The prediction of outcome of an acute episode of low back pain using the Fear Avoidance Model of Exaggerated Pain Perception.

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

Michael John Rose

April, 1994

To Ronald Edward Rose

ABSTRACT

Three hundred acute low back pain patients were the focus of a twelve months longitudinal study designed to test the validity of the Fear Avoidance Model of Exaggerated Pain Perception as an explanatory model of chronic low back pain.

The validity of the Fear Avoidance Model is supported by the results of the study. In addition, other physical and psychological variables were shown to be associated with outcome. This is interpreted as representing the complexity of low back pain experience. However, the variables which were most strongly associated with chronicity support the fear avoidance construct and a reformulated Fear Avoidance Model is presented.

The results also demonstrated that subjects with low back pain do not, in general, present with physical signs or significant levels of distress. In addition, in terms of pain complaint and disability, subjects who became chronic low back pain patients were significantly different from recovered subjects by two months after onset.

Limitations of the study were discussed and recommendations for future research were made. Finally, the implications of the results for the clinical management of low back pain patients were discussed.

ACKNOWLEDGEMENTS

I would like to thank my supervisor, Professor Peter Slade, for his support and guidance throughout the time that I have known him. I am especially grateful for the contribution he has made to my personal development and for the confidence that he has shown in me at all times.

I am also grateful to Mr Michael Dewey for the skill and patience he has demonstrated whilst advising me about data analysis and statistical methods.

I would like to thank my colleague Dr James Reilly for his support and comradeship during, what often seemed at the time, dark days for both of us.

I am indebted to Ms Lesley Atchison and Mrs Carol Foreman for their help with data collection and to Mr Bruce Pennie who has taught me much about the physical aspects of low back pain.

My heartfelt thanks go to Anita, my long suffering wife, who has encouraged me consistently and protected me from our four year old daughter, Nancy, whose attempts to help me write this thesis could have ended in disaster.

I consider myself most fortunate to have had the opportunity to spend four years as a member of the Department of Clinical

Psychology at the University of Liverpool. I have learned much from my colleagues and I am grateful for their help and encouragement. I am especially indebted to Ms Sue Kaney whose help at the early stage was invaluable.

I would also like to thank the doctors who referred patients to us and their staff who were so hospitable, often when our presence was most inconvenient.

The work upon which this thesis is based was funded by the Arthritis and Rheumatism Council.

Finally, I thank our patients for answering so many questions and showing such interest in our work.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Back pain is extremely common. During any one year, seven percent of 45 to 65 year old people experience low back pain of sufficient intensity to necessitate a visit to their general practitioner (G.P.) (Royal College of General Practitioners [morbidity statistics], 1972). This accounts for 2.2 million G.P. consultations every year, about 4% of all consultations. Various studies have shown that between 9.3% and 17.5 % of low back pain patients are referred by their GP for specialist advice (Dillane, 1966, Glass, 1979, Wood, 1983). Of those treated in hospital, 20% are not relieved of their pain (Fry et al,1986). The Office of Health Economics estimated that in 1982 back pain cost the Health Service £156 million and in the last decade, time off work due to low back pain has increased by 40% in comparison to 5.6% for all other complaints (DHSS, 1989). These figures have led Waddell (1987) to describe low back pain as an epidemic.

The pathology of the disorders that give rise to acute low back symptoms is obscure and we are still largely unable to identify the precise structures which cause pain. However, degenerative changes of the synovial and vertebral joints of the spine are generally held to be either responsible for making spinal structures vulnerable to injury or develop in response to injury (Farfan, 1972). Melin (1990) suggests that the capsular and ligamentous inelasticity which are associated

with degenerative changes may be the cause of reduced back mobility in those with low back pain. This inelasticity may increase the biomechanical loading on spinal structures which in turn may lead to an increase in degenerative changes in the structures of the spinal segment. Pathological changes of the structures in and around the spinal column may result in the stimulation of nerve endings in the area, either chemically or mechanically. Stimulation of these nerve endings results in the transmission of neural impulses along the axons of neurons, the bodies of which are located in the central nervous system. Grieve (1988) states that following initial injury of a structure it may never regain its original strength, thus becoming more vulnerable to reinjury. This hypothesis is supported by Troup et al (1987) who found that one of the best predictors of a future episode of acute low back pain is a previous history of low back pain. For a review of the pathological changes associated with low back pain see Grieve (1988).

Although, the large majority of episodes of acute low back pain resolve in about two months after onset of pain, 10% of individuals fail to recover and become chronic low back pain sufferers (Morrell and Wale, 1976, Scamler, 1981, Roland and Morris, 1982, Philips and Grant, 1991).

The experiences of acute and chronic low back pain differ in several ways. Acute low back pain, which may result from injury

of spinal structures, enforces inactivity and rest, which are often necessary to ensure recovery. Acute low back pain elicits an increased level of activity in the sympathetic nervous system with increases in cardiac output, respiration, and all other physiological responses associated with the 'flight or fight' reaction. These physiological responses occur roughly in proportion to the intensity of the noxious stimulus which results in the perception of pain. Acute pain varies in intensity from moment to moment, fluctuating until it goes completely as tissue healing occurs. Acute pain is linked with anxiety (Pilowsky, 1967). It requires a minimal change in social and occupational behaviour and has few consequences once the pathological changes leading to the perception of pain have ceased.

Chronic pain is usually defined as severe persistent pain which lasts longer than 6 months. Chronic pain patients complain of almost constant and unvarying pain in contrast to those suffering from acute pain. Chronic pain is usually destructive physically, socially and psychologically. It is characterised by depression, irritability, and bitterness. Sufferers are more preoccupied with their pain than are acute sufferers (Sternbach, 1974), engage in more intense, relief seeking behaviours and avoid family and social interactions to a greater extent than acute sufferers (Bond, 1979). Chronic pain patients demonstrate a pattern of vegetative signs including sleep, appetite and libido disturbances with

irritability and withdrawal of interests (Slade, 1984). Chronic pain patients also demonstrate common behavioural patterns. They can manifest a very high self reported level of pain over a long period of time, excessive use of analgesia with little benefit, high incidence of invasive therapy with worsening of symptoms afterwards and a withdrawal from all family, social and occupational responsibilities (Slade, 1984). The affective and behavioural symptoms associated with pain that has lasted for longer than the normal course of a disease are so consistent between individuals that the term 'chronic pain syndrome' has arisen.

In some cases the presence of chronic low back pain can be explained in pathological terms when the pain may result from an active disease state such as neoplasia or rheumatoid arthritis. However, in many instances the relationship between tissue damage and chronic pain complaint fails to hold up. Waddell (1987) contrasts the patient with serious spinal pathology and imminent paraplegia who has surprisingly little pain, distress or disability with the total 'cripple' in agony from a simple backache with very little objective physical abnormality. This inconsistency between the organic, nociceptive component of low back pain and the behavioural and affective components has been labelled by Lethem et al (1983) as 'desynchrony'.

At present there is no satisfactory or accepted method for assessing the severity of low back disorders. Traditionally assessment of severity is based on diagnosis. However, in the field of low back pain the word 'diagnosis' tends to lose its classical meaning, "in that on the one hand a frequently occurring and easily recognisable pattern of signs and symptoms may enjoy a different diagnosis for each day of the week depending upon the person examining the patient" (Grieve 1988). The Quebec Task Force on Spinal Disorders (Larocca, 1987), in a review of over 7000 publications concerning spinal pain, identified some 20 diagnostic terms. In practice it is common to find variation in severity between individual patients with identical diagnoses. Moreover, in most patients with low back pain, it is impossible to reach any definitive diagnosis while comparison of radiographs in patients with low back pain and asymptomatic normal people shows that clinical severity is not related to radiological degeneration. Eight controlled studies and two reviews have shown little relationship between symptoms and radiological changes of degeneration (Waddell,1987). It is important to recognise that symptoms and signs provide both information about physical disease but also a lot of information about the patient's emotional reaction and illness behaviour (Waddell, 1987). In addition, suffering is not the same as pain and is impossible to define or measure clinically. Therefore, it is an important principle to accept that only the sufferer can assess the severity of pain, accepting always the qualification that such a purely subjective assessment is open

to psychological or conscious bias. The patient's report may include physical sensation, distress, pain effects, pain expression and communication and in chronic low back pain, functional restriction due to pain may be more important than any anatomical or structural impairment (Waddell, 1993).

There exists a considerable literature concerning low back pain. The Quebec Task Force on Spinal Disorders (Larocca, 1097) identified more than 7000 articles related to spinal disorders published over the previous 10 years. Much of the work represented by the literature is concerned with explaining chronic low back pain either in psychological or physical terms. Opposing views have been expressed regarding the importance of physical and psychological variables in the natural history of acute low back pain and the management of chronic low back pain. Grieve (1988) states that "the fact that medical and surgical findings are negative, or are insufficient to explain the pain on an organic basis, does not justify a diagnosis of psychogenic pain. Such a diagnosis requires positive psychiatric findings" and "it is remarkable how frequently a patient, relieved by skilled treatment will seem to have shed their psychogenic aura and become 'reasonable' human beings again". From a different perspective Waddell (1987) writes that "there is no definite evidence that any treatment for low back pain is much better than a combination of natural history and placebo effect" and "physical treatment directed to a supposed but unidentified and possibly non-

existent nociceptive source may cause additional physical damage".

However, it is recognised that pain is a multi-faceted and complex phenomenon and can no longer be viewed in purely physiological terms (see Melzack and Wall, 1991). The gate control theory of pain (Melzack and Wall, 1965) provides a physiological basis for a biopsychosocial model of pain and explains how psychological and social influences may modulate individual perception and response to pain.

More recently, the Fear Avoidance Model of Exaggerated Pain Perception (Lethem et al, 1983) was developed in order explain the observation that a minority of acute, benign, low back pain sufferers fail to recover and go on to experience chronic low back pain syndrome. The model integrates physiological and psychological constructs and proposes that 'fear' of acute low back pain results in avoidance behaviour which in turn leads to a reduction in physical fitness, strength, spinal mobility and ultimately to chronic low back pain.

The central purpose of this study was to test the validity of the Fear Avoidance Model of Exaggerated Pain Perception as a theoretical model of chronic low back pain. The introductory chapter of this thesis describes the model, provides a critical analysis of its central constructs and describes its current status in the context of current research. In addition, a

description of other models of chronic low back pain are briefly presented. Chapter 2 articulates the aims, general objectives and specific research hypotheses of the study. Chapter 3 describes the longitudinal study of 300 acute low back pain patients designed, primarily, to test the Fear Avoidance Model and Chapter 4 is concerned with a study of the repeatability of the physical measures described in Chapter 2. Chapters 5 and 6 are concerned with the characteristics and natural history of the sample which, to the author's knowledge, represents the largest sample of acute low back pain patients presenting to their general practitioners to date. Chapter 7 contains the presentation and discussion of a series of factor analyses of the data which were carried out in order to explore the relationship between psychological and physical variables in patients presenting with acute low back pain and to derive a simplified set of variables for detailed analysis to be presented in the next chapter. Chapter 8, which forms the focus of the study, reports and discusses the results of statistical analyses designed to test the Fear Avoidance Model in terms of prediction of outcome of acute low back pain. In addition, statistical comparisons are also made between the Fear Avoidance Model and the other models of chronic low back described in earlier chapters. The ninth and final chapter contains a general discussion and reformulation of the Fear Avoidance Model at a conceptual level. This final chapter ends with a list of the conclusions of the thesis.

CHAPTER 1

THE FEAR AVOIDANCE MODEL OF EXAGGERATED PAIN PERCEPTION

1.1 INTRODUCTION

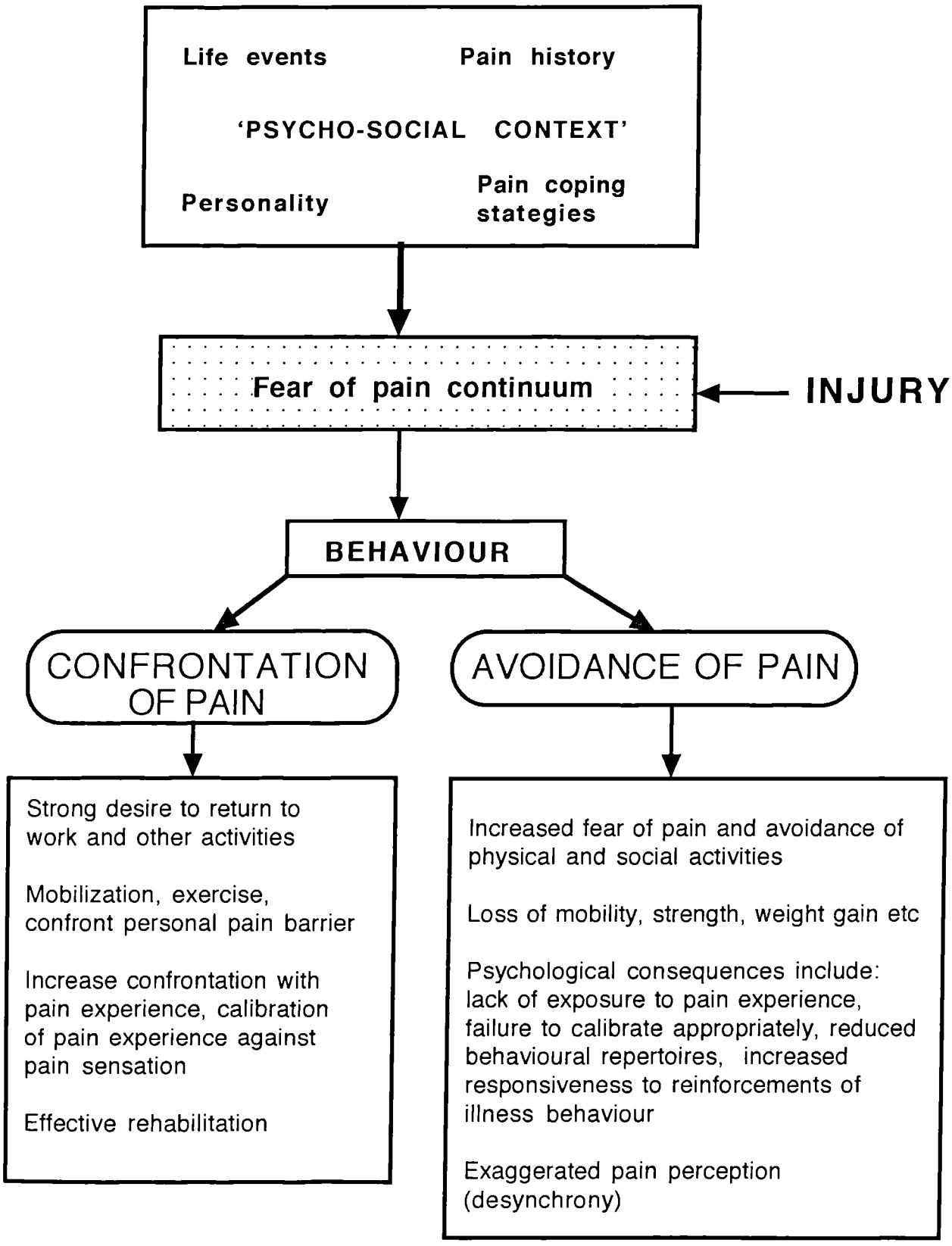
The Fear Avoidance Model of Exaggerated Pain Perception (Lethem et al, 1983 and Slade et al, 1983) was developed in order to explain the inconsistency between the observable organic components and behavioural components of chronic low back pain described in the preface. The Model emerged from the empirical observation of chronic low back pain patients by a multi-disciplinary team and represents the first theoretical model which integrates physiological and psychosocial variables in order to explain the development of benign chronic low back pain. This chapter is concerned with the description of the Fear Avoidance Model and its central constructs, the current status of the Model in terms of current research and a description of alternative models of benign chronic low back pain.

1.2 EXAGGERATED PAIN PERCEPTION

The Fear Avoidance Model (Figure 1.1) is concerned with the development of chronic low back pain syndrome following an episode of acute low back pain which results from injury or degenerative change.

Fig 1.1

The Fear Avoidance Model of Exaggerated Pain Perception (Lethem, et al. 1983)



Lethem et al (1983) employed the term 'exaggerated pain perception' in order to describe the inconsistency between pathological signs and current knowledge of biology and anatomy and the natural history, reported symptoms and functional disability of chronic low back pain patients. At the acute stage of low back pain, the term 'exaggerated pain perception' is not concerned with the physiological process of nociception, but is associated with 'abnormal illness behaviour'.

Illness behaviour has been defined by Mechanic (1962) as "the ways in which given symptoms may be differentially perceived, evaluated, and acted (or not acted) upon by different kinds of persons. It combines a clinical analysis of hypochondriasis with a psychosocial analysis of how and why the individual reacts to illness in the context of his environment. Normal illness behaviour may be defined as illness behaviour which corresponds to medicine's current knowledge regarding physical pathology and is congruent with the sick role offered to the patient by society. Pilowsky (1969) has defined abnormal illness behaviour as "the persistence of an inappropriate or maladaptive (i.e. ineffective to restore health and/or markedly deviated from normal) mode of experiencing, perceiving, evaluating, and acting in relation to one's own state of health. This is despite accurate reassurance after thorough examination and assessment."

The anatomical and time patterns of low back pain, its characteristics and the way in which patients present their symptoms usually fit in with common and recognisable disease patterns. However some patients give descriptions of signs and symptoms which do not fit in with these patterns. Physical signs and symptoms which appeared to have a non-organic basis were described early in this century following the introduction of the Compensation Act and were used to identify "malingerers" (McKendrick, 1912). Waddell et al (1979) standardized and reinterpreted a group of these non organic physical signs and symptoms and used them in an examination of a group of back pain patients. The study showed that 'non-organic signs and symptoms' were separable from, and did not correlate with, physical findings of organic pathology. Waddell et al (1984) found that low back pain patients received significantly more treatment if they demonstrated large amounts of illness behaviour. Treatment was in fact more influenced by illness behaviour than by the actual disease.

The Fear Avoidance Model proposes that exaggerated pain perception (or abnormal illness behaviour) at the acute stage of low back pain is mediated by fear of pain. Fear leads to avoidance of painful experience which in turn results in further painful degeneration of spinal structures (see below).

1.3 FEAR-AVOIDANCE

The study of fear is based on a powerful body of animal experimental work (Denny 1991) which for more than 40 years has emphasized classical conditioning and the learned nature of fear and avoidance behaviour. It has been suggested that classical conditioning of pain and muscle tension may occur when pain is acute, leading to chronic pain caused by sustained muscle hypertension (Gentry and Bernal, 1977). Avoidance of movement in an attempt to prevent further pain may lead to further muscle hypertension and more pain. In addition, avoidance of activity may lead to muscle weakness which, in the context of back trouble, may lead to further injury and disability. Central to the respondent model of chronic pain is the belief that chronic pain and raised EMG levels in associated muscle groups are related. This hypothesis has been supported by some workers (Grobel, 1973, Hoyt, 1982) but refuted by others (Basmajian, 1978).

Pain is one of the most powerful aversive drives in animals and humans and is closely allied to fear. As with any type of fear, there are two extremes of coping response available to the individual, namely confrontation or avoidance. The former type of response typically leads to a reduction (or total abolition) of fear with time, while the latter type of response usually leads to maintenance and exacerbation of fear, the end stage being a full blown phobic state. An individual who shows

the adaptive response of confrontation is likely to view low back pain as a temporary nuisance, is strongly motivated to return to work, social and leisure activities and is prepared to confront his personal pain barrier as the organic basis for pain resolves (Lethem et al, 1983). By contrast, the non-adaptive pain avoider is considered to be motivated to avoid any fresh exposure to pain and to minimize or avoid physical activity completely. Such extreme behavioural avoidance in turn leads to a number of physical and psychological consequences. It is this area which is of particular interest.

