Copyright is owned by the Author of the thesis. Permission is given for a copy to be downloaded by an individual for the purpose of research and private study only. The thesis may not be reproduced elsewhere without the permission of the Author.
THE INFLUENCE OF ILLNESS COGNITIONS ON DISEASE COURSE IN RHEUMATOID ARTHRITIS

A thesis presented in partial fulfilment of the requirements for the degree of Master of Arts in Psychology at Massey University

GLEN HADDON

MARCH 1995
ABSTRACT

This study investigated the relation between a set of illness cognitions and certain other related psychological factors, and disease course in rheumatoid arthritis (RA). The illness cognitions were knowledge of RA, appraisal of one's current condition and expectations for the future, thinking style relative to one's RA, and locus of control. Measures of dispositional optimism/pessimism, negative affect, general psychological distress, and various demographic factors were also included. Disease course specifically excluded onset and outcome factors. It was operationalised as changes in difficulty with daily activities, changes in symptoms, speed of changes, remissions, and fluctuations.

Participants comprised 82 RA sufferers, all members of the Arthritis Foundation who volunteered to complete a self-administered mailed questionnaire. Results showed that after controlling for the non-cognitive and demographic factors, the illness cognitions, as a set, had no influence on the course of RA. The results did demonstrate however, that the appraisal of present condition and expectations for the future cognition was meaningfully associated with RA disease course (on all disease course components) when its effect was assessed in isolation, and after taking into account the influence of the remaining cognitions. Some explanations are offered for the relative importance of this illness cognition together with possible reasons for the failure of the remaining cognitions to display any significant effect on disease course. The pervasiveness of the appraisal/expectations cognition and implications arising from the findings are discussed in terms of the roles of care-givers and their input towards more favourable disease course in RA.
ACKNOWLEDGMENTS

My sincere thanks go to my supervisor, Kerry Chamberlain, for his support and his example of a dedicated professional approach to research. I am especially appreciative of his being unrelenting in keeping me focused on the salient issues in my research, and in provoking me to thought and critical inquiry. I also gratefully acknowledge the assistance of Dr John Spicer from time to time, particularly with statistical analysis matters.

My appreciation also goes to all those members of the Arthritis Foundation, both in the Manawatu and Wanganui areas, who participated in the study by completing a lengthy questionnaire and consenting to their doctor’s involvement. My gratitude is also extended to the executives, office and field staff of the Manawatu and Wanganui branches of the Arthritis Foundation, without whose preparedness to be involved and many hours of practical assistance throughout, the research would have been difficult to undertake.

To those doctors who completed and returned doctors’ confirmation questionnaires, and particularly to Dr Gear, of the Rheumatology Department at Palmerston North Hospital, who attends a number of the participants, and whose comments were most helpful, I extend my sincere thanks also.

Finally, to my family, especially my sons, Matthew, Jeremy, Gareth, and Jonathan, and my friends, who allowed me much of their time, and who supported me in many ways throughout this endeavour, a very special thanks.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>CHAPTER</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Introduction</strong></td>
<td>1</td>
</tr>
<tr>
<td>Disease and the Mind-Body Connection</td>
<td>1</td>
</tr>
<tr>
<td>Purpose of the Study</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td><strong>Rheumatoid Arthritis: The Physical Disease</strong></td>
<td>5</td>
</tr>
<tr>
<td>Disease Description</td>
<td>5</td>
</tr>
<tr>
<td>Epidemiology</td>
<td>6</td>
</tr>
<tr>
<td>Aetiology</td>
<td>7</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>7</td>
</tr>
<tr>
<td>Treatment</td>
<td>9</td>
</tr>
<tr>
<td>Drugs</td>
<td>9</td>
</tr>
<tr>
<td>Physical Treatments</td>
<td>10</td>
</tr>
<tr>
<td>Surgical Intervention</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td><strong>Rheumatoid Arthritis: Psychological Aspects</strong></td>
<td>11</td>
</tr>
<tr>
<td>Personality</td>
<td>11</td>
</tr>
<tr>
<td>Psychosocial Factors</td>
<td>13</td>
</tr>
<tr>
<td>Psychopathology and Rheumatoid Arthritis</td>
<td>14</td>
</tr>
<tr>
<td>The Illness Cognition Approach</td>
<td>15</td>
</tr>
<tr>
<td>Thinking Style</td>
<td>17</td>
</tr>
<tr>
<td>Current Appraisals and Future Expectations</td>
<td>19</td>
</tr>
<tr>
<td>Belief in External Control</td>
<td>22</td>
</tr>
<tr>
<td>Knowledge and Understanding of Rheumatoid Arthritis</td>
<td>26</td>
</tr>
<tr>
<td>Summary</td>
<td>29</td>
</tr>
</tbody>
</table>
Demographic Variables .................................................. 72
Summary of the Relationship Between Illness Cognitions
and Disease Course .................................................. 72

8 Discussion .................................................................. 74
Disease Course as a Criterion Measure ......................... 75
Illness Cognitions and Disease Course in Rheumatoid
Arthritis ................................................................. 77
The Effect of Non-Cognitive Factors .............................. 82
Doctor’s Confirmation .................................................. 84
Implications Arising from the Study .............................. 85
Limitations ................................................................ 87
Future Research ......................................................... 89
Conclusion .................................................................. 90

References .................................................................. 92

Appendix I Participant’s Questionnaire ......................... 100
Appendix II Doctor’s Questionnaire ................................. 115
Appendix III Discriminant Analyses Results .................. 117
### LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>American Rheumatism Association criteria for the classification of rheumatoid arthritis</td>
</tr>
<tr>
<td>2</td>
<td>Descriptive characteristics of participants</td>
</tr>
<tr>
<td>3</td>
<td>Intercorrelations among independent variables</td>
</tr>
<tr>
<td>4</td>
<td>Intercorrelations among dependent variables</td>
</tr>
<tr>
<td>5</td>
<td>Intercorrelations between independent and dependent variables</td>
</tr>
</tbody>
</table>
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>FIGURE</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>The relationship between illness cognitions, non-cognitive psychological factors and disease course</td>
</tr>
<tr>
<td>2</td>
<td>Appraisal of present condition and future expectations patterns</td>
</tr>
</tbody>
</table>
CHAPTER ONE

Introduction

“We all know how physical illness or pain affects the way we think and feel. Now science is making exciting discoveries about how the way we think and feel affects all the rest of our bodily systems.”

(C. David Jenkins, 1994)

Disease and the Mind-Body Connection

The holistic approach to the mind-body connection has had an on-again off-again history. The 17th century philosopher, Rene Descartes, is generally credited with a return to the dualistic notion of the separate functioning of mind and body, and this Cartesian model has predominated in medicine, at least until recent times.

The scientific era of medicine, that developed with the discovery in the 19th century that micro-organisms caused various diseases, and the increase in physiological understanding, further entrenched the dualistic approach. As McMahon & Hastrup (1980) note, “psychosomatic” disorders were categorised as “nervous” and were dissociated from any physiological process. Such somatic complaints “of nervous origin” were seen as having no physical basis.

In the early part of the 20th century it was psychiatry that contributed to the understanding of the connection between psychological factors and physical illness, possibly because of poor integration of knowledge and practices between psychiatry and psychology (Gatchel, Baum, & Krantz, 1989). In the second half of this century however, with the development and expansion of psychology as a discipline, the input of psychologists into the arena of health and illness has steadily increased, until in the
last decade or so medical science has accepted the importance of the contributions of psychology and the need to treat patients as "whole" human beings. There is now a realisation that psychological factors are important in the course of almost any disease (Gatchel et al., 1989).

In 1950 Alexander (1950) classified Rheumatoid Arthritis (hereafter referred to as RA) as one of the seven major psychosomatic disorders, and since that time the role of psychological factors in RA has been the subject of considerable research interest. Such a classification was helpful to focus attention on the psychological component of RA, although an underlying premise of this thesis is that the clinical realities of the course of RA should not be eclipsed by an over-enthusiastic quest for psychological concomitants of the disease. Two important points emerge therefore. One is that the physical symptoms have an organic and clinical reality. The other is that, as Bakal (1979) suggests, the term "psychosomatic" is misleading in that it is difficult to isolate illnesses that are psychosomatic from those that are not, because few illnesses, if any, have either a separate emotional or physical cause. Most are multifactorial in origin.

A definition then, of a psychosomatic disorder is: "Physiological dysfunctions and structural aberrations that result primarily from psychological processes rather than from immediate physical agents like those involved in the organic disorders" (Lachman, 1972, cited by Bakal, 1979).

The mind-body connection is the holistic approach where, as Lipowski (1977) put it, an understanding of health and disease requires a view of people as "individual mind-body complexes ceaselessly interacting with the social and physical environment in which they are embodied" (p. 234). A caveat, or at least a warning should be registered however. The lay perception of holistic medicine has assumed a shallow fashionable dimension comprising a preoccupation with environmental stressors and "alternative treatments". This lay approach lacks an understanding of the clinical, psychological and intrapsychic stressor basis of a given illness and also lacks the
scientific and professional precision that psychosomatic medicine brings to the “true” holistic approach. It is not the anecdotal lay approach to holistic health care and medicine that is embraced in this thesis.

**Purpose of the Study**

The overall purpose of this study is to consider certain illness cognitions that are expected to be the relatively every day experience of RA sufferers and to investigate any influence these cognitions may have on the course the disease takes. Some related more general psychological factors are included in the investigation. Disease course includes how the disease progresses, and fluctuations and remissions. It specifically excludes onset and outcome factors. Broadly, the illness cognitions comprise thoughts, feelings and beliefs about one’s condition and its prognosis, as every day experiences, and are expected to be reasonably identifiable and self-recognisable by the average RA sufferer.

Accordingly, if these cognitions are found to be associated with disease course, their enhancement or modification, whichever is appropriate, should be a reasonably attainable goal for sufferers and their care givers, without the need for complex cognitive-behavioural interventions, strategies or therapies. In other words “treatment” derived from the recognition and understanding of the role of these cognitions could be very much on a self-help basis for the sufferer, and a practical, inexpensive and individually tailored adjunct to the care regimens of health professionals and other care givers.

An associated subsidiary purpose of the study is to help promote the understanding and acceptance of psychological concomitants of clinical organic RA by sufferers, care givers and health professionals alike.
The intention is, before describing the study and its findings, to move from the introductory outline of the mind-body connection in its historical context to providing some clinical understanding of RA, its aetiology, diagnosis and treatment. This is followed by an introduction to the psychological aspects of RA that have dominated the literature until recently. This leads into a review of the historical background to theory and research specifically with respect to illness cognitions in RA. There is also consideration of the dependent variables that have been used in RA research and the problems and confusions that have arisen. The specific issues to be addressed, and the hypotheses to be tested are also identified in conjunction with the theoretical and conceptual bases of the study.
CHAPTER TWO

Rheumatoid Arthritis: The Physical Disease

Disease Description

"Arthritis" means joint inflammation, from the Greek word "arthron" for joint and "itis" being a combining form meaning an inflammation. The terminology is inadequate on two fronts however. The first is that RA has systemic as well as articular (joint) manifestations and the other is that it involves autoimmunity. It is systemic in that it will often produce effects such as pyrexia, nausea, rigors, weight loss and anaemia, and in its advanced stages RA can affect the heart (by inflammation), blood vessels (vasculitis), lungs (pulmonary fibrosis), and various other organs (by rheumatic nodules), making it potentially the most serious arthritic condition. Pincus, Callahan, and Vaughn (1987) report a significant decrease in life expectancy for RA patients. Autoimmune dysfunction is where the immune system attacks the healthy tissues of the body as well as those of antigens. There are therefore, two basic mechanisms involved in RA - an inflammatory process with exudate (a mixture of fluid, protein, cells and cell debris) and cell proliferation, and a necrosis of tissue process which is independent of inflammation (Fassbender, 1975).

Commonly however, RA is a chronic disorder of the muscoskeletal system that involves the synovial joints. Synovial joints are best described as the freely moveable joints of which there are 187 in the human body, all of which can be affected by RA (Burckhardt, 1984). To the sufferer it is generally the progressive disablement from painful, stiff and deformed synovial joints that is the important manifestation of RA.

RA is a progressive condition with quite clearly defined stages. It seems to begin with a synovitis - an inflammatory condition of the synovial tissue which has enlarged and
become filled with lymphocytes. X-rays will show no destructive changes at this point other than inflammation of the synovium (the membrane enclosing the synovial fluid which cushions and lubricates joints). In the next stage, the inflamed synovial tissue will grow into the joint cavity gradually destroying the articular cartilage. X-rays will show a narrowing of the joint space due to cartilage loss. The next stage will evidence bone erosion around the margins of the joint with joint deformities becoming apparent. In the final stage the inflammatory process will be subsiding and fibrous or bony ankylosis (fixation) will end the functional life of the joint.

Common symptoms include pain, tenderness, warmth, swelling, stiffness and decreased range of motion of the affected joint. Pain and stiffness are especially likely in the early morning. RA is characterised by unpredictable fluctuations in symptomatology and spontaneous remissions (Young, 1992).

**Epidemiology**

After osteoarthritis, RA is the most common of over 100 arthritic conditions. In western societies the prevalence of RA in the general population is at least 1% and possibly up to 3% (Rimon, 1989). It is particularly common in the elderly population, affecting 2% of men and 5.5% of women over 65 years of age (Hall, MacLennan, & Lye, 1993). In the general population also, RA is approximately three times more common in women than in men (Lawrence et al., 1989), although severe RA appears to affect men and women more equally (Anderson, Bradley, Young, McDaniel, & Wise, 1985).

Approximately 70% of patients experience the unpredictable fluctuations and remissions which characterise RA, while 10% to 15% experience a progressive disabling disease course in spite of treatment. The remainder (10% to 20%) experience stable mild symptoms with remission within two years (Young, 1992). Over 50% of patients experience significant work disability within five years of disease

Typically RA begins around the age of 40 (Smith, Peck, & Ward, 1990) although onset may occur at any age with the 20 to 50 age range being common, and especially post-menopausally in women.

**Aetiology**

The cause of RA is not known. There has been considerable progress however in the understanding of possible aetiologic and pathogenetic mechanisms in the onset and perpetuation of the RA disease process (Anderson et al., 1985). Various aetiologic pathways have been considered. As yet there is little evidence to support the role of endocrine, metabolic or nutritional factors. Neither do occupation, climatic or other demographic factors appear to be involved (Anderson et al., 1985).

The triggering of the autoimmune reaction has been studied in terms of bacterial and viral agents, although no specific organism has yet been implicated. B- and T-lymphocytes which have been related to susceptibility to RA, appear to be involved in the autoimmune response (Maini, 1989). A genetic basis for the development of RA has also been delineated (Winchester, Dwyer, & Rose, 1992). In any event, whatever the triggering mechanism, there is evidence of intense immunological activity within the synovium of those with RA (Burckhardt, 1984). It is the immunological pathway towards an aetiologic understanding of RA that appears to have been the most productive to date.

**Diagnosis**

In New Zealand diagnosis of RA is a standardised procedure. That is, the medical profession generally adopts the American Rheumatism Association 1987 Revised
Criteria for the Classification of Rheumatoid Arthritis (Arnett et al., 1988). This comprises seven criteria (set out in Table 1). A definite diagnosis of RA is defined by the presence of four or more of the criteria with the proviso that criteria one to four must be present for at least six weeks. They are descriptive criteria rather than disease biology based due to a lack of knowledge about aetiology and the lack of a specific diagnostic test (Hakala, Pollänén, & Nieminen, 1993). Hakala et al. (1993) found the 1987 American Rheumatism Association (ARA) criteria to be valid diagnostic criteria. They applied the criteria to 193 subjects who had a clinician’s diagnosis of chronic RA, and found that the ARA criteria distinguished clinical cases of RA from non-clinical or non RA cases. 103 patients met the criteria.

Table 1 American Rheumatism Association criteria for the classification of rheumatoid arthritis

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Morning stiffness</td>
<td>Morning stiffness in and around the joints, lasting at least 1 hour before maximal improvement.</td>
</tr>
<tr>
<td>2. Arthritis of 3 or more joint areas</td>
<td>At least 3 joint areas simultaneously have had soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician. The 14 possible areas are right or left proximal interphalangeal joints, metacarpophalangeal joints, wrist, elbow, knee, ankle and metatarsophalangeal joints.</td>
</tr>
<tr>
<td>3. Arthritis of hand joints</td>
<td>At least 1 area swollen (as defined above) in a wrist, metacarpophalangeal joint or proximal interphalangeal joint.</td>
</tr>
<tr>
<td>4. Symmetric arthritis</td>
<td>Simultaneous involvement of the same joint areas (as defined in 2) on both sides of the body (bilateral involvement of proximal interphalangeal joints, metacarpophalangeal joints or metatarsophalangeal joints is acceptable without absolute symmetry).</td>
</tr>
<tr>
<td>5. Rheumatoid nodules</td>
<td>Subcutaneous nodules, over bony prominences, or extensor surfaces, or in juxtaarticular regions observed by a physician.</td>
</tr>
<tr>
<td>6. Serum rheumatoid factor</td>
<td>Demonstration of abnormal amounts of serum rheumatoid factor by any method for which the result has been positive in &lt;5% of normal control subjects.</td>
</tr>
<tr>
<td>7. Radiographic changes</td>
<td>Radiographic changes typical or rheumatoid arthritis on posteroanterior hand and wrist radiographs, which must include erosions or equivocal body decalcification localized in or most marked adjacent to the involved joints (osteoarthritis changes alone do not qualify).</td>
</tr>
</tbody>
</table>
Treatment

There is no known cure for RA to date (Young, 1992). Treatment is largely symptom management rather than prevention or treatment of the disease process (Scott, 1980; Stewart, 1991).

Drugs

Drug therapy is the major medical intervention. Systemic drugs administered comprise two main categories.

1. **Non-Steroidal Anti-inflammatory Drugs (NSAIDs)**
   These suppress inflammation which will relieve pain, so should obviate the need for analgesics. They are to be preferred to analgesics as to simply reduce pain encourages the use of swollen and inflamed joints which make damage to 'soft' joint structures much more likely. Pain reduction should therefore be accompanied by inflammation reduction (Pritchard, 1989). NSAIDs can produce some gastric side effects but are otherwise non-toxic (Stewart, 1991).

2. **Disease Modifying Antirheumatic Drugs (DMARDs)**
   Often referred to as "second-line" drugs that follow the anti-inflammatory drugs, DMARDs appear to suppress the disease process, thereby retarding the destructive processes of RA (Furst, 1990). Drug toxicity often limits their use however - these are powerful drugs that in some cases have been developed for the likes of cancer treatment and malaria prevention. Aggressive use of these drugs early in the course of RA to prevent joint destruction has been recommended (Wolfe, 1990).

The other major drug therapy comprises the injection of corticosteroids into the intra-articular space. While this may produce a reduction in symptoms, albeit temporarily, they do not appear to affect the underlying disease process
or its progression, and through osteomalacial change may exacerbate joint destruction (Pritchard, 1989).

**Physical Treatments**
Essentially physical treatments are of a physiotherapy and occupational therapy nature, both aimed at minimising joint damage and maximising functional capacity and personal independence. Physiotherapy will, essentially comprise ‘range of motion exercises’ and ‘strengthening exercises’ (Pritchard, 1989). The aim of the one is to prevent loss of movement and joint function, and of the other is to build and strengthen muscles supporting the joints and get involved in movement. Knowledge and experience in these therapies is essential - the inflamed and ‘active’ joint must be treated with special care (Pritchard, 1989).

Occupational therapy will provide advice and assistance in undertaking daily activities to improve functioning and minimise pain, damage, and development of deformity. It may also advise on the availability of devices and aids and assist with the use of practical aids such as splints prescribed by doctors and physiotherapists. Generally, and most importantly, these therapists aim to enhance a sufferer’s independence and quality of life.

**Surgical Intervention**
Surgical procedures include synovectomy (removal of the synovial membrane), arthroplasty (replacement or remodelling of damaged joints), and arthrodesis (fixation of a joint to prevent further movement) (Stewart, 1991). Early operative treatment in the form of synovectomy is reasonably common and can be effective for prevention and correction of deformity (Hall et al., 1993). Replacement of affected joints with artificial one’s however, is a last resort approach for extreme cases.
CHAPTER THREE

Rheumatoid Arthritis: Psychological Aspects

"It is more important to know what kind of a person has a disease than what kind of a disease a person has"
(Sir William Osler)

Until comparatively recently both the professional and lay perspectives have viewed the psychological aspects of RA largely in terms of personality, psychosocial, and psychopathological factors. Aspects of these factors are still relevant, but research should be selective to avoid those areas that have been well documented, and those that have been found wanting.

**Personality**

For many disorders it has been 'fashionable' to investigate the possibility of a personality specific to the disorder, or at least to identify psychological traits and conflicts that go with a specific somatic condition. Accordingly, over many years the possibility of the 'arthritic type' personality has attracted considerable interest. This interest may not have permanently subsided, however recent more methodologically sound studies have found little or no support for an arthritic personality that predates this disease, leading, in some way, to onset (Anderson et al., 1985; Friedman & Booth-Kewley, 1987).

