prevent their occurrence. For example, a healthy diet or moderate exercise can buffer against heart disease or adult onset diabetes. Similarly, following health and safety instructions can potentially prevent burn injuries. Finally the third cluster (Cluster III) included only lung cancer and AIDS. It seems meaningful to conclude that the events in this cluster are seen as preventable possibly due to the behavioural risk factors (e.g. smoking, unprotected sex etc.) relating almost causally to the events.

**Figure 8.5**: Dendrogram showing the three cluster solution yielded from the analysis of events rated on 'blame'.

When the three cluster solution was superimposed on the relevant MDS plot (Figure 8.6), the solution verified the interpretation given initially regarding the horizontal lay-out of events.
Figure 8.6: Cluster solution superimposed on MDS solution for 'blame' rating of events.

Following the same steps, the 14 events rated on responsibility were then examined. Here, the cluster solution was more ambiguous than in the previous case. As shown by the dendrogram in Figure 8.7, either a three or a four cluster solution could be retained. The four cluster solution resembled the MDS solution more than the three cluster one and was more interpretable. Thus, four clusters were analysed. As in the case of blame, Cluster I included child diabetes, breast cancer, rape, both examples of paralysis and domestic violence. These events were most likely seen as low on responsibility of the sufferer. Cluster II included adult diabetes, stroke, heart disease and high blood pressure. These events were seen as different from those in Cluster I and judging by their mean scores
were seen as involving more responsibility of the sufferer. Cluster III included only two events, burn injuries at home and at work, which in the corresponding analysis for blame were included in Cluster II. This cluster appears to demonstrate the separation between illnesses and accidents seen in the MDS plots described earlier. Finally, Cluster IV includes lung cancer and AIDS, the two events seen by participants as involving the highest degree of responsibility from the patient's perspective.

**Figure 8.7**: Dendrogram showing the four cluster solution yielded from the analysis of events rated on ‘responsibility’.

When this cluster solution was superimposed on the corresponding MDS plot (Figure 8.8), it fit the configuration of the plot well. Clusters matched the naturally occurring groupings of the point and overall the solution was very similar to that of blame ratings.
In a similar manner to that presented above, the concepts of avoidability and control were examined. Regarding avoidability three clusters were identified (Figure 8.9). Cluster I included child diabetes, breast cancer, rape, paralysis from a car accident, adult diabetes, and stroke. These events were seen as low on avoidability. Cluster II included heart disease, high blood pressure, domestic violence, burn injuries at work and paralysis from a sports accident. Compared to Cluster I, these events appeared to involve average levels of avoidability as indicated by their mean scores (Table 8.1). Finally, Cluster III included lung cancer, AIDS and burn injuries at home representing events high on avoidability.
**Figure 8.9:** Dendrogram showing the four cluster solution yielded from the analysis of events rated on 'avoidability'.

The MDS solution for the avoidability ratings matched the cluster solution satisfactorily. As shown in Figure 8.10, when the three clusters were superimposed on the relevant plot they appeared to follow the vertical separation of space given in Figure 8.3. A small difference was noted regarding adult diabetes and stroke which in the cluster solution appeared as similar to low responsibility events such as breast cancer and rape, while in the earlier interpretation of the MDS plot were seen as closer to average responsibility events such high blood pressure or domestic violence (depicted towards the middle vertical section of the plot).
Finally, a cluster analysis of the 14 events rated on participants’ perceptions of control that the sufferer would typically have over the event revealed three clusters (Figure 8.11). Cluster I included child diabetes, breast cancer, rape, domestic violence and paralysis from a car accident. As with previous analyses, this cluster was interpreted as representing events of low control of occurrence. Cluster II included both examples of burn injuries, heart disease, high blood pressure, adult diabetes, stroke and paralysis from a sports accident. This cluster was seen as including the events of average control. Cluster III included only AIDS and lung cancer as the events whose onset the sufferer has the highest level of control over.
Figure 8.11: Dendrogram of the three cluster solutions of events rated on 'control'.

Comparing the cluster solution with the MDS plot (Figure 8.12) showed a picture similar to that found in earlier comparisons. The clusters matched the vertical separation of space thus verifying the interpretation of one of the dimensions of the MDS analysis.

To summarise, four very similar three-cluster solutions were yielded from the cluster analysis of the 14 negative events for each of the four questions regarding the sufferer’s perceived responsibility, blame, control and avoidability.
From the above comparisons, it has been shown that people have specific ways of grouping negative events in their minds with a) most illnesses clearly perceived as different from other misfortunes and b) different representations of different illnesses when those are rated on the same scale. However, these results do not show whether there are significant differences between ratings of blame and responsibility or avoidability and control when the same event is considered. To test for significant differences between perceptions of a) blame and responsibility and b) control and avoidability for each event, two sets of 14 repeated measures t-tests were performed. In order to compensate for the increased chances of Type I error, the Bonferroni correction was applied and as a result the significance criterion was reduced to $\alpha = 0.0036$. Results are reported in the Table 8.3. Regarding perceived blame and
responsibility of the 14 negative events, no significant differences were found. When comparing perceptions of avoidability and control, only lung cancer, domestic violence and paralysis from a sports accident showed significant differences. Specifically, all three events were associated with higher avoidability than controllability.

Table 8.3: T-tests for differences in mean scores between i) blame and responsibility and ii) control and avoidability for each negative event.

<table>
<thead>
<tr>
<th>Event</th>
<th>t-values</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart disease</td>
<td>2.87</td>
<td>2.26</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.28</td>
<td>1.40</td>
</tr>
<tr>
<td>Adult diabetes</td>
<td>-2.24</td>
<td>-.75</td>
</tr>
<tr>
<td>Childhood diabetes</td>
<td>-1.84</td>
<td>1.52</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>1.92</td>
<td>3.34*</td>
</tr>
<tr>
<td>Breast cancer</td>
<td>-2.74</td>
<td>1.77</td>
</tr>
<tr>
<td>Stroke</td>
<td>.87</td>
<td>.000</td>
</tr>
<tr>
<td>AIDS</td>
<td>2.62</td>
<td>1.36</td>
</tr>
<tr>
<td>Rape</td>
<td>-1.72</td>
<td>1.21</td>
</tr>
<tr>
<td>Domestic violence</td>
<td>.09</td>
<td>6.27*</td>
</tr>
<tr>
<td>Paralysis (due to car accident)</td>
<td>-2.31</td>
<td>.32</td>
</tr>
<tr>
<td>Paralysis (due to sports accident)</td>
<td>-.78</td>
<td>3.39*</td>
</tr>
<tr>
<td>Burn injuries (in the kitchen)</td>
<td>1.45</td>
<td>1.47</td>
</tr>
<tr>
<td>Burn injuries (at work)</td>
<td>2.65</td>
<td>1.75</td>
</tr>
</tbody>
</table>

*p<.0036 following Bonferroni correction

8.2 DISCUSSION

This study has investigated people's representations of negative life events when issues of responsibility, blame, avoidability and control are considered. The aim was to investigate the plausibility of applying Janoff-Bulman's theory on different populations and attempting to generalize on the functionality of self-blame under the assumption that self-blaming
behaviour would be similar in different negative events. Furthermore, this study directly addressed the criticism of potential conceptual differences between the terms 'responsibility' and 'blame'.

The results yielded by this study generally support the idea of blame being event- or situation-specific suggested in the previous chapters of this thesis. As shown by the cluster analysis and the MDS plots, there is a tendency for people to group negative events according to the degree of blame, responsibility, avoidability and control associated with each event. Consequently, some events are seen as low on these four characteristics (e.g. rape, breast cancer), others as average (e.g. heart disease, burn injuries at work) and others as high (e.g. AIDS, lung cancer). Furthermore, on all MDS plots there is a clear distinction between illnesses—always appearing at the top of the plot—and other misfortunes—appearing at the bottom. This suggests that in people’s minds illness carries different properties from other misfortunes regardless of the degree of responsibility, blame, control or avoidability associated with them. There is therefore little point in attempting to generalize about issues of blame across illnesses and non-illness events. Some generalization however may be appropriate within groups of illness (or other events) as shown by the MDS plots and cluster analyses.

Janoff-Bulman’s (1979) work originated from her work with rape victims and people with spinal cord injuries from severe accident victims. In the present study, both of these events appear very close to each other on the MDS plots across all four ratings. If the properties that people perceive as common for these events are true for the actual victims of the events then the consistency in the applicability of the theory is expected. However, when similar
attempts were made by researchers to apply the theory on other events problems stated to arise. For example, the event of breast cancer -seen as equally blameless as rape on the horizontal dimension but at a distance from rape on the vertical dimension- has shown great inconsistencies in the literature of self-blame. There is therefore further evidence on that self-blame is event specific.

Regarding the potential differences between responsibility and blame suggested by Shaver and Drown (1986) results showed no significant differences between the two concepts on any of the 14 events. This suggests that in everyday conversation people do not differentiate between the terms ‘blame’ and ‘responsibility’ and using the terms interchangeably in self-report measures has no noteworthy effects on participants’ responses. These findings are in accordance with studies by Sholomskas, Steil & Plummer (1990) and Hall (2000).