The physical consequences can be summarised as follows: Reduced mobility may accelerate the onset of osteoarthrosis in those who are susceptible (Troup, 1988). When a joint is immobilized the capsule shrinks, the resting tension in the capsular ligament increases and the static compressive loading across the joint is enhanced. If a joint is immobilized at, or near, one extreme of its range of motion, the tension of articular ligaments is unilaterally increased and joint compression increases markedly. Such conditions lead to bone remodelling and soft tissue hypertrophy. A consequence of these changes is a reduction in pain free range of movement of the spinal joints affected. Protracted rest leads to a catabolic state with general malaise (Bortz, 1954). There is demineralization of bone (Sandstrom, 1985) and 3% loss of muscle strength per day (Spengler). There is decreased physical fitness (Mayer, 1985, Mayer, 1986)) and rest and inactivity may inhibit healing

(Bortz, 1984). There is evidence that increased activity not only promotes bone and muscle strength and improves disc and cartilage nutrition but may also increase systemic endorphin levels and reduce sensitivity to pain (Nachemson, 1983). Another side effect of physical inactivity is a decline in cardio-respiratory fitness. The associated reduction in mitochondria content in spinal musculature will lead to reduced exercise tolerance and earlier experience of muscle pain than in normal individuals. If this pain experience leads to further rest then the process of rest-pain-rest will continue (Troup, 1989). The final outcome of such an extreme non-adaptive response is likely to be an increase in organic pain perception and a concommitant increase in fear and avoidance. This can be viewed as 'physiological exaggerated pain perception'.

The psychological consequences of avoiding physical activity include limitation of exposure to a full range of painful stimulation with fewer opportunities for calibrating pain sensation against pain experience and a reduced behavioural repertoire. Thus invalid status becomes preeminent and there is an increased likelihood of response to various positive and negative reinforcers of invalidity. More generally, prolonged rest and inactivity leads to increased psychological distress and depression, loss of the work habit, increased difficultly in starting rehabilitation, progressive loss of job opportunity and decreasing probability of ever returning to work (Waddell, 1987).

Avoidance and its consequences in relation to pain has been addressed within the context of a behavioural framework. A behavioural model of chronic pain has been proposed by Fordyce (1978). This model has two essential conceptual features. One is that behaviour has significance in its own right and is not an extension of some underlying causative factor. The second feature is the emphasis placed on measurement and observation of the behaviour. When an illness (and the associated symptom complex) lasts for long intervals there is increased opportunity for operant conditioning effects to exert influence. Some parts of the set of symptoms involve behaviour. In the case of acute low back pain this behaviour may include limping, grimacing, moaning and the verbalisation of somatic distress. These behaviours are subject to influence by conditioning effects. Low back pain provides two of the three essentials for conditioning; namely that symptom behaviours occur and they occur over time. The third essential for conditioning, favourable circumstances, may or may not exist in the patient's environment. Fordyce (1978) emphasises that pain can be either respondent or operant in nature depending upon whether it is controlled by antecedents (nociception) or contingencies in the environment. Fordyce's initial emphasis was on positive reinforcement of pain behaviour. Subsequently, however, he considered 'avoidance-learning', ie, reduction of pain by avoidance behaviour resulting in negative reinforcement (Fordyce, 1982). Although, the operant model has made a significant contribution to the understanding and

treatment of chronic pain (for a review see Keefe and Williams, 1989) Fordyce has been widely criticised for "his somewhat cavalier attitude to pain experience" (Humphrey, 1989). The operant model does not account for subjective aspects of pain experience and the issue of whether the patients suffer less as a result of behavioural therapy based on the operant model is dismissed. In addition, the role of physiological variables as mediators of chronic pain and disability are not addressed.

The Fear Avoidance Model of Exaggerated Pain Perception is the first theoretical model which integrates several psychosocial and physiological constructs in order to explain chronic low back pain. Lethem et al (1983) propose that *extreme fear of* pain and consequent *avoidance and extreme confrontation of pain* lie at opposite ends of a continuum. They propose that an individual's position along this continuum is determined by the 'psychosocial context' in which their initial low back injury first occurs.

1.4 THE PSYCHOSOCIAL CONTEXT

In the context of the Fear Avoidance Model the concepts of fear of pain and anxiety in general are closely related. The 'psychosocial context' proposed by Lethem et al represents key elements of anxiety theory described in the literature. Speilberger (1972) states that "there is nothing to be gained in the conceptual distinction between anxiety and fear unless

the pattern of response in fear reactions differs from the response pattern in anxiety reaction". Several researchers have chosen to examine the relationship between anxiety and pain. Studies of the effects of induced anxiety on responses to acute laboratory pain suggest that anxiety related to pain increases ratings of perceived pain intensity (Weisenberg et al, 1984, Al Absi and Rokke, 1991). Results of Arntz et al (1991) suggest that attention to pain increases the impact of pain and that anxiety about pain directs attention to pain. Anxiety is a complex emotional reaction which cannot be characterized as a unitary phenomenon (Hugdahl, 1981). Lang (1968) identifies three sources of data from which anxiety may be inferred: physiological, behavioural and verbal indices. There is evidence that these indices do not correlate highly with one another (Rachman and Hodgson, 1974) and in the general anxiety literature failure to distinguish between these three aspects has led to confusion (Mathews and MacLeod, 1987). Fear of pain has been described in similar terms by McCracken et al (1992 who stated that "fear of pain can be conceptualized as a multidimensional response which may contribute significantly to the chronic pain experience. Fear is best construed as a set of loosely coupled components. The three most important components of fear are avoidance behaviour, physiological reactivity and cognitive reports of subjective fear". Lang (1971) and Rachman (1974) view the three components of fear as partially dependent but not three different ways of exposing the same phenomenon.

Lethem et al (1983) explain the psychosocial context in terms of four psychosocial constructs: previous stressful life events, memory for previous acute pain experience, behavioural coping strategies in response to acute pain generally and 'personality'. The interaction of these constructs determine where an individual is on the 'fear of pain continuum' at the time of spinal injury which in turn determines whether exaggerated pain perception results in avoidance or whether recovery is consistent with our knowledge of biology and anatomy.

i) Stressful life events. The notion of socially induced stress as a precipitating factor in chronic diseases is has gained acceptance among a wide spectrum of scientists. Dodge and Martin (1970) stated that "the chronic diseases are aetiologically linked with excessive stress and in turn this stress is a product of specific socially structured situations inherent in the organisation of technological societies". Even susceptibility to microbial infections is thought to be, in part, a function of environmental stress which leads to physiological stress on the individual. Stress is a broad and general concept describing the organism's reaction to environmental demands. In this context, the term stressful life events refers to any set of circumstances the advent of which signifies or requires change in the individual's ongoing life pattern (Holmes and Rahe, 1967). The adaptational approach in psychodynamic theory emphasizes the coping and defensive

strategies employed by the individual in adjusting to environmental demands. The Legitimization Motivation Theory (Meyers and Lyon, 1979) suggests that when an individual with personality problems is confronted with stressful life events, this may result in a disability which is unacceptable to the individual and society. However, if such individuals have an accident or become physically ill, especially if they are in pain, their inability to cope may become socially and personally acceptable.

Evidence for the potential of stressful life events to mediate fear and avoidance in the context of back pain can be found in the general anxiety literature. Given appropriate circumstances or life events (Finlay-Jones and Brown, 1981) high trait anxiety individuals may become sufficiently disturbed to meet the diagnostic criteria for anxiety states often characterised by avoidance behaviour. McKeon et al (1984) found that obsessive-compulsive patients rated as having highly anxious personality prior to the disorder had experienced fewer life events than did those with lower previous trait anxiety. These findings suggest that individuals with high trait anxiety who have experienced recent life events may, following an acute episode of low back pain become what Lethem et al describe as 'pain avoiders'.

Most investigators working in this field have adopted the Social Readjustment Rating Scale (SRRS) (Holmes and Rahe, 1967)

which consists of a 43 item checklist. The items (which are weighted) represent fairly common situations that require change in ongoing adjustment. Exposure to life stresses does not cause disease but may alter the individual's susceptibility and serve as a precipitating factor. In general, the purpose of life events research has been to demonstrate a temporal association between the onset of illness and a recent increase in the number of events that require socially adaptive responses on the part of the individual. Rose (1975) demonstrated that patients who presented to their General Practitioner with an acute attack of low back pain had experienced a higher incidence of stressful life events in the previous three months than pain free controls.

Lethem et al (1983) propose that the experience of recent stressful life events serve to undermine the personal coping strategies of acute back pain sufferers so that avoidance behaviour and consequent chronicity is more likely than confrontation and recovery. This view has been supported by the results of studies which demonstrate a relationship between stressful life events, depression and chronic low back pain. Feuerstein et al (1985) compared 35 chronic low back pain patients with 35 pain free controls across a range of variables concerning stress. They demonstrated that the mean score on the Social Readjustment Rating Scale of the chronic low back pain group was significantly higher than that of the pain free

controls. However, the results of this study need to be interpreted with caution given its cross-sectional design.

A positive relationship between depression and life events in the context of back pain has been demonstrated by Atkinson et al (1987) who compared a group of pain free, healthy controls with a depressed and non-depressed chronic low back pain patients. The depressed chronic low back pain patients reported significantly more recent adverse life events than non-depressed chronic back pain patients and controls ($p < 0.003$). Atkinson et al suggest that the depression experienced by the back pain group was secondary to the experience of stressful life events. The significance of this finding concerns the relationship between life events, *depression and* incongruent pain behaviour amongst chronic back pain patients. Waddell (1987) has demonstrated that depression and inappropriate illness behaviour are positively related. One interpretation of this data suggests that exaggerated pain perception at the chronic stage may be a consequence of depression, the level of which is, in part, determined by the number of previous stressful life events. This is supported by the results of Crauford et al (1990) who demonstrated that a group of acute back pain patients with an observable organic cause of their pain had experienced significantly fewer recent stressful life events than a group of patients with similar symptoms for whom no biological diagnosis could be made. However, a similar study by Leavitt et al (1979) failed to

demonstrate a significant difference in life events scores between three groups of low back pain patients categorised as having organic, probably organic and non-organic diagnoses.

The role of 'stress' generally in mediating back pain symptoms has been the focus of several studies. Wickstrom et al (1989) identified three 'personality types' in terms of competitiveness: Those who were identified as being competitive were described as having type A personalities. Those who fell into the moderately competitive category were described as having type AB personalities. Non-competitive individuals were described as having type B personalities. Type A subjects reported significantly more 'stress' in their lives than type B subjects. An increased incidence of back pain with leg pain was reported amongst the type A subjects and a significant association was seen amongst type A 'personality' and experience of radiating leg pain associated with nerve root involvement. Although the design of this study is cross-sectional, it is unlikely that back and leg pain could lead to the development of a stressed and competitive personality. The results suggest that competitive men are more likely to develop low back and leg pain. Explanations for this finding may be that i) the increased experience of stressful life events associated with type A personality may predispose the individual to illness, including low back pain or ii) the increased competitiveness associated with type A personality may lead the individual concerned to place himself in

potentially physiologically stressful situations, thereby becoming more likely to sustain a back injury. The results of a study by Gamsa and Vikis-Freibergs (1991) also demonstrate a relationship between occupational behaviour traditionally associated with competitiveness and back pain. They demonstrated that chronic pain patients scored more highly on measures of 'ergomania' (beginning work at an early age, frequent overtime work and infrequent holidays) than pain free controls. The authors of this study propose psychological and physiological explanations for their results. In psychodynamic terms, they propose that guilt and inner insecurity may lead to relentless activity and the development of a pain problem to fulfil the wish to be passive and cared for. In biological terms they suggest that consistent heavy work over a long period may lead to the development or aggravation of spinal problems.

The validity of the results of some studies which support the concept of a relationship between life events and illness has been challenged and several statistical and psychometric issues concerning life events research have been raised. The most immediate statistical issue concerns the size and practical significance of the correlation between number and nature of life events and subsequent illness episodes. Given the very large sample sizes characteristic of life events research, even very small correlations of no practical significance may pass tests of statistical significance (Rabkin

and Struening, 1976). In terms of the psychometric properties of the SRRS (the most widely used life events instrument) Rahe (1974) reports test-retest reliability correlations ranging from 0.26 to 0.90. Sarason et al have concluded that "the reliability of the SRRS is low". In terms of validity of measures of life stress, Brown (1974) has referred to the problem of "retrospective contamination" in which selective memory, denial of certain events and over reporting of events in order to justify illness are all potential sources of error. In prospective studies using the SRRS the subjective evaluation of the significance of a life event to the respondent is neglected.

However, research has demonstrated a relationship between the stressful life events construct and the onset and continuation of illness. In terms of low back pain a relationship has been shown between life events and stress and exaggerated pain perception, although, to the author's knowledge, life events have not previously been used as potential predictors of chronic low back pain in a longitudinal study. The SRRS, is, despite it psychometric shortcomings, the most commonly used instrument designed to measure stressful life events and is easily and quickly administered. It will therefore be used to measure the life events component of the psychosocial context in this study.

ii) **Personal pain history.** The behavioural component of anxiety (and fear) seems, at least partly, to be mediated by prior learning experience (Mathews and MacLeod, 1987). Lethem et al argue that fear of low back pain and consequent avoidance behaviour may be determined in large measure by the severity of previous episodes of acute somatic pain. Previous attacks of any type of acutely crippling pain may sensitize the individual to fear of pain and increase the probability of an avoidance response whenever more pain is threatened. Jones (1957) suggested that pain gets increasingly hard to cope with over time because later experiences elicit responses not only to current stimulation but also to the half buried memories of earlier pain.

The 'personal pain history' element of the Fear Avoidance Model is concerned with the relationship between previous pain experience and learned avoidance behaviour. However, retrospective measurement of severity of pain relies on memory for pain which is open to bias and distortion. The accuracy of memory for pain has been shown to be variable and in part dependent upon whether the pain is acute and novel or chronic. Tulving and Donaldson (1972) raised the distinction between episodic and semantic memory in order to conceptualise the difference between chronic and acute pain memory. In chronic pain memory the mood and lifestyle consequences of pain are registered, not just the pain itself. When asked to remember the intensity of chronic pain the patient's global view of the

pain problem will influence recall rather than the intensity of the pain itself. In recalling an isolated incident of pain patients are more likely to draw on episodic memory. Mood and affective state have also been shown to influence memory for pain (Kent, 1989, Roche and Gijsbers, 1985) and Eich et al (1985) demonstrated that memory for pain intensity is biassed by current pain state. In the context of a study designed to test the hypothesis that previous experience of severe, acute episodes of pain can lead to avoidance behaviour and chronic back pain (Lethem et al, 1983) it is desirable to minimise the bias and distortion associated with semantic memory and mood state. It is therefore preferable to use memories for acute, episodic pains which, in order to reduce the effect of mood state associated with back ache, were the result of non spinal pathology. However, this does not preclude the use of memory for back pain as part of other models of chronicity which are described below.

iii) **Personal response/coping strategies.** The central concept of the Fear Avoidance Model is fear of pain. Lethem et al (1983) propose that following single or repeated attacks of acute pain, the normal response to the threat of further pain is fear, to which there can be two responses; confrontation or avoidance. In practice most patients might be expected to exhibit a mixture of the two. Slade et al (1983) postulate that each individual develops a personal strategy for coping with pain, whatever its cause. Such strategies would span a

continuum from the purely active (eg the strategy in which pain is ignored, physical exercise is taken and activities are sought to distract from the pain) to purely passive (such as the resort to rest and to analgesics). Such active/passive strategies are considered by Slade et al (1983) to reflect the confrontation/avoidance responses to fear of pain.

A relationship between pain coping strategies and outcome has been demonstrated by Brown and Nicassio (1987) in a study of patients with rheumatoid arthritis. Level of pain was controlled for. Patients who reported the use of active coping strategies (such as 'engaging in physical exercise', 'staying busy' and 'participating in leisure activities') in response to exacerbations of their disease reported less pain and disability than those who adopted passive coping strategies (such as 'lying down to rest', 'restricting social activities' and 'taking a hot bath').

Although the Fear Avoidance Model is primarily concerned with low back pain, Lethem et al (1993) postulate that individuals have a coping style in response to acute pain generally and this will determine behaviour in response to acute back pain. In order to test this hypothesis it is necessary to use an instrument designed to measure the behavioural response to acute pain conditions with several different biological causes. This instrument, developed by Lethem et al (1983), is presented in Chapter 3.

iv) **Personality.** The relationship between personality characteristics and chronic pain has been the subject of much research. Pychodynamic theories of chronic pain propose that experiences in early life can predispose individuals to adopt a life-style in which suffering is a key element. Freud (1952) regarded pain as a common conversion reaction and proposed that chronic pain was a neurosis resulting from a compromise between the fulfilment of a forbidden drive and its punishment. Dependency strivings are often gratified in the context of pain experience. The Dependency Motivation Theory (Gentry, 1974) argues that chronic pain behaviour may be the sequel to unmet dependency needs. An injury or illness in adulthood provides the means for the individual to have their dependency needs met, especially if the individual had early parental models for pain and disability. Some evidence for this proposition comes from studies based upon retrospective reports showing that the parents and siblings of chronic pain patients suffer from more painful conditions than the families of controls (Block, 1981).

Psychodynamic theorists also propose theories of chronic pain which involve the relationship between the ego and body image. Engel (1959) suggested the concept of a 'body pain image' referring to the areas of the body previously involved in pain, and of pain memories which are the ideational complexes, conscious and unconscious associated with past pain experience, stimulation of which may later give rise to pain.

However, systematic investigations into the validity of psychodynamic theories of pain have been relatively uncommon (Pilowsky, 1978). As with all psychodynamic approaches, the theories generated are unfalsifiable. The evidence that is quoted lacks control groups and information about base rates of patients who suffer conflicts yet are not in pain (Ghadiali, 1987).

Much of the work done in the last 30 years concerning personality and pain has utilized standard psychometric tests such as the Minnesota Multiphasic Personality Inventory (MMPI) and the Eysenck Personality Inventory (EPI) (Slade, 1985). The MMPI has been the most widely used personality test applied to low back pain patients. The extensive use of the instrument has created a large data base wherein 'normal' scores are well established. The MMPI has been used to identify patients whose pain is 'functional' or non-organic, to describe the psychological features of chronic low back pain patients and predict outcome of treatment or natural history. It has been suggested that patients whose back pain is 'functional' have higher than normal scores on the hysteria, hypochondriasis and depression scales or high hysteria and hypochondriasis scores with a normal score on the depression scale (pain being converted into depression).

Lethem et al (1983) focused on the relationship between the MMPI hypochondriasis, hysteria and depression scales and

chronic pain. They related raised MMPI HS, Hy and D scores to the Fear Avoidance Model in three ways; a) The behaviour of an individual who is concerned about physical health (and consequently tends to score highly on the MMPI hypochondriasis scale) is likely to be more reinforced by concerns about acute pain than the individual who has low scores on the scale; b) the behaviour of those individuals who like to be the centre of attraction and tend to score highly on the MMPI scale for hysteria is likely to be more strongly reinforced by any attention at the time of their acute pain experience than that of individuals who tend towards lower scores on this scale; c) individuals who are currently depressed and tend to score more highly on the MMPI depression scale are likely to be suffering from reduced initiative and relative psychomotor retardation and they are more likely to respond by avoiding pain than by confronting it.

However, questions have been raised regarding the reliability and validity of a large number of widely used psychometric instruments such as the MMPI and EPI since the Fear Avoidance Model was proposed in 1983. Significant methodological questions concerning various instruments used in pain research involve poor discriminatory power, problems with statistical structure, ambiguities with clinical interpretation and overall level of sensitivity. This indicates that many questionnaires are used for purposes for which they were never originally designed and additionally have not been subjected to the kind

of rigorous statistical appraisal which is demanded by research inquiry (Main and Parker, 1989, Main et al, 1992, Ghadiali, 1987).

The inability to objectively identify pathological aetiology in the majority of patients has led to the development and employment of many psychometric instruments which a) were originally developed on psychiatric populations; b) attempt to identify pain prone personalities; and c) fail to address and evaluate the true psychological dynamics of low back pain (Main et al, 1992).