Personality characteristics which are not necessarily of a dispositional nature, such as depression, anxiety, and hostility, have been found to be correlated with RA (Friedman & Booth-Kewley, 1987). This is hardly evidence of the role of personality and
psychological factors in disease aetiology however. In non-disease-specific terms however, such personality factors may influence disease course.

The only personality characteristic of a dispositional nature of interest in this study is dispositional optimism/pessimism. There are two reasons for this. One is that dispositional optimism/pessimism is not a construct that is associated with a particular disease personality type. The other is that there is evidence that optimism impacts beneficially on physical well being (Scheier & Carver, 1985). For some years there has been an anecdotal and lay understanding that positive thinking is helpful (e.g., Peale, 1952). Research has essentially confirmed this and provided some understanding of the processes underlying these effects.

Scheier and Carver (1992) see the underlying process in dispositional optimism/pessimism as the idea that people's behaviour is greatly influenced by their expectations about the consequences of that behaviour. Scheier and Carver's (1985) model of behavioural self-regulation suggests that those who expect successful outcomes are more persistent and effective in their goal-directed behaviours. For the dispositionally optimistic RA sufferer this would translate into the tendency to have positive and optimistic expectations about, for example, treatment, therapy or disease prognosis generally. It would also translate into more health enhancing behaviours (Scheier & Carver, 1985).

Research supports the claim that optimism beneficially affects physical wellbeing. For example, in comparison to pessimists, optimists report fewer physical symptoms in the pre-examination period (Scheier & Carver, 1985) and recover more quickly following coronary bypass surgery (Scheier & Carver, 1987).

This argument is strengthened by research that examined the association between pessimistic explanatory style and health. Pessimistic people are those who explain bad events with stable, global and internal causes while those who are not pessimistic
attribute negative events to unstable, specific and external causes (Peterson, Seligman, & Vaillant, 1988). In terms of physical health Peterson et al.'s (1988) 35 year longitudinal study showed that those with a pessimistic explanatory style were more likely to display poor health 20 to 35 years later. In addition Lin and Peterson (1990) found that pessimistic explanatory style is associated with the reporting of more illnesses over the past year and more negative appraisal of health, than for an optimistic explanatory style.

Further research is needed however, especially in terms of specific illnesses, since the findings are not entirely consistent. Some studies have found no association between optimism and physical health (see Scheier & Carver, 1992).

**Psychosocial Factors**

Stress is one of the most commonly recognised and studied variables in psychosomatic medicine. It has also received considerable attention with regard to RA. Accordingly a brief explanation is warranted as to why stress as a variable was omitted from the present study.

First, in terms of major life events, no association with RA disease status, global or current, has been found empirically (e.g., Thomason, Brantley, Jone's, Dyer, & Morris, 1992). These findings are not limited to the influence of stress on the aetiology of RA but are also applicable to the concept of disease course.

Secondly, while Thomason et al. (1992) did find a relationship between minor daily stressors and current RA activity, minor events are not considered to be particularly relevant to the relatively long term nature of RA disease course. That is, this study is not concerned so much with day to day fluctuations which may follow daily hassles, but rather with changes that fit into a longer time frame.
Finally, there would be considerable methodological difficulty in isolating those daily stressors which are not an integral part of the experience of having a chronic disease such as RA which brings with it frustrations and difficulties in many aspects of daily life.

At best, any link between stress and RA remains equivocal, particularly given methodological problems in the research (Anderson et al., 1985; Stewart, 1991). In a substantive sense, Koehler (1985) was not optimistic about any association between stress and RA, citing inconsistency in findings, and major studies that find no relationship at all.

**Psychopathology and Rheumatoid Arthritis**

There has been considerable research confirming elevated levels of psychological distress or disturbance among RA sufferers (see Anderson et al., 1985 and Achterberg-Lawlis, 1982, for reviews). The evidence from a number of studies is that the qualitative nature of the psychological distress reaction is essentially a depressive one. A variety of instruments has been used consistently revealing elevated levels of depression in RA patients (Pritchard, 1989). For example, Liang et al. (1984) found that 41.2% of their sample of 160 RA patients had abnormally high scores on the depression scale of the MMPI.

The recurring question is whether psychological distress is antecedent to or is a response to the RA condition. The latter is more tenable from the evidence. Distress being implicated in the aetiology of RA is supported by some evidence, but is still a contentious issue (Pritchard, 1989). Psychological distress may be a response to the fact of having a condition such as RA with possible accompanying feelings such as negative affect and expectations, fear and shame, or it may be a response to symptomatology, such as constant pain and functional impairment. The former is
more pervasive than the latter and probably reflects more enduring trait or dispositional factors.

Other aspects of psychological distress which have attracted much less research interest with respect to RA are, inter alia, anxiety, hostility and psychoticism. For example, subjective clinical reports have suggested elevated anxiety is a result of RA, although there is little supporting research evidence (Anderson et al., 1985).

It is expected that the general measure of psychological distress employed in this study (the Hopkins Symptom Checklist -21) would reveal any significant psychopathological trends. Negative affect operationalised as daily mood, was included in the study essentially to control for the effect of current mood on self report of current health status.

Thus far, this chapter has briefly reviewed some ‘traditional’ approaches to the psychological aspects of RA. Of these factors, three aspects have been incorporated into the present study - dispositional optimism/pessimism, general psychological distress, and negative affect.

The Illness Cognition Approach

It is only comparatively recently that the psychosomatic arena has been the subject of scientific research. It has been suggested that this is as recent as the 1930’s with Cannon’s research demonstrating that emotions can cause physiological changes that could influence disease development (c.f., Brannon & Feist, 1992).

Research attention to the narrower focus of illness cognitions however, is an especially recent development of the last two decades. However, even the prior theoretical bases of illness cognition, for example, Beck’s (1974) health belief model, have been found wanting in the last decade (c.f., Leventhal & Nerenz, 1985).
In terms of RA, a 1985 review of psychological factors in RA (Anderson et al., 1985) made reference to studies of cognitive factors but only in terms of interventions. By 1992 in an updated review of psychological factors in RA (Young, 1992), various studies had been reported which had investigated illness cognitions in RA. Young (1992) refers to some aspects of illness cognitions as being “one of the most important new arenas of psychological research in RA” (p.621).

In the context of a somatic complaint, illness cognitions are broadly an individual’s cognitions or thoughts associated with having that condition. Croyle and Ditto (1990) define illness cognitions as “any mental activity (e.g., appraisal, interpretation, recall) undertaken by an individual who believes himself or herself to be ill, regarding the state of his or her health and its possible remedies” (p.31-32). Illness cognitions will include the meaning an individual ascribes to his or her disease. They may include understanding, perceptions and beliefs about the disease, attitude towards the disease and its consequences, or beliefs and attitudes about the fact of having the disease. This may also include a future element in terms of understanding and beliefs about how the disease develops and progresses, and about one’s own disease prognosis and the likely effect it will have on one’s life.

Illness cognitions may also be described as the way we think about our disease either specifically or about the concept of being ill generally. Thinking about one’s disease may include thoughts about how one contracted it, how one may have exacerbated it or slowed its progress, or how one may influence its future course. Thinking about one’s disease includes response to it and how that may become an agent affecting the progression of the disease.

The illness cognition approach comprises what Berkowitz (1986) refers to as “commonsense models of illness”. The four illness cognitions of interest in the present
study, as predictor variables, together capture this so-called commonsense approach. Individually they have each attracted some research attention in relation to RA.

**Thinking Style**

Essentially an individual’s thinking style, relative to the topic of RA, consists of their attitude to their RA on a positive to negative continuum. Attitude is to be understood in terms of an individual’s interpretations of their RA experience. More specifically it is seen as negative (or positive) thinking relating to the three elements of the cognitive triad identified by Beck (1967) - one’s self, the world and the future. This forms the basis of the measurement instrument used in this study.

The thinking style variable has its roots in Beck’s (1976) cognitive theory of depression, which has it that profoundly altered thinking is the most salient psychological symptom of depression (Wilkinson & Blackburn, 1981). These cognitive deficits operate at different levels: Thought content that is negative with respect to self, the world and the future; processing of external stimuli in an error based way affecting appraisal of one’s environment and condition; cognitive structures which include attitudes and personal rules and directives. This cognitive theory can be applied specifically to the RA sufferer, producing the theory that the way such a pervasive chronic illness progresses, will be related to the cognitive style of the individual. The theory is that something less than clinical depression, namely a negative style of thinking (or a positive one), will potentially have a sufficiently profound effect on behaviours, immune functioning and other somatic processes, to influence actual disease parameters.

By definition this variable necessarily refers to assessment of the thinking style of those already diagnosed with RA. This raises the question as to whether thinking style predisposes people to develop RA or whether it results from the disease. Pow (1987) appears to be the first reported study investigating thinking style relative to RA. Her
conclusion with respect to this question was that negative thinking style developed as a result of RA, because subjects did not evidence negative thinking about other topics.

Pow's (1987) contribution effectively validates thinking style as an illness cognition and supports this with the Rheumatic Thoughts Questionnaire developed for that study.

Beyond Pow (1987) the literature, in terms of thinking style, is limited. Smith, Peck, Milano, and Ward (1988) for example, contribute with their finding that cognitive distortion (which is a narrower conceptualisation of thinking style) is significantly associated with depression and physical disability in an RA population.

The relationship of cognitive distortion to psychological variables continues to attract research attention. For example, Smith, Christensen, Peck, and Ward (1994) investigated whether cognitive distortion and helplessness contributed to depression among RA patients, and found that they did. A subsidiary finding was perhaps of more interest that initial (or pre-existing) depression did not predict changes in the cognitive processes. This suggests that thinking is unlikely to be contaminated by psychological conditions such as depression. Neither did cognitions interact with disability in predicting change in depression, suggesting that severity of disease did not alter the effects of cognitive factors. From this may be drawn the conclusion that cognitive factors comprise a robust construct in the face of depressive symptoms and disease severity variation, both of which characterise an RA population.

Thinking style also reaches into research about coping with RA. This is qualified by a limitation to the conceptualisation of coping as an individual's ongoing attempt or effort to deal with or manage his or her RA. Thus, coping may have an effect on health. More specifically, thinking style may be conceptualised as a coping strategy and as such may influence the clinical course of RA, not just how much pain or disability is experienced.
Of specific relevance are coping strategies which are concerned with management of the thoughts and feelings that an individual has regarding his or her RA. These include both emotion-focused and problem-focused strategies. According to Young (1992) there is considerable consensus that emotion-focused strategies that are passive and avoidant are associated with poorer coping, and also with negative affect, lower self esteem and greater depression. Problem-focused coping attempts that are active (e.g., rational thinking and cognitive restructuring) are associated with better coping, positive affect and less depression.

Most research in the coping strategy area has taken the adjustment/adaptation disease outcome approach (e.g., Newman & Revenson, 1993). Some more recently however, has taken the coping effort disease-related variable approach using pain and disability as disease related variables. For example, Beckham, Keefe, Caldwell, and Roodman (1991) investigating whether coping strategies affected pain and disability, found that those scoring high on a measure of cognitive type pain control and rational thinking, had lower levels of physical disability and pain.

This represents a start into assessing the effect of cognitive factors such as thinking style on actual disease-related factors, although caution is required. It is arguable that pain and disability are subjective and as such are inherently confounded with other psychopathological and psychosocial variables, even when disability reports are supported by rheumatologists examination (as in Beckham et al., 1991). Research into coping strategies need not necessarily be influenced by the outcome approach. Pain and disability, while disease-related variables are outcome variables.

Current Appraisals and Future Expectations
People assess or interpret their clinical condition and functional impairment as either worse than it really is, or moving through an accurate assessment to an interpretation that is more positive than the clinical reality would indicate. This is not suggestive of
the severity of either a hypochondriacal approach at the negative end or an unrealistic or denial type approach at the positive end. It is a question of how an individual responds to and interprets psychologically their somatic condition - their representations of it, their beliefs about it and their emotions. Appraisal and expectation is therefore seen as something more than a state of mind of expecting the best or the worst (as in dispositional optimism or pessimism) - it relates more to the underlying processes that accompany such attitudes. Negative appraisals and expectations processes comprise fear in terms of pathogenesis and prognosis, while positive appraisals and expectations are likely to subsume high motivation for adherence to treatment and therapy and to self-help strategies.

Appraisals and expectations are also likely to affect daily mood and distress levels. This in turn may influence disease progression via various mechanisms such as illness behaviours, depressed immuno-competence and somatic responses such as increased bodily tension. Unrealistic appraisals and expectations could in theory, also lead to inappropriate behaviours, poor medical adherence, and then psychological distress upon reappraisal or after expectations fail to come to fruition.

Most commonly appraisal is part of, and dealt with in conjunction with coping. This is true both conceptually (e.g., Berkowitz, 1986) and empirically (e.g., Smith & Wallston, 1992). This is probably due to the influence of the conceptualisation of coping developed by Lazarus and colleagues over a number of years (e.g., Lazarus & Folkman, 1984). Appraisal is a fundamental component of his model. He sees three types of appraisal - primary, secondary, and reappraisal. Primary appraisal is the individual’s evaluation of the situation. Secondary appraisal is the individual’s assessment of what, if anything, can be done about the situation. Reappraisal is a further assessment based on new information, additional resources and one’s earlier responses.
With the exception of Felton and Revenson (1984), there have been few systematic investigations of coping in RA's (Pritchard, 1989). Accordingly appraisal in the RA context has similarly received little attention. Smith and Wallston (1992) saw appraisal of the disease and its consequences as an integral antecedent to coping attempts. For example, the interpretation of the disease as a permanent malady that one is unable to influence, will promote inadequate passive coping, while interpreting disease as a challenge to be overcome, promotes the more 'successful' active coping strategies. In this model an individual's current disease status is central to the process, generalised beliefs and expectancies regarding one's internal resources and abilities being an additional factor.

Thus the appraisal component of the coping model lines up with the conceptual basis of appraisal in the present study. The current study, however, is not a coping study - appraisal is not seen just as an antecedent to coping. Rather, appraisals and expectations are seen as subsuming not only current disease status and its consequences, but also the broader notion of an individual's representations and interpretations of their RA, their beliefs and emotions about it, and how this may influence the disease process or modify actual disease parameters.

This conceptualisation of appraisal and expectation includes some aspects of the 'personal models of illness' approach also. Skelton and Croyle (1991) (cited by Hampson, Glasgow, & Zeiss, 1994) define personal models of illness as peoples' representations of their disease, including their disease-related beliefs, emotions, knowledge and experiences. There appears to be no studies reported to date that apply personal models of illness to RA. Hampson et al. (1994) however, investigated those models with respect to osteoarthritis. Aspects of their findings may be generalisable to RA as a related condition.

Hampson et al. (1994) found that the shared beliefs of osteoarthritis sufferers included perceiving osteoarthritis as serious, painful, chronic, incurable, and manageable by
medical treatment. In the present study it was expected that RA sufferers would share a similar general appraisal of RA as a disease. A different aspect of appraisal and expectancy however, is how a person appraises their own experience of RA at a given time. The data collected by Hampson et al. (1994) potentially provided these two forms of appraisal, that is, of the condition generally, and of one's own condition, but the study did not appear to isolate the responses and make any distinction accordingly.

The present study was expected to replicate Hampson et al. (1994) in so far as certain shared beliefs were concerned, but for an RA population. However, it endeavoured to obtain appraisals and expectations that were subjective and psychological in nature, rather than those beliefs that were more objectively based, about the disease in general. The latter amounts more to knowledge about a particular disease, while the subjective approach accounts for the personal perception of one's own situation. As Pollock (1993) points out, a salient issue in relation to physical illness is evaluating the nature of the person through his or her response to his or her illness. This effect is seen as distinct from the effect of objective knowledge about the disease generally, and which is a separate issue investigated in the present study.

Belief in External Control

This should also be viewed conceptually as a component of the illness cognition model. It is important however to focus on the belief to avoid any confounding effect of the operation of the external control agent itself. For example, there may be some clinical effect from an unorthodox treatment, or there may be a remission that follows prayer. It is important to remember that the belief does not empower the agent. It may however, mobilise or enhance physiological mechanisms like the neuroimmunological and neuroendocrinological systems, or create adherence motivation for prescribed treatments and therapies.

Given the acknowledgment of the medical profession that it knows of no cause or cure for RA, excessive reliance on medical personnel, who are generally the powerful
others for RA sufferers, could also result in fewer self-help strategies such as diet experimentation and non-prescribed exercising. Ironically also, the psychology of externality may negate the self-discipline required to adhere to a medical regimen, especially when there are aversive side affects. Since human behaviour is complex and multidetermined, health locus of control beliefs alone can not be expected to predict very much of the variance in health behaviour (Wallston & Wallston, 1981). Nevertheless, there are sound theoretical bases for inclusion of this cognition in the model.

Internality on the other hand, should generate greater knowledge and understanding, hypothesised to be beneficial, and should result in taking more responsibility, and therefore being more proactive about one’s condition and treatment. Experimentation, involvement with other sufferers and helping agencies, and response to one’s supportive environment may influence disease course in practical ways, and bring an improvement in daily mood, outlook and attitude, and overall psychological wellbeing. Externality however, encourages blaming as the condition worsens which in turn causes alienation from those agents that may potentially have been of some help, together with increasing feelings of loneliness, bitterness, and resultant psychological distress. Another mechanism that theoretically may link control to disease course is that continuing externality reinforces belief that RA is an incurable disease that will press on inexorably no matter what. This will result in a plethora of negative illness behaviours and psychological distresses borne out of the notion that there is nothing one can do to help oneself.

A large body of literature exists concerning people’s beliefs regarding their control over their health. Inevitably disease specific research has resulted, and RA is no exception. Because the clinical course of RA and symptom occurrence is unpredictable, RA is characterised by a lack of actual control (Schiaffino & Revenson, 1992). It is the individual’s perceived control over their RA however, and how that
may influence the course their RA takes, that is of interest to psychology and the present study.

Both in a conceptual and an empirical sense the essence of belief (or disbelief) in powerful external agents is one of control. The concept of locus of control was developed to explain the beliefs and expectations that people have of controlling factors that may influence their disease (Pastor et al., 1993). There are two kinds of control. Internal is where people believe they can influence events by their own means. External is when they feel that this influence is due to outside factors. In health research, external locus of control has comprised either ‘powerful others’, where control is perceived to be in the hands of another agency, or ‘chance’, referring to the belief that events are modified or affected by uncontrollable factors such as chance, fate or luck.

In a comparatively early study, Gardiner (1980) found that those chronic RA patients who were dependent (external locus of control) displayed a more unfavourable disease course measured by time off work through illness. Various researchers have found that RA patients report greater belief in external control, than do ‘normal’ controls or those suffering from more predictable chronic illness, and to be consistently low scorers on measures of internal control (Wallston, 1993; Felton & Revenson, 1984). Pow (1987) found that seropositive RA patients were much more likely to have powerful others locus of control beliefs than internal beliefs. Skevington and Woolf (1984) however, found that acute RA patients held strong beliefs about the powers of others to help them, but as chronicity developed this changed to beliefs that nobody could help and that disease outcomes like pain were the result of chance or misfortune. Thus the two dimensions of externality, control by powerful others and control by chance, may be mutually exclusive aspects of the same concept.

Wallston (1993) reviewed the concept of locus of control in relation to physical health and the literature on the topic. His conceptualisation of control was that it is not
possible simply to equate internality with perceived control and externality with lack of control. For example, some with an internal locus of control hold themselves responsible for their poor health, but do not believe they can contribute to rectify the situation. Similarly, believing that the actions of others can influence one’s health does not necessarily imply loss of control. A conscious balanced decision to consult and comply with a health professional is a form of taking responsibility. Internality and externality are not opposite ends of a single dimension - people can hold both internal and external beliefs about their health.

The construct of locus of control has, in effect however, been called into question by Roskam’s (1986) conclusion that it is not locus of control that is important but the fact of control regardless of locus. This would suggest that the distinction between the cognitive styles of belief in others versus belief in self is irrelevant. RA patients in that study who were ‘believers in control’ (high on internal and powerful others but low on chance) became less depressed despite high disease activity. A criticism of Roskam’s (1986) conclusion however, is that because a person scores highly on both internal and powerful others scales does not necessarily mean that locus is unimportant. As already noted, internality and externality are not mutually exclusive - they can cohabit in a complementary way in a number of circumstances.

Wallston’s (1993) review is particularly helpful for a conceptual exploration of the related constructs of perceived self-efficacy and helplessness/hopelessness and their relationships with perceived control. The review concludes with reference to an unpublished dissertation study (Callahan, 1992), showing that helplessness (low control) beliefs are highly predictive of which RA patients would die and which would survive. Those who felt most helpless about their condition were more likely to die and die sooner than those who felt more in control. Mortality is the ultimate criterion measure! Callahan’s study (1992) is an exception in the literature - it examines the possibility of an independent effect of control on clinical disease.
Affleck, Tennen, Pfeiffer, and Fifield (1987) explored control among RA patients that were both internal (personal) and external (health-care provider), in terms of their interactions with varying targets of that control (disease course, symptoms and treatment). For the present study disease course as a target of control is of particular interest. Affleck et al. (1987) found that patients made a distinction between their ability to control daily symptoms and their capacity to influence the course of their disease. Their health-care providers exerted greater control over disease course than they did themselves (powerful other external locus of control). Daily symptoms however, were perceived as being personally controllable.