However, Shaver and Drown’s (1986) suggestions should not be entirely dismissed. The fact that people do not appear to differentiate between blame and responsibility in quantitative or qualitative self-report measures does not mean that they are not able to do so in different contexts. For example, people might give different responses if probed to concentrate on the differences between the two concepts. Alternatively, in situations where the above issues are discussed in depth or there is a variety of opinions stated (e.g. in focus groups) these differences may be more likely to come up. Therefore, a distinction must be made between people’s tendency to make such distinctions and their ability to do so if the situation supports this. While this study has shown that the former has little chance of occurring, more work is needed to investigate the latter.
Regarding the distinction between avoidability and control, only three events yielded significant differences. Specifically, lung cancer, domestic violence, and paralysis from a car accident were rated higher on avoidability than on control. However, looking at the table of means (Table 8.1) it can be observed that the differences between means for these events are very small. To illustrate, the difference between the avoidability and control mean ratings on a 5-point scale was for lung cancer 0.21, for domestic violence 0.52 and for paralysis from a sports accident 0.24. Although these differences are significant statistically, it is debatable whether they reflect significant psychological differences. Thus, until they are investigated further, the present results should be interpreted with caution.

These findings have specific implications for research. The fact that people tend to not differentiate among the four concepts of blame, responsibility, avoidability and control may simplify the interpretation of the relevant literature. Also, the way people tend to represent the negative events studied suggests that issues of responsibility for illness differ from those of other negative events and thus comparisons among events may be misleading. Of course, it must be noted that the sample used in this study constitutes of healthy individuals and not patients. There may be motivational differences between patients and non-patients that have yet to be identified. Perhaps, non-patients see no personal relevance in discussions about illness and can use relevant terms with less sophistication or complexity. In contrast, patients may place more importance in the precise meaning of the words and be more particular when using the terms studied here. The scarce evidence in the literature suggests that this is not the case but clearly more work is needed in this area.
There are a number of limitations in this study that should be considered. First, the relatively small response rate prevents the safe generalisation of results. Although the initial sample was a random sample of the south-east population of England, there was only a 14% response rate possibly due to lack of follow-up reminders and incentives. Second, due to financial and time constraints only non-patient populations could be recruited for this study. Therefore it is not clear whether the results regarding participants’ perceptions of blame for and control of negative events would apply to patient populations as well. In other words, it is important to establish the similarities and differences of such representations between patient and non-patient populations or alternatively ascertain the effects that the illness experience (or lack of) has on perceptions of blame when all other relevant characteristics of individuals are controlled for.
CHAPTER NINE
GENERAL DISCUSSION

SUMMARY
This chapter reviews the findings presented in the previous chapters and synthesises them to provide answers to the general research questions posed at the beginning of the thesis. The implications of the findings for psychological theory and practice are discussed. The issues of self-regulation as part of adjustment to the threats posed by illness are introduced as plausible mechanisms underlying and explaining the relationships reported in the present studies. The motivational and cognitive elements of self-blame are considered and ways to reconcile the contradictory evidence of past and present studies are discussed. The chapter closes with a review of the limitations of the research and a brief section of concluding remarks.

9.1 SUMMARY OF RESULTS
This thesis has investigated the concept of self-blame (placing more emphasis on the behavioural type) by addressing a number of methodological and conceptual limitations found in the literature. It attempted to place self-blame against a specific theoretical and conceptual background and give it a place in existing models of illness behaviour and cognition. More importantly, this thesis posed some novel research questions regarding self-blame and addressed them both empirically and theoretically.
The first two theoretical chapters (Chapters Two and Three) reviewed the literature on causal attributions and self-blame for illness and identified the main theoretical and methodological shortcomings regarding self-blame. They also proposed a more specific theoretical conceptualisation of the concept, by placing the, often confused, concepts of self-blame and causal attributions in the broader theoretical model of self-regulation theory proposed by Leventhal (Leventhal & Nerenz, 1985). In that model, self-blame is seen as a specific type of causal attribution for one's illness. Causal attributions, in turn, fall in the more general domain of illness perceptions, which is one of the main components of Leventhal's model. The usefulness of having a concrete position for self-blame in a theoretical model as well as its conceptualisation through attribution theory was introduced in these chapters.

Study 1 was designed to approach self-blame from a perspective close to the one seen so far in the literature while introducing a series of new ideas. Therefore, the study looked at self-blame and its relationship to psychological adjustment, this time comparatively across three patient groups (heart disease, diabetes, and breast-cancer patients). Other variables such as self-efficacy, number of perceived behavioural risk factors and diet and exercise levels were also included in the study and were investigated in relation to self-blame. Results showed significant differences between self-blame among the three patient groups and no overall relationship to adjustment. Also, the number of perceived behavioural risk factors reported by the patients was positively correlated with self-blame. Finally, diet and exercise were negatively correlated to self-blame but only in the case of diabetes patients. These findings provided support for the idea that self-blame may be situation specific and its level or role may differ from one patient group to the next. It
was, thus, necessary to investigate the possible predictors of self-blame and examine the conditions under which it occurs.

Study Two tested a theoretical model of the predictors and consequences of self-blame. The model was designed by bringing together findings from the attribution and self-blame literature and focused mainly on behavioural self-blame which is the type of self-blame that has yielded the most inconsistent results. A number of personal and situational characteristics were included in the model and an emphasis was placed in showing that causal behavioural attributions (i.e. a judgement between the cause and effect relationship between two events) can lead to behavioural self-blame (i.e. a moral evaluation of the potential role of oneself in causing an event) but should not to be confused with it.

Results from data collected in a survey of 160 heart disease patients showed that the proposed model including the predictors of self-blame fit the data well and revealed that gender, characterological self-blame and behavioural attributions were the best direct predictors of behavioural self-blame. Also, the type of diagnosis, prior risky health behaviour affect behavioural self-blame indirectly through their impact on behavioural attributions. The consequences of self-blame were studied using a more data-driven approach and showed that it is behavioural causal attributions, rather than self-blame, that predicted changes in some aspects of health behaviour. Psychological adjustment could not be predicted by either behavioural attributions or behavioural self-blame.

Studies Three and Four used a sample of non-patients in two ways. First, the sample was matched in all relevant dimensions with the heart-disease patient sample of Study Two and comparisons were made between patients' actual levels of self-blame and non-
patients’ levels of potential self-blame under the hypothetical event of a heart attack. Results indicated (though not entirely reliably) that non-patients engaged in higher levels of both behavioural and characterological self-blame than their patient counterparts. Also, unlike patients, non-patients did not show any relationship between behavioural self-blame and lifestyle risk factors, indicating that their self-blame scores regarding a potential diagnosis of heart disease resulted from factors other than an evaluation of their current lifestyle. Second, non-patients were asked to rate a series of negative life events including illnesses and accidents along the dimensions of responsibility, blame, controllability and avoidability. This was done to look at the way non-patients represent these events in their minds when such dimensions are considered and also to test for differences in scores that would signify a different understanding between the dimensions. Cluster and multidimensional scaling analyses showed that people group or rate these events in specific meaningful ways when they rate them on the above dimensions. They also differentiate between illnesses and other events, suggesting that an illness is perceived a type of misfortune unlike other negative events such as rape or severe accidents. Finally, this study also showed that people tend to treat the concepts of blame or responsibility and control or avoidability as very similar (at least quantitatively) and therefore criticisms regarding their interchangeable use should not warrant too much concern.

9.2 BRINGING IT ALL TOGETHER

When the above findings are combined, they can offer some interesting insight into the study of self-blame. This section will bring together the above results and to contribute to
addressing the research questions stated in the introductory chapters of the thesis (Section 3.4). They are dealt with here in the same order as in Chapter Three.

9.2.1 Sample Diversity

The extent to which the initial findings of Janoff-Bulman’s work (1979) should generalise – or be expected to hold – across different populations was criticised and tested in this thesis. Past literature had hinted at differences in self-blame across different sample groups (e.g. Forsythe & Compas, 1987) but findings have been rather incidental. Here, it was consistently found that self-blame varies among patients with different illnesses (i.e. heart disease, diabetes, breast cancer) as well as patients with different symptoms within the same illness category (i.e. angina vs. heart attack in heart disease). What appeared to be the explanatory pattern behind these differences was that in both cases the condition associated with the greatest number of behavioural risk factors was also associated with the highest levels of self-blame. This was also true for the non-patient samples that rated illnesses highly associated with lifestyle (e.g. AIDS) as more blame-relevant than illnesses unrelated to lifestyle (e.g. childhood diabetes).

Therefore, there is evidence that self-blame (or blame in general) is situation-specific and it is this characteristic of self-blame that may explain much of the inconsistent results in the literature. If, as shown here, self-blame is more prominent in some patient groups and less in others, then it is possible that its relationship to adjustment follows a similar pattern, whereby it is stronger in some patient groups (and thus more detectable by the often under-powered studies available) and weaker or non-existent in others.

183
9.2.2 Reasons for self-blame

This thesis proposed that the predictors of self-blame must be clarified if the relationship between self-blame and adjustment is to be meaningfully explained. The literature review showed that characterological self-blame is seen as an esteem-related reaction to a negative event, stemming from self-esteem deficits and tendencies towards self-criticism (Abramson, Seligman & Teasdale, 1978; Janoff-Bulman, 1979). It is more in line with attributional style and has been consistently linked to negative adjustment.