The MMPI is a case in point. The MMPI was designed originally to identify psychiatric profiles. However, partly as a result of the work of Hanvik (1951), it became the principal instrument for assessing the relationship between chronic low back pain and psychological variables. The first three clinical scales of the MMPI (Hypochondriasis, Depression and Hysteria), sometimes referred to as the "neurotic triad" have been shown fairly consistently to differentiate pain patients from non-pain-patients but are not sufficiently discriminating to permit decision making about individual patients (Cummings et al, 1979). Historically, the neurotic triad has been interpreted as a sign of malingering, gross psychological overlay and evidence of minimal physical pathology. Evidence suggests that this is not the case (Waddell, 1987). Main and Waddell (1987) assert that the use of the MMPI in order to dichotomize low back pain

patients into organic and functional groups is unhelpful as it is clearly possible for patients to have coexistent organic and psychological pathology. In addition, significant decreases in several key MMPI scales (Hy, D and Hs) have been shown to occur after successful treatment of chronic pain (Sternbach and Timmermans, 1975). Similarly, raised scores on the Eysenck Personality Inventory (EPI) scales which suggest neuroticism and extroversion among chronic pain sufferers have been shown to normalise following successful attempts at pain relief (Bond, 1978).

The MMPI is a personality inventory; yet attempts to diagnose, describe and predict the low back pain personality (France and Krishnan, 1985) have proved unsuccessful. As Main and Parker (1989) note on the overall clinical value of the MMPI for chronic pain groups: 'It lacks both diagnostic and descriptive accuracy....even the most elegant statistical superstructure is rendered vulnerable by weak theoretical foundations'.

The concept of psychological 'distress' in the context of low back pain personality research has been proposed by Main and Waddell (1984) in response to the concerns raised over the use of 'personality' measures such as the MMPI. Main and Parker (1989) argue that while the MMPI is clearly capable of identifying a measure of distress in patients with low back pain, this may be no more than anxiety attributed to a learned helplessness situation involving physical and psychosocial

disruption. At the acute stage of low back pain, distress is marked by anxiety, the most pervasive feature of which is a pattern of avoidance behaviour which may in the past have served to reduce anxiety, but which often generalises far beyond any originally adaptive level. The somatic symptoms of anxiety reflect physiological arousal associated with the consequences of increased adrenalin and noradrenalin secretion. Although clinical anxiety is not in general a feature of back pain patients (Sternbach, 1974, Wifling, 1981), most scales purporting to measure anxiety contain items reflecting both subjective agitation and somatic awareness (Main, 1983). Acute pain elicits escape or avoidance behaviour and heightened awareness of bodily functioning, one of the accompaniments of increased sympathetic activity which can be expected in any situation of threat or danger. Main (1983) developed the Modified Somatic Perception Questionnaire (MSPQ) which consists of thirteen items concerning awareness of somatic symptoms and experiences (eg sweating all over, dizziness feeling sick etc). The MSPQ was developed on chronic low back pain patients and differentiates between back pain patients and normals and between chronic and acute back pain patients (Main, 1983). Waddell and Main (1984) compared the MSPQ with other personality traits such as the MMPI and EPI in a study of two hundred chronic low back pain patients. Results indicated that the MSPQ was the most powerful predictor of patients' self reported disability. Other personality variables accounted for less than 12% of data variance.

The MSPQ is easy to administer, has high patient compliance and is much more sensitive than traditional measures of personality structure (Main, 1983). Furthermore, it can be argued that somatic awareness represents the somatic component of fear (anxiety) and is central to the Fear Avoidance Model, unlike personality constructs such as hysteria, depression and hypochondriasis. The MSPQ will, therefore, represent the 'personality' component of the Fear Avoidance Model in this study.

1.5 THE STATUS OF THE FEAR AVOIDANCE MODEL IN THE CONTEXT OF RESEARCH

The Fear Avoidance Model represents "the most specific model" of fear-avoidance to date (Waddell et al, 1993). The model has been tested in part by Slade et al (1983) and by Rose et al (1992). Perhaps more importantly, its development has contributed to the work of others who are also concerned with the relationship between fear-avoidance and chronic low back pain (eg Troup, 1987, Troup, 1988, Philips and Jahanshahi, 1985, Waddell et al, 1993).

The Fear Avoidance Model was supported, in part, by Slade et al (1983). A preliminary investigation of the relationship between personal pain history, personal coping strategies and back pain was made using a sample of normal subjects (165

university students). Subjects completed questionnaires consisting of items concerning history of low back pain, coping strategies for internally produced pain (headache, sore throat), severity of worst ever remembered externally produced pain (eg fractures, joint sprains) and the severity of worst ever remembered internally produced pain (eg stomach pain, sore throat).

Fifty-five percent of subjects reported that they had experienced back pain (back pain group). The average rating of the severity of externally produced pain of the 'back pain group' was significantly higher than that of the 'no back pain' group (P = 0.01). No significant differences were demonstrated between the back pain and no back pain groups in terms of strategies for coping with internally produced pain. However, the coping strategies of subjects who reported that the severity of their back pain was increasing were significantly more passive than those of the group who reported that the severity of their low back pain was decreasing (P = 0.01). Slade et al argued that the results of this study "justified the inclusion of personal pain history and coping strategies in the model for exaggerated pain perception" and "support for the hypothesis concerning the relevance of the model to chronic pain is therefore strong".

Although supportive of the Fear Avoidance Model, the results of the Slade et al study are limited. The study was cross-

sectional and consisted of a homogeneous sample in terms of age and social background. In addition, the sample consisted of subjects who were not experiencing back pain at the time of the study. Furthermore, only two of the components of the 'psychosocial context' were included (coping strategies and pain memory). The reasons for the exclusion from the study of 'personality' and life events were not given.

More recently, the Fear Avoidance Model has been supported by Rose et al (1992) in a cross-sectional study of three groups of chronic pain patients (Post-Herpetic Neuralgia (PHN), Reflex Sympathetic Dystrophy (RSD) and chronic low back pain) and their recovered controls (recovered shingles, recovered fractures and recovered acute low back pain). In the view of the author, an important result of this study is that the Model, developed to explain chronic back pain, has been shown to explain chronic pain generally, regardless of its aetiology.

Sixty-eight subjects (34 chronic pain patients and 34 recovered subjects) completed questionnaires consisting of the component parts of the Fear Avoidance Model's 'psychosocial context' (pain history, coping strategies, life events and 'personality'). Personality was measured using the MSPQ (Main, 1983).

The results of the study demonstrated that discriminant function analysis using the the four psychosocial context

variables as independent variables correctly predicted 82% of the sample in terms of recovery or chronicity. A two-way ANOVA on data from all the subjects revealed a significant main effect of condition (recovered or chronic) for highest externally produced pain, MSPQ and weighted life events. No significant main effect of nature of pain was demonstrated. Although not significant, chronic pain patients were shown to adopt a passive coping style in response to acute pain compared with recovered subjects. A serendipitous finding of the study suggests that chronic pain patients may be sensitized to previous pain experience which has the same nature of onset as their present pathology yet remain relatively untouched by previous pain experience with a different aetiology. External pain remained stable between pathological groups whereas RSD patients reported higher accidental pain than the *other groups* and PHN and chronic back patients reported higher internal pain than the RSD group.

Although cross-sectional in nature, the results of the study have important implications for chronic pain research. To date, chronic pain research has focussed on chronic back pain and headache, conditions in which the relationship between symptoms and organic pathology is not always clear. However, the results of this study, in addition to supporting the Fear Avoidance Model as a model of chronic back pain, suggest that pathologies such as RSD and PHN, which undoubtedly have a recognisable physiological basis, may also be mediated by psychological

variables. To the author's knowledge, this study represents the first demonstration of the relationship between psychological and observable organic variables in the context of chronic pain.

In summary, the preceding sections have described the Fear Avoidance Model and examined the constructs which determine the 'psychosocial context' in some detail. Evidence which supports the model has been presented although it is recognised that the cross-sectional nature of the studies concerned suggest the need for a longitudinal study in order to support the predictive utility of the model. The following section will briefly review competing models.

1.6 ALTERNATIVE MODELS OF CHRONIC LOW BACK PAIN

1.6.1 The physical model

Despite the widely held view that pain is a multi-faceted, diverse experience, defined by Melzack and Wall (1988) as:

"a category of experiences signifying a multitude of different unique experiences having different causes and characterised by different qualities varying along a number of sensory, affective and evaluative dimensions"

a considerable body of evidence exists which supports the notion that low back pain is a function of physical impairment alone. Reduced lumbar movement in sagittal and coronal planes, neurological deficit and aberrant or weak lumbar and abdominal muscle contraction have all been demonstrated to be positively associated with chronicity and disability. The implicit assumption behind the initiation of studies concerning physical variables and back pain is one which embraces specificity theory and the causal relationship between physical pathology and pain complaint, impairment and disability.

The issues of aetiology, prognosis and prediction of new episodes of low back pain have all been addressed in terms of physical measurements. The relationships between physiological variables and function, pain complaint and prognosis have been explored. Physical variables have also been used in an attempt to predict the outcome of an acute attack of low back pain, the onset of a first episode of low back pain and the outcome of treatment of low back pain. However, many studies are cross-sectional in design which makes the results difficult to interpret.

One such study was conducted by Pope et al (1985) who compared the anthropometric, postural, muscular and mobility characteristics of no back pain, moderate back pain and severe back pain males. They found the strongest associations to be between weakness of lumbar flexor and extensor strength and

severity of pain. No statistically significant differences were observed between the three back pain groups in terms of range of spinal movement.

In contrast to the findings of Pope et al, Pearcy et al (1985) demonstrated, in another cross-sectional study, that range of movement of lumbar flexion and lumbar extension did differentiate between chronic backs and non backs in a study of a pain free control group, a group with chronic low back pain only and a group with chronic low back pain and neural tension signs in the form of sciatica and limited straight leg raise. 'Movement analysis' showed that the control group had significantly greater ranges of lumbar flexion and extension than both back groups and the back pain only group had significantly more movement than the tension sign group.

One explanation for the contradictory observations of Pope et al and Pearcy et al may lie in the different techniques used by the two groups for measuring sagittal movement. Pope et al used the change in distance between surface anatomical landmarks to measure movement whereas Pearcy et al used a radiographic technique in which the angles of movement were measured directly from radiographs. The latter technique relies less on the palpation skills of the researcher and may be more accurate.

Burton and Tillotson (1989) were also concerned with the relationship between low back pain and sagittal spinal mobility. They hypothesized that mechanical lumbar instability results in lumbar hypermobility and is therefore associated with recurrent and chronic low back pain. They measured the range of lumbar flexion and extension of male and female non backs and recurrent backs. However, their results were in accord with those of Pearcy et al and Pope et al and failed to support Burton's hypothesis. Male and female recurrent back pain sufferers were shown to have reduced ranges of lumbar flexion and extension, rather than increased mobility. One reason for the failure of the data to support Burton and Tillotsons' hypothesis concerns their assumption regarding a positive relationship between range of sagittal movement and lumbar instability. This may not be so, and as they suggest, lumbar instability may be reflected in movements other than lumbar flexion and extension. Instability may be identified by measuring complex single-level movements. The technique of measurement used in the Burton and Tillotson study is incapable of measuring single level movement in even one plane and the movements associated with instability may indeed be conjoint multi-plane movements. However, even if lumbar instability had been demonstrated to be associated with chronic low back pain, no inferences could have been made regarding the relationship between these conditions because of the cross-sectional study design. Lumbar instability at the segmental level may be either

the cause of the degenerative changes associated with recurrent low back pain or the result of such changes.

Ahern et al (1990) were concerned with sagittal spinal movement in terms of the activity of the para-spinal musculature of the chronic low back pain patient. They demonstrated that the normal quiescence of the lumbar para-spinal muscles at approximately 40 degrees of flexion (flexion-relaxation response) of 39 chronic low back pain patients was absent. The authors hypothesized that the development of maladaptive postures such as guarding or bracing following persistent pain may in turn lead to aberrant neuromuscular patterns, which may further contribute to pain and a reduction in sagittal mobility.

Mellin (1990) also compared physical aspects of chronic back pain patients with those of pain free controls. He measured a range of physical movements of 103 male and female students, 48 of whom had no history of back pain. His results demonstrated that the control group had greater sagittal and coronal spinal movement and gleno-humeral movement than the chronic back pain group. It is of interest that Mellin demonstrated a reduced range of gleno-humeral joint movement amongst the chronic back pain group. This may suggest a general difference in the anatomy and/or physiology of the synovial joints between chronic low back pain patients and pain free controls generally, regardless of location in the body. However, in the

view of the author, this finding supports his own clinical experience that general joint stiffness is the result of inactivity associated with chronic back pain and resolves following rehabilitation.

Perhaps not surprisingly, it has been demonstrated that chronic low back pain patients are less physically fit than pain free subjects.

Naliboff et al (1985) compared several physical aspects of 68 chronic low back pain patients with those of 35 age matched controls. Age, gender and social status were controlled for in the analysis of the results of the study. The control group scored more highly than the chronic back pain group on a walking endurance test, trunk strength and flexibility, time spent working, time spent sleeping and the ability to control the low back while sitting, standing and walking.

McQuade et al (1988) were also concerned with the relationship between cardio-respiratory fitness and chronic back pain. They examined 96 chronic low back pain in terms of submaximal exercise tolerance, isometric strength and flexibility of back extensors and hamstrings. After controlling for age and gender, negative correlations were demonstrated between trunk strength and physical disability, between flexibility and disability and between exercise tolerance and physical

disability. None of the fitness elements were associated with level of pain.

The cross-sectional nature of studies of the kind reported above make interpretation of the results difficult. It may be, as the authors suggest, that muscular weakness and limitation of spinal movement signify spinal injury or degenerative pathology or, conversely, these findings may support the view of Troup (1987) concerning the physical sequelae to prolonged bed rest and avoidance of activity following acute back pain.

Lloyd and Troup (1983) identified five physical signs which predicted future low back pain in a study of 936 subjects returning to work following an episode of debilitating low back pain. Seven hundred and ninety of them were followed up 12 months later when information concerning recurrence of low back pain in the previous year was collected. The predictive signs were: restriction of the pain free range of straight leg raising by 15 degrees unilaterally or to 45 degrees bilaterally, inability to sit up from a position of supine lying, nerve root involvement, pain or weakness of resisted hip flexion seated and back pain on extension of the lumbar spine.

Troup et al (1987) carried out a further longitudinal study over one year of 2,891 volunteers from 12 different occupational groups. Physical data were collected at baseline and the subjects were interviewed at follow-up. Patients were

categorised as being 'non-backs', 'mild backs' and 'chronic backs' depending on the incidence and severity of back pain in the study period. Females who has become chronic back sufferers in the study period had been heavier at the beginning of the study than those in the mild and non-back groups. At the beginning of the study, male and female subjects who became chronic back pain patients were significantly less able to lift and less able to sit up from a lying position. Their sagittal spinal mobility and respiratory function were also significantly less than the subjects in the mild or non-back groups.

Roland and Morris (1983) carried out a longitudinal study of 230 acute low back pain patients who had presented to their general practitioner with acute low back pain. Patients were followed up four weeks after onset of pain. Patients were categorised as having a poor outcome at follow-up if a) they scored more than 14 out of 24 on a disability scale (Roland and Morris, 1983) or b) their pain was the same as or worse than at the acute stage or c) they had been off work for more than two weeks. Logistic regression analysis was used to determine whether clinical features had statistically significant prognostic value independent of one another. The results demonstrated that straight leg raise below 60 degrees was positively correlated with poor outcome in terms of disability but not in terms of severity of pain. Neurological involvement

and straight leg raise below 60 degrees were significantly related to poor outcome in terms of sick leave status.

The value of a physical examination and collection of physical data has been questioned.

Mellin (1986) examined 151 chronic low back pain patients before they were admitted to a rehabilitation centre. He demonstrated that lateral lumbar flexion, lumbar rotation, hip flexion and extension and trunk flexion strength correlated positively with a pain and disability index and outcome of treatment. However, correlations, although statistically significant, were low. Mellin interprets his findings as "suggesting that physical measurements are of limited value for assessments of disability and progress in chronic low back pain patients".

This view has been supported by Battie et al (1990) who studied 3,020 aircraft industry employees over a four year period. Initial examination consisted of a range of physical measurements. It was found that back pain elicited on straight leg raise was the symptom most significantly associated with subsequent reports of low back pain among men and women. The only variables to add predictive value in addition to this variable were age and weight in women and age and history of back problems in men. Predictive trends were demonstrated for a reduced or weak knee jerk and height. However, analysis of

data collected from subjects without a history of back pain failed to identify any physical variables associated with subsequent reports of a first episode of back pain. In addition, despite the fact that straight leg raise was a statistically significant predictor of back pain in the future, 80% of subjects who reported back pain elicited by SLR did not report back pain subsequently (up to 4 years after screening.)

In summary, although low back pain is recognized as being a complex and multi-faceted experience, a number of studies have demonstrated that a reduction in range of spinal movement, reduced fitness or muscle strength and symptoms and signs of neurological deficit are positively associated with chronicity and poor outcome of an acute attack of low back pain. However, many of the results of studies which purport to be concerned with physical variables are ambiguous. The findings can be viewed at face value and be interpreted as suggesting that limited physical function is indicative of the severity of spinal pathology. However, a converse view suggests that physical impairment may be, in large measure, a consequence of fear of pain and subsequent avoidance behaviour (Troup, 1987). This ambiguity is an inevitable result of failure to consider pain a diverse experience and neglecting to integrate physiological variables within a comprehensive theoretical framework of pain. However, in the experience of the author, the view that physical pathology is the main determinant of

pain report, disability and behaviour of back pain patients is still widely held amongst clinicians. Therefore, the physical model will also be tested as a predictor of outcome in this study.

1.6.2 The historical model

Variables concerned with the nature of previous attacks of low back pain have also been shown to be predictive of the course that future episodes of back pain will take. In general, few attempts have been made to integrate the results of studies using retrospective variables as predictors of outcome into a theoretical framework. However, as with the 'physical model' the type of research which is typically conducted and the view of clinical practitioners suggests that variables such as 'nature of onset' and severity of previous episodes are generally considered to be positively related to the severity of spinal pathology. The degenerative changes of osteoarthrosis and spondylosis are implicitly associated with the historical model. Degeneration is characterised by slow destructive changes that are not balanced by regeneration. Following initial injury of a structure it may never regain its original strength, thus becoming more vulnerable to injury (Grieve, 1988).

The results of a study by Lloyd and Troup (1983) support this view. They interviewed 936 patients returning to work following

an episode of debilitating low back pain. Seven hundred and ninety of them were followed up 12 months later. At follow up, information concerning recurrence of low back pain in the previous year was collected. Data analysis identified two historical variables which were associated with recurrence. These are falls onto the buttock or back as the cause of the initial low back pain and a history of 2 or more previous attacks. Conversely, Roland and Morris (1983), in a longitudinal study of 230 acute low back pain patients who had presented to their general practitioner with acute low back pain, demonstrated that gradual, none accidental, onset of pain was positively correlated with disability at four weeks.

Burton and Tillotson (1988) conducted a 12 months longitudinal study of 109 patients with low back trouble. Analysis of retrospective data showed that nature of onset, frequency of previous low back pain episodes and length of current spell were indicators of disability at three, six and twelve months follow-up. These findings were supported by Biering-Sorenson (1989) in a prospective, longitudinal study of 928 subjects who were pain free at entry into the study. The results demonstrated that previous history of low back pain in terms of frequency, recency of the last attack and increasing severity of symptoms were indicators of recurrence and persistence of low back pain.

In general, a positive relationship has been demonstrated between the severity and frequency of previous episodes of back pain and the poor outcome. The nature of onset (accidental or insidious) has also been shown to mediate the recurrence and natural history of low back pain. These variables are traditionally associated with physiological rather than psychosocial constructs. This may be justified in the case of nature of onset of low back pain where a mechanical explanation for the relationship is reasonable. Slow, insidious onset is said to be associated with disc prolapse whereas sudden onset, associated with trauma, is generally associated with soft tissue injury or facet joint dysfunction *(see Grieve, 1988)*. However, variables such as severity of previous episodes of pain rely upon memory for pain, a construct which has been shown to be open to psychological bias (Erskine et al, 1990). However, sufficient evidence exists for this loose collection of 'historical' variables to be considered to represent an explanatory model of failure to recover from acute back pain and as such, they will be included collectively in the predictive analyses reported in Chapter 8 of this thesis.