**Knowledge and Understanding of Rheumatoid Arthritis**

This variable concerns the possession of information about, and the understanding of the clinical nature of the disease - its aetiology, physiology, pathology, treatment and prognosis. It is acknowledged that conceptually the knowledge variable, in addition to any direct effects on disease course, is likely to interrelate with the other illness cognitions of interest in the study to influence disease course. For example, appraisal of current situation and expectations for the future would likely be influenced by knowledge and understanding of the disease. An increase in knowledge about one’s condition could help the appraisal process alleviate any fears that may have resulted from an unknown future or from excessively severe expectations. It may also reduce the threat by enabling reappraisal of the situation as not so hopeless after all (Pritchard, 1989).

Similarly the way people think about their disease and its consequences is likely to be influenced by the depth and accuracy of their knowledge and understanding of it. Realistic thinking has a knowledge component. Parker et al. (1988) raised the possibility that RA sufferers who are unable to think realistically about their situation and to restructure their life goals, experience considerable psychological difficulties.
In the compliance literature also there is considerable reference to the effect of knowledge. Generally the findings have been inconclusive, although poor methodology, such as poor measures of patient knowledge, characterises much of the literature. Where knowledge is specifically relevant to the treatment, however, compliance has been found to be strongly related to the knowledge (e.g., Svarstad, 1974, cited by Pritchard, 1989).

The effect of knowledge about RA has been investigated mainly in intervention studies. In a review conducted by Lorig, Konkol, and Gonzalez (1987), out of 76 studies of arthritis patients, predominantly with RA, only six were non-intervention studies. In most intervention studies the aim is primarily to assess the effectiveness of an educational intervention. They do not, therefore, investigate the underlying need for the knowledge and the benefits of having it. Furthermore, only a few of these educational interventions were expressly designed to increase patient knowledge about the disease per se - most were educating about management and coping. Interestingly, the medical profession prefer education programmes that focus on coping, while patients report education needs for disease process, diagnostic procedure and disease helping strategies like nutrition (Silvers, Melbourne, Hovell, Wiseman, & Mueller, 1985). Patients therefore, seem more interested in disease modification, while doctors seem more interested in symptom management.

Of the studies that have examined knowledge about the disease, only a few have assessed the effect of this knowledge. Some have assessed the effect of knowledge on psychological adjustment, with reports of a positive effect of knowledge. Wetstone, Sheehan, Votaw, and Peterson (1982), reported an ‘improved outlook on life’ and more optimism about disease prognosis among ‘knowledgable’ RA patients. Kaye and Hammond (1978) also reported positive changes in those receiving an information intervention. Both of these studies are limited methodologically however. Sample sizes were 36 and 48 respectively. In the first study no details were given as to
knowledge assessment methods or control conditions and in neither study was the validity or reliability of the assessments established.

Lenker, Lorig, and Gallagher, (1984) assessed inter alia, the effect of knowledge/awareness of one’s arthritis on “positive and negative health outcomes”, concluding that while there was no association between improved health behaviour (resulting from a knowledge intervention) and improved outcome, there was a significant difference between those who felt they had more control over their disease and positive emotional status and those with less control and more negativity, in terms of their health outcomes in arthritis. This was attributable to the education intervention.

The coping literature also provides some examination of the effect of knowledge of RA. Felton and Revenson (1984) for example, found that information seeking was associated with decreased negative affect, although Parker, Lorish, and Brown (1984) reported contradictory findings. They found that depression scores did not alter, but that educational intervention was associated with increased reports of pain. Parker et al. (1988) found that the use of information seeking as a coping strategy was of no psychological or functional advantage to RA patients. However, information seeking is not necessarily synonymous with knowledge. It is a coping strategy that is in itself a psychological process.

Pritchard’s (1989) study is a major contribution to the question of patient knowledge in RA. This is especially so for her comprehensive analysis of the content of RA patients’ knowledge and misconceptions, which was previously lacking in the literature. Pritchard (1989) found only one previous study that reported the content of patients’ knowledge. This was Grennan, Taylor, and Palmer, (1978) who found that while up to 87% of RA patients were aware of their diagnosis, only 16% were aware that their disease was one of joint inflammation with a tendency towards remissions and fluctuations. That study emphasised the responsibility of the medical profession to
provide patients with knowledge and understanding of their arthritis. Pritchard (1989) also found that most patients had a confused and incoherent understanding of their illness.

Summary

This chapter has briefly outlined earlier research attention to psychosocial, psychopathological, and personality factors. The latter included dispositional optimism/pessimism as the personality variable of interest in this study. The chapter then introduced illness cognitions as an approach to the psychological aspects of RA. For each of the illness cognitions that have been selected for the composite model, the chapter provided a description, a theoretical basis, and rationale for inclusion in the study. Previous research that has investigated these illness cognitions on an individual basis with respect to RA, where appropriate, was then considered.

The following chapter introduces disease course as the criterion variable, defining it, giving some theoretical and conceptual considerations, and examining how disease course has featured in previous research.
CHAPTER FOUR

Disease Course as a Criterion

Since Anderson et al.,'s (1985) review there has been a shift in research emphasis away from aetiologic factors towards clinical disease course (Young, 1992). This shift is not accompanied by any particular advances or changes in the theoretical and conceptual basis of the psychology of RA. Rather, it is a reflection of methodological difficulties in aetiological research, as consistently noted by Anderson et al. (1985). It is also a reflection of the continued lack of a definite clinical understanding of the organic causes of RA. In this study disease course as the criterion is a further development from onset/causality type studies. It is also seen as being of considerably more potential practical application in terms of symptom reduction or moderation and disease progression than the ‘ambulance at the foot of the cliff’ approach of outcome studies which have tended to dominate the clinical literature. If the progression of disease towards severity can be favourably influenced, this must be a higher ideal than symptom/outcome treatment or management.

The definition of disease course in the psychological literature is elusive. Clinically it is a distinctive concept however. In psychosomatic research it is important that clinical variables are demonstrable and accepted in a clinical sense. In the clinical context the course of RA has been divided arbitrarily into three patterns: Progressive disease - chronic disease with an invariable trend towards progression with some fluctuations in severity; Intermittent course - brief attacks often lasting less than one year with intermissions for variable periods; Long clinical remission cases - lasting for more than one year (Scott & Huskisson, 1992). In the present study disease course was seen as definable as in the clinical literature, although participants were not categorised as above. Rather, the intention was to gain a clear and definable understanding of the nature of the course RA had taken for each participant and to relate this to their illness cognitions.
To capture the true nature of the course of RA, its progression in terms of changes in functional ability and symptoms, including changes in speed of that progression, its fluctuations, and any remissions, must all be assessed. These are essentially the characteristics of the course of the disease.

Disease Course is conceptualised therefore as a multi-faceted variable although either one of the major components - progression, fluctuations and remissions - could be investigated alone. Fundamentally disease course is the journey between onset and outcome of disease. Theoretically disease course is modifiable by the operation of illness cognitions. Any such modification can not affect its starting point but it may influence the destination.

It has been suggested that psychological factors as criterion variables are more appropriate than disease related criteria. For example, Pritchard’s (1989) main study investigated the interrelationship of knowledge with other psychological variables. While the present study is a partial replication of Pritchard (1989) with respect to exploring the content and extent of RA knowledge and understanding, it differs from her design in that it investigates the potential direct effect of knowledge and understanding on RA disease course. Pritchard (1989) would probably describe this as approximating the traditional ‘psychosomatic hypothesis’ approach - “the idea that psychological stress or personality variables can affect disease onset or prognosis”. Pritchard (1989) sees her study as a move away from that approach towards more psychological content. The present study in discarding some personality variables and disease onset, does however, subscribe to the fundamental ‘psychosomatic hypothesis’ that psychological variables can affect disease-related variables. Pritchard (1989) is correct in saying that more psychological content is needed, but there is also a need for clinically feasible and sound disease related dependent variables that are both recognisable and assessable. The ‘psychosomatic hypothesis’ having recovered from the set back created by personality type and onset factors, is alive and progressing well.
A problem arises in the literature however. There is a confusion among terms for disease course, disease outcome and clinical disease activity. The result is that disease course has rarely been investigated without contamination by disease outcomes and activity factors.

**Disease Course Vs. Disease Outcome**

According to McFarlane, Kalucy, and Brooks (1987), in general, psychosomatic research with respect to RA has concentrated on two hypotheses. One is the specificity hypothesis, that specific psychological traits can be identified in RA sufferers prior to onset of manifest disease. The other is the onset hypothesis - that RA onset is associated with stressful life events. McFarlane et al. (1987) suggest that since it is "unarguable" that the experience of RA evokes a psychological response, there is a possibility that the course the disease takes is influenced by this reaction. Hence the third hypothesis - the disease course hypothesis. This hypothesis was originally proposed by Meyerowitz (1970), but has received little attention.

McFarlane et al. (1987) provide no formal definition of disease course however. They simply state that "this hypothesis examines the impact of psychological variables on the course of RA" (p.757). They proceed to speak of "primary impairments of a disease", as being distinct from "functional outcome"; "rapid progression"; poor outcomes and good outcomes; and disease activity. In extracting these terms from the literature the authors concluded, rightly, that there was "uncertainty concerning the validity of the disease course hypothesis", and this led them to test the null hypothesis that "the progression of RA would not be influenced by the psychological characteristics of the sufferers".

"Progression" appears to be the operationalisation of disease course. More specifically this is referred to as "progression of disease activity", and was measured two times three years apart. This is not however, a sensitive measure of progress of a
disease that can fluctuate even daily. McFarlane et al. (1987) acknowledge that their study may be more an investigation of predictors of remission than of progression, but even in this regard, the study measured remission at one point in time only. RA is characterised by multiple periods of remission of varying duration.

The lack of a definition of disease course leads McFarlane et al. (1987) to confuse disease course, disease outcome and disease activity. Pain, functional impairment and disability, and psychological status (where this is a result of the disease) are outcome variables. Support for the view that there is a confusion, is found in the clinical literature (c.f., Scott & Huskisson, 1992). These authors consider that the distinction is not made clear in the literature. “Serious attempts to describe disease course must take into account the transitory nature, the waxing and waning, of many features of clinical importance” (Scott & Huskisson, 1992, p.1).

Further clarification of the distinction is provided by Van Der Heide et al. (1994). They define RA outcome as the amount of suffering experienced throughout the course of the disease, and can be described as physical disability, physical and psychological discomfort, financial costs and mortality. This sets outcome apart from course, at least in the clinical context. The psychological context should be aligned with this.

**Disease Course and Disease Activity**

Similarly a confusion has arisen between disease activity and disease course. McFarlane et al. (1987), for example, recognised that few studies had used reliable, valid and quantitative measures of disease activity. They addressed this problem by having a trained observer supervise the completion of standardised pain, stiffness, joint tenderness and size measures, followed up three years later, to measure disease activity. Their claim was that this change score (total disease activity) quantified disease progression, which in turn equated with disease course. Changes in physical
disease activity however, comprise one way of measuring the fluctuation and remission components of disease course. But they are narrow in application, are out of step with the time frame of most psychological variables and lend themselves to confounding with current treatment regimens.

The dependent variable in Thomason et al. (1992) was ‘disease activity’, which was operationalised as inflammation, pain and functional impairment. The predictor variable was minor stress. The finding that minor stresses of the previous week were significantly related to inflammation only, is helpful but of limited application. It demonstrates that a disease activity measure is restrictive both conceptually and temporally. Stressful events are able to be defined and located in time as can specific disease activity factors like inflammation. Most other psychological factors, including illness cognitions, are not necessarily episodic and definable in time. Disease course provides more of an overview of the pattern of the disease over a period of time. This distinguishes disease course from disease activity and makes it a more all-embracing concept vis a vis psychological processes.

Thomason et al. (1992) also tend to confuse activity with outcome. Hassell et al. (1993) however, make the distinction plain. Activity, they say is “the process (or what happens along the way) whilst severity is the outcome (or end result) after a specified period of time” (p.601). In the present study disease course is seen as inclusive of activity and certain outcome aspects as part of the process of assessment and description of the life and progression (in either direction) of the disease in a given individual. Disease course is not limited by the finality of the outcome concept, nor by the temporal specificity of the activity measure.

McFarlane and Brooks (1988) support the concept of “illness course” as being a combination of factors in which they included disease activity, disability, and duration of illness. In that study disease activity assumed independent variable status and it was found that disease activity was a less significant determinant of disability than
psychological factors were. In effect that study supports the contention that disease activity is only a part of disease course, and its restriction as a determinant of disease outcome must restrict its efficacy as a dependent variable also.

Disability is a disease severity type measure and is therefore a disease-related variable as distinct from a psychological variable. Disability however, is not the most methodologically appropriate disease variable, especially when assessed by self report, although Smith et al. (1988) did employ a trained physical therapist to provide a backup disability assessment. Disability is potentially confounded with various psychopathological conditions including depression, helplessness and negative effect. This is both in terms of reporting and in motivation to actually attempt various activities.

**Measurement of Disease Course**

Disease course was operationalised as: Changes in difficulty with activities of daily living (ADL); changes in symptoms over the previous two years and over the life of the disease; speed of change - increasing or decreasing; periods of remission; and symptom fluctuation.

Clinical assessment in RA routinely includes consideration of ability to perform ADL and has been ranked the most important determinant of the course of RA in a survey of rheumatologists (Pincus, Sumney, Soraci, Wallston, & Hummon, 1983). A fundamental principle of the present study is to maintain a sound clinical basis with respect to disease variables. In terms of progression of RA, the change factor in capacity to perform ADL is seen as particularly relevant. Assessment of the change component is also the key factor in the symptom changes variable, as it is in the speed of change component.
Remission is another characteristic of disease course in RA which may be measured to assess disease course. Wolfe (1990) suggest that remission is the most hoped for outcome, and that frequent remissions, particularly in the early course of RA, may be expected. It must be stated that the defining of remission has its difficulties. Among rheumatologists there is substantial variation of what constitutes remission - a total absence of all features that might indicate disease activity, or a relative state where a patient has improved markedly or has essentially no symptoms and a low level of objective indications (Pinals, Masi, & Larsen, 1981). Self reports (as in the present study) would most probably reflect the latter definition. Similarly fluctuations in symptoms are conceptualised as representing a more favourable disease course. To be constantly chronic, particularly at a severe level, is probably less favourable than to have fluctuations in severity and impairment.

**Disease Variables**

A proper diagnostic basis for RA is critical for the efficacy of any research into the psychological influences on the disease. In considering pre-1988 studies regard must be had to the soundness of the diagnostic criteria. For example in McFarlane et al. (1987) the initial diagnosis included those with “classical” RA under the 1958 American Rheumatic Association diagnostic criteria. Criteria for classical RA are less rigorous than for definite RA. The 1958 criteria were amended in 1987 (Arnett et al., 1988). Under these criteria there is no “classical” category and there is greater confidence that people are correctly classified as having “definite” RA (Arnett et al., 1988; Dugowson, Nelson, & Koepsell, 1990; Smith & Arnett, 1991). Post-1987 studies should not suffer from this potential limitation and should negate many of the diagnostic uncertainties which Anderson et al. (1985) refer to as creating problems for much of the earlier literature.
A number of studies have utilised the presence of serum Rheumatoid Factor (RF) as a diagnostic criterion or have used this as the dependent variable. Pow's (1987) study for example, investigated the relationship of thinking style to RF type, finding that a negative style of thinking in relation to their RA was evidenced by those whose serum was positive for RF. Given that up to 80% of RA sufferers have serum that is positive (Aho & Kurki, 1994) it is suggestible that in general RA sufferers think negatively, or at least in a distorted fashion. This syllogistic approach would suggest that RF as a dependent variable is a rather 'dead end' channel of investigation. RF is a clinical status that is essentially unmodifiable. Thinking style cannot therefore affect RF status, whereas it is possible that thinking styles could influence disease course. Disease course, therefore, is a potentially more useful dependent variable than RF titer, and this, together with a conceptually wider notion of thinking style, sets the present study apart as a progression from Pow (1987).

Arnett et al. (1988) investigated the question of RF as a diagnostic criterion and concluded that the validity of the concept remained unproven. The need for clarification remains. Buchanan and Singal (1994) insist that a minimum of three negative tests for RF over at least three years should be required before confirming a person as RF negative. Generally RF negative patients tend to have a more favourable disease course than RF positive patients and many go on to complete remission (Aho & Kurki, 1994). Furthermore in early RA there is a 50% conversion rate from RF negative to RF positive, and anti-rheumatic drugs can reverse RF titer from positive to negative (Buchanan & Singal, 1994).

The RF issue is raised for two reasons. First, various studies have utilised RF as a dependent variable (e.g., Pow, 1987), so its implications for diagnosis and course of RA should not simply be ignored. Secondly, because of the continued uncertainty as to the biological parameters of RF and the rigorous testing requirements that are needed to maximise certainty, the present study did not distinguish among subjects
based on RF. The studies which have done so have not met the testing standards mentioned above. It may be that a study is confounded if it does and confounded if it does not include RF, but on balance, given clinical and measurement uncertainties, it is considered that it is valid for research to proceed without accounting for the distinction.
CHAPTER FIVE

Rationale, Theory and Hypotheses

Rationale and Significance of this Research

Reported research on the psychology of RA has essentially been dominated by investigations into the idea that psychological stress and personality can affect disease onset and prognosis - the classic 'psychosomatic hypothesis' (Pritchard, 1989). More recently however, there have been indications that research interest in RA is concentrating less on the methodologically difficult aetiological and personality arenas and more on identifying the psychological variables that impact on the clinical course of symptoms (Young, 1992). The evidence is that psychological characteristics and attitudes to one's illness (non-biomedical factors) are related to disease status and that further study of this relationship is needed (McFarlane & Brooks, 1988; Parker et al., 1991).

In the clinical literature there is also acknowledgment of a shift towards psychological factors in the treatment of patients with rheumatic disorders (Shipley & Newman, 1993). More specifically, cognitive factors and the cognitions adopted by sufferers during the course of their arthritis are seen as playing a central role. This is evidenced by the recent trend towards a multidisciplinary (biopsychosocial) approach to health care (Germond, Schomer, Meyers, & Weight, 1993). A fundamental rationale for the present study is, therefore, the need for research that acknowledges these shifts in emphases and examines the psychological, and more specifically, cognitive variables that impact on the clinical course of RA.
Prevention of a disease is always the ideal, but this is unlikely where the cause is unknown. Both in clinical and symptom terms RA is an unpredictably transitory and capricious disease. Limitation of duration and severity of periods of exacerbation, reduction of overall speed of progression, and increases in frequency and duration of remissions may be realistically achievable as a 'next best' goal to prevention.

The present study investigated whether certain illness cognitions, (with some related psychological factors) have any influence on these actual disease experience parameters, and if so to what extent. While to date the effects of some of these cognitive factors have been studied with respect to RA, they have been studied separately (e.g., Pow, 1987; Pritchard, 1989). They have also largely been explored for their mediational effects on the more general psychological aspects of RA sufferers, such as stress and depression rather than as more direct effects on the somatic condition.

The findings of this study are presented in the hope that those involved in the treatment and care of RA sufferers will recognise that in addition to disease outcome management through drug therapy, exercise and diet regimens, aids and devices, there may be the opportunity to influence the course of the disease by the recognition, understanding and addressing of cognitions adopted by sufferers throughout the course of the disease.

**Summary of the Theoretical and Conceptual Basis of the Study**

Broadly, the variables of interest in this study fall into four categories: Illness cognitions, non-cognitive psychological factors, and demographic factors as independent variables (IVs), and disease course operationalised as seven components, as dependent variable (DV).

The concept of illness cognitions as presented in this study encapsulates the way people think and feel about their illness - their representations of its current and future
severity and ramifications, what they know about it, and what they see as their own and others responsibilities regarding it. The theoretical basis of this study is that these illness cognitions may directly influence the course of a physical condition such as RA, without limiting their effect to a mediating role between psychological states and disease variables. There is support for the notion that the way individuals respond to their disease should influence the course thereof (c.f., Germond et al., 1993). This does not, however, exclude the possibility that other psychological or psychopathological variables may have a direct effect themselves on disease, or may have a mediational effect in the relationship between cognitions and disease course. Figure 1 summarises the model.

![Figure 1](image)

**FIGURE 1 THE RELATIONSHIP BETWEEN ILLNESS COGNITIONS, NON-COGNITIVE PSYCHOLOGICAL FACTORS AND DISEASE COURSE.**

Dispositional optimism/pessimism, psychological distress and negative affect are non-cognitive psychological factors that are considered to have some relevance to, or association with the operation of the illness cognitions of interest in this study. The underlying theoretical basis and justification for inclusion of these factors is their expected interrelationships with the illness cognition variables. Conceptually however, there are fundamental differences between the non-cognitive and the cognitive factors. The non-cognitive factors tend to be less illness specific, not necessarily a response to the illness, and either dispositional or possibly pre-existent for some other reason. They may also be complementary to the cognitive factors. For example, thinking style is conceptually closely related to dispositional optimism and pessimism and may be
borne out of such dispositional qualities. It may however be conceptualised as a response to the illness albeit with a predisposition to that response. Thinking style is therefore essentially a state rather than a trait factor.