Regarding behavioural self-blame, one of the most common theoretical assumptions behind its functionality is that it enhances feelings of control (e.g. Janoff-Bulman, 1979). Specifically, focusing on one’s own actions regarding a negative event means focusing on the changeable aspects of one’s behaviour and therefore increasing the perceptions of avoidability of similar future events. This assumption sounds plausible but has not been supported by conclusive evidence. In the present thesis, the issue of control was approached in various ways throughout the studies by measuring such variables as perceived controllability/avoidability of specific risk factors, behaviour, and character. General self-efficacy was also measured as an alternative, though indirect, way of tapping on one’s sense of control over difficult situations. None of the measures yielded any significant relationships with self-blame, behavioural attributions or adjustment. Indeed, there were other variables that predicted self-blame as shown by the measurement model in Chapter Six. Being male, engaging in characterological self-blame, having a diagnosis associated with a number of health behaviours and, indeed, having engaged in these risky behaviours predicts self-blame either directly or indirectly by affecting the type of behavioural attributions made. Therefore, behavioural self-blame seems to be a more
pragmatic approach to causal search, affected by various factors (e.g. sex, type of diagnosis, behavioural attributions). In order for a person to express behavioural self-blame, there needs to be a formation of behavioural attributions which in turn are a product of personal, rather stable factors as well as situational ones.

9.2.3 Timing

The time elapsed since diagnosis has often been presented as a possible explanation for the inconsistent findings regarding the relationship between self-blame and adjustment. In her systematic review, Hall (2000) concluded that the associations between attributions and adjustment diminish with time. However, she ascribes that to the fact that many of the studies she reviewed were conducted many years after the event. Indeed, the few longitudinal studies available on the subject show very little evidence of a decline in the associations between self-blame and adjustment over time (e.g. Downey, Cohen-Silver & Wortman, 1990). As Hall (2000) concludes, it is likely that the effects of time on self-blame depend on the content of attributions, the type of outcome assessed and the nature of the event.

This thesis did not present any longitudinal studies for practical reasons discussed in an earlier chapter. However, the cross-sectional study that compared self-blame in three different patient groups (Chapter Four) provides some support for Hall’s (2000) conclusions. Time since diagnosis was significantly correlated with self-blame in patients with diabetes with self-blame increasing with time. This was not true for patients with heart disease or breast cancer. As discussed in the relevant chapter, these findings make sense when the nature of the illness is taken into account. The everyday lifestyle management that diabetes demands of its patients often becomes more demanding with
time. It is often the case that people develop diabetic complications and/or start taking insulin (e.g. Type II diabetes patients not previously on insulin). Thus, patients see their condition as more serious and hence reflect more on their behaviour becoming increasingly aware of their role in managing their illness. They may therefore be more likely to express self-blame than other patients and their levels of self-blame will tend to increase with time. While this relationship does not include psychological adjustment, it does show once again the highly situation-specific nature of self-blame and the plausibility of a more pragmatic approach to certain aspects of the construct.

9.2.4 Self-blame and adjustment

While the focus of this thesis was on ‘unpacking’ the concept of self-blame (mainly behavioural self-blame) and clarifying the conditions under which it occurs, some attention was paid to the relationship between self-blame and adjustment. This was investigated both in the comparative study (Chapter 4) and in the model testing study (Chapter 5). In the former, no relationship was found between general self-blame and adjustment in the overall sample as well as each patient group separately. Similarly, in the latter study, neither attributions nor behavioural self-blame showed any statistical significance in predicting anxiety or depression. There was therefore, no evidence of a relationship between general or behavioural self-blame and adjustment. Only characterological self-blame was moderately correlated with anxiety verifying the fairly well established relationship between this type of self-blame and psychological adjustment. It is therefore the relationship between adjustment and behavioural self-blame that remains controversial.
However, the absence of a relationship between behavioural self-blame and adjustment might be explained by timing. The fact that the sample was captured many years after a diagnosis was made may account for the lack of evidence for a relationship. One may even argue that only if a study is conducted at the time of diagnosis or at most a few weeks later, could the relationship between behavioural self-blame and anxiety be investigated. This argument, however, has some basic flaws. In all samples, there were enough people diagnosed in the last year to allow for the detection of such a relationship. If time actually affects the relationship between self-blame and adjustment, one would expect a low to moderate correlation between self-blame and adjustment in the samples used in this thesis. No indication of such a relationship was found.

Furthermore, even if this relationship may only be detected in the very early stages of the illness experience, how informative would it really be regarding self-blame? In the first days after a diagnosis, patients are likely to experience symptoms of anxiety and these are likely to affect every aspect of their behaviour. Scores on any psychological adjustment scale are more indicative of the stressful situation than the person and it is likely that many of the correlations obtained at a time like this will change as the initial reactions to diagnosis begin to subside. Therefore, unless one is interested in the process of attributional search at the time of diagnosis, there is little point in aiming to capture people’s scores at that particular time. Scores captured at more stable times are likely to be more informative of the strength and nature of the relationship under investigation. This was the approach in the studies presented here and revealed no relationship between general or behavioural self-blame and psychological adjustment in heart disease, diabetes and breast cancer patients.
Parallel to uncovering specific relationships between self-blame and other situational or personal characteristics, this thesis has tested the plausibility of criticisms regarding the operationalisation and measurement of self-blame. One of the main criticisms has been the interchangeable use in the literature of the concepts 'cause', 'responsibility' and 'blame' (Shaver and Drown, 1986). Furthermore, the comparison of findings across different situations (e.g. illnesses vs. accidents) was also criticised in the early chapters of this thesis. Data collected to address these issues supported only the latter criticism (Chapter 7). Specifically, people do not differentiate between the concepts of blame and responsibility in any statistically significant way. The same was true for the concepts of 'control' and 'avoidability'. These findings have important practical implications for those investigating the literature on self-blame. Since the main concern behind this criticism was that the wrongful use of the terms interchangeably may account for the inconsistent findings, evidence provided here do not support these concerns and make the explanation of the situation-specificity of self-blame an even more plausible one.

Regarding the second criticism (i.e. the validity of comparison of findings across different situations), it was shown that people clearly differentiate illnesses from all the other misfortunes presented to them. There is therefore evidence that, in terms of the representations people use to make inferences regarding issues of responsibility and control, illnesses are perceived as different from other negative events. These differences could possibly refer to the personal meaning that the event may have, the threat for life it represents or that it does not entail the element of luck in the same way. Regardless of
what makes illnesses stand apart from other misfortunes in people's minds, it is a
distinction that should be reflected in the methodology of relevant studies and explored
further, preferably with more qualitative means.

9.3 IMPLICATIONS FOR THEORY

The most central aim of this thesis was to revisit the theory on self-blame, focusing
mostly on behavioural self-blame, and contribute to a more comprehensive description of
the processes involved.

With past research approaching behavioural self-blame as a motivational process and
evidence in the present thesis suggesting more cognitive properties, how can these rather
contradictory findings be explained? Is behavioural self-blame the outcome of a need for
control over future misfortune or is it a cognitive self-evaluation that is unrelated to
psychological adjustment? The answer lies in the theoretical and empirical approach one
adopts when studying self-blame.

Early in this thesis, Leventhal's self regulation model (Leventhal & Nerenz, 1985) was
presented as a potentially useful theoretical framework that can embody many of the
concepts and processes self-blame was expected to associate with. Indeed, this model can
be used as a prism through which the empirical findings presented earlier can be
interpreted and give rise to an alternative theoretical identity for self-blame. But simply
placing self-blame among the various causal explanations people include in their illness
representations is a rather superficial way of looking at this concept and does not
accommodate the findings presented in this thesis with much precision. Going back to Leventhal’s model as presented in Chapter Two, one could easily identify how illness characteristics can influence self-blame as reported by the measurement model in Chapter Six: all the relevant variables are included in the same box titled Threat representation. The same is true for the relationship between causal attributions and behaviour change, if the latter is interpreted as a form of coping: the model already includes the bi-directional arrow between ‘threat representation’ and ‘coping’. However, where would the personal characteristics (e.g. gender, characterological self-blame, prior risk) figure in the model? These are the sort of characteristics that not only predict behavioural self-blame, but could also affect other components of the self-regulation model such as symptom interpretation. How could the model account for this?

A plausible answer lies in the idea that the dynamic relationships described in Leventhal’s model are actually influenced by deeper processes involving the self. Leventhal, Idler and Leventhal (1999) recently acknowledged these processes as central to the illness experience. Specifically, when illness becomes apparent, it generates a series of psychological reactions. As already described in the model, an interplay begins between cognitive (i.e. illness interpretation and representations), emotional (i.e. fear, anxiety) and behavioural (i.e. coping) reactions. During this interplay, the self is required to assimilate the new status quo imposed by the illness experience and adjust to the situation to regain psychological balance. When an illness symptom or a diagnosis arises, people will attempt to interpret it and assign meaning and importance to it. This interpretation will be affected by the individual’s emotional reaction to the illness as well as his or her illness/threat representations. Within these representations, a causal search will begin for
the reasons behind this illness and the idea of self-blame may be entertained. It is at this stage that behavioural self-blame might be the result of motivational processes that aim to protect the self. Although blaming oneself may sound more like a self-mutilating than a self-protective mechanism, one has only to review the evidence on the complex structure of the self (Fiske & Taylor, 1991) to recognise that an apparently dysfunctional behaviour may indeed be serving a functional role. While self-blame is about placing the burden of fault on one’s behaviour, it may be, in that way, protecting other more important aspects of the self by providing a sense of stability and fairness. Indeed, aspects of self-regulation such as personal control (i.e. behaviour, cognitive, decision, information and retrospective control), the need for accuracy and consistency might all be better served by behavioural self-blame - an internal and potentially controllable explanation - than other or chance blame which are external and less controllable.