1.6.3 The demographic model

Demographic variables are held to play a role in the aetiology and outcome of episodes of low back pain. Variables which have been demonstrated to mediate the onset and outcome of back pain are, in general, associated with the social status of the

individual in terms of occupational and socio-economic indices. Variables such as nature of employment are concerned with the relationship between mechanical stress on the spine and the symptom of back ache. Socio-economic status, in addition to being a marker for nature of employment, is concerned with the relationship between social group membership and variables such as health service utilisation and illness behaviour.

Occupational related physical stress has been demonstrated to be associated with low back pain by Andersson (1981) who reviewed aspects of low back pain in industry, mentioning among other factors heavy physical work, static stooping postures, frequent bending and twisting, lifting and forceful movements, repetitive work and vibration. These findings were supported by Pope et al (1980), who, in a retrospective study of 3500 workers, demonstrated that low back pain sufferers had been subjected to higher exposures of vibrating machinery than those without back pain. Frymower also demonstrated a relationship between heavy work, especially when associated with vibration, and back pain. In a study of 1221 men between the ages of 18 and 55, they noted that among the 24% with severe back pain, associated factors included repetitive heavy lifting, the use of jack hammers or machine tools and the operation of motor vehicles.

Davis (1966) suggests that the 'lift and carry rate' in modern times is doubtless greater for modern man than in pre-medieval

times, and very much greater than in pre-Neolithic days when a rate of 50 lifts a day for the hunter gatherer may have been an average. Wood (1980) proffers this as a model for the progressive increase in spinal stress following the Industrial Revolution. He suggests that the incidence of low back pain has increased with an increase in the lifting rate at work. However, Horal (1969) compared 212 workers reporting back pain problems with 212 controls. No more than 25% blamed accidents at work for the onset of their pain, and a similar proportion blamed lifting and handling. Among the factors causing recurrences, trauma and heavy lifting were infrequent.

However, Davis and Troup (1986) suggested that low back pain has a bi-polar distribution, with heavy manual workers and sedentary workers being equally at risk. This view was supported by Magora (1973) who found that the earliest age of onset of low back pain was in bank clerks, heavy industrial workers, farmers and nurses. Prolonged sitting and prolonged stooping were salient risk factors.

The association between heavy manual work and back pain can be explained in terms of degeneration of spinal structures. With continued wear and increasing age, this degeneration can lead to spinal pathology and pain (Grieve, 1988). Trauma may occur as a single incident or it may be slow and repetitive with cumulative effects. It includes the effects of impact and vibration on the musculoskeletal system. The relationship

between back pain and sedentary occupation is, intuitively, less obvious especially when inactivity and rest are the mainstay of therapeutic intervention prescribed for low back pain. However, Janda (1980) hypothesises that inbalance between agonist and antagonist muscle groups around the spine may lead to biomechanical stress in the spinal structures which may in turn lead to pathology and pain. This muscle inbalance may be a result of static or dynamic postures which facilitate weakness of a particular muscle group.

Cigarette smoking has been claimed to be associated with low back pain (Biering-Sorenson 1984, Kelsey 1975). However, Dewey et al (1989) argue that "at present there is little compelling evidence that smoking is causally related to the symptom of low back pain". They hypothesise that "the fact that chest symptoms and low back pain appear to be closely related is as likely to stem from behavioural patterns created by the experience of back pain as it is from any pathological interaction between the two". In addition, it is likely that cigarette smoking is a marker for other socio-economic factors which are associated with back pain.

Socio-economic group membership has also been shown to be related to low back pain. This may be associated with pain behaviour and complaint in addition to being a marker for occupational content. Koos (1954) found that "upper class" persons were more likely to report themselves ill than were

"lower class" persons and that they were more likely to seek treatment when afflicted despite the fact that lower class people had more actual symptoms. Sigerist (1960) and Parsons (1951) provided the concept of the "social role" of the sick person where the person occupies a special role in society where he is relieved of usual demands and responsibilities. Some people may be motivated by this to maintain a sick role whereas others may be suspicious of doctors and avoid taking advice even when seriously ill. The assumption or otherwise of the sick role is dependent on variables such position in social group, necessity to work for financial reasons, ethnic background and role modelling and pressure from peers and spouse.

In conclusion, the demographic model is concerned with explaining low back pain in terms of its physical aetiology and consequent pain report and behaviour. The model proposes that occupational stresses provoke a spine made vunerable by age and consequent degenerative changes into causing pain. The response to this pain is determined, in large measure, by social group membership. When described thus, it is apparent that the demographic model is, in part, an alternative way of expressing the physical model and is embedded within the framework of specificity theory. However, the demographic model also takes into account the social context in which spinal pathology exists and recognises that outcome may be determined in part by sociological variables. It is therefore justifiable to consider

the demographic model as being independent from previously described models with which it will be used to test the predicitive utility of the Fear Avoidance Model.

In conclusion. this section has identified and described three alternative explanatory models of chronic low back pain which reflect the literature. Although it is apparent that the models share a common element which is concerned with explaining morbidity in terms of physical damage, the historical and demographic models also emphasise psychological and sociological aspects of back pain experience. The physical, historical and demographic models will therefore be included separately in statistical analyses designed to compare their predictive utility with that of the Fear Avoidance Model.

CHAPTER 2

RATIONALE OF THESIS

2.1 INTRODUCTION

The Preface and Chapter 1 have presented evidence to show that low back pain is a very common symptom which represents a source of considerable distress for sufferers and a drain on the resources of the Health Service and the Exchequer. The rate of increase of low back pain report over the last decade has resulted in this health problem being described as an epidemic (Waddell, 1987). Although it is thought that the majority of acute low back pain patients recover as tissue healing takes place, a small yet significant proportion go on to become chronic sufferers.

A number of opposing views regarding the nature of low back pain experience have been expressed. Some regard the natural history of the condition as being determined by physical pathology whereas others view psychological variables as the sole mediators of outcome of an acute episode. However, it is generally recognised that low back pain experience is a multi-faceted phenomenon and involves a complex interplay between physical, psychological and socioeconomic variables.

The Fear Avoidance Model of Exaggerated Pain Perception (Lethem et al, 1983), described in Chapter 1, was developed in response to the challenge to integrate psychological and physical variables within a theoretical model of chronicity. The central concept which underpins the Model is fear of pain. The model proposes that the amount of fear experienced by an individual with acute back pain will determine the extent to which s/he will confront their pain and regain physical function. Two studies (Slade et al, 1983, Rose et al, 1992) have provided evidence which supports the validity of the Fear Avoidance Model. However, these were cross sectional in design.

This longitudinal study was undertaken in order to test the predictive utility, and therefore to provide some further evidence for the validity, of the Fear Avoidance Model. The Fear Avoidance Model will be compared with other explanatory models of chronic low back pain which have been described previously (Chapter 1). In addition, a detailed description of a sample of acute back pain sufferers will be presented as will the natural history of the sample over a twelve months period.

To the author's knowledge, this is the first study of acute low back pain patients which fulfils the following criteria:-

1) The patient sample was drawn from a primary care setting.
2) The patient sample consisted entirely of individuals whose low back pain had begun no longer than two weeks before

recruitment into the study.

3) The research was based upon a current psychological model which was derived from theoretical principles.

4) Outcome measures were used which attempted to represent the complexity of low back pain experience.

In addition, data concerning physiological, psychological, historical and demographic variables were collected and the relationship between this data was explored in order to further develop theoretical knowledge concerning low back pain.

The following sections present the aims, objectives and the specific research hypotheses of the study.

2.2 AIMS OF THE STUDY

1) To test the validity of the Fear Avoidance Model of Exaggerated Pain Perception.

2) To describe a sample of acute low back pain patients presenting to their General Practitioners.

3) To describe the natural history of acute low back pain over a twelve months period.

2.3 OBJECTIVES OF THE STUDY

1) To recruit a sample of acute low back pain patients in order to fulfil the aims of the study. The study design is described in Chapter 5.

2) To collect data which represent the Fear Avoidance Model and the physical, historical and demographic models at different stages of the natural history of back pain. These data are presented in Chapter 5.

3) To identify appropriate outcome measures which could be used to define chronicity. These are presented in Chapter 6.

4) To use descriptive and inferential statistical techniques in order to:

a) test the validity of the Fear Avoidance Model as a predictor of outcome (Chapter 8).

b) describe the sample (Chapter 5) and the natural history of acute low back pain.

c) examine the relationship between other models and the development of chronicity (Chapter 8).

5) To interpret and discuss the findings of data analysis to:

a) comment on the validity of the fear avoidance construct in terms of outcome of acute back pain.

b) reformulate the Fear Avoidance Model if appropriate.

c) make a contribution to the theoretical basis of the management of low back pain.

d) identify foci for future research.

2.4 HYPOTHESES OF THE STUDY

Hypothesis 1

The Fear Avoidance Model of Exaggerated Pain Perception has utility in terms of predicting the outcome of an acute episode of low back pain and is therefore valid in terms of explaining chronic low back pain. This hypothesis is tested in Chapter 8.

Hypothesis 2

Low back pain is mediated in the long term by psychosocial variables rather than by physical pathology or impairment. This hypothesis is tested in Chapters 7 and 8.

Hypothesis 3

The degree of limitation of physiological movement is mediated in large measure by psychological variables rather than by physical pathology. This hypothesis is tested in Chapter 7.

Hypothesis 4

The large majority of acute low back pain patients are free of physical signs and psychopathology. This hypothesis is tested in Chapter 5.

Hypothesis 5

The large majority of acute low back pain patients recover within eight weeks of onset of pain. The remainder go on to become chronic low back pain patients. This hypothesis is tested in Chapter 6.

CHAPTER 3

METHOD

In order to achieve the stated aims and objectives of the study (Chapter 2), it was decided that a longitudinal design would be the most appropriate method (see Figure 3.1).

The study consisted of an initial interview, a further interview and physical examination at two months after onset of low back pain and a final assessment of outcome at one year after onset of low back pain.

Three hundred subjects suffering from a first or new episode of acute low back pain were recruited into the study. Inclusion criteria were as follows: a) that the subject was suffering from benign, musculoskeletal low back pain and b) that the subject's low back pain had begun no more than one week before presentation to their general practitioners (GPs).

Forty-seven doctors referred subjects from the following six General Practices in Merseyside:

Maghull Health Centre
Liverpool Road North, Maghull, Liverpool 31
'High Pastures' Health Centre
Liverpool Road North, Maghull, Liverpool 31
Park Road Health Centre

3, The Elms, Liverpool 8

Old Swan health Centre

St. Oswald's Street, Liverpool 13

Princes Park Health Centre

Bentley Road, Toxteth, Liverpool 8

Longmoor Lane Health Centre

Longmoor Lane, Walton, Liverpool 9

These practices were selected in order to give a broad socio-economic mix within the study and because they have close links with the University of Liverpool Department of General Practice.

The general practices were approached by the Professor of General Practice at Liverpool University and asked if they would be willing to cooperate in the study. The research workers in the study met the GPs concerned and discussed the rationale of the project and its proposed methodology.

Clerical staff within the practices were informed of the project methodology and a clerical worker in each practice was identified to take responsibility for communication with the research workers. This individual was usually the practice manager.

When a subject who fulfilled the inclusion criteria for the study was identified by a GP, he or she was given a letter

which explained the aims and method of the Low Back Pain Project (see Appendix A). If the subject then decided to participate in the study they were given an appointment to see a research worker on the premises, within one week. If the subject was confined to bed because of pain by the GP an appointment was made for a home visit by a research worker.

Subjects were formally interviewed by one of four research workers (two male, two female) either in a vacant consulting room at the health centre or, if necessary, in the subject's home. A simple verbal explanation about the aims of the project was given and their willingness to be re-interviewed and examined at six weeks and one year was ascertained.

The formal interview consisted of the completion of what became known by the Research Team as the 'Screening Questionnaire'.

3.1 CONSTRUCTION OF THE SCREENING QUESTIONNAIRE

The screening questionnaire was designed to collect data which a) represented each component part of the Fear Avoidance Model of Exaggerated Pain Perception (Lethem et al., 1983). b) provided data which has been shown by other workers to have relevance to the study of acute and chronic low back pain.

The questionnaire consisted of eight sections (A to H) and 119 items (see appendix B).

Section A consisted of demographic information and information regarding the subjects' history of low back pain. The age, sex, occupation, status and smoking habits of the subjects were recorded, plus the name of the subjects' referring GP.

Employment status was determined by asking subjects to explain how they were employed, whether part time or full time. If the subject was not employed their status (eg student, housewife, unemployed etc) was recorded. The occupations of employed subjects were coded according to the social group they represented. (Registrar General's Classification of Employment, 1989).

Information concerning the subjects' history of low back pain, its nature of onset (injury or no injury) and its severity at the time of interview and in the past (on a visual analogue scale of 1 to 10) was recorded. (It was found that subjects could only remember dates of onset and worst attack to the nearest year, rather than to the nearest month.) In the analysis of this section, time since onset and time since worst attack were converted into an ordinal scale of 1 to 5. This scale represents less than one year, 1-2 years, 2-3 years, 3-4 years, 4-5 years and more than five years. This section provided data concerning the initial onset and course of low back pain for each subject and the perceived severity of the present and earlier exacerbations.

Subjects were also asked whether or not they were off work at the time of interview because of their low back pain.

Sections B, C and E were designed to measure the subjects' perception of the severity of 'externally', 'internally' and 'accidently' produced pains experienced by the subjects throughout their lives. Subjects were asked to rate the severity (on a scale of 1-10) of each pain listed that they had experienced. If they had not experienced pain produced by a particular cause this was recorded as not applicable (NA). In terms of data analysis, the measures derived from sections B, C and E which were used i) were the highest rated 'externally' produced pain, ii) the highest rated 'internally' produced pain and iii) the highest rated 'accidentally' produced pain. Thus the pain history ratings were based on worst experience rather than average experience.

Validity has been claimed for the use of Visual Analogue Scales in both measuring and comparing both chronic and experimentally induced pain (Price et al, 1983); in the evaluation of loading of joint structures (Harms-Ringdahl et al, 1986); and in many studies comparing treatments or in the evaluation of outcome (Waddell et al, 1986). All visual analogue scales used in the questionnaires had anchors of 1 and 10. Subjects were told that 1 represented "no pain whatsoever" and that 10 represented the "worst imaginable pain possible".)

Data collected regarding the subjects' perception of the severity of previous episodes of low back pain and the severity of other painful experiences represents the 'pain history' element of the Fear Avoidance Model.

Section D consisted of five different, although not mutually exclusive, coping strategies which subjects would be likely to use in the event of experiencing the 'internally' produced pains listed in Section C. Subjects were asked to indicate, for each of the pains described which they had experienced, which of the following coping strategies they would use in response to the worst attack they could remember:

a) pain killers
b) physical exercise
c) go to the doctor/dentist
d) ignore the pain and carry on
e) rest

'Taking pain killers' and 'resting' were rated for the purpose of analysis as 'passive' strategies and 'taking physical exercise' and 'ignoring the pain and carrying on' were rated as 'active' strategies. The third listed coping strategy, 'go to the doctor/dentist', was included in the hope that data analysis would reveal which group, 'active copers' or 'passive copers', were more likely to seek the help of a doctor or dentist. Data collected by Section D represents the 'Personal Coping Strategies' element of the Fear Avoidance Model. An index of the coping strategies used by the individual subject

was derived for entry into data analysis as follows:

$$\% \text{ active coping strategies} = \frac{\text{no. of active strategies} \times 100}{\text{no. of active} + \text{no. of passive}}$$

The 'personality' component of the Fear Avoidance Model was represented by data collected in section F. This consisted of the Modified Somatic Perception Questionnaire (MSPQ) (Main, 1983). The MSPQ was devised specifically to be used with chronic pain subjects. The MSPQ has been shown to differentiate between acute low back pain subjects and chronic low back pain subjects (Main, 1983) and to correlate with the MMPI hypochondriasis, depression, hysteria, psychopathic deviate, paranoia, psychasthenia, schizophrenia and social introversion scales. It also correlates with the Zung Depression Inventory (Deyo et al., 1988). Its test/retest reliability has been demonstrated to be greater than 0.60 (Main, 1983) and its internal consistency has been shown to be high (Deyo et al., 1988). The MSPQ has also been described as a measure of anxiety. This view is based upon the relationship between anxiety and autonomic activity which often results in symptoms of the kind described in the instrument.

Section G represented the fourth component of the Fear Avoidance Model, stressful life events. The Social Readjustment Rating Scale (Holmes and Rahe, 1967) was used to assess the

ammount of stress the subject had endured during the year preceding the onset of the present episode of low back pain. Two measures were derived from this scale, namely i) total number of life events and ii) total weighted life events score using the weights given in the original article (Holmes and Rahe, 1967).

Section H consisted of the Roland and Morris Disability Questionnaire (Roland and Morris, 1982). This instrument was developed as part of a study of subjects who presented to their GP with low back pain and was based upon the Sickness Impact Profile (Bergner et al, 1981).

The research worker who administered the Screening Questionnaire read the questions to the subject and recorded the reponse rather than asking the subjects to complete the questionnaire themselves. This prevented the inaccuracy and embarrassment that might have ensued had a subject been unable to read or write. The total time taken to administer the Screening Questionnaire was 15 minutes. At the end of the interview subjects were reminded that they would be asked to attend their health centre in two months time so that they could be re-interviewed and examined.

3.2 TWO MONTHS FOLLOW-UP METHOD

If for any reason subjects could not attend the health centre they were seen at home. The two months follow-up interview and examination lasted for approximately 45 minutes. One hundred and sixty-two subjects attended for interview and 159 of these were examined.

The aims of the two month follow-up study were:

a) To determine which subjects from the original sample had completely recovered from their low back pain, which subjects had partially recovered and which subjects remained the same.

b) To test whether information collected in the first part of the study could be used to predict the condition of low back pain subjects two months after onset of pain.

c) To take the opportunity to collect further psychosocial data from the subjects which could be used both as measures of two-month outcome and potential predictors of chronic low back pain.

d) To conduct a physical examination of the subjects in order to collect physiological data which could be used as potential predictors of future outcome and allow the relationship between physical and psycho-social aspects of low back pain to be explored.

3.3 TWO MONTHS FOLLOW-UP QUESTIONNAIRE

The screening questionnaire was readministered in order to assess whether the subjects' condition had deteriorated,

remained static or inproved. This would also present the opportunity to assess the repeatability of several of the sub-sections of the screening instrument.

Pain Drawing: This test consisted of drawings of the anterior and posterior aspects of a human figure, upon which the subject was asked to mark the areas within which they felt pain. The scoring procedures recommended by Randsford et al. (1979) were used (Appendix C).

Modified Zung Depression Inventory: Main and Waddell (1984) developed this 23 item self-report scale which measures the degree of depressive symptomatology experienced by the subject. The instrument consists of 23 examples of depressive symptoms (see Appendix C). The subject is asked to indicate to what extent they experience each symptom. The possible reponses are rarely (no points), some of the time (one point), a moderate ammount of the time (two points) and most of the time (three points). Therefore, the range of possible scores is zero to sixty-nine.

Oswestry Low Back Pain Disability Questionnaire: Fairbank et al. (1980) developed this questionnaire in order to assess to what extent back pain had affected the subject's ability to perform compared with a fit person. The ten sections were identified to be those most relevant to subjects with low back pain. Each section contains six statements and each statement

describes a greater degree of difficulty than the last (Appendix C). Each section is scored on a 0 to 5 scale, 5 representing the greatest disability. The scores for all the sections are added and the total doubled and expressed as a percentage. Fairbank et al. (1980) validated the instrument against improvement in observed disability and symptoms and found it to be a valid measure of disability. Its test/retest reliability was demonstrated to be high ($r = 0.99$, $p < 0.001$).