Another function of the non-cognitive factors is in a quasi-control role. For example, in interpreting results caution would be necessary where there were indications of severe psychological distress, pessimism or negative affect. Not only could other factors have contributed to them, but they may cause various response biases in self reports of thinking towards and attitudes about the experience of illness.

Factors that may differentiate subgroups of RA sufferers and that may influence their responses to questionnaire items (e.g., level of education), require controlling for (Anderson et al., 1985). Level of education, for example, has been found to be associated with disease outcome in RA. Pincus and Callahan (1993) found that poorer clinical status was associated with lower levels of formal education in two studies. These findings were not explained by age, illness duration, ethnicity or other clinical factors.

Educational level attained is expected to influence disease course via: An individual’s interest in and ability to assimilate knowledge about their disease; their ability and motivation to think and act proactively about their condition and to interact with caregivers and treatment regimens; their ability to make accurate and realistic appraisals and expectations; and their understanding of their own responsibilities and potentials in dealing with their disease and situation. Similarly age will likely impair these functions also, especially in conjunction with a long disease term. These factors are likely to set up an ongoing cycle which may include the dulling of optimism, reinforcement of the inadequacy of treatments to date leading to increased feelings of hopelessness and various psychopathological conditions like depression.
Age is also a potentially confounding variable which requires accounting for. For example, Raja, Williams, and McGee (1994) suggested that older individuals were more likely than younger individuals to acknowledge the importance of external control. Age, education level, and other demographic variables that are considered likely to confound or mediate in the association between the psychological variables and disease course, are included in the study.

Conceptually the distinction between disease course and disease outcome is important. It emanates from the unpredictable and transitory nature of the disease. Measurement of outcome variables does not capture this fundamental feature. For example, disability is an end result which will only change in one direction - for the worse. Impairment on the other hand, may fluctuate either way and as such is a function of disease course. Pain is a major aspect of chronic RA but while it fluctuates it is conceptualised as an outcome and not a disease course factor. This is because pain is not in itself a component in the clinical progression of the disease. It is a reaction to clinical and disease activity aspects, treatments and therapies, or the lack of them.

**Hypotheses**

RA is a disease which not only appears to have multiple aetiologic factors (Anderson et al., 1985), but the course which it takes appears to be multidetermined as well. It is hypothesised that among the determinants of the course which an individual’s RA takes are the individual’s cognitive responses to their RA. This is not a hypothesis about pre-onset causal factors of RA. It refers to the association between an RA sufferer’s cognitive response to his or her condition and the course their disease takes. A specific combination of cognitions has been selected to investigate the cognitive effect. In general terms the hypothesis may be stated as follows: The cognitions that sufferers of chronic RA adopt relative to their RA, in terms of the way they think about it, what they know about it, how they appraise their present and future status,
and whether they accept responsibility for their disease status, will be associated with the clinical course that their RA takes.

More specifically the primary hypothesis proposed is: Negative thinking style in relation to one’s RA, poor general knowledge and understanding of RA, more negative appraisal of present disease status and future expectations, and belief in external agents rather than internal beliefs, are associated with a more unfavourable disease course in RA.

A secondary yet associated hypothesis pertains to the non-cognitive variables and may be stated as follows: Dispositional pessimism (or low optimism), negative affect, and elevated psychological distress levels in combination with the cognitive variables are associated with a more unfavourable disease course in RA.

The hypothesis is stated as separate hypotheses mainly for syntactical reasons and also to facilitate the reporting (and interpreting) of results in both bivariate and multivariate forms. The underlying premises of the hypothesis, which are reflected in the selection of multivariate analyses, are firstly that the individual illness cognitions form a composite illness cognition model. Secondly, the non-cognitive variables are conceptually interrelated with the illness cognitions, and that accordingly their effect should be assessed in conjunction with the effect of the illness cognitions. Similarly the demographic factors selected, given both the nature of the IVs and the condition itself, are considered likely to interrelate with various of the IVs, and their effect should be assessed jointly with all other variables.
CHAPTER SIX

Research Design and Method

Research Design

The present study was designed as between subjects, retrospective, cross-sectional, and correlational.

While participants were asked to report on their illness cognitions and other psychological factors at a particular point in time, disease course measurement included retrospective reports of the previous six months, two years, and the total life of their RA.

Participants

Participants were those members of the Manawatu branch of the Arthritis Foundation Inc., and some from the Wanganui branch, who were registered with the Foundation as being RA sufferers. Questionnaires were mailed to 128 Manawatu branch members and 11 Wanganui members. 6 of those returned were excluded as being incorrectly registered as RA sufferers. A total of 82 valid completed questionnaires were entered in the analysis, 74 from Manawatu and 8 from Wanganui. This represents an overall response rate of 64%. Table 2 summarises the descriptive characteristics of participants.

There were both advantages and disadvantages in selecting participants from the membership of the Arthritis Foundation. Disadvantages may be as follows: These may have been people who are more proactive about their disease than are sufferers who have not taken any steps to associate with other sufferers and/or gain the benefits of a specialist organisation. This could also account for a higher ratio of females to
males than is generally the case epidemiologically. They may also be sufferers who have benefited by the various training and assistance programs offered by the Foundation. These factors could reduce the representativeness of the sample.

### Table 2. Descriptive Characteristics of Participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Descriptive Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (N=82)</td>
<td>72 (87.8%) females; 10 (12.2%) males</td>
</tr>
<tr>
<td>Age (N=81)</td>
<td></td>
</tr>
<tr>
<td>16-25 years</td>
<td>2 (2.4%)</td>
</tr>
<tr>
<td>26-35 years</td>
<td>2 (2.4%)</td>
</tr>
<tr>
<td>36-45 years</td>
<td>3 (3.7%)</td>
</tr>
<tr>
<td>46-55 years</td>
<td>8 (9.8%)</td>
</tr>
<tr>
<td>56-65 years</td>
<td>23 (28%)</td>
</tr>
<tr>
<td>Over 65 years</td>
<td>43 (52.4%)</td>
</tr>
<tr>
<td></td>
<td>80.4% were over the age of 55</td>
</tr>
<tr>
<td>Ethnicity (N=81)</td>
<td>90.2% European</td>
</tr>
<tr>
<td></td>
<td>4.9% Maori</td>
</tr>
<tr>
<td></td>
<td>3.7% Other</td>
</tr>
<tr>
<td>Education (N=81)</td>
<td>12.2% Primary School Only</td>
</tr>
<tr>
<td></td>
<td>63.4% Secondary School</td>
</tr>
<tr>
<td></td>
<td>23.2% Some Tertiary</td>
</tr>
<tr>
<td>Duration of RA (time since diagnosis) (N=75)</td>
<td>Mean: 18.6 years</td>
</tr>
<tr>
<td></td>
<td>SD: 13.3 years</td>
</tr>
<tr>
<td></td>
<td>Range: 1.3 to 52 years</td>
</tr>
<tr>
<td></td>
<td>Median: 14 years</td>
</tr>
</tbody>
</table>

On the other hand, by drawing participants from the Arthritis Foundation, more certainty as to diagnosis was achieved. It is important not to rely solely on a participant's perception of their condition when, as with RA, it is one of over 100 related conditions, and often has various 'aches and pains' loosely attributed to it. Another advantage was that in supporting the study, the Arthritis Foundation
engendered confidence in participants to persevere with the lengthy and sometimes personal task of completing the questionnaire.

**Ethical Considerations**

Participants were volunteers from whom informed consent was obtained by way of an Information Sheet provided by the researcher and a letter written by the Vice President of the Manawatu branch of the Arthritis Foundation.

All participants were assured of total anonymity throughout the study - only Arthritis Foundation staff were privy to names at any time.

The measures were not likely to create any difficulties or concerns for the participants of an emotional, stressful, or clinical nature. Neither should the study have raised any aversive thoughts or worries about their physical condition. Nevertheless, participants were given the opportunity to ask any questions before, during, or after their participation, and to decline to answer any questions.

Before commencement the study was granted approval by the Massey University Human Ethics Committee.

**Measures**

All measures were self-report by self administered questionnaire, although confirmation from participants' doctors of fact and date of diagnosis, and fluctuations and remissions was sought.

**Measures of Participant Characteristics**

The following biographical information was obtained: Gender, ethnicity, level of education, age group, whether taking medication for RA, and date of doctor's diagnosis of RA. Date of diagnosis was scored in months elapsed since diagnosis.
Dependent Measures
Disease course was operationalised as seven separate variables each designed to measure a component of the overall dependent variable, disease course.

Change in difficulty over the last six months in performing activities of daily living (ADL) was assessed using a part of the Modified Stanford Health Assessment Questionnaire (MHAQ) (Pincus, Summey, Soraci, Wallston, & Hummon, 1983). The MHAQ is an eight item arthritis specific version of the twenty item HAQ. The HAQ has been found to be a valid and sensitive measure of physical disability in RA (Peck, Smith, Ward, & Milano, 1989). The eight item version provides an acceptable alternative (Pincus et al., 1983). Because the same eight ADL questions are used in each component of the MHAQ, it is considered that the validity of the scale is not compromised by utilising the one segment of the scale which is applicable to the dependent variable in the present study.

Scoring of the scale was modified in the present study so that ‘no change’ was scored as 0, ‘less difficult now’ as -1 and ‘more difficult now’ as +1. This more appropriate scoring system was made possible by using this segment of the MHAQ in isolation, and aligned this measure with the scoring of the following measure. (N.B. The four segments of the MAHQ were included in the questionnaire but only the change in difficulty with ADL segment was scored and utilised).

Changes in symptoms and the direction of any change over the last two years was assessed by asking participants to select from three graded statements the one which best described any change over that period. These items were developed by Stewart (1991). There are no psychometric data available.

Remissions were assessed with a single specially prepared item which asked participants to indicate whether there had been at least one period of time when their RA seemed to be in remission.
Fluctuations over the life of the disease were similarly assessed with a single question developed for this study which asked if symptoms seemed to fluctuate in intensity over the life of the disease.

Fluctuations over the previous two years were separately assessed with a single question drawn from Stewart's (1991) disease course questionnaire.

Change in symptoms over the life of the disease was measured with two items (in the alternative), developed for this study, one reflecting negative change, and the other no change. Participants were asked to indicate which applied to them. Overall improvement was not provided for for two reasons. One was that steady improvement is not a characteristic of RA, and the other was that general improvement would almost certainly be attributable to, and therefore confounded by, medication regimens. 85.4% reported taking medication for their RA.

Speed of change (worsening RA) was measured in terms of whether it was increasing or decreasing, by two items in the alternative, modified from Stewart’s (1991) RA onset questionnaire. Participants were asked to indicate either increasing or decreasing speed of change.

Independent Measures
Knowledge and understanding of one’s RA was measured with the 29 item Patient Knowledge Assessment Questionnaire (Pritchard, 1989), an instrument that specifically assesses illness knowledge in RA patients. It includes questions on prognosis, physiology, treatments, and the disease process. One response out of five options is correct, so scoring was on a right or wrong basis. No response was taken as a ‘don’t know’ and scored as an incorrect answer.
Pritchard (1989), in an attempt to control for guessing, asked participants to indicate how certain they were of their response. In the present study this was omitted to help reduce item quantity. In any event, an assumption was made that uncertainty usually resulted in no response. Furthermore, a one in five chance of guessing the correct answer would reduce the impact of intermittent guessing.

Psychometric data is not particularly appropriate for a scale such as this, although it may benefit from an assessment of a panel of rheumatologists as to accuracy and whether the scale represents an appropriate coverage of RA general knowledge. Pritchard (1989) presents no background on the construction of the scale.

Appraisal of present condition in terms of severity and stage of progression was assessed by requiring participants to circle a number on a five-point Likert type scale where 1 represented extremely mild and 5 represented extremely severe. This measure was prepared for the purposes of the present study.

Expectations for the future (RA outcomes in the next 12 months) were similarly assessed with a specially prepared question using a five-point Likert scale ranging from much better than now through about the same to much worse than now.

Thinking style in relation to one's RA was measured using the Rheumatic Thoughts Questionnaire (RTQ) (Pow, 1987). This is a twelve item scale with six items referring to pleasant events and six to unpleasant, with four possible responses for each. In addition, each set of pleasant and unpleasant events are couched in terms of the self, the world, and the future, in relation to negative thinking. These are presented randomly, as are the gradings of statements from negative to positive within each item.

There are no reliability or validity data for the RTQ itself, although Pow (1987) stated studies were in progress to calculate this. Nothing has emerged in the literature as yet. Face validity however, was tested by four psychologists identifying and ranking items
from most negative to most positive. Kendall’s coefficients of concordance (W) were satisfactory (Pow, 1987). Since, however, the Cognitive Style Test (CST) (Wilkinson & Blackburn, 1981), from which the RTQ was modified to make it RA specific, is a well established measure, the results based on its use can be assumed to have similar validity (Pow, 1987). The validity and reliability of the CST has been found to be satisfactory (Williams, 1984).

**Locus of control** was measured primarily with the powerful others externality subscale of the Multidimensional Arthritis Locus of Control Scale (MALC) (Wallston, 1989), an RA specific version of the Multidimensional Health Locus of Control scales (MHLC) (Wallston, Wallston, & DeVellis, 1978, cited by Wallston, 1989). The full scale consists of three 8-item subscales assessing internal locus of control, powerful others externality, and chance locus of control. The items of each subscale are presented randomly in a 5-point Likert format ranging from *strongly disagree* to *strongly agree*. The three subscales were administered. Each subscale was separately scored as it is inappropriate to use the three dimensions to produce a single overall score (Wallston & Wallston, 1981). Furthermore, since powerful others externality is an important dimension in health research, this subscale should be used as a separate measure (Wallston & Wallston, 1981). The rationale for including all subscales is at least twofold. One is to enable the externality items to be positioned randomly amongst other items to avoid response bias. Another is to account for the possibility raised by Roskam (1986) that it may not be a question of externality or internality but control versus no control. Internality and externality are both facets of control. Administering all subscales helps account for this possibility.

There appears to be no psychometric data available for the MALC scales, however since it is simply the MHLC scale made arthritis specific, the psychometric data for the latter should be substantially applicable. Reliability in terms of test-retest for the powerful others subscale of the MHLC is respectable (.71), as it is for the other two subscales (.66 for internality and .73 for chance subscales) (Lefcourt, 1991).
In terms of convergent validity, the MHLC has been compared with Levenson’s I, P, C, scales. The internality subscales were positively related at .57, while powerful others externality displayed a milder positive association (r = .28) with Levenson’s P scale (Lefcourt, 1991). These data were obtained using a student sample of unspecified health status. The authors of the MHLC scale acknowledge however that further studies are required to determine validity with different samples (Raja et al., 1994).

Non-Cognitive Variables

Dispositional optimism (and pessimism) was measured using the Life Orientation Test (LOT) (Scheier & Carver, 1985). The scale consists of twelve items, four phrased optimistically, four phrased pessimistically, and four filler items. Participants indicate the extent of their agreement with each item along a 5-point Likert scale ranging from strongly agree to strongly disagree.

The standard scoring method consists of reversing scores on the pessimism items and summing to produce an overall score, with a higher score indicating greater optimism. This reflects the conceptual perspective that optimism and pessimism are opposite poles of a unidimensional continuum. Marshall, Wortman, Kusalas, Hervig, and Vickers (1992) however, confirmed their hypothesis that optimism and pessimism are empirically differentiable. A factor analysis of the structure of the LOT confirmed the two factor model (Marshall et al., 1992). Accordingly, in the present study, the LOT was scored bidimensionally as well, producing separate optimism and pessimism scores. This enabled examination of the predictive validity of each of these distinct constructs.

As a unidimensional measure, Scheier and Carver (1985) report acceptable internal reliability (Cronbach’s alpha = .76) and satisfactory test-retest reliability (r = .79 over a 4-week interval, .72 over a 13-week interval). In terms of convergent and discriminant validity, Scheier and Carver (1985) evaluated the LOT against a number
of relevant scales, and reported moderate correlations in the appropriate direction (Scheier & Carver, 1987). The psychometric qualities of the LOT as a bidimensional scale require further examination. It would be reasonable however, to expect reliability to be equivalent, and of course, the items retain their high face validity. Similarly specific validity studies would be expected to confirm at least equivalent convergent and discriminant validity with the unidimensional model.

**Negative affect** was measured using the 10 item negative affect (NA) subscale of the Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988). The scale is suitable for use as a trait measure of affect (Watson et al., 1988). Participants were asked to rate their negative feelings and emotions as to the extent to which they had felt that way during the past few weeks on a 5-point Likert scale ranging from *very slightly or not at all* to *extremely*.

Positive and negative affect are highly distinctive dimensions and the PANAS scales permit separate administration where appropriate. Watson et al. (1988) claim considerable psychometric soundness for the scales. Internal consistency for the NA scales is high (Cronbach’s alpha for ‘past few weeks’ time frame = .87). Test-retest reliability over an 8-week retest interval for the ‘past few weeks’ time frame was .48 but increases as the rated time frame increases. Convergent and discriminant validity are highly acceptable in terms of both scale and item validity.

**General psychological distress** was assessed using the Hopkins Symptom Checklist-21 (HSCL-21) (Green, Walkey, McCormick, & Taylor, 1988). This is a 21-item version of the 58-item Hopkin’s Symptom Checklist (HSCL) (Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974). The HSCL-21 comprises three 7-item subscales: General feelings of distress, somatic distress, and performance difficulty. The scales are summed to obtain an overall psychological distress score.
Reliability of the HSCL-21 is high (corrected split-half reliability = .91, alpha coefficient = .90) (Green et al., 1988). These compare favourably with longer versions of the HSCL. Green et al. (1988) did not attempt clinical validation but they demonstrated the robust and stable factor structure of the scale. It is expected to retain the excellent psychometric properties of its forerunners. Deane, Leathem, and Spicer (1992) investigated the psychometric properties of the HSCL-21, producing evidence of construct validity by comparing clinical norms with those of non-clinical samples, and by assessing the change in total distress scores over the course of psychotherapy. Evidence for concurrent validity was demonstrated by significant and moderate to strong correlations between the HSCL-21 scales and the A-State and A-Trait scales of the STAI-Y (Spielberger, 1983).

**Procedure**

**Questionnaire**

Each potential participant (identified by the relevant branch of the Arthritis Foundation as having RA) was mailed a pack containing the following: a letter from the Arthritis Foundation (Manawatu Branch) Vice President supporting the research; a letter from the researcher introducing the research; an Information Sheet; an Informed Consent Form; a form authorising doctor's disclosure of patient information; the questionnaire (see Appendix 1); and a prepaid return envelope.

At the request of the Manawatu Branch of the Arthritis Foundation, absolute participant anonymity was maintained. This was achieved by the Arthritis Foundation staff addressing the envelopes and retaining a confidential list of names and addresses with ID numbers for participants, and mailing the packs. Questionnaires were then returned to the Arthritis Foundation where they were opened, the two completed consent forms removed and retained, the master list noted, and the questionnaire handed to the researcher. Participants who had not responded after one month were sent a reminder letter following the same mailing procedure. Arthritis Foundation field
staff encouraged those participants whom they visited during the relevant period to complete the questionnaire and assisted those who were impaired or disabled. Participants were sent a summary of the findings upon completion of the study debriefing them and thanking them for their participation.

**Doctor’s Confirmation**

A fundamental requirement of this study was that participants were definitely RA sufferers. Being members of the Arthritis Foundation added considerably to the certainty of actual diagnosis, however to avoid possible confusion with other arthritic conditions, self diagnosis, and/or poor memory as to diagnosis and duration, this information was also sought from participants’ doctors. For similar reasons it was considered that it would be helpful to obtain doctors’ confirmation of fluctuations and remissions in terms of frequency and duration. Even if doctors’ response rate did not match that of the participants, it would provide a sample from which the accuracy of participants self-reports could be gauged. Doctors’ response rate was 61.5%. This questionnaire is reproduced as Appendix II.

Upon receipt of the signed doctors’ authorisation form from participants, Arthritis Foundation staff attached a pre-signed letter from the researcher, and a doctors’ questionnaire (with participants ID on it), and mailed it to the doctor whom the participant had named. Upon completion, the doctor retained the authorisation form and returned the completed questionnaire. Upon completion of the study, doctors were sent a summary of the findings.

**Data Analysis**

The statistical analyses were completed using SPSS/PC and SPSS/X. Alpha levels used in intercorrelations were .05 and two-tailed, except that for intercorrelations between independent and dependent variables significance levels up to .10 are reported. \( P \) values of .15 are common default values for the inclusion of variables in
multiple regression models (Parker et al., 1991). The same authors describe p values between .05 and .10 as "marginally significant" and utilise <.15 as a definition of consistent correlations. In the present study, while all variables are entered in the multivariate analyses, (including those displaying non-significant bivariate correlations), it is acknowledged that in health related research it is important to detect any associations, and that p values between .05 and .10 may have some meaning.

Intercorrelations to assess the degree of the relationships among the various combinations of variables were obtained using Pearson product-moment correlations for intercorrelations among the dependent variables and among the independent variables. Point-biserial correlation was used for associations between dependent and independent variables where the dependent measures produced dichotomous data. Some of the dependent measures produced data of a trichotomous nature however, and these relationships were assessed using Spearman's rank order correlations. Pearson product-moment correlation was used for the one disease course component (change in difficulty with ADL) that comprised continuous data.