All these dynamic processes are more relevant at the earlier stages of an illness when the search for explanation is novel and quite crucial. As time progresses, however, most people manage to adjust to the new reality and adopt a new (or modified) ‘illness-related’ self. They reach this stage after a long, complicated process of self-regulation and adaptation to illness. With time, and as patients reach the standard milestones of medical care (i.e. diagnosis, treatment plans, follow-up assessments) more information becomes available and, with time, several aspects of the illness is re-assessed. Always through an interplay among interpretations, representations, emotional reactions and coping mechanisms, individuals find a way to balance the pre-diseased and the diseased self, possibly in the face of the new illness-related self. The illness explanation will inform and be informed by the person’s current identity, and behavioural self-blame, if present,
will reflect this causal search. At this stage, motivational processes may be less active. Indeed, they may work in a way opposite to that in earlier stages of the illness, and restrain people from fully accepting their role in contracting their illness. In contrast, the cognitive characteristics of self-blame are more evident and clearly relate to causal attributions and behaviour change. According to the model tested in Chapter Six, a patient might ask the following questions: As a man (woman) what has my role been in falling ill? What does this diagnosis mean to me? What risky behaviours did I engage in before my illness? Do I believe these behaviours caused my illness? To what extent am I to blame for my illness? This process can explain the rather pragmatic basis of behavioural self-blame revealed in the studies reported earlier.

Viewing self-blame through the broader prism of self-regulation may explain the lack of a relationship between behavioural self-blame and adjustment and its new-found relevance to behaviour change. Psychological adjustment to a threat is achieved when progression through the various stages of the self-regulation process runs smoothly, and a functional new or modified self emerges (Kanfer & Hangeman, 1981; Rosenbaum & Ben-Ari, 1985). It is in essence the resolution of the threat posed by illness and the incorporation of the new status quo in everyday life. This is done irrespective of whether the person has engaged in behavioural self-blame. Interpreting illness as the product of controllable or uncontrollable factors will have no impact on adjustment as long as that interpretation fits well with one’s sense of self. On the other hand, having constructed a self that is seen as instrumental in causing the illness (or in not trying to avoid it) can be the first step in motivating oneself to change one’s lifestyle and control illness recurrence. Therefore the proposed theoretical framework can account for the relationship between causal
attributions and reported behaviour change found in this thesis as well as the relationship between behavioural self-blame and behaviour change reported by Christensen et al (1999).

The differences in self-blame across different patient groups can also be explained through this theoretical framework. In this case too, self-blame reflects the search of causal explanations for one’s illness which is influenced by both the pragmatic (e.g. is my cancer/diabetes/heart disease hereditary?) and the psychological (e.g. what does it mean to be a cancer/diabetes/cardiac patient?) aspects of an illness or illness identity. These aspects will tend to be homogeneous within an illness but heterogeneous across different illnesses and thus give rise to different levels of self-blame across medical conditions.

Furthermore, the idea of self-reconstruction can explain the differences in self-blame between patients and non-patients. What these differences may reflect is really the presence or absence of the process of self-regulation. Non-patients replied to a hypothetical scenario, and had not experienced the presence of a real threat. Thus, they most likely responded to the questions using information they had accumulated from indirect sources rather than personal experience with heart disease. The consequence of that is that, while non-patients answered the relevant questions free of any self-regulatory motivations or cognitions that a real illness would put in motion, patients responded in the face of real threat to their self-perception. This would explain the lower levels of behavioural self-blame observed in patients. A personal experience with a given illness may make any personal claim regarding the illness less threatening. In other words, a patient may be more threatened by the admission to health-threatening behaviours and/or
self-blame than a non-patient, precisely because of the personal relevance that such an admission carries. Patients may therefore be motivated to ‘downplay’ their self-blame compared to non-patients because of the potential impact self-blame may have on their identity.

Overall, behavioural self-blame can prove to be as dynamic in nature as any other aspect of the self-regulation concept. And its dynamic nature can become even more complex when the social network within which the illness experience unfolds is considered. For example, both the cognitive and the motivational aspects of self-blame are likely to be influenced by current social representations of illness. The way a given illness is portrayed by peers, media, and scientific communities may influence the degree to which an individual will adopt or deny self-blame. These limited degrees of freedom of self-regulation have yet to be defined and measured appropriately in studies involving the illness experience.

To conclude, a theoretical framework in which self-blame (especially behavioural self-blame) is seen as the product of the dynamic self-regulation process that is put in motion by an illness experience may be more helpful than the unidirectional, unidimensional approach adopted in past literature. Of course, a lot of work remains to be done before this theoretical framework becomes a theory. As with the self-regulation model in general (Leventhal, Idler & Leventhal, 1999), the key variables need to be specified and many sub-models of relevant variables examined in detail. Factors such as self-efficacy and self-esteem not mentioned above but with known relationships to identity should be included and their relationship to self-blame explored from the point of view of illness
identity. Finally, it is important for future researchers to incorporate *explicitly* as many aspects of the illness experience as possible at least at the level of theoretical understanding of an illness related concept. Narrowing down to few specific variables may be practical from an empirical point of view but it may be a hindrance in understanding the implications of the findings.

### 9.4 IMPLICATIONS FOR PRACTICE

Research on self-blame can contribute significantly to psychological practice in health. While so far the focus has been on identifying the effects of self-blame on adjustment, the need for a shift of focus on other outcomes has been supported here. One of the most important findings in this thesis is the relationship between behavioural attributions and behaviour change. As proposed by Christensen et al (1999) and Grove (1993), evidence in this area may be particularly useful for those medical populations whose illness aetiology and progression depends largely on health behaviour. It may allow for the identification of individuals at greater risk of engaging in health damaging behaviours due to their causal explanations and overall representations of their illness. Clearly, however, more work is needed in this area before this relationship can be offered as a useful tool to health professionals working with in the area of chronic illness.

From a more general perspective, this thesis has demonstrated the highly individual nature of chronic illness. Illness explanations, representations and most likely identities can differ not only across different illnesses but also across different conditions within the same illness category. This points towards the need for highly personalised interventions
for any kind of psychological support to patients with chronic illness. Less directly, it also raises important points, previously discussed in the literature by Donahue & McGuire (1995) and Kirkwood & Brown (1995), regarding the communication of illness causes and the awareness of responsibility in health and illness. In future studies, it would be worth to investigate how much of the self-blame displayed by patients is a result of a wider culture of blame created in an attempt to motivate people to adopt healthier lifestyles.

9.5 LIMITATIONS

The limitations relevant to each study have already been mentioned in previous chapters and, thus, will only be summarised here. On several occasions there were some considerations regarding the samples used. Specifically in the first study (Chapter 4) the patient groups may not have been large enough to test for differences of potentially smaller effect. Also, with one of the conditions being breast cancer, there was an unavoidable imbalance in the men/women ratios across the three patient groups, and sex differences could not be tested in that study. In the studies involving non-patients (Chapters Six and Seven), sample considerations involved the low response rate which consequently affected the generalisability of results. Regarding the adequacy of measures used, it was acknowledged that, in the first study, the developed measure was one of general self-blame and did not differentiate between behavioural and characterological self-blame. Furthermore, in the second study (Chapter Five) the measure of behaviour change was based on participants’ subjective estimates and was thus vulnerable to biases. Finally, in the same study, the model tested can only be seen as plausible—not true. Many
different versions of the model must be tested on new data before more general conclusions can be drawn. Also, the model should ideally be more parsimonious (i.e. have greater degrees of freedom). In this study, reduced parsimony was a result of lack of a clear theoretical basis for self-blame which lead to some ambivalence regarding the parameters to be estimated.

Apart from the limitations relevant to each study, there are several shortcomings of the overall work presented in this thesis that must be noted. First, the findings discussed here rely entirely on quantitative data collected with the use of questionnaires. While the use of such data has the potential of making more global claims with confidence, it tends to oversimplify the psychological processes underlying the scores. In retrospect, the use of qualitative data in addition to the quantitative data available could have been more informative of the reasons behind self-blame.

Another shortcoming is the use of cross-sectional designs throughout this thesis. A longitudinal design could have shed light on the changes that people undergo following an illness diagnosis and consequently the way those changes affect self-blame. Unfortunately the adoption of such a design was not possible for practical reasons (see Chapter Three).

Finally, the measurement of self-blame is an important methodological limitation of the studies presented here. In the pilot work presented in Chapter Four, an attempt was made to use a multi-item measure of self-blame. While that measure was sufficient for the purposes of that study, it did not differentiate between behavioural and characterological
self-blame and was therefore not used in subsequent studies. Consequently, the majority of the studies presented earlier rely on single-item measures of behavioural and characterological self-blame and thus face the criticisms related to the validity and reliability of the measures. While the present findings are comparable to past research using the same items, there is still the need for a standardised multi-item measure of self-blame and its sub-types that will ideally differentiate between self/behavioural attributions and self-blame. The limitations of the available alternatives were acknowledged early in the thesis but construction and standardisation of such a measure was beyond the its scope.

9.6 CONCLUSION

The studies presented in this thesis focused on the concept of behavioural self-blame and attempted to ‘unpack’ its role in the illness experience. According to the findings, behavioural self-blame is a situation-specific representation of the role one has played in acquiring a chronic illness. It appears to have a more ‘pragmatic’ nature than that proposed in the literature and is generally unrelated to adjustment. Both theoretically and empirically, it fits well within the framework of illness representations and illness identity, which is offered as a new way of understanding and conceptualising self-blame.


American Heart Association (2002). Risk Factors and Coronary Heart Disease [Internet


adults. *Social Science and Medicine, 43*(10), 1453-1460.