Non-organic Physical (or 'Inappropriate') Symptoms: Waddell and Main (1984) have described seven 'inappropriate' symptoms which are distinguishable from standard symptoms of physical disease (Appendix D). They have also been shown to be closely related to psychological distress measured by the MSPQ and Zung Depression Inventory. Affirmative responses to the following questions are associated with inappropriate symptomatology, except question 5. In this case, a negative response is considered to be an inappropriate symptom.

1) Do you get pain at the tip of your tailbone?
2) Does your whole leg ever become painful?
3) Does your whole leg ever go numb?
4) Does your whole leg ever give way?
5) In the past year have you had any spells with very little pain?
6) Have you ever been made worse by treatment?

7) Have you ever been admitted as an emergency into hospital because of back pain?

An inappropriate symptom carries a score of 1, an appropriate symptom scores 0.

3.4 PHYSICAL EXAMINATION

A formal physical examination of the subjects in the study was undertaken by the author. The examination consisted of standard physical tests routinely used in the clinical assessment of subjects with low back pain and those that have been used to some advantage by other research workers. An attempt was made to quantify ranges of movement by the use of measuring instruments. In clinical practice ranges of movement are often estimated. The examination techniques were applied in the same order to each subject and the results recorded directly onto the examination sheet (Appendix D). Subjects were asked to undress sufficiently to facilitate access to their low back and legs.

Non-organic (inappropriate) physical signs

Waddell et al. (1980) described eight standardised inappropriate clinical signs associated with low back pain. The measurement of these signs has been shown to be reliable between examiners and stable over time. The signs have also been shown to be associated with the 'neurotic triad' of the MMPI. The signs are:-

Tenderness

Tenderness related to physical disease is usually localised to a particular skeletal or neuromuscular structure. Inappropriate tenderness may be superficial or non anatomical.

1) Superficial tenderness - This test was considered positive if the subject's skin was tender to a light pinch over a wide area of skin in the lumbar area. A localised band in a posterior ramus distribution may have been caused by nerve irritation and was discounted.

2) Non anatomical tenderness - This test was considered positive if deep tenderness was reported by the subject over a wide area and not localised to one structure. Inappropriate non anatomical tenderness often extends to the thoracic spine, pelvis and sacrum.

Simulation - These tests give the naive subject the impression that a particular examination technique is being carried out when in fact it is not.

3) Axial Loading - If low back pain was reported on vertical loading on the top of the subject's head the test was considered to be positive. (Neck pain was discounted.)

4) Rotation - If low back pain was reported when the pelvis and trunk were rotated together about a longitudinal axis the test was positive. (No rotation of the lumbar spine takes place in this test but if nerve root irritation is present leg pain may be reported. This was discounted.)

5) Distraction Test in sitting - If the straight leg raise test (see below) had resulted in a report of pain the subject was asked to sit up on the examination bed or floor with knees and legs extended. If the angle between trunk and femur was as great or greater in this position than the angle recorded for straight leg raise and the subject did not complain of pain the test was considered to be positive.

Regional Disturbances

The essential feature of these tests is divergence from accepted neuroanatomy.

6) Sensory - If a subject reported sensations associated with neurological deficit or referred pain which covered a wide, non dermatomal area the test was considered to be positive.

7) Weakness - If muscle weakness was demonstrated on normal testing that could not be explained by neuroanatomy (ie 'cogwheel giving way' or weakness in both flexor and extensor groups around a joint) the test was considered to be positive.

8) Overreaction to the examination

This may take the form of disproportionate verbalisation, facial expression, muscle tension, collapse, sweating etc. Care is taken to avoid observer bias and account is taken of cultural variations.

Scoring

If an inappropriate sign was observed the test scores one point. The scores for inappropriate symptoms and inappropriate signs tests were summed to give an overall score for inappropriate signs and symptoms. The maximum possible inappropriate signs and symptoms score was therefore 15.

Distribution of symptoms

The areas of pain, paraesthesia and numbness reported by the subject were recorded on a body chart.

Weight and height

Height and weight were measured using a wall mounted height gauge and bathroom scales. In order to derive an index of obesity, Body Mass Index (Slade and Brodie, 1988) was computed using the following formula:

$$\text{Body mass Index} = \frac{\text{Weight (Kg)}}{\text{Height (m)}^2}$$

Neurological tests

Reflexes

The subjects quadriceps reflex was tested with the subject positioned on the edge of the examination bed,legs hanging free with knees passively flexed to 90 degrees. The patella tendon was struck with a patella hammer and the 'strength' of reflex elicited assessed and recorded. Any apparent reduction in the quadriceps reflex is clinically associated with involvement of the fifth lumbar (L5) nerve root on the affected side.

The Tendo Achilles reflex was tested with the subject positioned in prone lying. The foot was passively dorsiflexed to plantigrade by the examiner and the Tendo Achilles struck with a patella hammer. Any apparent reduction of this reflex is clinically associated with involvement of the first sacral (S1) nerve root on the affected side. In both cases standard facilitatory techniques were used if the reflex proved difficult to illicit.

Muscle strength

All lower limb myotomes were assessed for weakness with the subject supine. The appropriate muscle groups were resisted manually in their middle range. Their strength was subjectively rated using the Oxford Scale. This scale ranges between zero which represents an absence of muscle contraction and five

which represents 'normal' strength. The subject was asked to demonstrate their ability to walk on their heels and toes. An inability to do so or obvious weakness of the associated muscle groups is clinically associated with L5 and S1 nerve roots respectively on the weak side.

Sensory loss

Sensory loss was determined by a comparison of left and right lower limbs in terms of sensation to touch by the author's hand. If a subject reported that they were experiencing a reduced sensation to touch in one or more dermatomes, the test was considered to be positive. This procedure depends upon the subjective report of the subject and a positive result is at best indicative of the need for further examination. However, it was felt that this procedure best suited the aims of the study in terms of time and efficiency.

Any signs of muscle weakness, inhibition of spinal reflexes, muscle wasting or sensory loss are clinically thought to be associated with nerve root entrapment. For the purpose of data analysis, a new 'neurological deficit' variable was created. This variable was treated as being positive if any one individual neurological test was thought by the author to be positive.

Straight leg raise test

The starting position for this test was supine. The subjects' legs were extended at the knee by the author and a hydrogoniometer (Medesign UK) was placed on the leg approximately half way between the ankle and the knee. The hydrogoniometer was set at zero degrees with the subjects' legs resting on the examination bed. The extended leg was flexed at the hip until

i) the author felt resistance

ii) the subject experienced pain behind one or both legs

iii) the subject experienced an increase in low back pain.

The angle of inclination was recorded and the process repeated on the other leg. A positive straight leg raise test may be associated with involvement of the fifth lumbar and first and second sacral nerve roots. The average value of left and right straight leg raise was computed for the purpose of data analysis.

'Slump' or adverse mechanical tension (AMT) test

This test procedure is concerned with dynamic nature of the central and peripheral nervous system in its anatomical relationship with the spinal column. The test is designed to stretch the nervous system in order to detect adhesions in and around lumbar nerve roots as they pass through intervertebral foramen.

The subject sits on the side of the examination bed with both hands resting on the bed, behind him/her. The thoracic, lumbar and cervical spine, are flexed, the knee extended and the ankle dorsiflexed. This position puts maximal physiological 'tension' on the nervous system. If, during the sequence of movements, low back or leg pain is reported the position is maintained and the cervical spine extended. Cervical extension reduces nervous system tension. If this movement reduces pain, the test is deemed to be positive. A positive 'slump' test is suggestive of adhesions in and around the lower lumbar nerve roots.

The test, and the rationale behind it have been described in detail by Troup (1986).

Prone knee bending

A positive result of this test is associated with 'short' knee extensor muscles or involvement of the upper three lumbar nerve roots. The subject was positioned in prone lying upon the examination bed and one knee passively flexed until

i) resistance was felt by the author

ii) anterior thigh pain was reported by the subject

iii) an increase in low back pain was reported by the subject.

The angle of the knee joint was measured using a goniometer. The arms of the goniometer were two feet in length. One arm was held by the author parallel to the bed, the other arm was held along the midline of the leg. The angle was recorded and the procedure repeated on the other leg. The average value of left

and right knee flexion was computed for the purpose of data analysis.

The 'sit-up' test

The starting position for this test was supine lying with hips and knees flexed and feet, held by the author, placed flat upon the examination bed. The subjects were instructed to clasp their hands behind there heads and sit up. The ability to appose chest to knees was considered to be a positive test result.

Lateral flexion

This method provides an overall measurement of spinal movement in a coronal plain and is not specific to the lumbar spine. The subjects were instructed to stand upright with their feet together and hands placed on the outside of their thighs. A mark level with the end of the subjects' middle fingers was made by the author on both legs. The subjects were instructed to side flex until further movement was prevented by pain or soft tissue resistance. A second mark was made level with the subjects' middle fingers. This was repeated on the other side. The distances between the neutral position and the limits of lateral flexion to left and right was measured with a tape measure and recorded.

Sagittal movement of the lumbar spine

This method is complicated in comparison with common clinical

methods of assessing lumbar movement in a saggital plain. However, it has the advantage of isolating the lumbar spine from the rest of the vertebral column. The method has been described in full by Burton (1986). The surface positions of the spinous processes of the 12th thoracic, 4th lumbar and 2nd sacral vertebrae were marked. A draughtsman's flexible curve was 'moulded' over the fully flexed lumbar spine, the location of the spinous processes of the 12th thoracic, 4th lumbar and 2nd sacral segments were recorded on the flexible curve. The curve was transferred to paper, a line drawn round the curve and the positions of the three spinous processes marked. Tangents were drawn to the curve at the levels of the spinous processes and the angles between them measured. This method produced angular values for upper and lower lumbar flexion. The procedure was repeated with the subjects in lumbar extension.

Orthopaedic interview and examination

In addition to the formal examination conducted by the author, the subjects were interviewed and examined by a Senior Orthopaedic Registrar. The main aim of this interview and examination was to identify subjects whose low back pain may have had a sinister cause. This procedure served not only to protect the methodology of the study but also to provide a service to the subjects in the study and their referring doctors. In addition, the Senior Orthopaedic Registrar made a clinical diagnosis based upon his examination of the subjects.

The Senior Orthopaedic Registrar also applied the Waddell inappropriate signs tests (Waddell et al., 1980) to each subject examined.

3.5 TWELVE MONTHS FOLLOW-UP METHOD

The aims of the twelve months follow-up study were:

a) To determine which subjects from the original sample had completely recovered from their low back pain, which subjects had recovered from their low back pain but had suffered from further intermittent episodes of low back pain and which subjects had become chronic low back pain subjects.

b) To test whether information collected in the first parts of the study could be used to predict the condition of low back pain subjects one year after onset of pain and so predict a chronic low back pain outcome.

c) To explore the relationship between psychosocial and physiological variables at different stages of the natural history of low back pain.

The examination procedure and questionnaire were identical to those used at two months follow-up. However, subjects were not examined by the Senior Orthopaedic Registrar.

The original study design called for all twelve months follow-up subjects to attend their health centres so that the screening questionnaire, two months follow-up questionnaire and physical examination could be repeated. This would have enabled the author to explore the relationship between psychosocial and physiological variables at different stages of the natural history of an episode of low back pain.

In practice, fifty-eight subjects attended their health centres to be formally examined by the author and to complete the twelve month follow-up questionnaires.

It therefore became apparent that the attrition rate was such that insufficient twelve month follow-up data could be generated to make a meaningful statistical analysis possible. It was therefore decided to collect twelve months follow-up data by sending postal questionnaires to all the subjects who had been recruited into the study, regardless of whether or not they had attended their health centres to be followed up at two months after onset of low back pain (Appendix E). The data collected from subjects at twelve months follow-up in this way consisted of the minimum information necessary to allow categorisation of the respondent subjects. One hundred and thirty eight subjects returned completed (or partially completed) questionnaires.

3.6 THE TWELVE MONTHS FOLLOW-UP POSTAL QUESTIONNAIRE

The questionnaire consisted of:

The Roland and Morris (1983) Disability Questionnaire

Present pain scale: A visual analogue scale with anchors one (no pain) to ten (the worst pain imaginable) upon which the subjects identified the severity of their low back pain now.

Course of the low back pain over a twelve month period: Three alternative courses that the subjects' low back pain may have taken since its onset. These were 'stayed away completely', 'bothered me on and off' and 'painful all the time'. Subjects were instructed to mark the appropriate alternative.

Work status: The subjects were also asked to indicate whether or not their low back pain was preventing them from attending work.

The twelve months follow-up postal questionnaire was sent to subjects with an envelope which was stamped and addressed to the author.

The information measures collected on subjects at initial screening, two months follow-up and twelve months are summarised in Table 3.1.

3.7 MISSING DATA

An aim of the study was to collect complete sets of data from each of the 300 subjects recruited at the acute stage. However, this aim was not achieved, not least because of the attrition rate which has been discussed previously. In addition, several data sets of subjects who did attend for interview and examination were incomplete. Three subjects who attended at two months follow-up refused to be examined by the author. Other data sets are incomplete because of errors in the completion of questionnaires and transcription of data by the author and data collectors and the refusal of subjects to co-operate with physical examination test procedures. These ommisions in data collection explain any inconsistencies between the total number of subjects included in cross-tabulation tables and the number of subjects who were interviewed or examined at each stage of the study. The frequencies of missing data per variable are expressed in table 3.1

Table 3.1

Frequency of responses for each variable measured in the study at the acute stage (total number of subjects = 300)

Variable	Frequency
Referring practice	300
Referring GP	300
Sex	299
Age	297
Employment category	300
Marital status	300
Smoking habits	300
Nature of onset	294
History of previous back pain	300
Time since first episode	300
Severity of present pain	294
Severity of worst episode	294
Number of previous episodes	293
Severity of first episode	290
Severity of worst accidental pain	296
Severity of worst internal pain	294
Severity of worst external pain	297
Percent active coping strategies	291
MSPQ	299
Life events	299

Table 3.1 (continued)

History of legal advice	295
Disability	298
Whether on sick leave*	207

Two months follow-up data (162 subjects interviewed, 159 examined)

Variable	Frequency
Severity of present attack	160
Disability	161
Whether on sick leave*	108
Oswestry disability	160
Pain drawing	153
Zung	159
Neurological deficit	159
Inappropriate signs and symptoms	159
BMI	157
Side flexion	159
Sagittal movement	159
Straight leg raise	159
Ability to sit up	159

Twelve months follow-up data (196 subjects)

Severity of present pain	196
Disability	196
Sick leave status*	179
History of pain	196

* Question concerning sick leave status is only appropriate for those in paid employment at the time of interview.

Figure 3.1: Information/measures collected on patients at the three data collection points.

VARIABLES	SCREENING QUESTIONNAIRE	TWO MONTHS FOLLOW-UP	TWELVE MONTHS FOLLOW UP	POSTAL FOLLOW-UP
	Age Sex Doctor Employment Marital status Smoking habits History and severity of previous LBP Severity of present LBP Onset	Screening questionnaire Plus Pain drawing Modified Zung Disability (Oswestry) Non-organic symptoms " " signs Body Mass Index	Two months follow-up variables Plus Course of LBP	Level of pain Disability Whether off work Course of LBP
FAM* -	Worst accidental pain	Neurological tests		
FAM* -	Worst internal pain	Straight leg raise		
FAM* -	Worst external pain	Prone knee bend		
FAM* -	Coping strategies	Hip flexion		
FAM* -	Stressful life events	Sit up		
FAM* -	MSPQ	Lateral flexion		
	Disability	Sagittal movement		
	Whether off work because of pain	Area affected		
		Clinical diagnosis by Doctor		
NUMBER OF PATIENTS	300	162 Interviewed (159 examined)	58 plus 196	138

* FAM = Fear Avoidance Model variable

CHAPTER 4

THE INTRA-OBSERVER REPEATABILITY OF THE ANTHROPOMETRIC VARIABLES MEASURED IN THE STUDY.

4.1 INTRODUCTION

Measurement of lateral lumbar flexion, straight leg raise, prone knee bending and lumbar sagittal movement is commonly undertaken in clinical practice. The results of these measurements are usually used to add to the diagnostic power of other observations and special tests or to aid in the evaluation of clinical outcome. Therefore, they are are often estimated by eye. However, the physical measurements used in this study were chosen so that the relationship between physical, functional and psychological variables in patients with low back pain could be explored. Several studies have been conducted in order to test the reliability and repeatability of physical measurements related to spinal function (Reynolds, 1975, Anderson and Sweetman, 1975, Mayer et al., 1984, Burton, 1986, Mellin, 1989, Porter, 1989, Newton and Waddell, 1991). However, these studies evaluated several different measurement techniques and methods differed between studies. It was therefore thought necessary to conduct a study designed to test the intra-observer repeatability of the anthropometrics to be used in the present low back pain study.

The physical measurements concerned were straight leg raise (SLR), prone knee bending (PKB), lateral lumbar flexion and sagittal flexion and extension.

4.2 METHOD

Eighteen physiotherapy students (15 female, 3 male, mean age 19.5 years, SD = 4.617 years) were assessed by the author. They were seen in three groups over three consecutive days. The measurement techniques have been described in Chapter 3.

The measurements were taken again three weeks later in the same order, again over three days, and at the same time of day. (The subjects had been instructed to try to reproduce their 24 hour pre-measurement activity level in order to avoid bias resulting from 'exercise induced' stiffness. However the subjects from the second day's follow-up had been subjected to a rigorous exercise class the day before.)

Thirty-six measurements for each technique were entered into the analysis except left straight leg raise at re-test. This technique had one missing value as a result of a hamstring injury in one subject. (The missing value was assigned the mean result for that measurement.)

4.3 STATISTICAL ANALYSIS

Descriptive statistics were calculated for all measurement techniques (Table 4.1).

Pearson's Correlation Coefficient (r) was calculated for each test/retest pair of measurements.

The Least Significant Difference (LSD) was calculated for each technique using the following formula:

LSD = t * sd

(Where t is derived from t test tables (two tailed) at, in this case the 5% significance level, with degrees of freedom equal to number of subjects minus one. For 18 subjects t = 2.110. SD = standard deviation of test/retest differences.)

LSD has been shown to be a meaningful statistical technique when used to evaluate physical measurements in terms of repeatability (Bland and Altman, 1986, Tillotson and Burton, 1990). It has been argued that a statistically significant correlation coefficient can conceal serious differences between repeated measures. A correlation coefficient expresses the strength of association between two measures. LSD, however, represents the closeness of both measures to a graphical line

of equality where test and re-test represent the x and y axes (Bland and Altman, 1986).

Another feature of LSD concerns the fact that it is expressed in the appropriate units of measurement. A given LSD for a physical measurement represents the extent to which repeated measures must differ for the difference to be statistically significant; conversely test/retest variations less than the value of the LSD cannot be considered to be different (in this case at the 5% significance level).

4.4 RESULTS

Correlation were high and statistically significant (r = 0.61 to 0.89) for all measures except upper lumbar flexion (r = 0.13) which was not statistically significant (Table 4.2).

However, LSD was also high for most measurement techniques (Table 4.2).

The results were similar on the left and right side for SLR, lateral flexion and prone knee bending (Tables 4.1 and 4.2).

It was confirmed that a high value for LSD does not mean that a low level of correlation exists between the test/retest of that measure (Bland and Altman, 1986). A high r can in fact conceal substantial variability between repeated measures.

4.5 DISCUSSION

The results of this study suggest that the repeatability of techniques which measure the ranges of SLR, PKB, lateral flexion and sagittal mobility of the lumbar spine is lower than that found by other workers using similar techniques (Porter, 1989, Tillotson, 1991). This may in part be explained by the error introduced by the effects of the exercise class that several of the subjects took part in the day before they were re-tested. An additional source of reduced repeatability may involve the length of time between test and re-test. For example, Tillotson and Burton (1991) only allowed between five and 30 minutes between test and re-test. However, the author considered a rest/retest period of three weeks to more appropriate considering that the test/re-test time in the low back pain study was intended to be 10 months.