These bivariate analyses were followed by a standard multiple regression regressing the one continuous dependent measure against all of the IVs to examine the extent to which each in the set of IVs independently predicts that disease course component. Multiple regression was used to predict the value of the disease course variable (change in difficulty with ADL) from our knowledge of the values of the several predictor variables. Direct (standard) discriminant function analyses (DFA) were carried out with the remaining DVs (because of their dichotomous and trichotomous nature) to determine whether the combination of predictor variables was associated with the different categories of the various criterion variables, and to enable prediction of values of the criterion variables given values of predictor variables. The direct-entry DFA procedure, where all variables are entered simultaneously, was used as there was no theoretical basis for entering IVs individually or in any specified priority order as in stepwise DFA. This was also the reason for using forced entry standard
multiple regression. While the study was primarily interested in the influence of illness cognitions, due to the expected interrelatedness of the non-cognitive variables with the cognitive and the expected effect of the demographic variables, these variables required accounting for. All variables were therefore entered simultaneously as discriminating variables in the DFA procedure and as predictor variables in the multiple regression procedure.

Discriminant analysis has two main purposes. One is where it is used for determining and interpreting group differences, and the other is where it is used to determine the probability of cases falling into a particular group. In the present study, discriminant analysis was utilised for examining group differences. For example, the groups created by the remissions component of disease course are 'remissions' or 'no remissions'. The analysis was used to answer the question 'whether these groups differ from one another on dimensions of illness cognitions while controlling for certain non-cognitive psychological factors and certain demographic factors'.
CHAPTER SEVEN

Results

Data Checking and Treatment of Missing Values

Before commencing analysis the data were checked for accuracy of data entry, missing values and directionality of scoring. Some missing values were to be expected in a lengthy self-administered questionnaire with a predominantly elderly sample. For continuous data a prorated score was calculated where appropriate by multiplying the number of completed items by the number of items in the scale and dividing the result by the number of completed items. On scales comprising 8 items, one missing item was permitted (12.5%). For 10 to 12 item scales two missing items were permitted (20% and 17%) and three missing items were permitted for prorating on a 21 item scale (14.5%). This procedure captured almost all of the missing cases among the independent variables. For the knowledge of RA questionnaire where one option was the correct response and the others incorrect, no response was treated as a "don't know", therefore an incorrect answer. In the dependent measures (except for changes in difficulty in ADL) where response options were limited and required marking if applicable, a conservative approach was adopted where no response was treated either as not evidencing that quality (e.g., no response = no remissions, while a response = remissions), or as a missing value, as appropriate. Missing values reduced the sample size noticeably on some of the DV's. Examination of cases with missing values did not however, disclose evidence that missing values were associated with any particular characteristics of the cases.

Outline of Results Presentation

Presentation of results broadly falls into three sections. In the first some illness cognition patterns are illuminated on a univariate basis. This is followed by a series of
tables showing the bivariate correlational relationships among variables. The third section presents the results of the multiple regression analysis, which examines the predictive effect of all variables on the continuous component of the DV, and the discriminant function analysis which enables assessment of the differences between the response groups (either two or three) with respect to all predictor variables simultaneously. It was the multivariate analyses which essentially tested the composite illness cognition approach.

**Observed Patterns Within Illness Cognition and Non-Cognitive Variables**

Examination of the IV data disclosed some noteworthy patterns within these variables. These are presented in this section.

Previous research (Pritchard, 1989) has shown that RA sufferers display the poorest knowledge about their RA in the areas of pathology, clinical symptoms, medical treatment and the disease process. This is also the pattern displayed in the present study. The ratio of incorrect to correct responses was 716:330. This area is essentially the domain of the medical profession. The ratio of incorrect to correct responses in the area of lifestyle and self-help type of treatments however was 183:317. The pattern is reversed for those aspects of their RA that sufferers can be taught by non-medical caregivers.

Participants' appraisals of their present condition demonstrated some heterogeneity within the sample in terms of severity, with a trend slightly towards severity. To some extent this profile was mirrored in participants' expectations of their future condition, although more expected an improvement than a worsening - a slightly more optimistic approach. Figure 2 demonstrates this.

A pattern emerging from the thinking style data was that the most consistently positively endorsed items related to the future. Of the four future oriented items the
percentage endorsing the two most positive options were 86.6%, 86.6%, 61% and 80.5% respectively. The highly positive future orientation points to a fundamental optimism inherent in this sample which is surprising, given the negative trend in appraisal of present condition. The basic optimism however is confirmed by the results of the bidimensionally scored dispositional optimism measure (LOT). The mean optimism score was 10.5 (SD = 2.4), while the mean pessimism score was 5.7 (SD = 2.8), the possible score for both being 16.

The generally optimistic outlook was reflected also in the measure of daily mood. For negative affect, 82% of responses fell within the very slightly or not at all to a little categories.

At 37.4 (SD = 11.3) the mean score for psychological distress was slightly elevated above the mean for a normative sample of the general population in Palmerston North, which was 32.8 with a standard deviation of 8.74 (Deane & Chamberlain, 1992), but
similar to the mean score for second year psychology students at 36.4 (SD = 6.77) (Deane, 1993). Comparison with a clinical sample with a mean of 44.3 (SD = 11.3) (Dean et al., 1992) confirms levels of distress closer to a normal population.

**Doctors' Confirmation**

**Diagnosis and Duration**

Of the doctors' responses (45), four reported no formal diagnosis of RA. For three of these, doctors reported generalised osteo-arthritis. This represents 7% of those participants for whom doctors responded, as not having RA. These participants were however, included in the analyses as doctors responses were received after the analysis was completed.

The mean illness duration disclosed by doctors confirmation was 14.5 years, while the mean duration reported by the same participants was 16.9 years. Overall self-reported mean was 18.5 years.

**Fluctuations and Remissions**

There was doctors' confirmation that fluctuations had occurred in 82% of cases. Of these 62% were confirmed as having “occasional” fluctuations and the balance as “frequent”. 62% were also reported as having fluctuations of variable duration with the balance of several weeks duration. Doctors reported more fluctuations than the same participants did by a margin of 34%.

In 67% of cases doctors confirmed remissions. 62% of these had experienced more than one period of remission. For 60%, remission periods had been measurable in months, while 27% had experienced remission periods measurable in years. Doctors’ reports of remissions exceeded the same participants’ reports by 28%.
A correlation of doctors' responses with participants' responses was not undertaken since some of the doctors treated more than one participant.

**Correlational Relationships**

**Relationships Among Independent Variables**

Intercorrelations among all IVs are presented in Table 3. The correlations involving illness cognitions that are significant are generally low. Their directionality however is as expected.

Correlations between knowledge and certain non-cognitive variables are in line with the hypothesised role of knowledge as a positive psychological factor. For example, while knowledge does not appear to be associated with optimism, pessimism does tend to decrease as knowledge increases ($r = -.34$). Also as expected, as education level increases so does knowledge of RA ($r = .31$). Knowledge is also associated with reduced negative affect ($r = -.27$) and reduced psychological distress ($r = -.34$).

Appraisal of current condition also discloses some hypothetically consistent associations, also mainly with non-cognitive factors. It was expected that as appraisal moved towards more negativity, so negative affect would increase ($r = .24$) and similarly for psychological distress ($r = .37$). Understandably also, as illness duration increased so did negativity in appraisal ($r = .30$). These associations however, did not carry through to expectations for the future, which was surprising, especially since expectations were significantly associated (mildly) with beliefs that one's condition was determined by chance ($r = .27$).

The only illness cognition that thinking style displayed any significant association with was internal control ($r = .30$). While only a mild association, there was no corresponding result in the opposite direction for external control (or for chance).
# TABLE 3: INTERCORRELATIONS AMONG INDEPENDENT VARIABLES (N = 82)

<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>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
<th>15</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Knowledge</td>
<td>1.00</td>
<td>- .16</td>
<td>- .14</td>
<td>- .19</td>
<td>- .09</td>
<td>- .01</td>
<td>- .14</td>
<td>- .02</td>
<td>- .34*</td>
<td>.20</td>
<td>- .27**</td>
<td>- .34**</td>
<td>.31**</td>
<td>- .17</td>
<td>- .20</td>
</tr>
<tr>
<td>2. Appraisal</td>
<td>1.00</td>
<td>0.09</td>
<td>0.17</td>
<td>0.28*</td>
<td>- .14</td>
<td>0.08</td>
<td>0.15</td>
<td>0.11</td>
<td>- .01</td>
<td>0.24*</td>
<td>0.37**</td>
<td>- .09</td>
<td>0.14</td>
<td>0.30**</td>
<td></td>
</tr>
<tr>
<td>3. Expectations</td>
<td>1.00</td>
<td>0.14</td>
<td>0.10</td>
<td>- .24*</td>
<td>0.27*</td>
<td>0.09</td>
<td>0.06</td>
<td>0.04</td>
<td>0.13</td>
<td>0.21</td>
<td>- .08</td>
<td>0.01</td>
<td>- .03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Thinking Style</td>
<td>1.00</td>
<td>- .01</td>
<td>- .30**</td>
<td>0.05</td>
<td>- .25*</td>
<td>0.23*</td>
<td>- .34**</td>
<td>0.39**</td>
<td>0.24*</td>
<td>- .18</td>
<td>- .01</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. External Control</td>
<td>1.00</td>
<td>0.22</td>
<td>0.28*</td>
<td>0.21</td>
<td>0.17</td>
<td>0.07</td>
<td>0.08</td>
<td>0.03</td>
<td>0.02</td>
<td>0.25*</td>
<td>0.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Internal Control</td>
<td>1.00</td>
<td>0.00</td>
<td>0.10</td>
<td>0.10</td>
<td>0.10</td>
<td>0.11</td>
<td>0.13</td>
<td>0.12</td>
<td>0.03</td>
<td>0.17</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. Chance</td>
<td>1.00</td>
<td>0.09</td>
<td>- .24*</td>
<td>0.21</td>
<td>0.21</td>
<td>0.01</td>
<td>0.08</td>
<td>0.11</td>
<td>0.00</td>
<td>0.06</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Optimism</td>
<td>1.00</td>
<td>- .19</td>
<td>0.74**</td>
<td>- .13</td>
<td>- .04</td>
<td>- .02</td>
<td>0.16</td>
<td>0.11</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Pessimism</td>
<td>1.00</td>
<td>- .71**</td>
<td>0.24*</td>
<td>0.12</td>
<td>0.21*</td>
<td>0.09</td>
<td>0.07</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Total Optimism</td>
<td>1.00</td>
<td>- .30**</td>
<td>- .19</td>
<td>0.16</td>
<td>0.16</td>
<td>0.02</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Negative Affect</td>
<td>1.00</td>
<td>0.54**</td>
<td>- .02</td>
<td>- .34**</td>
<td>0.09</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Psychological Distress</td>
<td>1.00</td>
<td>- .01</td>
<td>0.03</td>
<td>0.36**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13. Education +</td>
<td>1.00</td>
<td>- .54**</td>
<td>0.09</td>
<td>0.59**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Age ++</td>
<td>1.00</td>
<td>0.64**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15. Illness Duration</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p ≤ .05; ** p ≤ .01 (two-tailed)
+ Spearman's rank-order correlation
++ Point Biserial correlation
This points to the efficacy of the argument that the question of control versus no control is important to people rather than the question of locus. As expected, thinking style was significantly although mildly to moderately associated with optimism, daily mood and psychological distress. Directionality of these associations are all appropriate. For example, as negativity in thinking style increases, optimism decreases \((r = -0.34)\) and negative affect increases \((r = 0.39)\) as does psychological distress \((r = 0.24)\). Some evidence of an overlap between the trait variables with a more state measure was expected.

External control as the remaining cognitive variable disclosed an expected significant, although mild, association with age \((r = 0.25)\). As age increases it is understandable that reliance on medical and other care givers would increase also.

Among the non-cognitive and demographic variables there are no unexpected or unexplainable correlations. The moderate to high correlations between optimism and pessimism scored bidimensionally and the conjoint total optimism score \((0.74\) and \(-0.71\) respectively) are in the appropriate direction in each case, and were logically expected as functions of the overlap of items in the two subscales. The correlations between age and level of education follow a normal pattern with older people showing a lower level of formal education \((r = -0.54)\) and longer illness duration \((r = 0.64)\). Interestingly while there is some association between illness duration and psychological distress \((r = 0.36)\), there is no association between age and distress. This would suggest that psychological distress is not necessarily a function of age for this population.

**Relationships Among Dependent Variables**

Disease course comprised seven essentially separate criterion measures combined to assess different aspects of the course of RA. The intercorrelations among these criteria are presented in Table 4.
The moderate negative correlation (-.36) between changes in difficulty with ADL and symptom changes over the last two years, and the negative correlation (-.26) with speed of change, were as expected. Difficulty with ADL has increased as symptoms have worsened. No association was expected or found between ADL changes and fluctuations, remissions and speed of change however. The association (r = .47) between symptom changes over the last two years and over the life of the disease was as expected.

**TABLE 4: INTERCORRELATIONS AMONG DEPENDENT VARIABLES (N=82)**

<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>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>1.00</td>
<td>-.36**</td>
<td>.13</td>
<td>.19</td>
<td>-.26*</td>
<td>-.02</td>
<td>.09</td>
</tr>
<tr>
<td>2.</td>
<td>1.00</td>
<td>.02</td>
<td>.47**</td>
<td>.11</td>
<td>.17</td>
<td>-.21*</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>1.00</td>
<td>.15</td>
<td>-.02</td>
<td>.10</td>
<td>.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>1.00</td>
<td>-.12</td>
<td>.82**</td>
<td>.86**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>1.00</td>
<td>.34**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.00</td>
</tr>
<tr>
<td>7.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p ≤ .05, ** p ≤ .01 (two-tailed)
+ Spearman's rank-order correlation

The moderate to high relationships among the changes over the life of the disease variables (ranging from .67 to .90) suggest that each of these, especially where the correlation is high, may be a measure of the same disease course construct. An argument can be made, at a theoretical level however, that each is measuring a different, albeit related, aspect of disease course. For example, it is expected that those reporting periods of remission would also report fluctuations. It is common for
these two characteristics to coexist. Similarly those reporting changes in symptoms would also be expected to report fluctuations and possibly remissions. The relationships among the DV's are therefore taken as indicating that each is measuring a different aspect of disease course.

**Relationships Between Individual Independent and Dependent Variables**

Table 5 shows the relationships between individual predictor variables and criterion measures.

The degree of association between individual illness cognitions and disease course is mostly low or non-existent in each case.

Knowledge of RA for example, discloses a very small relationship with changes in difficulty with ADL ($r = .12$). The directionality of the association however, suggests that knowledge influences people to be more cautious of changes for the worse (increasing difficulty). This was unexpected, although the association was non-significant. No relationship with the other components of disease course was found. This indicates that knowledge, in isolation, does not influence the course of RA, except insofar as it may influence the way sufferers approach everyday tasks.

Appraisal of present condition was mildly associated with change in difficulty with ADL ($r = .20, p = .07$). This association may be a reflection of the temporal proximity of the variables - they are both assessing a “now” situation. Consequently, the hypothesised distinction between predictor and criterion is equivocal. Appraisal of present condition was however, significantly and moderately associated with change in RA over the last two years ($r = -.44, p < .001$). As appraisal moved towards severity, RA symptoms over the last two years were reported as having worsened. This trend
<table>
<thead>
<tr>
<th></th>
<th>Change in difficulty with ADLs</th>
<th>Symptom change over 2 years</th>
<th>Fluctuations over 2 years</th>
<th>Symptom change over disease life</th>
<th>Speed of change</th>
<th>Remissions</th>
<th>Fluctuations over disease life</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Knowledge (N=82)</td>
<td>.12</td>
<td>.01</td>
<td>-.04</td>
<td>-.02</td>
<td>.01</td>
<td>-.05</td>
<td>-.02</td>
</tr>
<tr>
<td>2. Appraisal (N=81)</td>
<td>.20*</td>
<td>-.44***</td>
<td>.12</td>
<td>.13</td>
<td>-.12</td>
<td>-.07</td>
<td>.02</td>
</tr>
<tr>
<td>3. Expectations (N=82)</td>
<td>.02</td>
<td>-.24**</td>
<td>-.11</td>
<td>-.06</td>
<td>-.16*</td>
<td>-.21*</td>
<td>-.16</td>
</tr>
<tr>
<td>4. Thinking Style (N=82)</td>
<td>-.02</td>
<td>-.08</td>
<td>.09</td>
<td>.00</td>
<td>-.05</td>
<td>-.04</td>
<td>-.11</td>
</tr>
<tr>
<td>5. External Control (N=81)</td>
<td>-.04</td>
<td>-.16**</td>
<td>.02</td>
<td>.02</td>
<td>.00</td>
<td>-.02</td>
<td>.00</td>
</tr>
<tr>
<td>6. Internal Control (N=82)</td>
<td>.01</td>
<td>.09</td>
<td>.00</td>
<td>.03</td>
<td>.02</td>
<td>.00</td>
<td>.02</td>
</tr>
<tr>
<td>7. Chance (N=82)</td>
<td>.06</td>
<td>-.14</td>
<td>.12</td>
<td>.01</td>
<td>-.13</td>
<td>-.05</td>
<td>.04</td>
</tr>
<tr>
<td>8. Optimism (N=82)</td>
<td>.02</td>
<td>-.05</td>
<td>-.05</td>
<td>.11</td>
<td>-.17**</td>
<td>-.04</td>
<td>.03</td>
</tr>
<tr>
<td>9. Pessimism (N=82)</td>
<td>-.09</td>
<td>-.10</td>
<td>.09</td>
<td>-.17</td>
<td>-.23**</td>
<td>-.09</td>
<td>-.14</td>
</tr>
<tr>
<td>10. Total Optimism (N=82)</td>
<td>-.04</td>
<td>.10</td>
<td>-.07</td>
<td>.22**</td>
<td>.13</td>
<td>.09</td>
<td>.14</td>
</tr>
<tr>
<td>11. Negative Affect (N=79)</td>
<td>.13</td>
<td>-.05</td>
<td>-.09</td>
<td>-.01</td>
<td>-.12</td>
<td>-.04</td>
<td>-.02</td>
</tr>
<tr>
<td>12. Psychological Distress (N=82)</td>
<td>.29**</td>
<td>-.17*</td>
<td>.10</td>
<td>-.03</td>
<td>-.15*</td>
<td>-.12</td>
<td>-.09</td>
</tr>
<tr>
<td>13. Education (N=81)</td>
<td>.10</td>
<td>.02</td>
<td>-.08</td>
<td>.12</td>
<td>-.01</td>
<td>.10</td>
<td>.06</td>
</tr>
<tr>
<td>14. Age (N=81)</td>
<td>.19*</td>
<td>-.20**</td>
<td>-.03</td>
<td>-.17</td>
<td>-.08</td>
<td>-.22**</td>
<td>-.22**</td>
</tr>
<tr>
<td>15. Illness Duration (N=75)</td>
<td>.19</td>
<td>-.11</td>
<td>.02</td>
<td>-.21</td>
<td>-.13</td>
<td>-.21*</td>
<td>-.22*</td>
</tr>
</tbody>
</table>

* p ≤ .10; ** p ≤ .05; *** P ≤ .001
+ Spearman's rank-order correlation
++ Point biserial correlation
was perpetuated although very mildly and insignificantly, for symptoms over the life of the disease, suggesting that the hypothesised direction of the effect was indicated—that those who are generally experiencing worsening RA are those whose belief about their condition is negative.

Expectations for the future and appraisal of present condition are conceptually closely related variables, and between them they account for most of the association between illness cognitions and disease course. For future expectations the relationship with remissions is one of the strongest ($r = .21, p = .06$). The direction of this association suggests that those whose expectations are generally of a positive nature are those whose RA is characterised by the remission component. Similarly those with positive expectations tend to be those whose RA demonstrates more stability, that is, they report a slowing down of progression. This association is also very mild however ($r = -.16, p = .08$). Also, those with more negative future expectations tend to report less fluctuations. Again, the correlation is very low ($r = -.16$) and insignificant but directionality is associated with a less favourable disease course. The association of negativity with disease course is further perpetuated with the significant, albeit low, correlation between expectations and symptom changes over the last two years. As negativity increases symptoms are reported as worsening ($r = -.24$).

Thinking style discloses no significant associations with any of the disease course components, which was an unexpected result. The largest correlation (-.11) was with fluctuations over the life of the disease. The more negative the thinking, the fewer the fluctuations.

Similarly the locus of control variables disclose only very low correlations with disease course variables. External control, which is specifically of interest, displays the highest correlation ($r = .16, p = .05$), which is with symptom changes over the last two years. To a small extent therefore, as feelings of reliance on others have increased, RA has been perceived as worsening. For both externality and internality there were no other
associations with disease course. Interestingly, chance factors (no control), display
more consistent (although very mild) associations with disease course.

The non-cognitive variables evidence some association, albeit low, with certain disease
course components. Pessimism is generally the most noticeable, correlating -.17
(p = .13) with symptom changes over disease life and -.23 (p = .05) with speed of
change. These suggest that as pessimism increases symptoms worsen, and
development has speeded up. While the associations are small, they are consistently
higher than they are for optimism, suggesting that a pessimistic outlook on one’s RA
is likely to have more negative effect on its course than any positive effect of
optimism. The result also lends some support to the efficacy of scoring the LOT
bidimensionally.