206


Rosenbaum, M., & Ben-Ari, K. (1985). Learned helplessness and learned resourcefulness:


Turnquist, D. C., Harvey, J. H., & Andersen, B. L. (1988). Attributions and adjustment to


APPENDICES
## APPENDIX I

### MALE AND FEMALE DEATH RATES FROM SELECTED CAUSES

Indicators of the Nations Health: Male death rates by selected causes

<table>
<thead>
<tr>
<th>England</th>
<th>Rates per 100,000 population</th>
</tr>
</thead>
<tbody>
<tr>
<td>All causes</td>
<td>1,060.5</td>
</tr>
<tr>
<td>All Malignant Neoplasms (ICD 140-208)</td>
<td>282.2</td>
</tr>
<tr>
<td>Stomach (ICD 151)</td>
<td>16.4</td>
</tr>
<tr>
<td>Colon, rectum, rectosigmoid junction &amp; anus (ICD 153-4)</td>
<td>30.9</td>
</tr>
<tr>
<td>Pancreas (ICD 157)</td>
<td>10.8</td>
</tr>
<tr>
<td>Lung (ICD 162)</td>
<td>79.7</td>
</tr>
<tr>
<td>Prostate (ICD 185)</td>
<td>34.3</td>
</tr>
<tr>
<td>Diabetes mellitus (ICD 250)</td>
<td>11.1</td>
</tr>
<tr>
<td>All Circulatory diseases (ICD 390-459)</td>
<td>450.9</td>
</tr>
<tr>
<td>Ischaemic heart disease (ICD 410-14)</td>
<td>281.7</td>
</tr>
<tr>
<td>Cerebrovascular disease (ICD 430-8)</td>
<td>85.9</td>
</tr>
<tr>
<td>Pneumonia (ICD 480-6)</td>
<td>83.7</td>
</tr>
<tr>
<td>Bronchitis and allied conditions (ICD 490-8)</td>
<td>63.4</td>
</tr>
<tr>
<td>Chronic liver disease and cirrhosis (ICD 571)</td>
<td>8.3</td>
</tr>
<tr>
<td>All accidents and adverse effects (ICD E800-E949)</td>
<td>22.5</td>
</tr>
<tr>
<td>Road vehicle accidents (ICD E810-29)</td>
<td>8.6</td>
</tr>
<tr>
<td>Suicide (ICD E950-9, E980-9, excluding E988.8)</td>
<td>14.9</td>
</tr>
<tr>
<td>---------</td>
<td>------</td>
</tr>
<tr>
<td>All causes</td>
<td>1,102.4</td>
</tr>
<tr>
<td>All Malignant Neoplasms (ICD 140-208)</td>
<td>249.4</td>
</tr>
<tr>
<td>Stomach (ICD 151)</td>
<td>10.3</td>
</tr>
<tr>
<td>Colon, rectum, rectosigmoid junction &amp; anus (ICD 153-4)</td>
<td>29.0</td>
</tr>
<tr>
<td>Pancreas (ICD 157)</td>
<td>11.5</td>
</tr>
<tr>
<td>Lung (ICD 162)</td>
<td>42.0</td>
</tr>
<tr>
<td>Breast (ICD 174)</td>
<td>47.2</td>
</tr>
<tr>
<td>Uterus (ICD 179-82)</td>
<td>9.9</td>
</tr>
<tr>
<td>Diabetes mellitus (ICD 250)</td>
<td>12.5</td>
</tr>
<tr>
<td>All circulatory diseases (ICD 390-459)</td>
<td>471.0</td>
</tr>
<tr>
<td>Ischaemic heart disease (ICD 410-14)</td>
<td>225.9</td>
</tr>
<tr>
<td>Cerebrovascular disease (ICD 430-8)</td>
<td>140.9</td>
</tr>
<tr>
<td>Pneumonia (ICD 480-6)</td>
<td>125.7</td>
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<td>Bronchitis and allied conditions (ICD 490-6)</td>
<td>43.0</td>
</tr>
<tr>
<td>Chronic liver disease and cirrhosis (ICD 571)</td>
<td>5.4</td>
</tr>
<tr>
<td>All accidents &amp; adverse effects (ICD E800-E949)</td>
<td>16.0</td>
</tr>
<tr>
<td>Road vehicle accidents (ICD E810-29)</td>
<td>3.7</td>
</tr>
<tr>
<td>Suicide (ICD E950-9, E980-9 excluding E988.8)</td>
<td>4.9</td>
</tr>
</tbody>
</table>

Source: Department of Health (http://www.doh.gov.uk)
Our Ref: EC35/98

26 October 1998

Ms Irene Manaras
School of Human Sciences
Department of Psychology
University of Surrey
Guildford GU2 5XH

Dear Ms Manaras

Self-Blame: a pilot study

Thank you for your letter of 24 September enclosing a revised patient information document.

I am now able to enclose a signed copy of the Application Form which confirms the Committee's approval of your study.

Yours sincerely

Dr J W Wright
Chairman

Enc
This is a questionnaire exploring peoples feelings about their illness. Please, read each statement carefully, and tick the box that best describes how you feel about the statement. Please be as honest as you can. Your answers are strictly confidential. If at any time you feel any distress or anxiety while completing the questionnaire please feel free to say so, and I will be more than glad to discuss any relevant issue with you. Alternatively, for any queries or comments contact me at 01483-300800, ext.2892 during office hours.

Thank you for agreeing to take part in this study.

Before you go on with the questionnaire, I need some general information from you that will help me understand your answers better.

Age: .................................

Male / Female (delete as applicable)

Reason for being in the hospital/clinic: .................................................................

.................................................................

Occupation: .................................
<table>
<thead>
<tr>
<th></th>
<th>Strongly Disagree</th>
<th>Disagree</th>
<th>Slightly Disagree</th>
<th>Neither Agree nor Disagree</th>
<th>Slightly Agree</th>
<th>Agree</th>
<th>Strongly Agree</th>
<th>I don't know</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) I don't know what caused my illness.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>2) I could have prevented getting my illness.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>3) I am responsible for my own health.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>4) Whenever I see the doctor I feel guilty.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>5) I should have looked after myself better.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>6) People around me blame me for my illness.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>7) I deserve to be in this situation because of who I am.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>8) I am to blame for my illness.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>9) I don't deserve to be looked after.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>10) I should have lived a more moderate life.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>11) My illness is God's way of punishing me.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>12) Getting my illness was just a matter of bad luck.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>13) I don't think I had anything to do with getting ill.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>14) I am not the only one responsible for my illness.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>15) My illness could have happened to anybody.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>16) Apart from me, a lot of other things (factors) led to my illness.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>17) I don't feel responsible for my illness.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>18) Getting ill was God's will.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>19) I should have taken better care of myself or I would have never fallen ill.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>20) I couldn't help getting sick.</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>
APPENDIX IV
QUESTIONNAIRE USED IN PHASE II OF STUDY 1 (CHAPTER 4)

ABOUT YOU....

Male ☐ Female ☐

Age: ..............................................................

Occupation: ..........................................................

Diagnosis /Reason for appointment: ..............................................

Time since initial diagnosis: ..........................................................

PART ONE

Please tick any of the following items that you believe have contributed to your medical condition.

Age
Alcohol
Heredity
Medication
Smoking
social class
Gender
Occupation
Hormones
Food additives
Personality
virus/germs etc

Diet
Pollution
Emotions
Environment
High blood Pressure
Being Overweight
Sexual Behaviour
Depression
Cholesterol
Stress

other ........................................

224
PART TWO

Take a minute to think about your medical condition in general. Then, read carefully the following questions and answer as honestly as possible by either filling in the blanks or by putting a circle around the number that best describes your answer.

<table>
<thead>
<tr>
<th></th>
<th>Strongly Disagree</th>
<th>Strongly Agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) I could have prevented my condition</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>2) I am responsible for my condition</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>3) I deserve to be in this situation because of choices I made</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>4) People around me blame me for my condition</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>5) I deserve to be in this situation because of who I am</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>6) I am to blame for my condition</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>7) My condition could have happened to anybody</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>8) My condition was God's way of punishing me</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>9) I don't feel responsible for my condition</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>10) I should have taken better care of myself</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>11) I couldn't prevent my condition</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>12) I had nothing to do with my condition</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>13) I should have behaved more moderately</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>14) My condition was just a matter of bad luck</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
</tbody>
</table>
PART THREE

Please, take a minute to think about a negative incident related to your medical condition that occurred within the last three months (e.g., bad test results, alarming symptoms, etc.).

Please, name that negative incident: ........................................

<table>
<thead>
<tr>
<th>Statement</th>
<th>Strongly Disagree</th>
<th>Strongly Agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) I could have prevented the incident</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>2) I am responsible for the incident</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>3) I deserved to be in that situation because of choices I made</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>4) People around me blamed me for the incident</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>5) I deserved to be in that situation because of who I am</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>6) I am to blame for the incident</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>7) This incident could have happened to anybody</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>8) The incident was God's way of punishing me</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>9) I don't feel responsible for the incident</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>10) I should have taken better care of myself</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>11) I couldn't prevent the incident</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>12) I had nothing to do with that incident</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>13) I should have behaved more moderately</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
<tr>
<td>14) The incident was just a matter of bad luck</td>
<td>1 2 3 4 5 6 7</td>
<td></td>
</tr>
</tbody>
</table>
PART FOUR

Below is a list of statements dealing with your general feelings about yourself. Please, put a circle around the answer that best describes how you feel.