It was intended that the results of this test/retest study would be used in the interpretation of the results of the back pain study. However, the attrition rate between two and 12 months led to a change in the study design which involved the use of postal questionnaires to collect 12 months data. Subjects were not examined a second time at the twelve months stage. The results of the predictive analyses, presented in Chapter 8 demonstrate that the physical measurements used in this test/retest study can be useful in terms of prediction despite their apparently low repeatability. This may be due to

the nature of the analyses in which the measurements of over 100 subjects are included. This may lead to the 'cancelling out' of the low repeatability demonstrated by their LSDs.

However, despite the change in method after the test/retest study was completed, the results are still of interest. It is worthy of note that these techniques are widely used in orthopaedic and physiotherapy departments and contribute to the process of clinical decision making. The results of this study suggest that care needs to be taken in the interpretation of these measurement techniques in a clinical environment.

TABLE 4.1

Descriptive statistics of lateral flexion, straight leg raise, prone knee bend and sagittal movement of the lumbar spine test and re-test results.

	TEST		RETEST	
Measurement	Mean	SD	Mean	SD
Lateral flexion				
Right	22.94 cm	3.07	22.72 cm	3.06
Left	22.72 cm.	2.54	22.44 cm	3.01
Straight Leg Raise				
Right	74.05 deg	16.10	74.44 deg	12.78
Left	73.70 deg	15.87	72.58 deg	12.58
Prone knee bend				
Right	141.61 deg	9.81	140.88 deg	10.47
Left	141.33 deg	9.18	140.00 deg	10.38
Lumbar Flexion				
Upper	14.27 deg	3.83	15.94 deg	3.97
Lower	7.88 deg	3.83	8.83 deg	3.24
Lumbar Extension				
Upper	28.27 deg	8.51	29.22 deg	11.75
Lower	15.66 deg	6.94	16.61 deg	6.40

TABLE 4.2

Intra-observer repeatability of lateral flexion, straight leg raise test, prone knee bend and sagittal movement of the lumbar spine using Pearson's correlation coefficient (r) and least significant difference (LSD)

Measurement	r (P)	LSD
Lateral Flexion		
Right	0.89 (P = 0.000)	3.0 cm
Left	0.78 (P = 0.000)	4.0 cm
Straight Leg Raise		
Right	0.86 (P = 0.000)	17.4 deg
Left	0.83 (P = 0.000)	18.9 deg
Prone Knee Bend		
Left	0.61 (P = 0.004)	18.9 deg
Right	0.69 (P = 0.001)	16.5 deg
Lumbar Flexion		
Upper	0.80 (P = 0.000)	5.2 deg
Lower	0.13 (P = 0.302)	9.9 deg
Lumbar Extension		
Upper	0.64 (P = 0.002)	19.2 deg
Lower	0.69 (P = 0.001)	11.1 deg

CHAPTER 5

DESCRIPTIVE RESULTS

5.1 INTRODUCTION

The second aim of the study was to describe a sample of acute low back pain patients presenting to their General practitioners. This chapter represents the fulfillment of that aim. In addition, this chapter reports the test of the fourth hypothesis of the study, 'the large majority of acute low back patients are free of physical signs and psychopathology'.

To the author's knowledge, the data set collected in this study represents the largest of its kind to date. In addition, subjects were interviewed at an earlier stage in the course of their episode of back pain than in any other study and, unlike many studies of low back pain, the sample consist entirely of patients presenting to general practice. For these reasons the descriptive statistics of the sample at each stage of the study are presented in full in this chapter.

In addition, sub-groups within the sample were compared in terms of potential predictor variables in order to i) assess the sample in terms of homogeneity and ii) make inferences from any demonstrated sub-group differences in terms of the

aetiology of low back pain. Therefore, the chapter includes a comparison of males and females, age groups, occupational and social groups, work status groups, smokers and non-smokers and subjects with and without a history of back pain and attenders and non-attenders at each stage of the study.

Finally, the repeatability of retrospective Fear Avoidance Model variables and retrosective pain history variables was tested, along with the inter-observer repeatability of the Waddell Innapropriate Signs variable.

Analysis approach

Descriptive statistical analyses were carried out on the sample at each stage of the study. These results are presented and discussed in this chapter.

In the analyses of sub-groups, multivariate procedures were carried out in order to identify overall group differences. If omnibus procedures identified significant differences between groups, univariate analyses were conducted. Significant results are presented and discussed in the text of this chapter. All univariate sub-group analyses results are presented in Appendices F anf G.

5.2 SCREENING DATA DESCRIPTIVE STATISTICS

Three hundred acute low back pain subjects (151 males, 149 females) were included in the study.

Sources of referral: Patients were referred from six health centres. Forty two per cent of the subjects were referred from one practice, Maghull Health Centre. Forty seven GPs referred subjects into the study. Fifity eight per cent of the subjects were referred by a group of eight GPs. Over twenty-three percent of the subjects were referred by two of the GPs (C and H) (Table 5.1).

Age: The mean age of the subjects included in the study was 42.7 years (SD = 14.0 years), the age range was 65 years, the minimum age being 13 years and the maximum 78 years (Table 8.3). The mean age of male subjects was 42.9 years (SD = 15.2 years) and the mean age of female subjects was 42.5 years (SD = 12.7 years). Sixty percent of the subjects were aged between 29 and 55 years

Social Group: Seventy three percent of the subjects interviewed were in full or part time paid employment. Categorization of subjects into social groups was based upon the Registrar General's Classification of Employment (1989). The categories identified and the frequency distribution are presented in Table 5.2.

Sick leave: Fifty eight per cent of those in the sample who were in paid employment were on sick leave at the time of interview because of their back pain. (Table 5.18).

Marital status: Sixty-nine percent of the sample were married and 20% were single at the time of entry into the study. The remaining 11% consisted of subjects who were divorced (6%), widowed (2%), separated (1%) and cohabiting (2%). Frequencies of marital status are presented in Table 5.3.

Smoking: Sixty three percent of the sample were non smokers. 13.7 percent smoked between one and 10 cigarettes per day, 18.3% between 11 and 20 per day, 3% between 21 and 30 per day and 1.7% between 31 and 40 per day. Only one subject smoked more than 40 cigarettes per day (Table 5.4).

Previous low back pain: Two hundred and forty-five (81.7%) of the sample reported that they had suffered from previous episodes of low back pain.

First attack of low back pain: Eighteen percent had never experienced back pain before the present episode. Nine per cent had experienced their first attack of back pain less than one year previously and 24% had experienced their first attack of low back pain between one and five years previously. Forty nine percent of the sample had suffered their first attack of low back pain more than five years previously (Table 5.5).

Severity of first attack: The mean severity of the first attack of low back pain was 6.5 (SD = 2.3) on a scale of 1-10 (Table 5.6)

Severity of worst attack: The mean severity of the worst attack of back pain was 8.4 (SD = 1.6) on a scale of 1-10 (Table 5.6).

Severity of present attack: The mean severity of the present attack of low back pain was 6.3 (SD = 2.5) on a scale of 1-10 (Table 5.18).

Cause of onset: One hundred and eighty-eight (63.9%) of the sample reported that the onset of the present episode of low back pain had been insidious. The remaining 106 (36.1%) attributed the onset of their back pain to some kind of injury.

Number of previous attacks of low back pain: More than seventeen per cent (17.7%) of the sample had never experienced back pain previously whereas 35.8% had experienced more than nine previous episodes (Table 5.7).

'Internally' produced pain (1-10): Table 5.8 shows that the mean 'highest ever internally produced' pain was 7.8 (SD = 2.2).

'Externally' produced pain (1-10): Table 5.8 the mean 'highest ever externally produced' pain was 6.9 (SD = 2.3).

'Accidentally' produced pain(1-10): Table 5.8 shows that the mean 'highest ever accidentally produced' pain was 7.3 (SD = 2.1).

Legal advice: Only nine subjects (3%) had ever taken legal or Trade Union advice regarding their low back pain.

Active coping strategies: The mean 'per cent active coping strategies' was 38.4% (SD = 29.4%). The scores ranged between 0% and 100%.

Life events: The mean number of stressful life events experienced in the preceding 12 months was 5.3 (SD = 3.5). The mean weighted life events score was 152.7 (SD = 105.2).

Modified Somatic Perception Questionnaire: The mean MSPQ was 5.3 (SD = 4.1). The maximum possible MSPQ score is 39. The scores ranged between 0 and 19.

Roland and Morris Disability Questionnaire: Table 5.17 shows that the mean disability measured by this questionnaire was 12.7 (SD = 5.7). The maximum possible value of this variable is 24.

5.3 TWO MONTH FOLLOW-UP DATA DESCRIPTIVE STATISTICS

One hundred and sixty-two subjects (88 males, 74 females, mean age = 44.9 years [sd = 13.9 years]) were re-interviewed two months after onset of low back pain. One hundred and fifty-nine of these were examined.

The results of two months screening measures are as follows:-

Sick leave: Of those who were in paid employment, 88.9% were not on sick leave because of their back pain at two months follow-up. Of the total group at two months follow-up, 59.3% were not on sick leave (Table 5.17).

Modified Somatic Perception Questionnaire: The mean MSPQ at two months after onset was 3.8 (SD = 3.7). The scores ranged between 0 and 19.

Roland and Morris Disability Questionnaire: The mean disability measured by the Roland and Morris Disability Questionnaire was 6.5 (SD = 6.3) (Table 5.17). The maximum possible score is 24.

Oswestry Disability Questionnaire: The mean disability measured by the Oswestry Disability Questionnaire was 16.3% (SD = 15.1%). Scores ranged between 0% and 56%.

Severity of present attack of low back pain: The mean severity (on a scale of 1-10) of low back pain at two months after onset was 3.1 (SD = 2.5) (Table 5.17). Forty percent of the group were no longer in pain.

Zung Depression Inventory: The mean depression score measured by the Zung Depression Inventory was 17.3 (SD = 10.7). The maximum possible score is 69. Scores ranged between 0 and 48.

Pain drawing: The mean pain drawing score was 6.717 (SD = 7.592). The range of scores was 58 (Minimum = 0, maximum = 58).

Physical examination descriptive statistics

Body mass index: The mean body mass index of the subjects examined was 25.5 (SD = 3.8).

'Inappropriate signs and symptoms': Forty-two per cent of the subjects examined two months after onset of back pain were free of inappropriate signs and symptoms. Only 10% of the group exhibited more than three inappropriate signs or symptoms out of a possible 15. The mean value was 1.5 (SD = 2.2) (Table 5.9).

Reflex strength: Eighty-nine percent of the subjects examined two months after onset of low back pain had normal lower limb reflexes (Table 5.10).

Muscle strength: Lower limb muscle strength was found to be normal in every patient examined at two months after onset of low back pain (Table 5.10).

Lower limb sensory loss: Almost fifteen per cent (14.5%) of the subjects examined two months after onset of low back pain were found to have some lower limb sensory loss in one or more dermatomes (Table 5.10).

Neurological deficit: Patients were considered to have neurological deficit associated with nerve root entrapment if they were found to have one or more of the previously described neurological signs. Table 5.10 shows that 75.5% of the group failed to exhibit any sign associated with neurological deficit.

Area of pain: Of the subjects still in pain at two months after onset, symptoms were equally distributed between the central, right and left areas of the low back (Table 5.11).

'Slump' test: Approximately one third (34%) of the group examined at two months after onset of pain had a positive slump test.

Straight leg raise: The mean left straight leg raise was 64.2 degrees (SD = 16.2 degrees) and the mean right straight leg raise was 63.2 degrees (SD = 16.0 degrees). Straight leg raise

on both sides was limited by soft tissue tightness rather than pain in 80% of cases (Table 5.12). For the purpose of analysis, the mean total straight leg raise was divided by two to give the mean straight leg raise.

Side flexion: Table 5.13 shows that the mean values for this variable were 17.4 cm (SD = 4.4 cm) on the left and 16.8 cm (SD = 4.8 cm) on the right.

Prone knee bending: Table 5.14 shows that the mean values for this measurement were 131.1 degrees (SD = 23.8 degrees) on the left and 133 (SD = 21.7 degrees) on the right.

Sagittal movement: Table 5.15 shows the mean values for upper and lower lumbar extension and upper and lower lumbar flexion. The mean value of lumbar extension was 38 degrees (SD = 11.2 degrees) and the mean value of lumbar flexion was 21.8 degrees (SD = 8.3 degrees). Mean overall sagittal movement was 59.7 degrees (SD = 15 degrees).

Sit up test: Over sixty-five per cent (65.4%) of subjects examined were able to sit up from the supine position.

Orthopaedic diagnosis: The orthopaedic surgeon examined 103 subjects at two months follow-up. Of these, 71 (69%) failed to present with signs and symptoms from which a differential diagnosis could be made (Table 5.16)

Outcome variables: At two months follow-up, the mean severity of present pain was 3.1(2.5), mean disability was 6.6(6.3) and 11% of subjects in work were on sick leave because of back pain. These results are presented in Table 5.18.

5.4 TWELVE MONTHS FOLLOW-UP DATA DESCRIPTIVE STATISTICS

One hundred and ninety six subjects (98 males, 98 females, mean age 43.3 years, [SD = 14.1 years]) were followed up one year after onset of low back pain.

The results in terms of outcome measures are as follows:-

Course of low back pain: Sixteen subjects (8.2%) followed up at 12 months after onset reported a history of constant pain since screening. Thirty-five subjects (18.1%) reported that their pain had "stayed away all the time" after the acute phase had resolved. The remaining 142 (73.8%) had been bothered by low back pain "on and off" (Table 5.17). Three subjects failed to complete this item on the postal questionnaire

Sick leave: Of those who were in paid employment (N = 179), 154 (85.1%) were not on sick leave because of their back pain at twelve months follow-up (Table 5.18).

Low back pain: The mean reported pain twelve months after onset was 3.44 (SD = 2.44) (Table 5.18).

Roland and Morris Disability Score: The mean score of this variable was 5.76 (SD = 5.50) (Table 5.18).

Table 5.1

SOURCES OF REFERRAL

Maghull	126 (42.0%)
High Pastures	4 (1.3%)
Old Swan	34 (11.3%)
Park Road	85 (28.3%)
Aigburth Road	1 (0.3%)
Prince's Park	31 (10.3%)
Longmoore Lane	19 (6.3%)
Total	300
Doctor A	20 (6.7%)
Doctor B	10 (3.8%)
Doctor C	32 (12.2%)
Doctor D	21 (8.0%)
Doctor E	13 (4.9%)
Doctor F	13 (4.9%)
Doctor G	15 (5.7%)
Doctor H	30 (11.4%)
Total	154 (57.6%)
Remainder (N=39)	146 (42.4)
Total	300

Table 5.2

SOCIAL GROUP BASED ON OCCUPATION

I	16 (5.3%)
II	39 (13.0%)
III	64 (21.3%)
IV	48 (16.0%)
V	53 (17.7%)
VI	25 (8.3%)
VII	18 (6.0%)
VIII	1 (0.3%)
IX	5 (1.7%)
X	31 (10.3%)
Total	300

I = Professional, II = Intermediate, III = Skilled manual, IV = Semi-skilled, V = Unskilled, VI = Home maker, VII = Unemployed, VIII = Student, IX = Disabled (not because of back pain), X = retired

Table 5.3

MARITAL STATUS

Married	207 (69.0%)
Single	61 (20.3%)
Divorced	17 (5.7%)
Widowed	7 (2.3%)
Separated	3 (1.0%)
Cohabiting	5 (1.7%)
Total	300

Table 5.4

SMOKING

Non-smokers	189	(63.0%)
1-10/day	41	(13.7%)
11-20/day	55	(18.3%)
21-30/day	9	(3.0%)
31-40/day	5	(1.7%)
41+	1	(0.3%)
Total	300	

Table 5.5

TIME SINCE FIRST ATTACK OF LOW BACK PAIN

No previous	55	(18.3%)
Less than 1 year previously	25	(8.4%)
1-2 years previously	30	(10.0%)
2-3 years previously	17	(5.7%)
3-4 years previously	12	(4.0%)
4-5 years previously	14	(4.7%)
More than 5 years previously	147	(49.0%)
Total	300	

Table 5.6

SEVERITY OF PREVIOUS ATTACKS OF LOW BACK PAIN (1-10)

	Mean	SD	Min	Max
First attack	6.5	(2.3)	1	10
Worst attack	8.4	(1.6)	3	10
(Present attack)	6.3	(2.5)	1	10

Table 5.7

NUMBER OF PREVIOUS ATTACKS OF LOW BACK PAIN

0	52 (17.7%)
1	33 (11.3%)
2	33 (11.3%)
3	20 (6.7%)
4	15 (5.1%)
5	19 (6.5%)
6	9 (3.1%)
7	3 (1.0%)
8	4 (1.4%)
9+	105 (35.8%)
Total	293

Table 5.8

PAIN HISTORY

	Mean	SD
Highest 'internally' produced pain (1-10)	7.8	2.2
Highest 'externally' prod·· ·ᵈ pᵃin (·-)	6.9	2 3
Highest 'accidentally' produced pain (1-10)	7 ᵌ	2.1

Table 5.9

FREQUENCY OF INAPPROPRIATE SIGNS AND SYMPTOMS

0	69	(42.8%)
1	40	(25.2%)
2	19	(11/9%)
3	15	(9.4%)
4	5	(3.1%)
5	1	(0.6%)
6	3	(1.9%)
7	3	(1.9%)
8-13	0	
14	1	(0.6%)
15	1	(0.6%)
Total	157	

Table 5.10

NEUROLOGICAL SIGNS

Reflex strength	normal	143 (89.9%)
	abnormal	16 (10.1%)
Muscle strength	normal	159 (100%)
Sensation	no deficit	136 (85.5%)
	deficit	23 (14.5%)
Neurological deficit	absent	120 (75.5%)
	present	39 (24.5%)

Table 5.11

AREA OF PAIN

No pain	64 (40.3%)
Right side pain	23 (14.5%)
Left side pain	22 (13.8%)
Central pain	48 (30.2%)
Bilateral leg pain	2 (1.3%)
Total	159

Table 5.12

STRAIGHT LEG RAISE (IN DEGREES) LEFT (L) AND RIGHT (R)

	Mean	SD	Range	Min	Max
L	64.2	16.1	90	10	100
R	63.2	16.0	90	10	100
Average	63.7	15.5	90	10	100

Table 5.13

SIDE FLEXION LEFT (L) AND RIGHT (R)

	Mean	SD	Range	Min	Max
L	17.4cm	4.5cm	28cm	7cm	35cm
R	16.7cm	4.8cm	28cm	4cm	32cm
Average	34.3cm	8.8cm	55cm	12cm	76cm

Table 5.14

PRONE KNEE BENDING (IN DEGREES) LEFT (L) AND RIGHT (R)

	Mean	SD	Range	Min	Max
L	131.1	23.8	130	30	160
R	133.0	21.7	130	30	160
Average	132.3	21.8	130	30	160

Table 5.15

SAGITTAL MOVEMENT (IN DEGREES)

	Mean	SD	Range	Min	Max
Upper lumbar extension	21.4	8.3	50	0	50
Lower lumbar extension	16.6	6.7	41	1	42
Lumbar extension	38.0	11.2	68	3	71
Upper lumbar flexion	12.8	6.2	31	0	31
Lower lumbar flexion	8.9	6.2	31	0	50
Lumbar flexion	21.8	8.3	50	0	50
Total	59.7	15.0	84	20	104

Table 5.16

DIAGNOSTIC CATEGORIES OF SUBJECTS EXAMINED BY ORTHOPAEDIC REGISTRAR

Prolapsed intervertebral disc with nerve root entrapment	11	(10.7%)
Probable prolapsed intervertebral disc with nerve root entrapment	21	(20.4%)
Low back pain of unknown cause	71	(69.0%)

Table 5.17

COURSE OF LOW BACK PAIN FROM ONSET TO TWELVE MONTHS FOLLOW-UP

Pain stayed away	35	(19.2%)
Pain 'on and off'	142	(73.5%)
Constant pain	16	(8.3%)
Total	193	

Table 5.18

COMPARISON OF THREE OUTCOME VARIABLES AT SCREENING, TWO MONTHS AND ONE YEAR

	Severity of present back pain (0-10)	Disability	Sick leave
Screening (n = 300)	6.3 (SD = 2.5)	12.7 (SD = 5.7)	58% (n = 207)
2 months (n = 162)	3.1 (SD = 2.5)	6.6 (SD = 6.3)	11% (n = 108)
12 months (n = 196)	3.4 (SD = 2.4)	5.6 (SD = 5.5)	14% (n = 179)

5.5 SUBGROUP DIFFERENCES IN SCORES OF SCREENING VARIABLES

Sex differences

A Multivariate Analysis of Variance (MANOVA) was performed, using sex as the independant variable and the Fear Avoidance Model variables, functional variables and historical variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between males and females in terms of the variables included in the analyses (Hotellings = 0.245, df = 266, P = 0.001).