Psychological distress was mildly associated with increasing difficulty with ADL
(r = .29, p = .05), and there tended to be an association with recent worsening of
symptoms (r = .13), and with an increasing rate of disease development (r = -.14).

Age and illness duration are the only demographic factors where there is some
association with disease course. For example as age increases symptoms are reported
as worsening over the last two years (r = .20, p = .05). Also with increasing age
comes fewer fluctuations and remissions (r = .22, p = .05 in each case). A similar
pattern emerged for duration of illness.

The Influence of Illness Cognitions and Related Psychological Factors on Disease
Course

It was hypothesised that a composite group of illness cognitions would predict disease
course in RA. It was further hypothesised that certain more dispositional
psychological factors may also influence RA disease course as being interrelated with,
or predispositional of the more illness response cognitive factors. Age, level of
education and illness duration also required controlling for. It was considered that these factors were likely to influence a person's perception of or attitude towards their illness.

To examine these hypotheses a series of analyses were conducted comprising standard multiple regression and standard discriminant function analysis. The bivariate correlations addressed the question: how important are the various IV's when each of them alone is used to predict the various disease course components, describing a simple linear association. The multivariate analyses address the more complex question: how important are the individual IV's when, along with each other, they are used to predict disease course?

The Influence of Illness Cognitions
Standard multiple regression was used to ascertain the extent to which scores on the illness cognition measures predicted RA disease course as assessed by the ADL change component. All variables, including the non-cognitive and demographic variables were entered in the analysis. Multiple regression was the appropriate analysis, as the ADL measure produced continuous data. None of the illness cognitions however, contributed significantly to prediction of disease course as assessed by change in ADL ability. Furthermore, adjusted $R^2$ showed that only 9.5% of the variance in change in ADL was explained by the combined effect of all predictor variables. Knowledge of RA was the only illness cognition variable that tended towards any association ($B = .191, p = .13$).

Discriminant analysis was the appropriate analysis for the remaining disease course components since they comprised either dichotomous or trichotomous data. These analyses were carried out to establish whether disease course and illness cognitions were related to each other by examining how well the set of illness cognitions discriminated between the groups created by each of the disease course components while controlling for the effects of the non-cognitive and demographic factors.
None of the possible dimensions of discrimination calculated for the grouping variables disclosed statistically significant separation among the groups of the respective disease course components. The DFA procedure, like the multiple regression analysis, has shown that there is no composite effect of these illness cognitions in disease course in RA. Various factors may have contributed to the insignificance of the discriminant functions however. For example, the inclusion in those dimensions of a number of variables which the bivariate correlations had disclosed as having no influence on disease course, may have generated unnecessary noise in the analysis. Knowledge is an example of a variable that consistently disclosed no effect in the bivariate analyses. This may have been compounded also by the sample size in relation to number of variables, further reduced by missing values.

As would be expected, examination of the relationships among the discriminant functions, the groups, and the discriminating variables disclosed trends which were consistently supportive of those disclosed by the bivariate analyses.

The standardised discriminant function coefficients, which determine the relative importance of the cognitive variables to the separation of the respective disease course component groups, were consistently moderate to high for the appraisal cognition, and/or the closely related future expectations variable. The structure coefficients also confirmed the importance of the appraisal and expectation variables, relative to the other IVs, in the relationship with disease course. Furthermore, the generally low correlations among IVs adds meaning to these findings in that appraisal and expectations, as being variables of some relative importance, are not strongly confounded by other IVs. All DFA results are shown in Appendix III.

The Effect of the Non-Cognitive Variables
In the multiple regression analysis only psychological distress was significantly associated with the ADL change component of disease course ($B = .352, p = .03$).
Among the non-cognitive discriminating variables, psychological distress, and optimism and pessimism appear to make the most consistent contributions to the discriminant functions for most of the grouping variables. For example, for fluctuations over the last two years, and speed of symptom progression, the most important predictor separating participants experiencing fluctuations and those not, was psychological distress. For remissions, optimism and pessimism were the most important predictors separating those who experienced remissions from those who did not, in the hypothesised direction in each case. The relative importance of all discriminating variables are displayed in the DFA results reproduced in Appendix III.

**Demographic Variables**

Level of education, especially education to primary level only, was, relative to the other discriminating variables, of some importance in the separation of groups of the disease course components. For example, for fluctuations, primary education followed appraisal as the variable of most importance. While however, education to primary level appears to be an important variable relative to the others, examination of the respective group means reveals no meaningful or consistent pattern even though there was clear separation between groups. For example, all participants who reached primary level reported fluctuations while the same participants reported no remissions. While it was expected that lower educational level would be associated with more unfavourable disease course (no fluctuations, no remissions), an explanation might be that these particular components have little theoretical link with educational status.

No other demographic variables displayed any meaningful association with disease course in the discriminant analyses.

**Summary of the Relationship Between Illness Cognitions and Disease Course**

Subject to the caveat imposed by the overall insignificance of the multivariate results, these results do reveal a meaningful relationship between appraisal and expectations.
and disease course component groups. Of the variance that is shared between illness cognition and disease course, the moderate to high standardised coefficients for appraisal and expectations suggest that the variance is mostly attributable to the relationship of these variables with disease course. Put another way, the separation or difference between groups on disease course components are largely a function of differences in appraisal and expectations. This is further supported by the magnitude of the correlations between appraisal and expectations and various disease course components as reflected in the structural coefficients for these variables.

The standardised and structural coefficients further reveal that psychological distress, optimism and level of education are also important in the separation of groups, acting with appraisal to influence disease course. Furthermore the moderate and significant correlation between appraisal and psychological distress, suggests that the importance of appraisal to the separation of groups is partly attributable to (lower) levels of psychological distress.

The present results, at both bivariate and multivariate levels, are consistent in a trend towards appraisal and expectations being meaningful factors in the course that RA takes. Psychological distress and optimistic outlook tend also to influence this relationship.
CHAPTER EIGHT

Discussion

The aim of the research described in this thesis was primarily to explore the relationship between illness cognitions and disease course for chronic RA sufferers, while acknowledging and accounting for the effects of certain other psychological factors of both a dispositional nature (such as optimistic or pessimistic outlook) and of a more general responsive nature (such as mood and psychological distress).

The fundamental hypothesis is that a negative style of thinking about one’s RA, together with a more negative appraisal of present condition and future expectations, belief in external agents rather than personal responsibility, coupled with poor general knowledge of RA, is associated with a more unfavourable disease course.

The concept of disease course has been ill-defined and somewhat confused in the literature. The hypothesis in this study was not a disease onset hypothesis, nor was it an outcome hypothesis, both of which have, in the literature, been attributed to disease course. Rather, it related to the profile of the disease during its life - how it changes and progresses, both negatively and positively. This change or progression was not therefore, assessed by impairment, disability, or disease activity measures. It was assessed in terms of generalised symptom improvement or worsening, and the patterns and speed of these changes, fluctuations, remission, and changes in ability with ADL.

Essentially the hypothesis was concerned with the effect of the integrated model of illness cognitions on disease course. No association was found between the composite illness cognition model (i.e., the hypothesised set of illness cognitions) and any of the components of disease course. The results did however, disclose differences in the relative importance of the individual illness cognitions within the set.
Appraisal of present condition and the related cognition, future expectations, were the most important in the relationship with disease course when the effect of the other cognitions was accounted for, and the other related psychological and demographic factors were controlled for. It is noteworthy also, that all components of disease course disclosed meaningful association with one or other of these variables.

The relative importance of these cognitions was the logical result of the finding that appraisal and expectations were the only cognitions directly associated with disease course when assessed in isolation. Similarly, of the non-cognitive variables, psychological distress and dispositional optimism and pessimism demonstrated the most association with disease course.

**Disease Course as a Criterion Measure**

One of the intentions of the present study was to establish disease course as a sound criterion. The disease course hypothesis was essentially as proposed by McFarlane et al. (1987) - that the experience of RA evokes a psychological response which may influence the course the disease takes. Unlike McFarlane et al. (1987), the present study has maintained a clear distinction between disease course and disease outcome and activity. It has therefore been confined to that time frame after onset and before final outcome when the disease is unpredictable and transitory. This is a distinction which previous studies have not adequately recognised (Scott & Huskisson, 1992). In the present study a number of criterion measures were brought together to provide an assessment that would capture the special capricious nature of the course of RA.

The relationships among the disease course components support the conceptual basis of the measurement of disease course employed in this study. Where there were strong associations between disease course components (e.g., between remissions and fluctuations) this did not negate the conceptual basis of the distinction between these components. The seven components were assessing fundamentally different aspects of
disease course and any apparent overlap was as expected. For example, those reporting periods of remission would also have experienced fluctuations, although not necessarily vice versa. Similarly, those reporting changes in symptoms over disease life (which was also a component strongly associated with remissions and fluctuations), would have included those who experienced fluctuations and possibly remissions, although, the reverse does not necessarily hold. Similarly, two of the measures (symptom change and fluctuations) were assessed both over two years and over the life of the disease as separate criterion variables. Clearly for some, changes will have occurred both over the last two years and over a longer time span, while for others only one or the other will be applicable. It was important to consider the possibility that changes were a recent phenomenon only or had ceased to occur in recent times. The moderate correlation between time frames suggests that for many sufferers symptom changes and fluctuations have characterised their condition throughout its life, and still do.

A further consideration is that there is no theoretical or somatic basis for expecting any particular combination of these components. Different sufferers may experience a different combination, yet any one of the components, or any combination, could be an operationalisation of disease course. Predicting disease course is important, not predicting the specific components which are variable and unpredictable across sufferers.

The disease course model as operationalised by the seven criterion components utilised in this study, has no particular empirical support, and the present study does not purport to provide this. Conceptually however the disease course construct has received considerable support in the literature (e.g., McFarlane et al., 1987, and Scott & Huskisson, 1992), and occupies a logical place between disease onset and outcome.
Assessment of the efficacy of the operationalisation of disease course in this study is exacerbated however, by the study's failure to find consistent association between illness cognitions and the disease course components. It cannot be stated with confidence that this result is a function of the measurements for either or both of the independent and dependent variables, or whether either or both of the illness cognition and disease course models is flawed conceptually, or whether, simply, the influence of illness cognitions on disease course is of a limited nature. The latter position is suggested as representing the actual situation, however the first two must be acknowledged as being possible limitations of this study.

It is of some comfort however, that the disease course DVs performed consistently in terms of their relationships with the IVs. For example, fluctuations over two years displayed no association with any of the fifteen independent measures. Where DVs did evidence some association with IVs there was also a degree of consistency - the particular independent measure would be associated with a number of dependent measures, appraisals and expectations, psychological distress and age being examples.

Illness Cognitions and Disease Course in Rheumatoid Arthritis

The results did not support the hypothesis that the proposed composite model of illness cognitions is associated with disease course in RA. Stated another way, no significant associations were found between the combination of illness cognitions (where the effect of all predictors was taken into account) and any disease course component.

Further examination of these results however, suggested that some illness cognitions, namely appraisal of present condition and expectations for the future, are more important than others, and individually do evidence meaningful associations with disease course when the effect of the remaining cognitions is taken account of along with certain other psychological and demographic factors. It is noteworthy that all
disease course components disclosed meaningful association with one or other of the appraisal and expectations variables. The remainder of this section offers some explanations for the significance and relative importance of the appraisal and expectation cognitions and for the insignificance of the remaining cognitions.

One explanation for the consistent effect of these cognitions is that they are the most directly related to the somatic condition. They are effectively asking, "how is your RA today and how is it likely to be in the future?". Knowledge, thinking style, and control beliefs however, are essentially questioning about thought processes, feelings and attitudes. The metacognitive aspect of these variables may have required more thought and introspection than a predominantly elderly population is accustomed to being questioned about. Not unexpectedly, longer illness duration was associated with more negative appraisal. Intuitively it is arguable that the negative appraisal resulted from the illness duration. It is also arguable however, that chronicity is influenced by negative appraisal. This is supported by the finding that future expectations are not associated with illness duration. Any negativity would be expected to be reflected in reports of future expectations.

Similarly, while appraisal of current condition is significantly associated with negative affect and psychological distress, appraisal of the future is not. If negative affect and distress were enduring factors that were sharing the variance between appraisal and disease course, it is expected that the psychological states would similarly influence future appraisal.

Further confirmation of the independence of the appraisal and expectations variables is the unexpected finding of no relationship between expectations and dispositional optimism especially since expectations tended to be positive. Intuitively one would expect a relationship - optimistic people generally expect positive outcomes. Yet such relationship would have altered the conceptual proximity of the expectation variable to
the appraisal variable by moving expectations out of the illness response cognition category towards the personality variable category.

A further explanation for the relative importance of appraisal and expectations is that they are not measured as illness-specific variables as are the thinking style and locus of control variables. The attempt to make the latter responsive to the specific illness may account for their lack of significant effect in this study.

From a more conceptual perspective, the present study did not see appraisal as limited to being an antecedent of coping, which has generally been the case to date (e.g., Berkowitz, 1986; Smith & Wallston, 1992). The present study has more in common with the personal models of illness approach taken by Hampson et al. (1994) in their investigation with respect to osteoarthritis. An important distinction however, between Hampson et al. (1994) and the present study is this study's concentration on appraisal of one's own disease experience as opposed to appraisal of RA generally as a disease. Thus, a personal model of illness approach became a personal response to illness model. This was then applied to the potential of this response model to modify disease course in RA. This application of appraisal to actual disease parameters is a progression and a contribution made by the present study.

There are two components to appraisal. One is simply assessment of current disease status. The other is the assessment of disease status against the background of one's knowledge and understanding about the disease itself. This points to the more integrative and possibly interactional role of knowledge with other cognitions, rather than a direct effect on disease course. The possession of knowledge per se is of little use and therefore of little effect. It is the appropriate utilisation of that knowledge that is important. Because the reasonable level of knowledge displayed by this population related mainly to non-clinical aspects of RA and its treatment, utilisation would have excluded the clinical arena, from where any direct effect could be expected to come.
As, not unexpectedly, knowledge of RA is possessed by those who reached a higher level of education, then a reasonable assumption is that the knowledge is put to good use. To some extent this assumption is assisted by the influence knowledge displayed in reducing pessimism, negative affect, and psychological distress. The suggestion that knowledge while having no direct effect on disease, interrelates with other psychological variables that are important in RA. coincides with Pritchard’s (1989) finding in her pilot study that a lack of knowledge contributed to psychological distress. Pritchard’s (1989) main study however, did not support the pilot study finding. Even levels of optimism/pessimism showed no association with knowledge.

A possible explanation for the difference in Pritchard’s (1989) findings and those of the present study, is the difference in patient knowledge scores. The “confused and incoherent” understanding of their RA demonstrated by Pritchard’s 1989 sample was not so for the present sample. Poor illness knowledge may not translate into increased pessimism and psychological distress because not knowing something does not necessarily create distress. A higher level of understanding however brings with it peace of mind or at least certainty which could translate into decreased pessimism and psychological distress. Conceptually the findings may not conflict. Pritchard’s finding that patients tend to overestimate the risks of their RA and underestimate the value of their treatment seemed to be a function of their poor knowledge about RA. In the present study such a negative bias was not evident, at least in terms of appraisals and expectations, which may have been attributed to the generally higher level of knowledge and understanding.

The lack of any significant association with disease course was unexpected for thinking style. Pow (1987) found that those who were sero-positive for Rheumatoid Factor showed a negative style of thinking in relation to their illness. By equating sero-positive with more unfavourable disease course, the expectation was for a similar finding. It is arguable however, that Pow’s (1987) findings were confounded by her sero-positive group being older, more disabled, and of lower socio-economic status.
that the sero-negative group. These potential confounds were not present in this study. While socio-economic status was not measured, informal enquiries suggest little variability.

A possible explanation for the apparent lack of importance of thinking style is that its effect was masked by the effect of dispositional optimism or pessimism because of their close relationship conceptually. There may not be sufficient separation between general dispositional outlook on life factors and illness specific response factors which essentially comprise RA thinking style. Conceptually it was important to separate the effect of pre-existent dispositional traits from the effect of illness cognitions and to control for these trait personality factors. They are distinctive factors which should be addressed and treated quite differently. In practice however, this separation may not have been achieved.

A further blurring of the distinction could arise from thinking style being determined by underlying personality traits like dispositional optimism or pessimism - thinking style may simply be an extension of these personality factors. The significant findings that as negative affect and psychological distress levels increase so does negativity in thinking style, suggest that this cognition is responsive also to these psychological factors. It may therefore be too closely associated with or affected by these factors to be a useful determinant of disease course.

The failure of locus of control to demonstrate any relationship with disease course was possibly a function of the validity of the measurement device. Validity research on the non-illness-specific MHLC has resulted in various opinions, but have generally found validity to be greater when used as a dependent measure (Lefcourt, 1991). An assumption as to the validity of the arthritis specific version may be presumptuous.

A possible explanation from a conceptual perspective is that fundamentally it was the intention in this study to distinguish between taking personal responsibility for how
one's health or illness is, and passing that responsibility on to some other person or agent. This may in fact be control versus no control, rather than external control versus internal control. Roskam (1986) suggested the former distinction was more important. Accordingly, by utilising the MALC scales scored separately, the study may not have focussed as intensely on this distinction as it should have. A combining of the effects of externality and chance, as representing something other than personal responsibility and control, may have helped to illuminate the distinction and reveal some association with disease course. Alternatively the development of a more focussed scale may be necessary.

A further possible explanation is that chronic RA sufferers are on regular medication that requires careful monitoring for side effects and the fluctuating nature of the disease. They are therefore often locked in to reliance on and control by external agents, even though their personal preference may be for personal responsibility and control. A revised scale could take account of this.

The Effect of Non-Cognitive Factors

The contribution of the non-cognitive psychological and demographic factors was of interest primarily in the composite sense where the effect of all other IVs was accounted for. Their independent effect on disease course however, also provided some understanding of the psychological concomitants of disease course in RA. Of the non-cognitive variables, distress was the most important predictor, surpassing appraisal as the most important variable separating those who experienced fluctuations over the last two years from those who did not. The distress variable however, is essentially a control variable and should not be interpreted in a predictive sense. It is a measure of current (over the last seven days) distress and therefore may itself be a result of the illness and the course it is taking. It is also illogical to assess the retrospective influence on a long term criterion of a predictor that is measured for its present effect.
For the association between psychological distress and change in ADL difficulty it is unclear which comes first. This is possibly due to the temporal proximity of the two variables - change in difficulty with ADL refers to the current situation which is the same for distress. It would be unwise to interpret this association in terms of psychological distress as the predictor variable. Intuitively it is more plausible to think of ADL change as predictive of distress.

The only component of disease course that optimism/pessimism tended to have any association with was speed of change in RA and this was brought to light only by the bidimensional scoring of the LOT. It is understandable, in terms of the hypothesis that as pessimism increases so does the speed of progression of RA. There seems to be no obvious explanation however, for the finding (albeit a lower and less significant correlation) that increased optimism is also associated with a speeding up in progression. One possibility is that optimism has the effect of reducing commitment to medical advice and other treatment or self-help strategies.

Among the demographic variables education to primary level was the only variable of any importance and then only for fluctuations and remissions. The only explanation for the seemingly contradictory results between these criterion variables is that the reporting of these variables requires more memory input and analytical type thinking about one's life with RA, which may have caused some inconsistency in response across a lower educated sample. It is possible also that there may have been some difficulty understanding the questions. The results did not indicate however, that level of education had any confounding effect that may have contributed to the lack of significance of the results.

Age discloses some consistent associations with disease course components however. Understandably people find increasing difficulty with ADL as age increases. It is arguable however, that RA hastens the change, since the items assessed change over
the last six months. The association between age (increasing) and symptoms worsening over the last two years, is also not necessarily a function of age. The negative correlations between age and remissions and fluctuations suggest that with increasing age comes fewer fluctuations and remissions. This in turn may suggest an overall imbedding of chronicity rather than age being predictive of unfavourable disease course. Not unexpectedly, a similar picture emerges for illness duration. Generally, as time since diagnosis increases, so does the perception of unfavourableness in disease course.

**Doctor's Confirmation**

The level of doctors' response to providing confirmation of participants' self reports as to the diagnosis, illness duration, fluctuations and remissions was encouraging. Doctors substantially confirmed their patients' self reports, although disclosed a distinct negative orientation on the part of the participants in terms of duration, fluctuations and remissions. Patients' reports of longer illness duration and fewer fluctuations and remissions than their doctors reported represent the perception of a more unfavourable disease course than their doctors have recorded.

This orientation could logically be expected to operate for appraisal and future expectations also. For appraisal at least, there was a trend towards severity. As a predictor variable, appraisal is not concerned however, with actual clinical status. It is concerned with sufferers' perceptions and beliefs about their status. This raises the possibility that participants' reports of these cognitions may in fact be distorted. On the other hand, patients may be better judges of their own condition. The doctors' confirmation data however, do not permit any firm conclusions to be drawn regarding the relationship between appraisal and disease course based on the more favourable self reports of disease course. This is primarily because the doctors' sample is inadequate in relation to the patient population, especially since some doctors represented more than one patient. There are also other questions that would need
addressing, such as, where the doctors' information came from - patients reports to him/her, or his own comprehensive observations. There is also the question, already raised, as to whether participants fully understood the disease course items. Studies relying on self reports of illness status, past and present, may need to account for the negative orientation, or consider the need for more objective assessment procedures. To objectively assess past changes in disease status however, longitudinal studies may be necessary.