<table>
<thead>
<tr>
<th></th>
<th>Not at all true</th>
<th>Exactly true</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) I can always manage to solve difficult problems if I try hard enough</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>2) If someone opposes me, I can find the means and ways to get what I want.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>3) It is easy for me to stick to my aims and accomplish my goals.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>4) I am confident that I could deal efficiently with unexpected events.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>5) Thanks to my resourcefulness, I know how to handle unforeseen situations</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>6) I can solve most problems if I invest the necessary effort.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>7) I can remain calm when facing difficulties because I can rely on my coping abilities.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>8) When I am confronted with a problem, I can usually find several solutions.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>9) If I am in trouble, I can usually think of a solution.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>10) I can usually handle whatever comes my way.</td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
</tbody>
</table>
PART FIVE

Please read each of the following items and circle the reply that comes closest to how you have been feeling in the past week.

1) I feel tense or 'wound up':
   i) Most of the time
   ii) A lot of the time
   iii) From time to time/Occasionally
   iv) Not at all

2) I still enjoy the things I used to enjoy:
   i) Definitely as much
   ii) Not quite so much
   iii) Only a little
   iv) Hardly at all

3) I get a sort of frightened feeling as if something awful is about to happen:
   i) Very definitely and quite badly
   ii) Yes, but not too badly
   iii) A little, but it doesn't worry me
   iv) Not at all

4) I can laugh and see the funny side of things:
   i) As much as I always could
   ii) Not quite so much now
   iii) Definitely not so much now
   iv) Not at all

5) Worrying thoughts go through my mind:
   i) A great deal of the time
   ii) A lot of the time
   iii) From time to time, but not too often
   iv) Only occasionally

6) I feel cheerful:
   i) Not at all
   ii) Not often
   iii) Sometimes
   iv) Most of the time

7) I can sit at ease and feel relaxed:
   i) Definitely
   ii) Usually
   iii) Not Often
   iv) Not at all

8) I feel as if I am slowed down:
   i) Nearly all the time
   ii) Very often
   iii) Sometimes
   iv) Not at all

9) I get a sort of frightened feeling like 'butterflies' in the stomach:
   i) Not at all
   ii) Occasionally
   iii) Quite Often
   iv) Very Often

10) I have lost interest in my appearance:
    i) Definitely
    ii) I don't take as much care as I should
    iii) I may not take quite as much care
    iv) I take just as much care as ever

11) I feel restless as I have to be on the move:
    i) Very much indeed
    ii) Quite a lot
    iii) Not very much
    iv) Not at all

12) I look forward with enjoyment to things:
    i) As much as I ever did
    ii) Rather less than I used to
    iii) Definitely less than I used to
    iv) Hardly at all

13) I get sudden feelings of panic:
    i) Very often indeed
    ii) Quite often
    iii) Not very often
    iv) Not at all

14) I can enjoy a good book or radio or TV program:
    i) Often
    ii) Sometimes
    iii) Not often
    iv) Very seldom
PART SIX

The questions below ask you about your self-care activities over the last 7 days. If you were ill during that time, please think back the last 7 days that you were not ill. Answer the questions as honestly and accurately as you can. Tick the answer that applies to you.

1. How often did you follow your recommended diet or general dietary guidelines over the last 7 days?

2. What percentage of the time did you successfully limit your calories as recommended in healthy eating for weight control?
   a. 0%  b. 25%  c. 50%  d. 75%  e. 100%

3. During the past 7 days, what percentage of your meals included high fibre foods such as fresh fruits, fresh vegetables, whole grain breads, dried beans and peas, bran etc?
   a. 0%  b. 25%  c. 50%  d. 75%  e. 100%

4. During the past 7 days, what percentage of your meals included high fat foods such as butter, ice cream, oil, nuts and seeds, mayonnaise, avocado, deep-fried food, salad dressing, bacon and other meat with fat or skin?
   a. 0%  b. 25%  c. 50%  d. 75%  e. 100%

5. During the last 7 days, what percentage of your meals included sweets and deserts such as pie, cake, jelly, soft drinks (regular, not diet drinks), cookies, biscuits?
   a. 0%  b. 25%  c. 50%  d. 75%  e. 100%

6. On how many of the last 7 days did you participate in at least 20 minutes of physical exercise?
   0 1 2 3 4 5 6 7

7. What percentage of the time did you participate in exercise sessions (other than what you do around the house or as part of your job)?
   a. 0%  b. 25%  c. 50%  d. 75%  e. 100%
Dear Ms Manaras

Self-blame in chronic patients

I am pleased to be able to inform you that at its meeting on 14 September 1999 the Ethics Committee approved the above study subject to the following conditions:

(i) The title of the study should be changed to "Feelings and thoughts related to medical conditions". This avoids any conflict between the information on the Patient Information Sheet and the study title.

(ii) No deviations from or changes of the Protocol should be initiated without prior written approval of the Committee.

(iii) The Committee should be provided with a copy of the report on the outcome of the study or a copy of any published document.

(iv) If the start of the project is delayed more than one year from the date of approval the Protocol should be resubmitted to the Committee for further review.

Yours sincerely

JOHN KERSLAKE
Co-ordinator
A. Scree plots from Principle Components Analyses carried out on a) the general and b) the specific self-blame items.

a) general self-blame

b) specific self-blame
B. Pattern matrices for a) four and c) three factor solutions of general self-blame

a) Four factor solution

<table>
<thead>
<tr>
<th>Component</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>could prevent condition</td>
<td>.800</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>am responsible</td>
<td>.801</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>choices I made</td>
<td>.841</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>people blame me</td>
<td>.699</td>
<td>.440</td>
<td></td>
<td></td>
</tr>
<tr>
<td>who I am</td>
<td>.511</td>
<td></td>
<td>.779</td>
<td></td>
</tr>
<tr>
<td>I am to blame</td>
<td>.799</td>
<td>.440</td>
<td></td>
<td></td>
</tr>
<tr>
<td>god's punishment</td>
<td></td>
<td>.366</td>
<td>.447</td>
<td>-.440</td>
</tr>
<tr>
<td>not responsible</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>should have cared for self</td>
<td>.748</td>
<td>.447</td>
<td></td>
<td></td>
</tr>
<tr>
<td>couldn't prevent condition</td>
<td>.962</td>
<td></td>
<td></td>
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<tr>
<td>should have behaved more moderately</td>
<td>.731</td>
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<td>-.360</td>
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<td>had nothing to do with</td>
<td></td>
<td>.853</td>
<td></td>
<td></td>
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<tr>
<td>could happen to anybody</td>
<td></td>
<td></td>
<td>.821</td>
<td></td>
</tr>
<tr>
<td>was just bad luck</td>
<td>-.485</td>
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</table>

Extraction Method: Principal Component Analysis.
Rotation Method: Oblimin with Kaiser Normalization.
a Rotation converged in 14 iterations.

b) Three factor solution

<table>
<thead>
<tr>
<th>Component</th>
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</thead>
<tbody>
<tr>
<td>could prevent condition</td>
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<tr>
<td>am responsible</td>
<td>.787</td>
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<td></td>
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<tr>
<td>choices I made</td>
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<td></td>
</tr>
<tr>
<td>people blame me</td>
<td>.691</td>
<td></td>
<td></td>
</tr>
<tr>
<td>who I am</td>
<td>.676</td>
<td>.587</td>
<td></td>
</tr>
<tr>
<td>I am to blame</td>
<td>.833</td>
<td></td>
<td>.698</td>
</tr>
<tr>
<td>god's punishment</td>
<td>.452</td>
<td>.587</td>
<td></td>
</tr>
<tr>
<td>not responsible</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>should have cared for self</td>
<td>.799</td>
<td></td>
<td></td>
</tr>
<tr>
<td>couldn't prevent condition</td>
<td>.870</td>
<td></td>
<td></td>
</tr>
<tr>
<td>should have behaved more moderately</td>
<td>.619</td>
<td>-.534</td>
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</tr>
<tr>
<td>had nothing to do with</td>
<td></td>
<td>.769</td>
<td></td>
</tr>
<tr>
<td>could happen to anybody</td>
<td></td>
<td></td>
<td>.490</td>
</tr>
<tr>
<td>was just bad luck</td>
<td>-.352</td>
<td></td>
<td></td>
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</tbody>
</table>

Extraction Method: Principal Component Analysis.
Rotation Method: Oblimin with Kaiser Normalization.
a Rotation converged in 5 iterations.
C. Pattern matrices for three factor solution of specific self-blame items.

<table>
<thead>
<tr>
<th>Pattern Matrix</th>
<th>Component</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1</td>
</tr>
<tr>
<td>could prevent condition</td>
<td>.776</td>
</tr>
<tr>
<td>am responsible</td>
<td>.795</td>
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<tr>
<td>choices I made</td>
<td>.867</td>
</tr>
<tr>
<td>people blame me</td>
<td>.731</td>
</tr>
<tr>
<td>who I am</td>
<td>.734</td>
</tr>
<tr>
<td>I am to blame</td>
<td>.805</td>
</tr>
<tr>
<td>could happen to anybody</td>
<td>.571</td>
</tr>
<tr>
<td>god's punishment</td>
<td></td>
</tr>
<tr>
<td>not responsible</td>
<td>.729</td>
</tr>
<tr>
<td>should have cared for self</td>
<td>.671</td>
</tr>
<tr>
<td>could'nt prevent condition</td>
<td>.656</td>
</tr>
<tr>
<td>had nothing to do with it</td>
<td>.868</td>
</tr>
<tr>
<td>should have behaved more moderately</td>
<td>.560</td>
</tr>
<tr>
<td>was just bad luck</td>
<td>.512</td>
</tr>
</tbody>
</table>

a Rotation converged in 11 iterations.
APPENDIX VII
LETTER OF ETHICAL APPROVAL FOR STUDY 2 (CHAPTER 5)
SOUTH WEST SURREY LOCAL RESEARCH ETHICS COMMITTEE
Postgraduate Medical Centre,
The Royal Surrey County Hospital, Egerton Road
Guildford, Surrey GU2 7XX
Tel: 01483 571122 ext 4382 Fax: 01483 303691

Chairman: Professor D L Russell-Jones MD FRCP Co-ordinator: Mr J Kerstake

Our Ref: EC49/00

17 May 2000

Ms I Manaras
Department of Psychology
University of Surrey
GUILDFORD
GU2 5XH

Dear Ms Manaras

Predictors of self-blame in chronic patients

Thank you for submitting the above study which was considered by the Ethics Committee at its meeting held on 16 May 2000.