Univariate tests (two-tailed T-tests and Fisher's exact tests) were performed in order to examine univariate differences between males and females.

The 'percent active coping strategies' scores for males were significantly higher than those of females (t = 2.04, P = 0.042).

The reported severity of the worst ever 'internally produced pain' and 'externally produced pain' were significantly higher for females than males. (t = -3.14, p = 0.002 and t = -2.16, P = 0.01).

The reported severity of the present and worst ever attacks of low back pain was significantly higher for females ($t = -2.51$, $P = 0.013$ and $t = -3.52$, $P = 0.001$ respectively).

Females had significantly higher MSPQ scores than males ($t = -5.56$, $P = 0.001$).

The referring practice variable was collapsed and dichotomised in order to perform a Fisher's exact test, testing referring practice with sex in order to identify significant differences.

The referring practice categories consisted of Maghull Health Centre ($n = 125$) and the remaining practices ($n = 175$). Significantly fewer females were referred by doctors at Maghull Health Centre than by doctors at the remaining practices ($P = 0.001$).

The employment category variable was collapsed into three categories in order to identify sex differences for this variable. The employment categories consisted of groups I to III, IV and V and those not at work. No significant differences were demonstrated between the sexes in terms of this variable.

Injury was more likely to be the cause of onset of the present episode of low back pain amongst the male group than the female group ($P = 0.039$).

Females reported a significantly higher incidence of low back pain in the past (P = 0.038).

No other statistically significant differences were demonstrated.

These results are presented in Appendix F (Tables F1 and F2).

Work status differences

A MANOVA was performed, using work status as the independant variable and the Fear Avoidance Model variables, functional variables and historical variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between subjects who were on sick leave and those who were not in terms of the variables included in the analyses (Hotellings $= 0.171$, $df = 188$, $P = 0.001$).

Univariate tests (two-tailed T-tests and Fisher's exact tests) were performed in order to examine univariate differences between those on sick leave and those who were not.

The reported severity of the present attack of low back pain and the Roland and Morris disability score were significantly

higher for the individuals who were on sick leave (t = -2.34, P = 0.021 and t = -4.34, P = 0.001 respectively).

A higher proportion of those on sick had experienced back pain in the past than those not on sick (P = 0.026).

A higher proportion of subjects who belonged to occupational groups IV and V were on sick leave from work than subjects from groups I to III.

No other statistically significant differences were demonstrated.

These results are presented in Appendix F (Tables F3 and F4).

Smoking habits

A MANOVA was performed, using smoking as the independant variable and the Fear Avoidance Model variables, functional variables and historical variables measured at screening as dependent variables.

This procedure demonstrated no statistically significant differences between smokers and non-smokers in terms of the variables included in the analyses (Hotellings = 0.532, df = 232, P = 0.345).

Univariate tests (two-tailed T-tests and Fisher's exact tests) were performed in order to examine univariate differences between smokers and non-smokers.

These results are presented in Appendix F (Tables F5 and F6).

Age differences

The screened sample was divided into three groups according to age. The first group was aged between 13 and 40 years (47.1%), the second group was aged between 41 and 59 years old (36.7%) and the third group was aged between 60 and 78 years old (16.2%). These groups were selected to represent the age groups whose low back pain may have different aetiologies and pathologies.

A MANOVA was performed, using age as the independant variable and the Fear Avoidance Model variables, functional variables and historical variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between subjects in the three age groups in terms of the variables included in the analyses (Exact F = 0.257, df = 265, P = 0.001).

Univariate tests (one-way analysis of variance (ANOVA) and Fisher's exact tests) were performed in order to examine univariate differences between the three age groups described above.

ANOVA revealed significant differences between the age groups on scores of 'highest ever internally produced pain' (F = 3.27, df = 2,288, P = 0.039). The intermediate age group had the highest mean score.

Younger subjects scored more highly on weighted life events. ANOVA revealed significant differences between the groups (F = 12.16, df = 2,293, P = 0.001).

The proportion of the intermediate age group who were females was higher than in the younger and older age groups (p = 0.021) and the patients referred by Maghull Health Centre tended to be older than those from the other practices (P = 0.001).

The younger patients were more likely to belong to professional and skilled occupational groups (P = 0.001).

Injury was the cause of onset of present pain in 75% of the intermediate and older groups. However injury was the cause of pain in only 37% of the younger patients. This difference was statistically significant (P = 0.004).

Subjects in the oldest age category were significantly more likely to have had a long history of repeated episodes of low back pain (P = 0.001, P = 0.002).

No other statistically significant differences were demonstrated

These results are presented in Appendix F (Tables F7 and F8).

History of low back pain differences

The screened sample was divided into three groups according to time since the onset of their first attack of low back pain. The first group had never had back pain before (18%), the second group had experienced back pain for the first time between one and five years previously (32%) and the third group had experienced their first attack of back pain more than five years previously (49%).

A MANOVA was performed, using time since onset as the independant variable and the Fear Avoidance Model variables,

functional variables and historical variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between time since onset groups in terms of the variables included in the analyses (Exact F = 6.90, df = 268, P = 0.001).

Univariate tests (ANOVA and Fisher's exact tests) were performed in order to examine univariate differences between time since onset groups.

The remembered severity of past episodes of pain and weighted life events scores was higher amongst the subjects with a history of low back pain. ANOVA revealed significant differences between the time since onset groups and score of 'highest ever internally produced pain' (F = 6.057, df = 2,291, P = 0.003), 'highest ever externally produced pain' (F = 3.57, df = 2,294, P = 0.029), severity of worst attack of low back pain (F = 22.23, df = 2,289, P = 0.001) and weighted life events score (F = 4.11, df = 2, 296, P = 0.017).

Smoking was positively related to the length of time since the first episode of low back pain (P = 0.047).

The number of previous episodes was also positively related to the time since the first episode of low back pain (P = 0.001).

No other statistically significant results were demonstrated.

These results are presented in Appendix F (Tables F9 and F10).

Occupational differences

The sample was divided into three occupational groups. The first group consisted of those individuals who were categorised as belonging to social groups I to III (40.1%). The second group consisted of those who belonged to social groups IV and V (33.7%) and the third group consisted of the individuals who were not in paid employment (26.2%).

A MANOVA was performed, using occupational group as the independant variable and the Fear Avoidance Model variables, functional variables and historical variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between occupational groups in terms of the variables included in the analyses (Exact F = 2.57,, df = 265, P = 0.001).

Univariate tests (ANOVA and Fisher's exact tests) were performed in order to examine univariate differences between males and females.

One-way ANOVA revealed significant differences between the groups on scores of severity of 'highest ever internally produced pain' ($F = 3.77$, $df = 2,291$, $P = 0.024$).

The weighted life events score was highest for those in social groups I to III and lowest for those not at work ($F = 4.08$, $df = 2,296$, $P = 0.018$).

Subjects in social groups I, II or III were less likely to be on sick leave from work than subjects in social groups IV and V ($P = 0.009$).

These results are presented in Appendix F (F11 and F12).

5.6 SUBGROUP DIFFERENCES IN SCORES OF PHYSICAL VARIABLES

Sex differences

A MANOVA was performed, using sex as the independant variable and the physical variables as dependent variables.

This procedure demonstrated statistically significant differences between males and females in terms of the variables included in the analyses (Hotellings = 0.111, $df = 147$, $P = 0.046$).

Univariate tests (two-tailed T-tests and Fisher's exact tests) were performed in order to examine univariate differences between males and females.

The "innapropriate signs and symptoms" score of females was significantly higher than that of males (P = 0.001).

No other statistically significant differences were demonstrated.

These results are presented in Appendix F (Tables F13 and F14).

Age differences

A MANOVA was performed, using age group as the independant variable and physical variables as dependent variables.

This procedure demonstrated statistically significant differences between males and females in terms of the variables included in the analyses (Exact F = 5.55, df = 156, P = 0.001).

Univariate tests (ANOVA and Fisher's exact tests) were performed in order to examine univariate differences between males and females.

One way ANOVA revealed statistically significant differences between the three age groups on all measures of mobility and passive knee flexion. Age was inversely related to mobility.

The Body Mass Index of the intermediate age group was greater than that of the young and old age groups ($P = 0.002$).

Fisher's exact test revealed that subjects who were able to sit up from a supine position were significantly younger than subjects who could not ($P = 0.001$).

These results are presented in Appendix F Tables F15 and F16).

5.7 REFERRING PRACTICE DIFFERENCES

In view of the different demographic characteristics of the area in which patients from Maghull Health Centre live, and the influence of referring practice on attendance (see section 5.8) it was decided to compare patients from Maghull with patients from the city practices in terms of screening variables.

A MANOVA was performed, using referring practice as the independant variable and the Fear Avoidance Model variables, functional variables and historical variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between subjects from Maghull and subjects from city practices in terms of the variables included in the analyses (Hotellings = 0.123, df = 252, P = 0.003).

Two-tailed T-tests revealed statistically significant differences between Maghull patients and city patients on severity of worst ever attack of low back pain (t = -0.56, P = 0.047), MSPQ (t = -4.05, P = 0.001), severity of worst accidentally produced pain (t = -2.73, P = 0.007) and weighted life events score (t = -2.14, P = 0.033). Maghull Health Centre patients had lower scores on all of these variables.

No other statistically significant differences were demonstrated.

These results are presented in Appendix F (Table F17).

5.8 DIFFERENCES BETWEEN ATTENDERS AND NON ATTENDERS AT EACH STAGE OF THE STUDY

In view of the attrition rate at each stage of the study, and the decision to collect twelve months follow-up data by post and 'face to face', attenders were compared with non-attenders and non-respondents. Subjects who visited their GP practice to be re-interviewed and examined at two months follow-up and twelve months follow-up were labelled 'attenders'. Subjects who failed to visit their GP practice to be re-interviewed at two months or twelve months follow-ups were labelled as 'non-attenders'. Non-attenders who returned the postal questionnaire at twelve months follow-up were labelled as 'respondents'. Comparisons were made in terms of demographic, physiological and psychological data.

Comparison of attenders and non-attenders at the two months follow-up:

A Multivariate Analysis of Variance (MANOVA) was performed, using attendance as the independant variable and demographic, physiological and psychological variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between attenders and non attenders in terms of the

variables included in the analyses (Hotellings = 6.90, df = 268, P = 0.001).

Univariate tests (two-tailed T-tests and Fisher's exact tests) were performed in order to examine univariate differences between two months attenders and non-attenders.

Attenders were statistically significantly older (t = -2.85, P = 0.005) than non-attenders.

Patients from Maghull Health Centre were less likely to drop out of the study at the two months follow-up stage than patients from city practices (Exact P = 0.001).

A higher proportion of non attenders smoked than attenders at the two months follow-up. (Exact P = 0.001).

No other significant differences were demonstrated between attenders and non-attenders at two months follow-up.

These results are presented in Appendix G (Tables G1 and G2).

Comparison of subjects from whom twelve months follow-up data were collected by post or attendance (providers) with subjects for whom no follow-up data were collected (non-providers):

A MANOVA was performed, using provision of data as the independant variable and demographic, physiological and psychological variables measured at screening as dependent variables.

This procedure demonstrated statistically significant differences between providers and non-providers in terms of the variables included in the analyses (Hotellings = 0.245, df = 266, P = 0.001).

Univariate tests (two-tailed T-tests and Fisher's exact tests) were performed in order to examine univariate differences between two months attenders and non-attenders.

Subjects who were not working at the screening stage provided proportionately fewer 12 month follow-up data sets than subjects who were working (P = 0.019).

The mean sagittal mobility of non-providers was statistically significantly higher than that of providers of data (P = 0.006).

Married subjects were significantly more likely to provide data at 12 months follow-up (P = 0.035).

No other significant differences were demonstrated between the screening data collected from subjects who provided follow-up data and those who did not.

These results are presented in Appendix G (Table G3 and G4).

Comparison of postal respondents and attenders at twelve months follow-up:

One hundred and ninety six data sets were collected at twelve months follow-up. One hundred and thirty eight of these were collected by post.

Attenders were significantly older than postal respondents (t = 2.47 P = 0.017).

The mean severity of the worst attack of low back pain reported by attenders was significantly lower than that of postal respondents (t = -2.26, P = 0.025).

The attendance rate of Maghull Health Centre patients at twelve months follow-up was significantly higher than that of patients from city practices (Exact P = 0.001).

Married subjects were significantly more likely to be attenders ($P = 0.028$).

Number of previous episodes of low back pain was positively related to attendance at twelve months, with attenders reporting a higher incidence of previous episodes ($p = 0.001$).

Of the total twelve months follow-up group, 114 had attended for physical examination at two months. Of these, 55 attended for twelve months follow-up. The range of sagittal mobility of the subjects who attended at twelve months was significantly higher than the range of sagittal mobility of the postal respondents ($t = -4.10$, $P = 0.001$).

No other statistically significant differences between postal respondents and attenders at twelve months follow-up were demonstrated.

These results are presented in Appendix G (Table G5 and G6).

Twelve months outcome measures and differences between postal respondents and attenders:

Four variables formed the basis of the evaluation of twelve months outcome. These were the natural history of the low back pain since entry into the study, whether on sick leave because

of back pain, severity of present pain, and disability score (see Chapter 3).

Sixteen subjects reported experiencing constant pain over the 12 months study period. All of these subjects were postal respondents ($P = 0.001$).

The mean severity of present attack of low back pain and mean disability score of postal respondents were statistically significantly higher than those of attenders ($t = -4.13$, $P = 0.001$ and $t = -2.38$, $P = 0.010$ respectively).

These results are presented in Tables 5.19 and 5.20.

Table 5.19.

Comparison of twelve month attenders and twelve months postal respondents in terms of twelve month outcome measures (P = postal, A = attenders)

Variable		N	Mean	SD	t value	P
Severity of present LBP	A	58	2.40	1.97	-4.13	0.001
	P	137	3.91	2.46		
Disability score	A	57	4.31	4.52	-2.38	0.010
	P	137	6.35	5.77		

Table 5.20.

Comparison of twelve month attenders and twelve months postal respondents in terms of twelve month outcome measures (P = postal, A = attenders) (nominal variables)

	Postal	Attenders	
Not on sick leave	114	40	
On sick leave	22	3	
			Exact P = 0.140
Recoverd completely	14	21	
Pain "on and off"	107	35	
Constant pain	16	0	
			Exact P = 0.001

5.9 THE TEST/RETEST REPEATABILITY OF FEAR AVOIDANCE MODEL VARIABLES AND PAIN HISTORY VARIABLES AND THE INTER-OBSERVER REPEATABILITY OF INAPPROPRIATE SIGNS SCORE

The test/retest repeatability of highest ever externally, internally and accidentally produced pains and the severity of first and worst ever attacks of low back pain was evaluated.

In addition, the inter-observer repeatability of the inappropriate signs score was evaluated.

The repeatability of these variables was evaluated because the pain experience variables were concerned with events which happened in the past. These events were considered unlikely to recur between the acute and two months follow-up stages, whereas variables such as severity of present pain score or disability score were considered likely to change.

The physical examination of subjects had been conducted by the author and the Senior Registrar in Orthopaedics at two months follow-up. Both examiners had administered the tests designed to evaluate inappropriate signs (see Chapter 3) so that interobserver repeatability of this variable could be evaluated.

Repeatability was evaluated using Pearson's correlation coefficient.

Results

Pearson's correlation coefficients between each pair of variables were statistically significant ($P < 0.001$). Coefficients ranged from 0.799 (severity of worst attack of low back pain) to 0.292 (percent active coping strategies). These results are presented in Table 5.44.

Table 5.44

Repeatability of pain history and inappropriate signs variables using Pearson's correlation coefficient.

Variable	r	P
Severity of worst attack (1-10)	0.80	< 0.001
Severity of first attack (1-10)	0.73	< 0.001
Highest externally produced pain (1-10)	0.61	< 0.001
Highest internally produced pain (1-10)	0.53	< 0.001
Highest accidentally produced pain (1-10)	0.52	< 0.001
Percent active coping strategies (0-100%)	0.29	< 0.001
Inappropriate signs (0-8)	0.65	< 0.001

5.10 DISCUSSION

5.10i Descriptive data

The purpose of the collection and analysis of descriptive data was twofold.

First, the generalizability of the results of the study depend upon the representativeness of the sample in terms of the population of acute low back pain patients who visit their general practitioners.

Second, as the data set collected represents the largest study of acute (less than two weeks after onset) low back pain patients presenting to general practice to date it is important to describe the characteristics of subjects so that the level of physical and psychological dysfunction of this pathology group can be elucidated.

It is probable that general practitioners see a sample of patients with back pain which is not representative of the experience of this symptom in the general population (Morrell and Whale, 1976); the 10% of patients who chose to consult their GP may be different in terms of the nature of the pain and levels of anxiety about its significance.

The results of the descriptive analyses suggest that the sample is representative of the population of acute low back pain patients who present to their general practitioner.

The study was designed to recruit back pain sufferers from practices which, together, were broadly representative of the population of the Mersey Region. However, the recruitment of subjects was uneven in terms of the practices from which they were recruited and the GPs who referred them into the study. Nevertheless, in terms of age, gender, smoking habits and social class the resulting sample is representative of the general population (The General Household Survey, 1988 and 1989). Roland and Morris (1983) studied 252 acute low back pain patients. Their average age was 40.6 years (42.7 in this study), eighty seven percent were employed (73.3% in this study). Fifty three percent were engaged in manual occupations (46% in this study).

The sample comprises subjects whose low back pain 'career' was well established and subjects who were experiencing their first attack. The mean severity of the present attack of low back pain was similar to the mean severity of the first attack. Both were less severe than the worst attack. This suggests that, for those who had experienced low back pain in the past, the condition was not becoming more severe in terms of the perception of the severity of pain.

Over half of the sample reported an insidious onset of low back pain. This supports the findings of Horal (1969) and Troup (1979). Horal demonstrated that no more than 50% of a sample of manual workers blamed accidents or lifting at work for the onset of their low back pain. In a study of workers returning to work following an episode of low back pain, Troup demonstrated that 52% of the individuals reported no back injury.

Only 3% of subjects had taken legal advice concerning their low back pain. However, subjects were only recruited into the study if their present pain was acute, of less than one weeks duration and if they had been pain free prior to onset. It is chronic low back pain patients who may be expected to seek legal or trades union advice and this group were excluded from the study.

The mean score on the MSPQ in this study was 5.3 (SD = 4.1). This is in contrast with a study by Main (1983) of a sample of 79 'routine GP referrals' where the score was 3.96 (4.21). This may reflect the fact that the subjects in Main's study completed the MSPQ at a later stage in the natural history of their back pain. As a consequence their level of anxiety, which may have been similar at the early acute stage to that of the subjects of this study, may have reduced with time.