**Implications Arising from the Study**

There are various implications of these findings. RA sufferers appear to be reasonably in touch with their condition. This is evidenced, for example, by their interest in learning about their disease and the treatment of it, particularly about how their own actions and lifestyles might help them. Yet there is an imbalance between their clinical knowledge and what they can learn in a lay environment, as was evidenced by the pattern emerging from an examination of the knowledge scores. While the study did not test for an interactional effect of knowledge of RA, the indication emerged that knowledge is interrelated with other illness cognitions and may therefore play an important interactional role with illness cognitions and other psychological factors in their relationship with disease course. Accordingly attention by doctors to patient education about the clinical aspects of the disease, not simply symptom management, may address the imbalance. The probable beneficial effects of this are both psychological in terms of better adjustment to the disease process, and practical in terms of better adherence to treatment regimens. The suggestion that doctors pay attention to patient education is not a criticism that they do not. It may be that patients are not eager to learn the clinical details or may be quick to forget or reject them.

The overall educational and encouraging role of the likes of the Arthritis Foundation emerges as an implication of the findings as well. Appraisal of one's condition and
expectations of the future, which have been shown to be of some importance in disease course, are most likely to be modifiable through education and support based interventions.

Perhaps more importantly, the study demonstrates the need for an integrated team approach comprising doctor, specialised non-medical care giver/support agency (such as the Arthritis Foundation) and the sufferer. Doctors must recognise the role of psychological factors. Non-medical support givers must recognise their ability to interact with and modify or strengthen the cognitive approach people have to their RA. Their role as encouragers and motivators to a positive approach is part of this. The sufferer must be eager to learn about his or her condition and its treatment and respond with self help strategies and commitment to becoming and doing whatever he/she can to promote a more favourable disease course.

Appraisal of present condition and expectations for the future are reasonably pervasive factors that subsume aspects of the other cognitions and psychological factors. For example, appraisals and expectations are based on knowledge of the disease; they will be flavoured by disease response thinking style and dispositional outlook on life; level of belief in one’s own ability to influence progress of the disease and one’s own responsibilities regarding it will have a bearing on how one sees one’s current and future condition; and daily mood and level of psychological distress will impact one’s approach to RA, and vice versa. Treatment and therapy strategies and approaches to RA, in taking account of all these factors, may be able to influence the course RA takes in a given individual by modifying the individual’s approach to his or her condition in terms of current appraisal and future expectations. The flow-on effect of positive appraisal and expectations for immunocompetence, treatment adherence, lifestyle modification and the like, is likely to favourably influence disease course and quality of life in general.
Limitations

The most obvious limitation of the present study is its correlational design which precludes the suggestion of causal relationships between variables. This is in addition to those instances where directionality of effect is ambiguous conceptually, and where time frames in which variables are measured are the same.

A longitudinal design would overcome much of the limitation of the retrospective design of this study. In obtaining information about medical status at various points in time over a long time frame, self report is subject to various confounding factors. Self reports of physical symptoms are influenced by cognitive, psychological and cultural factors which may affect their reliability (Pennebaker, 1982). To some extent doctors’ confirmation alleviated this problem, but it also highlighted the problem. Not utilising examination by doctors to determine disease status, which is a common approach, is not seen as a limitation in this study however. Disease activity is not seen as an aspect of disease course, and even if it were, that assessment procedure relates only to the present. Such information is irrelevant for assessment of changes if it cannot be compared with prior examinations. Doctors’ records, in a limited way, have been shown in this study to provide the necessary data. The extension of this procedure into a longitudinal design where doctors know in advance the type of information to be included in their notes would add considerably to a study such as the present one.

A further limitation emanates from the use of some dependent and independent measures for which no psychometric data have been obtained, or for which doubts have been raised as to validity. The RTQ and MALC scales are in these respective categories.

There is also potential difficulty inherent in the appraisal and expectation measures. While it was intended that they reflect a person’s usual and enduring attitude to their illness and its future, it cannot be stated unequivocally from the present data that this
was achieved. They may reflect only current attitude, which would affect the
distinction between predictor and criterion. These measures are also likely to be
affected by the deficiencies of self report and a response set that has participants
placing themselves in the 'average' category.

For some of the dependent measures, questionnaire design may have been confusing
or ambiguous. The forced choice method was specifically avoided to reduce the
possibility of prompting, or extracting a response which was artificial. The design of
these items was intended to permit freer self expression without forcing a choice to be
made about a possibly irrelevant or non-existent circumstance for a given participant.
Some clarity of what was required of participants, and therefore certainty of response,
may have been sacrificed. Design of these measures could be improved.

A possible confound which would be common in psychosomatic research is the effect
of any other illness a participant might be suffering from. While all measures were RA
specific, the psychological effect of any other serious or chronic condition would be
difficult to isolate and control for.

It is acknowledged that generalisability of the present results may be limited by the
selection of participants from among those who belong to the Arthritis Foundation.
Foundation membership may reflect psychological factors not present in the wider
community of RA sufferers, such as socio-economic status and a self help attitude. It
may also reflect the considerable benefits of the support and practical assistance
provided by the Foundation.

In terms of the statistical analysis of the results, the size of the sample may have
limited the power of the study to detecting only the strongest effects. The sample size
may also have been too small for the number of measures used, which may have
limited the power of the effects that were found.
**Future Research**

Previous research that has examined disease course in RA has failed to produce consistent findings (Mcfarlane et al., 1987). This, and the tentative findings of the present study, suggests the need for further examination of the role that psychological factors play in the disease course of RA. The present study has highlighted however, the need to clearly differentiate between disease outcome, disease activity, and disease course. If disease course is conceptualised and measured as a separate construct in future research, this in itself will be progress.

More specifically, future research with illness cognitions would benefit by assessing interactional and mediating effects of illness cognitions, especially for the knowledge variable. The probability of these types of effects has been demonstrated in the present study.

Given the relative importance of the appraisal and expectation variables disclosed in this study, attention should be given in future research to the development of a more detailed measure for these factors. The Likert scale format may encourage an “average” response set. A questionnaire format may elicit a more accurate and meaningful response. Similarly, the disease course measures would benefit from more attention to their design to reduce ambiguity.

The indication from this study is that illness cognitions may not necessarily be as illness specific as was expected. Accordingly, it may be more beneficial for future studies to utilise measures that are applicable to the specific peculiarities of RA, but which tap into broader aspects of attitudes towards illness generally. A further indication is that the influence of illness cognitions could be investigated on an individual cognition basis rather than on a composite basis.
Finally, the indication that doctors are interested in the psychological aspects of RA, and are prepared to be involved in the research process, suggests that their assistance in validating self reports should be investigated as a worthwhile strengthening process in a study. Their involvement also helps in the melding of the disciplines of medicine and psychology, which is fundamental to the psychosomatic approach.

**Conclusion**

Alexander's (1950) classification of RA as one of the seven classic psychosomatic disorders must be seen in terms of disease progression rather than onset and causality. The findings of the present study, while tentative, do suggest that one's response psychologically, to one's RA, in terms of assessment of current condition and view of the future with the disease, may influence the pace of, and extent to which the disease progresses, and its characteristics along the way. The study demonstrates the need for further examination of the role psychological, and especially cognitive factors, may play in the course of RA. Part of this is its demonstration of the continued erosion of the bio-medical model. The composite illness cognition model was not found to be a significant predictor of disease course. The appraisal and expectations cognitions, which arguably subsume aspects of the others in the model, did appear to be associated with the disease course however. This emphasises the need to see the progression of RA in multifactorial terms which accounts for the interaction of biological, psychological and social factors. Engel's (1977) biopsychosocial approach, with special emphasis on the response to others in the psychological component, is vital in future research.

The results of this study point to the conclusion that the course of a disease such as RA may be influenced and modified by the psychological response of sufferers to their condition. Three possible mechanisms are suggested by which this may operate. One concerns a direct effect of the psychological response on actual disease parameters via the likes of improved immunocompetence and regenerative properties. Another is
simply that a positive response may evoke a lifestyle and physical activity levels which are in themselves therapeutic, (and unexpectedly achievable) resulting in the breaking down of preconceived barriers and myths that have been generated about the limitations of RA. A third pertains to an increased awareness and acceptance of, and motivation to adhere to treatment regimens and pain reduction strategies. This in turn will result in increased activity and mobility bringing both physical and psychological therapy. These mechanisms can all be said to be part of the psychosomatic aspect of RA. They have in common the impetus produced by a positive appraisal of one's present condition and one's future prognosis. Thus, Alexander's (1950) classification of RA as having an important psychosomatic component may be confirmed.
REFERENCES


APPENDIX I

Participant's Questionnaire
RHEUMATOID ARTHRITIS RESEARCH QUESTIONNAIRE

IN CONFIDENCE

Listed below are four sets of the same eight questions about activities of daily life. Each set asks you about a different aspect of the activities.

Set 1 Please tick the column that best indicates the degree of difficulty you have with these activities at the present time.

Are you able to:

<table>
<thead>
<tr>
<th></th>
<th>Without Any Difficulty</th>
<th>With Some Difficulty</th>
<th>With Much Difficulty</th>
<th>Unable To Do</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Dress yourself including tying shoe laces &amp; doing up buttons</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Get in &amp; out of bed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Lift a full cup or glass to your mouth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Walk outdoors on flat ground</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Wash &amp; dry your entire body</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Bend down to pick up clothing from the floor</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Turn taps on &amp; off</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Get in &amp; out of a car</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Set 2 Please tick the column that indicates whether you are satisfied or dissatisfied with your ability to perform these activities.

How satisfied are you with your ability to:

<table>
<thead>
<tr>
<th></th>
<th>Satisfied</th>
<th>Dissatisfied</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Dress yourself including tying shoe laces &amp; doing up buttons</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Get in &amp; out of bed</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Lift a full cup or glass to your mouth</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Walk outdoors on flat ground</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Wash &amp; dry your entire body</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Bend down to pick up clothing from the floor</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Turn taps on &amp; off</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Get in &amp; out of a car</td>
<td></td>
</tr>
</tbody>
</table>
**Set 3** Please tick the column that best indicates any change in difficulty over the last six months.

Compared to six months ago, how difficult is it now to:

<table>
<thead>
<tr>
<th></th>
<th>Less Difficult Now</th>
<th>No Change</th>
<th>More Difficult Now</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Dress yourself including tying shoe laces &amp; doing up buttons</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Get in &amp; out of bed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Lift a full cup or glass to your mouth</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Walk outdoors on flat ground</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Wash &amp; dry your entire body</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Bend down to pick up clothing from the floor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Turn taps on &amp; off</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Get in &amp; out of a car</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Set 4** Please tick the column that indicates whether or not you need help to perform these activities.

Do you need help to:

<table>
<thead>
<tr>
<th></th>
<th>Do Not Need Help</th>
<th>Need Help</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Dress yourself including tying shoe laces &amp; doing up buttons</td>
<td></td>
</tr>
<tr>
<td>2.</td>
<td>Get in &amp; out of bed</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Lift a full cup or glass to your mouth</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Walk outdoors on flat ground</td>
<td></td>
</tr>
<tr>
<td>5.</td>
<td>Wash &amp; dry your entire body</td>
<td></td>
</tr>
<tr>
<td>6.</td>
<td>Bend down to pick up clothing from the floor</td>
<td></td>
</tr>
<tr>
<td>7.</td>
<td>Turn taps on &amp; off</td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Get in &amp; out of a car</td>
<td></td>
</tr>
</tbody>
</table>
The following set of questions is not specifically about arthritis. As you answer these questions be as accurate and honest as you can. Try not to let your answer to one question influence your answer to other questions.

There are no right or wrong answers.

Circle a number for the one answer that best indicates the extent to which you agree with the statement.

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>0</strong></td>
<td>Strongly Disagree</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>1</strong></td>
<td>Disagree</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>2</strong></td>
<td>Neutral</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>3</strong></td>
<td>Agree</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>4</strong></td>
<td>Strongly Agree</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In uncertain times, I usually expect the best 0 1 2 3 4
It's easy for me to relax 0 1 2 3 4
If something can go wrong, for me it will 0 1 2 3 4
I always look on the bright side of things 0 1 2 3 4
I'm always optimistic about my future 0 1 2 3 4
I enjoy my friends a lot 0 1 2 3 4
It's important for me to keep busy 0 1 2 3 4
I hardly ever expect things to go my way 0 1 2 3 4
Things never work out the way I want them to 0 1 2 3 4
I don't get upset too easily 0 1 2 3 4
I'm a believer in the idea that "every cloud has a silver lining" 0 1 2 3 4
I rarely count on good things happening to me 0 1 2 3 4
The following are a number of words that describe different feelings and emotions. Consider each word and then circle a number which best indicates to what extent you have felt this way during the past few weeks.

<table>
<thead>
<tr>
<th>Word</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distressed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upset</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guilty</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scared</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hostile</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irritable</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ashamed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nervous</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jittery</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Afraid</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In the following set of 29 questions, please circle the number of the option which you consider to be the correct answer. Please answer these questions from your own knowledge of RA.

1. Rheumatoid arthritis:
   1. Only affects the joints
   2. Only affects the joints and immediately surrounding tissues (eg. muscles and tendons)
   3. Only affects the joints and blood
   4. Always affects the joints and can also affect many different organs and systems throughout the body
   5. Always affects the bones and in some cases also affects the blood and eyes but nothing else

2. Generally speaking, in RA, exercise:
   1. Should be avoided as far as is possible because it wears the joints out more quickly
   2. Is good because it keeps you generally more healthy and fit and therefore better able to cope with a disease like RA
   3. Should be avoided as far as possible, as it inflames joints that were previously alright
   4. Is important as it helps, ultimately, to reduce the pain and inflammation
   5. Is important, done correctly, as it maintains the movement and usefulness of the joint.
3. The thing that most characterises RA is:
   1. The inflammation or growth of the joint lining
   2. The loss or drying-up of the joint fluid
   3. Chemical changes in the joint fluid (eg. increased acidity or crystallisation)
   4. Loss of bone tissue
   5. Growth of abnormal bone

4. Some RA patients are given splints to wear at night which hold the hand and wrist in a set position. What do you think the main purpose of these splints is?
   1. To repair deformity
   2. To stop you lying on your hands and thereby stopping the blood getting to the joints
   3. To make your joints more comfortable through the night
   4. To prevent long-term deformities
   5. To hold the joints still so that they can be repaired overnight

5. When do you think it would be most important to wear night splints?
   1. When the joints are cool, not swollen or painful
   2. When the joints are very painful, swollen, hot, and inflamed
   3. When you have not used those joints much during the day
   4. When you have used those joints a lot during the day
   5. Every night is equally important, therefore, it does not matter when you wear them just so long as you wear them fairly often

6. RA most commonly starts:
   1. In childhood
   2. In adolescence (the teens)
   3. In early adulthood (under 25)
   4. Between the ages of 25 and 55
   5. In the over 55s

7. RA is:
   1. A chronic or long-term disorder
   2. An acute, or short-term disorder
   3. A disorder of intermediate length

8. What sort of exercise is best for the rheumatoid arthritic?
   1. Any sort of exercise done in moderation
   2. Exercises that put a strain or tension on the affected joints and thereby strengthen them (eg. archery for the hand)
   3. No exercise is good - joints should be used as little as possible
   4. Exercises that involve quick jerky movements (eg. squash, jogging)
   5. Exercises that put each joint through its full range of movement without putting a strain on the joints

9. Most of the pain experienced with RA is caused by:
   1. The acidity of the joint fluid eating into the joint tissues and bone
   2. The joint not having enough fluid to lubricate it
   3. The inflammatory process and the grinding together of bones
   4. The joint fluid leaking into the muscle
   5. Sharp crystals that have formed in the joint damaging the joint tissues
10. Drugs used in the treatment of RA:
1. Carry a low risk of side-effects (less than 5% of people on these drugs develop side-effects)
2. Carry a fairly low risk of side-effects (around 5% to 10% of the people on these drugs develop side-effects)
3. Carry a moderate risk of side-effects (10% to 20% of people on these drugs develop side-effects)
4. Carry a fairly high risk of side-effects (around 20% to 50% of people on these drugs develop side-effects)
5. Carry a very high risk of side-effects (over 50% of people on these drugs develop side-effects)

11. Deformity in RA is caused primarily by:
1. Damage to the tendons and ligaments and loss of bone
2. Loss of muscle tissue
3. Abnormal growth and twisting or warping of the bones
4. Loss of muscle tone and strength
5. Loss of bone and abnormal bone growth

12. What proportion of RA patients become completely disabled?
1. Over 70%
2. Between 50% and 60%
3. Between 30% and 50%
4. Between 10% and 30%
5. Less than 10%

13. Morning stiffness is produced by:
1. The joint fluid getting or getting more sticky or viscous overnight
2. An accumulation of fluid in the joint tissues overnight
3. A loss or reduction in the amount of joint fluid overnight
4. The muscles involved in movement seizing-up due to inactivity
5. The tendons and ligaments contracting or shrinking overnight

14. Which of these statements is true?
1. There are two main categories of drug used in the treatment of RA - those which reduce the symptoms of pain and inflammation and those that also slow the disease down
2. The only effect of drugs in the treatment of RA is to reduce the pain experienced
3. There are drugs used in the treatment of RA which can actually cure the disease
4. While there are drugs which actually reduce the symptoms of swelling and inflammation, as well as the pain, there is none which actually affects the eventual damage that RA does to the body
5. All of the drugs used in the treatment of RA affect the speed with which the disease damages the joints

15. Approximately what proportion of RA patients have little or no disability?
1. Over 75%
2. Between 55% and 75%
3. Between 35% and 55%
4. Between 15% and 35%
5. Less than 15%
16. In RA, bone tissue:
   1. Turns to crystals at the outer edges
   2. Is not affected
   3. Is swollen or enlarged
   4. Is eroded or destroyed chemically and mechanically
   5. Is first softened by joint fluid being absorbed into it and then crystallised by the chemical action of the joint fluid in the bones

17. Deformity in RA:
   1. Occurs in every case and cannot be prevented or lessened at all
   2. Is only avoided or lessened by the use of drugs
   3. Can only be avoided by stopping using the joint altogether
   4. Can probably only be avoided or lessened by adopting a very strenuous exercise programme
   5. Can probably be avoided, to some extent at least, by the careful use of the joints and the appropriate use of exercise and rest

18. In RA the amount of time that joints are very hot, inflamed, and tender:
   1. Tends to increase with the length of illness
   2. Tends to lessen after a number of years and in some cases seems to stop altogether
   3. Tends to lessen after a number of years, but never stops altogether
   4. Does not alter with the duration of illness
   5. Tends to stay about the same or get worse

19. A hot inflamed joint:
   1. Should be exercised as much as possible and ideally more than normal
   2. Should be exercised very frequently throughout the day using special exercises that involve moving the joint fully without straining it
   3. Should be rested completely except for moving it through its range of movement twice a day
   4. Should be rested completely
   5. Should be used as normal despite the pain

20. In RA, when a joint is hot, inflamed, and very tender and painful, rest:
   1. Makes you feel better but does not really help in any other way
   2. Helps prevent long-term deformities, reduces damage to the joint, and helps reduce the hotness, inflammation and pain
   3. Makes you feel better but actually makes long-term deformities and problems more likely than if the joint is used normally
   4. Makes the hotness and inflammation stay longer than if the joint is used normally
   5. Is bad as it makes the joint very stiff and reduces its mobility

21. In RA the joint membrane:
   1. Becomes thinner and more delicate
   2. Reduces in elasticity or stretch
   3. Thickens, becomes inflamed and produces abnormal tissue
   4. Is eaten away and destroyed
   5. Contracts or shrinks, reducing the space between the bones
22. In RA the joint fluid:
1. Is lost or decreased
2. Is crystallised
3. Is thinner and often increased
4. Gels or thickens
5. Is absorbed into the bones making them soft

23. In what way, if at all, does the way symptoms first occur predict the result of RA?
1. Rapid onset of symptoms in many joints suggests a more disabling arthritis
2. Rapid onset of symptoms in many joints suggests a less disabling arthritis
3. A gradual onset of symptoms suggests a less disabling arthritis
4. A gradual or slow onset of symptoms suggests a more disabling arthritis
5. Type of onset of symptoms (rapid or gradual) is not associated with the degree of disability

24. A Rheumatoid Factor is:
1. A gene type (an inherited factor or unit)
2. Virus (type of germ)
3. A type of blood cell
4. Joint fluid cell
5. Antibody (a part of the body's defence system)

25. High levels of rheumatoid factor in the blood:
1. Are associated with severe RA and more non-joint complications
2. Are associated with mild RA but more non-joint complications
3. Are associated with severe RA but less non-joint complications
4. Are associated with mild RA
5. Are not associated with the course of RA or frequency of non-joint complications

26. The cause of RA is:
1. Unknown
2. A virus (type of germ)
3. Genetic
4. The cold and damp
5. Wear and tear (the over-use or bad use of the joints, accidents or old age)

27. Which of these general guidelines about exercise do you think is correct?
1. If after exercise your joints ache more than before, the sort of exercise is wrong or you have overdone it
2. Doing as much exercise as is possible, regardless of the effects afterwards, is best for the arthritis
3. A level of exercise that does not make your joints ache more (or for less than one hour) is probably right
4. A level of exercise that makes the joints ache more the next day, but no longer, is likely to do the joints most good
5. No exercise is good for RA, whether it makes the joints ache more or not.