The Committee made the following comments.

(i) With regard to the initial approach to patients, the Committee would welcome a more detailed explanation of the actual process involved. It would not be appropriate for you to be put in contact with patients without their prior consent. This means that the initial approach would need to come from the consultant or a member of his team working on his behalf. You may, therefore, need to revise your documentation to cover that point. For example, by revising the Patient Information Sheet for dispatch by the consultant or a member of his team or by producing, for his signature, a covering letter for the Patient Information Sheet. Patients responding positively could be asked to make contact with you direct.

(ii) The Patient Information Sheet states in the third paragraph that "No-one (except me) will be able to identify which questionnaire is the one you answered". Why do you need the "except me" qualification? If you do not need it, it should be removed.

If you would care to respond to the above points, together with any necessary revised documentation, I will ask the Committee Chairman to consider taking Chairman’s Action to approve the study if he considers your responses satisfactory.
PATIENT INFORMATION SHEET

‘Feelings and thoughts related to medical conditions’

Dear Sir/Madam,

As a PhD student at the University of Surrey carrying out research in Health Psychology, I am investigating some aspects of people’s feelings about their medical condition in order to understand the thoughts and emotions involved in various diseases such as heart disease. Knowing more about patients’ experiences is critical for health care improvement. It allows health professionals to provide better physical and psychological care for their patients by identifying possible signs of distress early and handling them effectively. This is why I need your help.

If you agree to participate in this study, you will be asked to complete a questionnaire regarding your thoughts and feelings towards your medical condition. This will require about 30 minutes of your time. If at any time before, during or after the completion of the questionnaire you need to ask me any questions, please do so. I would very much like to hear any comments and thoughts you may have about the questionnaire.

Please try to answer all the questions included in the questionnaire. The information you provide will be treated in strict confidence. It will not be disclosed to third parties and no-one (except me) will be able to identify which questionnaire is the one you answered.

Participation is completely voluntary. Refusing to take part in the study will NOT result in any penalties for you, and you will continue to receive the same care offered by your doctor. Also, once you have agreed to participate in the study, you are free to withdraw at any time.

However, I hope you will agree with me that it is important to understand better how patients feel about their illness and that you will be willing to take part in this study. Your help is very important for the success of this research.

If you decide not to take part in the study, please return the questionnaire to me.
If you decide to participate, please go on to the next page

Thank you for your time

The Researcher

Irene Manaras (MSc)
CONSENT FORM

'Feelings and thoughts related to medical conditions'

I have received and read a copy of this consent form and have had the opportunity to read and ask questions about the project. I realise that my participation is voluntary and that I may withdraw from the project at any time without penalty, and still receive the same care offered by my doctor. I am aware and agree that my GP will be notified about my participation in this study. By signing below, I agree to participate in the questionnaire survey carried out by Miss Irene Manaras, from the University of Surrey, under the terms stated above.

Participant’s name: _______________________

GP’s name: _______________________

Signature: _______________________

Date: _______________________

Before you start, please fill in the following:

a) Age:.........................

b) b) Sex: Male □ Female □

c) Diagnosis:.................................................................

d) When were you first diagnosed?......................................

e) What type of treatment have you had for your condition?

□ None □ Medication

□ Angioplasty □ By-pass

□ Other

f) Is there an incidence of the same condition in your family? □ Yes □ No

g) Are you currently on any medication? □ Yes □ No

If yes, what for? .................................................................

h) What is the highest qualification you obtained since high-school?

.................................................................

i) Occupation: ..............................
BEFORE the onset of my condition:

1) I was overweight.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

2) I was following a calorie controlled diet.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

3) My meals included high fat foods such as butter, ice cream, oil, nuts, mayonnaise, deep fried food, bacon and other meat with fat or skin.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

4) My meals included high fibre foods such as fresh fruit, fresh vegetables, whole grain breads, beans, peas etc.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

5) My meals included sweets and deserts such as pie, cake, cookies, biscuits etc.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

6) I exercised regularly.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

7) I led a physically active life.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

8) I used to smoke cigarettes.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

9) I led a stressful life.
   □ Not at all  □ Somewhat  □ A lot  
   □ A little  □ Quite

1) Smoking
   □ Not at all due to it  □ Mostly due to it  
   □ A little due to it  □ A lot due to it  
   □ Somewhat due to it

2) Unhealthy diet.
   □ Not at all due to it  □ Mostly due to it  
   □ A little due to it  □ A lot due to it  
   □ Somewhat due to it

3) Being overweight.
   □ Not at all due to it  □ Mostly due to it  
   □ A little due to it  □ A lot due to it  
   □ Somewhat due to it

4) Very little or no exercise.
   □ Not at all due to it  □ Mostly due to it  
   □ A little due to it  □ A lot due to it  
   □ Somewhat due to it

5) Stressful lifestyle.
   □ Not at all due to it  □ Mostly due to it  
   □ A little due to it  □ A lot due to it  
   □ Somewhat due to it

6) Heredity.
   □ Not at all due to it  □ Mostly due to it  
   □ A little due to it  □ A lot due to it  
   □ Somewhat due to it

7) Chance.
   □ Not at all due to it  □ Mostly due to it  
   □ A little due to it  □ A lot due to it  
   □ Somewhat due to it
C. Please, read the following statements and then tick (✓) the box that best describes how you feel.

1) I believe a bad diet is a habit I can avoid.
   □ Strongly disagree  □ Agree
   □ Disagree          □ Strongly agree
   □ Neither agree nor disagree

2) I believe smoking is a habit I can avoid.
   □ Strongly disagree  □ Agree
   □ Disagree          □ Strongly agree
   □ Neither agree nor disagree

3) I believe I can avoid a stressful lifestyle.
   □ Strongly disagree  □ Agree
   □ Disagree          □ Strongly agree
   □ Neither agree nor disagree

4) I believe that I can change my behaviour if I want to.
   □ Strongly disagree  □ Agree
   □ Disagree          □ Strongly agree
   □ Neither agree nor disagree

5) I believe I can change my character (personality) if I want to.
   □ Strongly disagree  □ Agree
   □ Disagree          □ Strongly agree
   □ Neither agree nor disagree

6) I believe I can avoid hereditary conditions.
   □ Strongly disagree  □ Agree
   □ Disagree          □ Strongly agree
   □ Neither agree nor disagree

7) I believe I can avoid conditions that happen by chance.
   □ Strongly disagree  □ Agree
   □ Disagree          □ Strongly agree
   □ Neither agree nor disagree

D. Please, assign one of the following numbers to each of the statements below:
   1 = strongly disagree
   2 = disagree
   3 = neither agree nor disagree
   4 = agree
   5 = strongly agree

1. My illness is a serious condition.  
2. My illness has had major consequences on my life.
3. My illness has become easier to live with.
4. My illness has not had much effect on my life.
5. My illness has strongly affected the way others see me.
6. My illness has serious economic and financial consequences.
7. My illness has strongly affected the way I see myself as a person.

F. Please put a tick in the box that best describes your answer.

SINCE the onset of my condition:

1) I have been overweight.
   □ Not at all  □ Somewhat  □ A lot
   □ A little  □ Quite

2) I am following a calorie controlled diet.
   □ Not at all  □ Somewhat  □ A lot
   □ A little  □ Quite
3) My meals include high fat foods such as butter, ice cream, oil, nuts, mayonnaise, deep fried food, bacon and other meat with fat or skin.

- Not at all
- Somewhat
- A lot
- Quite

4) My meals include high fibre foods such as fresh fruit, fresh vegetables, whole grain breads, beans, peas etc.

- Not at all
- Somewhat
- A lot
- Quite

5) My meals include sweets and deserts such as pie, cake, cookies, biscuits etc.

- Not at all
- Somewhat
- A lot
- Quite

6) I have been exercising regularly.

- Not at all
- Somewhat
- A lot
- Quite

7) I have been leading a physically active life.

- Not at all
- Somewhat
- A lot
- Quite

8) I have been smoking cigarettes.

- Not at all
- Somewhat
- A lot
- Quite

9) I have been leading a very stressful life.

- Not at all
- Somewhat
- A lot
- Quite

G. Please read the following items and tick the reply that comes closest to how you have been feeling in the past week.

1) I feel tense and wound up.

- most of the time
- from time to time
- a lot of the time
- not at all

2) I still enjoy the things I used to enjoy.

- definitely as much
- only a little
- not quite so much
- hardly at all

3) I get a sort of frightened feeling as if something awful is about to happen.