28. Rheumatoid arthritis occurs:
1. About twice as often in men as in women
2. About twice as often in women as in men
3. About ten times as often in men as in women
4. About ten times as often in women as in men
5. With equal frequency in both men and women
29. What percentage of the population has RA?
1. About 2%
2. About 10%
3. About 25%
4. About 40%
5. About 55%

The following statements refer to how you see your arthritis as changing over time for the last couple of years. Please tick any of the statements which you think apply to you. Remember, you may tick more than one.

- Except for some day to day variation, my arthritis symptoms have remained about the same intensity for the last couple of years
- Except for some day to day variation, my arthritis symptoms have become steadily worse
- Except for some day to day variation, my arthritis symptoms are not as bad now as they were two years ago
- My arthritis seems to get gradually better or worse over a matter of months - it is like it has a cycle

The following statements are similar to the previous five, except that they cover the whole of the time that you have had arthritis. Please tick any of the statements which you think apply to you. Remember, you may tick more than one. Ignore these if you have had arthritis for only two years or less.

- Since I first got arthritis my symptoms have remained about the same
- Since I first got arthritis my symptoms have steadily worsened
- After I first got arthritis my symptoms did not seem too bad until more recently when they started worsening
- After I got arthritis my symptoms developed quite quickly and then seemed to stabilise
- Since getting arthritis there has been at least one period of time when it seemed to be in remission
- Since getting arthritis, my symptoms have been worse at some times than at others - they seem to fluctuate in intensity
Now, we would like to know how you have been feeling over the past seven days, including today. Below is a list of things you may have been feeling over this time. Please circle the appropriate number to describe how distressing you have found these things over this time.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Not at all</th>
<th>A little</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difficulty in speaking when you are excited</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Trouble remembering things</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Worried about sloppiness or carelessness</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Blaming yourself for things</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Pains in the lower part of your back</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Feeling lonely</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Feeling blue</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Your feelings being easily hurt</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Feeling others do not understand you or are unsympathetic</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Feeling that people are unfriendly or dislike you</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Having to do things very slowly in order to be sure you are doing them right</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Feeling inferior to others</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Soreness of your muscles</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Having to check and double check what you do</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Hot or cold spells</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Your mind going blank</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Numbness or tingling in parts of your body</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>A lump in your throat</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Trouble concentrating</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Weakness in parts of your body</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Heavy feelings in your arms and legs</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>

**Appraisal of your Present Condition**

On a scale of 1 to 5 where 1 represents extremely mild and 5 represents extremely severe, show how you would rate your arthritis (by circling the appropriate number).
Your Expectations for the Future

Now think about what you are expecting to happen with your arthritis in the next 12 months. Please circle the number below which best describes how you think your arthritis pain and disability are likely to be in 12 months time.

1 2 3 4 5

much better about the much worse
than now same than now [ ]

Below you will find a series of events that could happen to you. After each situation, are alternative ways that people might think about it, marked A, B, C, and D. Imagine that these events are happening to you. Then choose the alternative that best describes how you would think about the situation. (If your reaction is different from the alternatives provided choose the thought that is nearest to your own. If you agree with more than one, choose the one which would run through your mind most often.) When you have chosen the thought, put a circle around the letter next to it.

There are no right or wrong answers. Work through the questions quickly and try to pick the thought that is nearest to your immediate reaction.

1. You manage a fairly long walk. I think:
   A. I am a physically able person
   B. I wonder how I managed that
   C. Walking is one of the things I can still manage
   D. It was just luck that I was able to complete that walk

   [ ]

2. You can't manage to open a jam jar and have to ask for help. I think:
   A. I often find it difficult to do things like that
   B. I sometimes find it difficult to do things like that
   C. I just can't manage simple everyday tasks
   D. These sort of tasks are always easier with help

   [ ]

3. You are about to go into hospital for an operation which will relieve a lot of your pain. I think:
   A. It may not work
   B. It should work, but I may still have some pain
   C. I don't think there's much chance of it working
   D. It will be a success and I will be able to do all the things I used to do

   [ ]

4. You attend a new arthritis support group and make new friends. I think:
   A. It is nice to meet and mix with other arthritis sufferers
   B. This may not be a good idea
   C. Support groups are generally disastrous
   D. The support group are very pleased that I have joined them

   [ ]
5. You have a lot of pain today. I think:
A. I should have taken more care today
B. I am just incapable of helping my arthritis or handling my pain
C. I have taken as much care and handled my pain the best I could today
D. Maybe I could have been more careful

6. You used to go walking with some friends, but now that you can’t manage it they don’t seem to visit so often. I think:
A. My condition is a major strain on our friendship
B. Our friendship will suffer a bit, temporarily
C. It won’t make any difference to our friendship
D. These friendships are ruined for good

7. You are elected president of the local Arthritis Foundation. I think:
A. This is a mixed blessing
B. This is an impossible task/position
C. It is an important and interesting position
D. This position may be too difficult

8. You have won a dream holiday for next year but it seems that you may be too disabled by then to enjoy it. I think:
A. This is going to be an absolute disaster
B. It shouldn’t be too bad
C. If I plan properly, I’ll probably have a very enjoyable time
D. It will be a reasonably difficult time

9. You find that an important appointment is up three flights of stairs with no lift, and you have a lot of pain. I think:
A. This is an unfortunate coincidence
B. Why do problems like this happen to me
C. It really doesn’t matter
D. There are always obstacles in my way, whatever I want to do

10. You arthritis did not give too much trouble today. I think:
A. I didn’t do anything that would have helped it
B. It was because of the medication my doctor has given me or the advice the Arthritis Foundation gave me
C. I might have contributed in some way to its improvement
D. I helped to improve my arthritis today

11. You have been told that your arthritis may get a lot worse over the next few years. I think:
A. I will shortly become severely disabled and in constant pain
B. My arthritis isn’t going to get much worse
C. When it does get worse, I hope the pain and disability won’t be more than I can handle
D. There’s a chance my arthritis will get worse but I’ll manage okay

12. You read about a miracle drug which the developers promise will help you. It will be available soon. I think:
A. I look forward to it eagerly
B. It will probably not happen
C. If it happens I shall be pleased
D. It’s better not to make plans as it might not happen
Read the following statements and circle a number for the one answer that best indicates the extent to which you agree with the statement.

0 1 2 3 4

<table>
<thead>
<tr>
<th>Strongly Disagree</th>
<th>Disagree</th>
<th>Neutral</th>
<th>Agree</th>
<th>Strongly Agree</th>
</tr>
</thead>
</table>

If my arthritis worsens it is my own behaviour which determines how soon I feel better again 0 1 2 3 4

No matter what I or anyone else does, if my arthritis is going to get worse, it will get worse 0 1 2 3 4

If I see a doctor regularly, I am less likely to have problems with my arthritis 0 1 2 3 4

Most things that affect my arthritis happen to me by chance 0 1 2 3 4

Whenever my arthritis worsens, I should consult a medically trained professional 0 1 2 3 4

I am directly responsible for my arthritis getting better or worse 0 1 2 3 4

Other people play a big role in whether my arthritis improves, stays the same, or gets worse 0 1 2 3 4

Whatever goes wrong with my arthritis is my own fault 0 1 2 3 4

Luck plays a big part in determining how soon my arthritis improves 0 1 2 3 4

Health professionals are responsible for seeing that my arthritis improves 0 1 2 3 4

Whatever improvement occurs with my arthritis is largely a matter of good fortune 0 1 2 3 4

The main thing which affects my arthritis is what I do myself 0 1 2 3 4

If my arthritis takes a turn for the worse, it is because I have not been taking proper care of myself 0 1 2 3 4

In order for my arthritis to improve, it is up to other people to see that the right things happen 0 1 2 3 4

Even when I take care of myself, things outside of anyone's control can make my arthritis get worse 0 1 2 3 4

If my arthritis worsens, it's a matter of fate 0 1 2 3 4

If I take the right actions, my arthritis should improve or at least not get any worse 0 1 2 3 4

Regarding my arthritis, I should only do what my doctor tells me to do 0 1 2 3 4

I deserve the credit when my arthritis improves and the blame when it gets worse 0 1 2 3 4

Following doctor's orders to the letter is the best way to keep my arthritis from getting any worse 0 1 2 3 4

If I am lucky, my arthritis will get better 0 1 2 3 4

I'm the one with the responsibility for what happens with my arthritis 0 1 2 3 4

The type of help I receive from other people determines how soon my arthritis improves 0 1 2 3 4

As to my arthritis, what will be will be 0 1 2 3 4
Finally, please answer the following general questions by circling or ticking as appropriate.

I am Male / Female  

I am European / Maori / Other (Please state ___________________)  

The level of formal education I reached was:

Primary School  
Secondary School  
Tertiary  

The age group I am in is:

16-25 years  
26-35 years  
36-45 years  
46-55 years  
56-65 years  
more than 65 years  

I am currently on medication for some aspect of my arthritis  

Yes / No  

The approximate date that a doctor diagnosed me as definitely having Rheumatoid Arthritis was:

THANK YOU!
APPENDIX II

Doctor’s Questionnaire
(a) Has this patient been formally diagnosed as having RA? Yes/No

(b) If so when was that diagnosis made?

(a) Have you noted any fluctuations in this patient's RA condition? Yes/No

If yes, please indicate by ticking the appropriate box.

- Frequently
- Occasionally

(b) Have you noted any periods of remission in this patient's RA condition? Yes/No

If yes, please indicate by ticking the appropriate box.

- measured in months
- measured in years
- of variable durations

I have provided the above information upon the request and authority of my patient. I understand that it will be kept confidential at all times and that my patient will not be identifiable in any material resulting from the study.

Signed: ____________________________

I would like to receive a copy of the summary of findings that will be sent to participants. Yes/No
APPENDIX III

Results of Discriminant Analyses
Grouping Variable: Symptom changes over last 2 years (N=71)

<table>
<thead>
<tr>
<th>Canonical Discriminant Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Function</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Standardised Discriminant Function Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knowledge</td>
</tr>
<tr>
<td>Knowledge</td>
</tr>
<tr>
<td>Appraisal</td>
</tr>
<tr>
<td>Expectations</td>
</tr>
<tr>
<td>Thinking Style</td>
</tr>
<tr>
<td>Internal Control</td>
</tr>
<tr>
<td>Optimism</td>
</tr>
<tr>
<td>Pessimism</td>
</tr>
<tr>
<td>Chance Control</td>
</tr>
<tr>
<td>External Control</td>
</tr>
<tr>
<td>Negative Affect</td>
</tr>
<tr>
<td>Psychological Distress</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Structure Matrix</th>
</tr>
</thead>
<tbody>
<tr>
<td>Function 1</td>
</tr>
<tr>
<td>Appraisal</td>
</tr>
<tr>
<td>Internal Control</td>
</tr>
<tr>
<td>Thinking Style</td>
</tr>
<tr>
<td>Chance Control</td>
</tr>
<tr>
<td>Pessimism</td>
</tr>
<tr>
<td>Knowledge</td>
</tr>
<tr>
<td>External Control</td>
</tr>
<tr>
<td>Psychological Distress</td>
</tr>
<tr>
<td>Expectations</td>
</tr>
<tr>
<td>Optimism</td>
</tr>
<tr>
<td>Negative Affect</td>
</tr>
</tbody>
</table>
### Grouping Variable: Fluctuations over the last 2 years (N=71)

#### Canonical Discriminant Functions

<table>
<thead>
<tr>
<th>Function</th>
<th>% of variance</th>
<th>Canonical Correlation</th>
<th>After Function</th>
<th>Chi-Square</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100.00</td>
<td>.3841</td>
<td>0</td>
<td>9.814</td>
<td>.8313</td>
</tr>
</tbody>
</table>

#### Standardised Discriminant Function Coefficients

<table>
<thead>
<tr>
<th>Function 1</th>
<th>Knowledge</th>
<th>.06221</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Appraisal</td>
<td>-.43913</td>
</tr>
<tr>
<td></td>
<td>Expectations</td>
<td>.35731</td>
</tr>
<tr>
<td></td>
<td>Thinking Style</td>
<td>-.20101</td>
</tr>
<tr>
<td></td>
<td>Internal Control</td>
<td>-.08278</td>
</tr>
<tr>
<td></td>
<td>Optimism</td>
<td>.20984</td>
</tr>
<tr>
<td></td>
<td>Pessimism</td>
<td>-.11863</td>
</tr>
<tr>
<td></td>
<td>Chance Control</td>
<td>-.34806</td>
</tr>
<tr>
<td></td>
<td>External Control</td>
<td>.18216</td>
</tr>
<tr>
<td></td>
<td>Negative Affect</td>
<td>.71963</td>
</tr>
<tr>
<td></td>
<td>Psychological Distress</td>
<td>-.77848</td>
</tr>
</tbody>
</table>

#### Structure Matrix

<table>
<thead>
<tr>
<th>Function 1</th>
<th>Psychological Distress</th>
<th>-.52191</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Appraisal</td>
<td>-.38605</td>
</tr>
<tr>
<td></td>
<td>Optimism</td>
<td>.36390</td>
</tr>
<tr>
<td></td>
<td>Chance Control</td>
<td>-.32547</td>
</tr>
<tr>
<td></td>
<td>Thinking Style</td>
<td>-.27220</td>
</tr>
<tr>
<td></td>
<td>Pessimism</td>
<td>-.22587</td>
</tr>
<tr>
<td></td>
<td>Knowledge</td>
<td>.20417</td>
</tr>
<tr>
<td></td>
<td>Expectations</td>
<td>-.14661</td>
</tr>
<tr>
<td></td>
<td>External Control</td>
<td>.07315</td>
</tr>
<tr>
<td></td>
<td>Internal Control</td>
<td>.05918</td>
</tr>
<tr>
<td></td>
<td>Negative Affect</td>
<td>.03718</td>
</tr>
</tbody>
</table>
Grouping Variable: Speed of change (N=68)

### Canonical Discriminant Functions

<table>
<thead>
<tr>
<th>Function</th>
<th>% of variance</th>
<th>Canonical Correlation</th>
<th>After Function</th>
<th>Chi-Square</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>80.75</td>
<td>.5853</td>
<td>0</td>
<td>31.119</td>
<td>.4096</td>
</tr>
<tr>
<td>2</td>
<td>19.25</td>
<td></td>
<td>1</td>
<td>6.792</td>
<td>.9424</td>
</tr>
</tbody>
</table>

### Standardised Discriminant Function Coefficients

<table>
<thead>
<tr>
<th></th>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knowledge</td>
<td>-.15846</td>
<td>.25952</td>
</tr>
<tr>
<td>Appraisal</td>
<td>.58230</td>
<td>-.29586</td>
</tr>
<tr>
<td>Expectations</td>
<td>.50644</td>
<td>-.41726</td>
</tr>
<tr>
<td>Thinking Style</td>
<td>.10001</td>
<td>.03830</td>
</tr>
<tr>
<td>Internal Control</td>
<td>.35152</td>
<td>.13351</td>
</tr>
<tr>
<td>Optimism</td>
<td>.71952</td>
<td>.23492</td>
</tr>
<tr>
<td>Pessimism</td>
<td>.42155</td>
<td>.12717</td>
</tr>
<tr>
<td>Chance Control</td>
<td>.31969</td>
<td>.75947</td>
</tr>
<tr>
<td>External Control</td>
<td>.74824</td>
<td>-.48097</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.10035</td>
<td>.72037</td>
</tr>
<tr>
<td>Psychological Distress</td>
<td>-.75497</td>
<td>-.06223</td>
</tr>
</tbody>
</table>

### Structure Matrix

<table>
<thead>
<tr>
<th></th>
<th>Function 1</th>
<th>Function 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Expectations</td>
<td>.35585</td>
<td>-.32180</td>
</tr>
<tr>
<td>Optimism</td>
<td>.35314</td>
<td>-.01137</td>
</tr>
<tr>
<td>Pessimism</td>
<td>.23536</td>
<td>.17458</td>
</tr>
<tr>
<td>Appraisal</td>
<td>.17960</td>
<td>-.00256</td>
</tr>
<tr>
<td>Internal Control</td>
<td>.13840</td>
<td>.13037</td>
</tr>
<tr>
<td>Knowledge</td>
<td>.06206</td>
<td>.03815</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.04807</td>
<td>.42508</td>
</tr>
<tr>
<td>Chance Control</td>
<td>.20233</td>
<td>.31759</td>
</tr>
<tr>
<td>Psychological Distress</td>
<td>-.20648</td>
<td>.29671</td>
</tr>
<tr>
<td>External Control</td>
<td>-.12199</td>
<td>-.15135</td>
</tr>
<tr>
<td>Thinking Style</td>
<td>.03958</td>
<td>.11178</td>
</tr>
</tbody>
</table>
### Grouping Variable: Remissions (N=68)

#### Canonical Discriminant Functions

<table>
<thead>
<tr>
<th>Function</th>
<th>% of variance</th>
<th>Canonical Correlation</th>
<th>After Function</th>
<th>Chi-Square</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100.00</td>
<td>.4931</td>
<td>:</td>
<td>16.295</td>
<td>.3627</td>
</tr>
</tbody>
</table>

#### Standardised Discriminant Function Coefficients

<table>
<thead>
<tr>
<th>Function 1</th>
<th>Knowledge</th>
<th>Appraisal</th>
<th>Expectations</th>
<th>Thinking Style</th>
<th>Internal Control</th>
<th>Optimism</th>
<th>Pessimism</th>
<th>Chance Control</th>
<th>External Control</th>
<th>Negative Affect</th>
<th>Psychological Distress</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-.04006</td>
<td>.37205</td>
<td>.20269</td>
<td>-.02411</td>
<td>-.00686</td>
<td>.57600</td>
<td>-.63139</td>
<td>.28822</td>
<td>.05044</td>
<td>.29369</td>
<td>.04038</td>
</tr>
</tbody>
</table>

#### Structure Matrix

<table>
<thead>
<tr>
<th>Function 1</th>
<th>Optimism</th>
<th>Pessimism</th>
<th>Appraisal</th>
<th>Expectations</th>
<th>External Control</th>
<th>Chance Control</th>
<th>Negative Affect</th>
<th>Internal Control</th>
<th>Psychological Distress</th>
<th>Thinking Style</th>
<th>Knowledge</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>.49453</td>
<td>-.37491</td>
<td>.34676</td>
<td>.26828</td>
<td>.20703</td>
<td>.18861</td>
<td>.11393</td>
<td>-.10194</td>
<td>.07178</td>
<td>-.05936</td>
<td>.03368</td>
</tr>
</tbody>
</table>
Grouping Variable: Fluctuations over life of disease (N=68)

### Canonical Discriminant Functions

<table>
<thead>
<tr>
<th>Function</th>
<th>% of variance</th>
<th>Canonical Correlation</th>
<th>After Function</th>
<th>Chi-Square</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100.00</td>
<td>.4753</td>
<td>0</td>
<td>14.982</td>
<td>.4527</td>
</tr>
</tbody>
</table>

### Standardised Discriminant Function Coefficients

<table>
<thead>
<tr>
<th>Factors</th>
<th>Function 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knowledge</td>
<td>.03571</td>
</tr>
<tr>
<td>Appraisal</td>
<td>.59403</td>
</tr>
<tr>
<td>Expectations</td>
<td>.44616</td>
</tr>
<tr>
<td>Thinking Style</td>
<td>-.45007</td>
</tr>
<tr>
<td>Internal Control</td>
<td>.34789</td>
</tr>
<tr>
<td>Optimism</td>
<td>.22322</td>
</tr>
<tr>
<td>Pessimism</td>
<td>.03653</td>
</tr>
<tr>
<td>Chance Control</td>
<td>-.26319</td>
</tr>
<tr>
<td>External Control</td>
<td>-.38303</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.05554</td>
</tr>
<tr>
<td>Psychological Distress</td>
<td>.13400</td>
</tr>
</tbody>
</table>

### Structure Matrix

<table>
<thead>
<tr>
<th>Factors</th>
<th>Function 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appraisal</td>
<td>.40056</td>
</tr>
<tr>
<td>Thinking Style</td>
<td>-.31937</td>
</tr>
<tr>
<td>Psychological Distress</td>
<td>.30600</td>
</tr>
<tr>
<td>Chance Control</td>
<td>-.21325</td>
</tr>
<tr>
<td>External Control</td>
<td>-.19312</td>
</tr>
<tr>
<td>Optimism</td>
<td>.18842</td>
</tr>
<tr>
<td>Internal Control</td>
<td>.12795</td>
</tr>
<tr>
<td>Expectations</td>
<td>.11466</td>
</tr>
<tr>
<td>Negative Affect</td>
<td>.11403</td>
</tr>
<tr>
<td>Pessimism</td>
<td>-.10471</td>
</tr>
<tr>
<td>Knowledge</td>
<td>-.01017</td>
</tr>
</tbody>
</table>