- most of the time
- from time to time
- a lot of the time
- not at all

4) I have lost interest in my appearance.

- definitely
- I may not take as much care as I should
- I don't take as much care as I should
- as ever.

5) I can laugh and see the funny side of things.

- As much as I always could
- definitely not so much now
- not quite as much now
- not at all

6) Worrying thoughts go through my mind.

- a great deal of the time
- from time to time
- a lot of the time
- only occasionally

7) I feel cheerful.

- not at all
- not often
- sometimes
- not at all

8) I can sit at ease and feel relaxed.

- definitely
- usually
- not often
- not at all

9) I feel as if I am slowed down.

- nearly all the time
- sometimes
- very often
- not at all

PLEASE TURN OVER
10) I get a sort of frightened feeling like butterflies in my stomach.
☐ not at all ☐ occasionally ☐ quite often ☐ very often

11) I feel restless as if I have to be on the move.
☐ very much indeed ☐ not very much ☐ quite a lot ☐ not at all

12) I look forward with enjoyment to things.
☐ as much as I ever did ☐ definitely less than I used to
☐ rather less than I used to ☐ hardly at all

13) I get sudden feelings of panic.
☐ very often indeed ☐ not very often ☐ quite often ☐ not at all

14) I can enjoy a good book or radio or TV program.
☐ often ☐ not often ☐ sometimes ☐ very seldom

THE END ! ! !

Thank you very much for your participation in this study.
Thank you very much for participating in this survey.

If you have any questions or comments regarding this project please feel free to ask me now or contact me at the following phone/address.

Please keep this page for future reference.

Irene Manaras
Departments of Psychology
University of Surrey
Guildford
Surrey GU2 5XH
Tel. (01483) 876946
Fax. (01483) 259553
Dear Sir/Madam,

The best way to find out about public views and opinions regarding physical and mental health is to communicate with people themselves. Gathering such information is essential for making effective health policies. This is what this survey is about: it aims to collect information about people’s beliefs about various physical illnesses and misfortunes.

This survey is conducted by a research team at the University of Surrey. Below we try to answer some of the questions you may have about this survey.

**How was I selected?** Our Department occasionally obtains representative samples of the British population from market research support companies for research purposes. The people in the sample are selected through a complex statistical process: the communities are divided into neighbourhoods, the neighbourhoods are selected at random, and for each neighbourhood households are selected at random. Please note that the lists of names we obtain are usually large enough to support a number of studies over a long period of time. Unfortunately, this means that we may miss changes in occupancy that happened since we first obtained the sample. If this is the case with you, please accept our apologies. You can still participate in this survey if you want to.

**Who will have access to the information I give?** Your responses will be treated in strict confidence. We are required by law to keep any information you give us confidential. All data gathered during this survey is kept safe and secure at all times. Since your name and address will not appear on the questionnaire you will return, your anonymity is guaranteed.

**What is involved in participating?** Simply fill in the questionnaire and return it in the addressed, Freepost, envelope provided. This means that you do not need to attach a stamp on the envelope. Please return the questionnaire by March 20, 2001.

**Who can I contact if I have questions?** For any queries regarding the survey please write to: Irene Manaras, Department of Psychology, University of Surrey, Guildford, Surrey, GU2 7XH.

Thanking you in advance,

Irene Manaras

Irene Manaras
Take a few minutes to IMAGINE yourself in the following situation: You have recently been experiencing disturbing chest pains. After being admitted to the hospital and undergoing the appropriate examinations, you are diagnosed as suffering from heart disease.

Keeping in mind that in all other respects your life is exactly the same as in reality, please read the following statements carefully and respond by ticking (✓) the box that best describes how you feel.

<table>
<thead>
<tr>
<th></th>
<th>NOT AT ALL</th>
<th>A LITTLE</th>
<th>SOMEWHAT</th>
<th>VERY MUCH</th>
<th>COMPLETELY</th>
</tr>
</thead>
<tbody>
<tr>
<td>How much would you blame yourself for your medical condition?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much would you blame your condition on things you did (your behaviour)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>How much would you blame your condition on the kind of person you are (your character)?</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Section B.

**In order to better understand your answers in this questionnaire, I need some information about your lifestyle. There are no right or wrong answers, just your own view of things.**

**Please put a tick (✓) in the box that best describes your answer:**

<table>
<thead>
<tr>
<th>Statement</th>
<th>NOT AT ALL</th>
<th>A LITTLE</th>
<th>SOMEWHAT</th>
<th>QUITE</th>
<th>A LOT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I am overweight.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I am following a calorie controlled diet.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>My meals include high fat foods such as butter, ice cream, oil, nuts, mayonnaise, deep fried food, bacon and other meat with fat or skin.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>My meals include high fibre foods such as fresh fruit, fresh vegetables, whole grain breads, beans, peas etc.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>My meals include sweets and deserts such as pie, cake, cookies, biscuits etc.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I exercise regularly.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I smoke cigarettes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I lead a stressful life</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I lead a physically active life</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Back to reality now....
Please, read the following statements and then tick (√) the box that best describes how you feel about them.

<table>
<thead>
<tr>
<th>Statement</th>
<th>STRONGLY DISAGREE</th>
<th>DISAGREE</th>
<th>NEITHER AGREE NOR DISAGREE</th>
<th>AGREE</th>
<th>STRONGLY AGREE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I believe a bad diet is a habit I can avoid.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I believe smoking is a habit I can avoid.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I believe I can avoid a stressful lifestyle.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I believe that I can change my behaviour if I want to.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I believe that I can change my character (personality) if I want to.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Thank you for getting this far!
Section D.

The first column of the grid below (words in capital letters) contains a list of events or medical conditions. For each of these events or conditions, please answer the questions at the top of the grid and put your answer in the corresponding empty cell. Choose your answers from the following scale:

1= not/none at all  2= a little  3= somewhat  4= quite a lot  5= very much

If, for example you think that for ‘heart disease’ the answer to the question ‘How much is the sufferer responsible for causing this?’ is 3 = somewhat, you should write 3 in the first empty cell.

**There should be no empty cells when you have finished.**

<table>
<thead>
<tr>
<th><strong>HEART DISEASE</strong></th>
<th><strong>How much is the sufferer responsible for causing this?</strong></th>
<th><strong>How much control does the sufferer have over causing this?</strong></th>
<th><strong>How much is the sufferer to be blamed for causing this?</strong></th>
<th><strong>How much could the sufferer have avoided this?</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HIGH BLOOD PRESSURE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ADULT DIABETES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>CHILDHOOD DIABETES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>LUNG CANCER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BREAST CANCER</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>STROKE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>AIDS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>RAPE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>DOMESTIC VIOLENCE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>PARALYSIS DUE TO CAR ACCIDENT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>PARALYSIS DUE TO SPORT ACCIDENT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BURN INJURIES AT WORK</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>BURN INJURIES FROM A KITCHEN ACCIDENT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*When answering this question for the event of 'rape', think about 'managing the aftermath of the event'.

Have you experienced any of the conditions or events listed in the first column?

No □  Yes □  If yes, which one(s)? .................................
Please fill in the following:

AGE: .................. YEARS
SEX: MALE □  FEMALE □

What is the highest qualification you have obtained either while at high school or since leaving school? .................................................................

Occupation: (previous occupation if retired) .....................................................

Do you suffer from any major medical conditions? No □ Yes □
If yes, please specify: .............................................................................................

Do any of your close family members suffer from any major medical conditions?
No □ Yes □ If yes, please specify: ..............................................................................

THE END ! ! !

Thank you very much for your participation in this study.
Agglomeration schedule from Hierarchical Cluster Analysis of ‘blame’ ratings.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Cluster combined</th>
<th>Sage Cluster First Appears</th>
<th>Next stage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cluster 1</td>
<td>Cluster 2</td>
<td>Coefficients</td>
</tr>
<tr>
<td>1</td>
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<td>6</td>
<td>71</td>
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<td>2</td>
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<td>3</td>
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<td>4</td>
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<td>2</td>
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<td>4</td>
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<td>168.5</td>
</tr>
<tr>
<td>9</td>
<td>12</td>
<td>13</td>
<td>209</td>
</tr>
<tr>
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<tr>
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<td>1</td>
<td>12</td>
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<td>4</td>
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</table>
Agglomeration schedule from Hierarchical Cluster Analysis of ‘responsibilities’ ratings.

<table>
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<tr>
<th>Stage</th>
<th>Cluster combined</th>
<th>Sage Cluster First Appears</th>
<th>Next stage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cluster 1</td>
<td>Cluster 2</td>
<td>Coefficients</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
<td>6</td>
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<td>7</td>
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<td>5</td>
<td>1</td>
<td>2</td>
<td>144</td>
</tr>
<tr>
<td>6</td>
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<td>1</td>
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<td>10</td>
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</table>
Agglomeration schedule from Hierarchical Cluster Analysis of ‘control’ ratings.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Cluster combined</th>
<th>Sage Cluster First Appears</th>
<th>Next stage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cluster 1</td>
<td>Cluster 2</td>
<td>Coefficients</td>
</tr>
<tr>
<td>1</td>
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<td>13</td>
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<td>12</td>
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<td>511.42</td>
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</table>
Agglomeration schedule from Hierarchical Cluster Analysis of 'avoidability' ratings.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Cluster combined</th>
<th>Sage Cluster First Appears</th>
<th>Next stage</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cluster 1</td>
<td>Cluster 2</td>
<td>Coefficients</td>
</tr>
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<td>4</td>
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<td>4</td>
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