The mean disability score of 12.7 in the subjects of this study is similar to the mean score demonstrated by Roland and Morris (1983) in the development of the instrument using a similar sample. This suggests that, in terms of disability, the sample used in this study is representative of acute low back pain patients who present to their general practitioner

The design of the study involved the measurement of some psychological and functional variables and all physical variables for the first time at two months. This made analysis of these variables difficult in terms of comparison with variables measured at the acute stage and comparison of variables measured by other workers at the acute stage. However, many other studies have labelled subjects as 'acute low back pain patients' at two or more months after onset and the literature reflects a lack of methodological rigor on this issue. Therefore, it was justifiable to compare the descriptive results of two months follow-up with the results of other workers.

The mean Oswestry disability score at two months was 16.3% (SD = 15.1). Fairbank et al (1980) demonstrated a mean score of 20%, three weeks after inclusion in their study of acute low back pain patients. However, the time since onset of pain at inclusion into the study was not stated. Fairbank et al (1980) interpret scores between 0%-20% as representing minimal disability.

In terms of affect, the subjects who attended at two months follow-up were more distressed than the non-back population but similar to other samples of low back pain patients.

The representativeness of the sample is further evidenced by the findings of Main and Waddell (1983). The mean depression score measured by the Zung Depression Inventory was 17.3 (SD = 10.7). The mean Zung score of 'normal' subjects demonstrated by Main and Waddell (1984) was 11.2 (SD = 7.8) and the mean Zung score for 'routine referral' backs was 19.7 (SD = 7.0). 'Abnormality' in terms of Zung score has been defined as a score of over 29 for females and 32 for males (Main and Waddell, 1983).

The mean MSPQ score of the sample in this study was 3.8 (SD = 3.7). The mean MSPQ score of 'normal' subjects has been demonstrated to be 1.79 (SD = 2.13) and the mean 'GP backs' to be 3.96 (SD = 4.21) (Main and Waddell, 1983). 'Abnormality' in terms of MSPQ score has been defined by Main and Waddell (1983) as a score of over 10 for females and 7 for males.

Severity of low back pain, Roland and Morris disability score and sick leave status at two and twelve months follow-up will be discussed in Chapter 6.

The results of the physical examination of subjects in this study at two months follow-up are similar to those reported in the literature.

The problem of diagnosis (Chapter 1) is evidenced by the large number of subjects who were diagnosed by the orthopaedic surgeon as having 'low back pain of unknown cause' (69%). The diagnostic categories used in this study are physiopathological hypothesis focused.

The mean Body Mass Index of 2,975 industrial workers recruited into a study by Battie et al (1990) was 25.4 compared with 25.5 in this study.

The mean inappropriate signs and symptoms score of the subjects of this study, at two months after onset was 1.50 (SD = 2.2). The mean score of a group of 'GP backs' was demonstrated by Main and Waddell (1983) to be 1.48 (SD = 1.42). Abnormality in terms of inappropriate signs and symptoms was defined by Main and Waddell (1983) as scores of over 4 (males) and 8 (females).

The mean straight leg raise of subjects in this study was 64 degrees compared with a mean score of 68 degrees demonstrated by Pope (1985) in a study of 151 chronic low back pain patients.

Spinal mobility was demonstrated to be comparable with that found by other workers in studies of subjects with low back pain. The mean side flexion of 476 low back pain patients was demonstrated by Mellin (1989) to be 16cm (compared with 17cm in this study). Burton (1986) measured the range of sagittal mobility of 140 recurrent low back pain sufferers using the flexible curve technique used in this study. He found the mean sagittal mobility of his sample to be 61 degrees (60 degrees in this study), the mean sagittal flexion to be 24 degrees (22 degrees in this study and the mean sagittal extension to be 37 degrees (38 degrees in this study).

Burton (1992) demonstrated that 61.5% of a sample of low back pain patients presenting to an osteopath were unable to perform a sit up from a supine position compared with 65% in this study.

Hypothesis 4 of the study, which proposes that the large majority of acute low back pain patients are free of physical signs and psychopathology, is supported by the descriptive results of the study. The mean disability, somatic awareness (MSPQ), depression and inappropriate signs and symptoms were below the cut-offs indentified as representing morbidity and the proportion of those with neurological impairment was small.

5.10ii

Sub-group differences

Several statistically significant differences between sub-groups of the sample were demonstrated. The notable differences between sub-groups were those between males and females in terms of psychological distress and perception of severity of previous and present episodes of pain.

These findings are in accord with the literature concerning gender differences in terms of pain perception. Most studies concerned with this issue usually involve laboratory induced pain rather than pain which results from pathology. Lipman et al (1990) demonstrated that males 'behaved slightly more stoically' than females when subjected to heat-beam dolorimetry and had significantly higher pain tolerance levels when pain was induced over the breasts.

Different causal mechanisms have been proposed in order to explain observed differences between males and females in terms of pain perception. One view suggests that the emotional reaction to pain experience mediates the reported pain severity. Robin et al (1987) hypothesised a causal effect of anxiety on raised pain perception amongst females. In a laboratory experiment on male and female students, they demonstrated a significant positive correlation between anxiety

score (measured by the Cattell's anxiety test) and pain perception. In addition, they demonstrated that female subjects had significantly higher anxiety scores and significantly higher pain perception scores. An opposing view of the mediating factors in sex differences in the perception of pain concerns physiological mechanisms. Feine et al (1991) demonstrated than females rated laboratory induced pain as being significantly more severe than males regardless of the gender of the experimenter. However, their data also suggested that females discriminated between painful heat intensities better than males. Feine et al conclude that the sex-related variation in pain perception is probably related to sensory factors rather than differences in attitude or emotional response. The findings of Mogil et al (1993) suggest that sex hormonal variation may affect the neuronal mechanisms of analgesia, thereby explaining gender differences in pain perception.

However, not all studies have demonstrated differences between males and females in terms of pain perception. Lander et al (1989) compared the reported pain arising from clinical conditions. Two groups of adults and one group of children were studied and no sex differences in reported pain were demonstrated. These findings have been supported by Bush et al (1993) in a study of subjects with temporomandibular disorders. Their results demonstrated few differences between males and females in terms of ratings of pain and illness behaviour.

Previous research and the results of this study fail to explain the observed differences in reported pain perception between males and females. If physiological differences do exist between males and females which lead to a reduction in pain tolerance or an increase in the severity of pain perception, emotional consequences, such as raised anxiety levels, may be expected. However, the multi-faceted and complex nature of pain perception evidenced by studies of single gender samples does not support a simplistic physiological model of differences in pain perception between males and females.

In terms of the implications of the observed gender differences on the longitudinal study it was decided to note these differences but to include both groups in later analyses. The reasons for this are discussed in Chapter 8.

Perhaps not surprisingly, subjects who were on sick leave at the acute stage reported that the severity of their pain and disability levels were significantly higher and they were more likely to be members of the lower social groups than those who continued to work. It is difficult to determine a causal mechanism which explain these findings, given the cross-sectional nature of the analysis. One interpretation involves the use of reports of severe pain and dysfunction as justification for remaining on sick leave. Conversely, the severity of the physiological pathology may have resulted in

heightened pain perception and consequent disability which made return to work impossible.

Several age differences were demonstrated. In general they reflected demographic and social changes and the reduction in physical mobility associated with ageing. Only one pain perception variable, highest ever internal pain, was significantly different between groups. This may be either a chance finding or an expression of an increased incidence in pains associated with middle age such as toothache or chest pain. A counter-intuitive observation concerns the increase in reported back pain caused by injury associated with getting older. One may have expected younger subjects to be more at risk of injury and older subjects to develop back pain as a result of an exacerbation of advanced osteo-arthrotic changes. In physical terms, it is noteworthy that range of movement decreases with age, as does the ability to sit up from a supine position. This observation supports the view that the sample were suffering from a degenerative condition. However, straight leg raise is not increasingly impaired with age. This suggests that angle of straight leg raise may be mediated by other factors in addition to physiological range of movement. It is surprising that neurological signs did not increase with age, given the cumulative effects of repeated episodes of low back pain experienced by older subjects.

The observed differences between the time since onset groups in terms of pain perception are difficult to interpret. In terms of back pain, the time since the first episode was positively related to the severity of the worst ever episode. This may either signify a gradually deteriorating pathology which leads to an increase in nociception, or a change in the interpretation of nociception which is related to the experience of repeated exacerbations.

Several differences were demonstrated between subjects recruited into the study from Maghull Health Centre and other practices. Although difficult to interpret, the results may be an expression of the differences between individuals for which membership of Maghull Health Centre is a marker. The catchment area of Maghull Health Centre is mainly suburban and relatively privileged in socio-economic terms. The catchment areas of the other practices in the study are traditionally associated with social disadvantage. This may explain the significantly higher weighted life events score of subjects from other practices. The lower MSPQ score of Maghull subjects may be associated with the higher percentage of female subjects recruited by other practices, as several items in the MSPQ are female specific (eg menstrual pain) or more likely to be experienced by females (eg migraine). However, raised anxiety levels may also be associated with other factors which in turn are more likely to be experienced by subjects recruited by the other practices. It is the author's view that the raised

anxiety levels demonstrated among subjects from other practices has more to do with socio-economic and sex-related factors than with pain perception. In support of this view is the observation that the severity of present pain and disability were not significantly different between the groups.

In terms of the other pain variables and the Fear Avoidance Model variables Maghull Health Centre subjects and subjects from other practices did not differ significantly. It is therefore justifiable to include data collected from Maghull Health Centre subjects and data collected from subjects recruited by other practices in the longitudinal and predictive analyses.

5.10ii Attenders and non-attenders

As with any study of the natural history of a relatively common and largely self-limiting illness the drop out rate of subjects over the course of the study was high. Central to the results reported in later chapters is the extent to which attenders at two and twelve months differed from non-attenders, in terms of potential predictor variables or variables associated with pain severity and dysfunction. The results suggest that non-attenders differ significantly from attenders in demographic terms only, and these findings resemble other reports in the literature about defaulted appointments in general practice.

Appointment defaulters have been shown to be significantly younger than attenders (Barron, 1980, Bickler, 1985), more likely to be members of lower socioeconomic groups (Barron, 1980) and more likely to be registered with particular GPs (Bickler, 1985). The latter two observations may be accounted for by the statistically significant difference in this study between attenders and non-attenders in terms of referring practice. Volunteers for research studies have been shown to have higher educational levels and occupational status (Rosenthal and Rosnow, 1969). The significant differences between non-attenders and attenders in terms of referring practice and the difference in terms of social group (which approaches significance) support these findings.

Postal respondents at twelve months had significantly higher pain severity and disability scores than attenders and the 16 subjects who reported 'constant pain' were all postal respondents.

These observations may be explained in several ways. First, the context in which information regarding pain experience is gathered may effect the results. It may be, for example, that subjects were less willing to complain of severe pain or disability in person than through the anonymous medium of the postal service. Conversely, subjects may have felt more able to exaggerate pain experience when completing postal questionnaires.

The findings may be also be explained in the context of the effects of chronic pain and the Fear Avoidance Model of Exaggerated Pain perception. The literature suggests that chronic pain and depression (with a reduction in proactive behaviour) are positively correlated. In addition, if attendance at twelve months follow-up is viewed as an 'active' behaviour, the avoidance of active behaviour by subjects in constant (or chronic) pain is in accord with the Fear Avoidance Model.

5.10iii The reliability of predictor variables

Despite a rigorous study design (in terms of the length of time between test and retest) the measures included in the analysis were demonstrated to be highly reliable.

The only variable to have a Pearson's correlation coefficient below 0.5 is 'percent active coping strategies'. This may reflect the psychometric properties of the instrument itself which contains a limited number of pain situations. A change from a 'positive coping strategy' to a 'negative coping stategy' in the same pain situation between test and retest may result in a large change in percent active coping score. Alternatively, the relatively low correlation coefficient may be explained by the effects of change in current pain and disability state over the two months test-retest period which

may alter the way in which subjects report or remember their general pain coping style.

The results of this study suggest that the inter-observer reliability of inappropriate signs is high and support the findings of Waddell et al (1980). However, a difference in statistical methods prevents a direct comparison of the results of this study and those of Waddell.

5.11 CONCLUSION

The results of the descriptive analyses presented in this chapter, including the differences between sub-groups within the sample, have been presented and examined. Hypothesis 4 of the study has been supported and a sample of acute low back pain patients presenting to their GP has been shown to be generally free of physical signs and psychopathology. The results lead the author to conclude that the study group is broadly representative of patients with acute low back pain attending their GPs. Some sub-group differences have been found. However, these differences are comparatively small and can be explained in terms which do not affect the inferences which can be made from the results of the longitudinal and predictive analyses which are reported in the following chapters. In addition, several measures which will be used in the predictive analyses have been shown to be highly reliable.

CHAPTER 6

NATURAL HISTORY AND OUTCOME MEASURES

6.1 INTRODUCTION

This chapter represents the fulfillment of the third aim of the study, to describe the natural history of acute low back pain over a twelve months period. In addition, Hypotheis 4 of the study - 'ninety percent of acute low back pain patients will recover within eight weeks of onset of pain' is tested.

This chapter describes the natural history of the subjects' acute attack of low back pain over the twelve months period of the longitudinal study. The number of subjects included in the analysis is less than the total number of subjects interviewed at the acute stage and less than the total number who provided 12 months follow-up data. Subjects are included in the analysis if they had either attended for assessment and examination or responded to postal questionnaires at each of the three stages of the study (screening, two months follow-up and twelve months follow-up). Therefore the number of subjects in this sub-group was 123. However, all screening (N = 300), two months (N = 162) and twelve month data (N = 196) sets have been included in other analyses (see Chapters 5, 7 and 8).

In addition, the development of combined measures of outcome at two months after onset of pain and twelve months after onset of pain are described. This process included analysis of all data collected at two months (N = 162) and twelve months (N = 196)

6.2 NATURAL HISTORY IN TERMS OF SINGLE MEASURES OF OUTCOME

6.2i Outcome measures

Four variables which were thought to represent the 'outcome' of an acute episode of low back pain were included in the study. These variables have also been widely used by others (see Chapter 3).

These variables are:

1) Disability (measured by The Roland and Morris (1982) disability questionnaire).

2) Severity of present low back pain (on a scale of 1-10 where 1 represents no pain and 10 represents 'the worst imaginable pain').

3) Whether on sick leave from work because of low back pain.

4) Course of low back pain since entry into the study. The three alternative courses were 'pain stayed away completely', 'pain on and off' and 'constant pain'.

Information concerning course of low back pain since entry into the study was collected at twelve months (N = 196) follow-up only. Information concerning disability, severity of pain and sick leave was collected at two (N = 162) and twelve months (N = 196) follow-up.

A large proportion of subjects were not in formal employment a the time of the study. This led to a reduced sick leave data set at each stage of the study (Table 6.2)

6.2ii Natural history

Course of low back pain:

Table 6.1 demonstrates that 9 (7.3%) subjects reported that they had experienced constant pain since inclusion in the study, twelve months previously. The remaining 114 (92.7%) subjects had either experienced intermittent low back pain or had not experienced a further episode over the twelve months period.

Sick leave:

Table 6.2 demonstrates that by two months after onset of low back pain, most subjects who had been on sick leave at the acute stage (60.9%) had returned to work, leaving only 10.8% of those who were employed on sick leave. By twelve months this figure was 13.9%.

Severity of pain:

The mean severity of pain score at the acute stage was 6.5 (SD = 2.3). At two months follow-up this score was 2.9 (SD = 2.4) and at twelve months mean severity of pain was 3.1 (SD = 2.4). The natural history of severity of pain over twelve months is presented graphically in Figure 6.1.

At the acute stage, 62.3% of subjects scored 6 or above on severity of present pain (on a scale of 1 to 10). At two months follow-up 11.5% of subjects scored 6 or above and by twelve months follow-up 17.7% of subjects scored 6 or above.

Disability:

The mean disability score at the acute stage was 13.2 (SD = 6.2). By two months follow-up this score had reduced to 6.4 (SD = 6.3) and at twelve months remained about half of the acute

mean score at 5.5 (SD = 5.6). The natural history of disability over twelve months is presented graphically in Figure 6.2.

At the acute stage, 50.8% of subjects scored 15 or above on the disability questionnaire (on a scale of 0 to 24). At two months follow-up 14.9% of subjects scored 15 or above and by twelve months follow-up 8.1% of subjects scored 15 or above.

6.3 NATURAL HISTORY OF 'PAIN STAYED AWAY', 'PAIN ON AND OFF' AND 'CONSTANT PAIN' GROUPS

The analysis of the natural history of the subjects' low back pain over twelve months was broken down into separate analyses of the three 'course of pain over twelve months' groups in an attempt to identify differences between the groups in terms of natural history of pain, disability and sick leave.

Sick leave:

Fifty five per cent of the 'pain stayed away' group who were employed were on sick leave at the acute stage. Fifty six per cent of the 'pain on and off' group were also on sick leave at the acute stage. A similar percentage of the 'constant pain group' were on sick leave at the acute stage (60%).

By two months, none of the 'pain stayed away' group and 11% of the 'pain on and off' group were on sick leave. Only three of the 'constant pain' group who attended at two months follow-up were employed. Two of these were on sick leave.

At twelve months, none of the 'pain stayed away' group, 10% of the 'pain on and off' group and 69% of the 'constant pain' group were on sick leave. These results are presented in Figure 6.3.

Severity of pain:

At screening, the mean severity of present pain score of the 'pain stayed away' group was 6.4 (SD = 2.3), the mean score of the 'pain on and off' group was 6.3 (SD = 2.4) and the mean pain score of the 'constant pain' group was 7.6 (SD = 2.7).

By two months, the mean severity of present pain scores of the 'pain stayed away' and 'pain on and off' groups were 1.8 (SD = 1.6) and 2.9 (SD = 2.3) respectively. However, the mean pain score of the ten members of the 'constant pain' group who attended at two months follow-up was 5.4 (SD = 3.4).

At twelve months follow-up, the mean severity of present pain scores of the 'pain stayed away' group was 1.8 (SD = 1.6). The mean pain score of the 'pain on and off' group was 3.0

(SD = 2.0) The mean pain score of the 'constant pain' group at twelve months was 8.1 (SD = 1.4).

These results are presented graphically in Figure 6.4.

Disability:

At screening, the mean disability score of the 'pain stayed away' group was 11.9 (SD = 6.2), the mean disability score of the 'pain on and off' group was 12.7 (SD = 5.8) and the mean disability score of the 'constant pain' group was 15.25 (SD = 5.5).

By two months, the mean disability scores of the 'pain stayed away' and 'pain on and off' groups were 3.5 (SD = 4.2) and 6.5 (SD = 6.2) respectively. However, the mean disability score of the ten members of the 'constant pain' group who attended at two months follow-up was 12.5 (SD = 7.6).

At twelve months follow-up, the mean disability scores of the 'pain stayed away' and 'pain on and off' groups were 0.8 (SD = 1.3) and 5.9 (SD = 4.9) respectively. The mean disability score of the 'constant pain' group at twelve months was 13.5 (SD = 5.1).

These results are presented graphically in Figure 6.5.

Two way analyses of variance (ANOVA) were performed, using pain history and time (acute stage, two months follow-up and twelve months follow-up) as independent variables. Pain and disability were the independent variables.

Significant main effects were demonstrated for pain history ($F = 23.24$; $df = 116,2$; $P = 0.001$) and time ($F = 46.46$; $df = 232,2$; $p = 0.001$) using pain as the independent variable. A significant interaction was demonstrated between time and pain history (Wilks Lambda = 0.843, $P = 0.001$).

Significant main effects were also demonstrated for pain history ($F = 16.55$; $df = 119,2$; $P = 0.001$) and time ($F = 36.14$; $df = 238,2$; $P = 0.001$) using disability as the independent variable. A significant interaction was demonstrated between time and pain history (Wilks Lambda = 0.900, $P = 0.015$).

Analysis of variance (ANOVA) was performed on the pain and disability scores of the three outcome groups at the acute stage, two months follow-up stage and twelve months follow-up stage.

No significant differences were demonstrated between the pain history groups in terms of pain and disability at the acute stage.

Figure 6.1

Mean severity of pain (1-10) at acute stage, 2 months and 12 months follow-ups (N = 123)

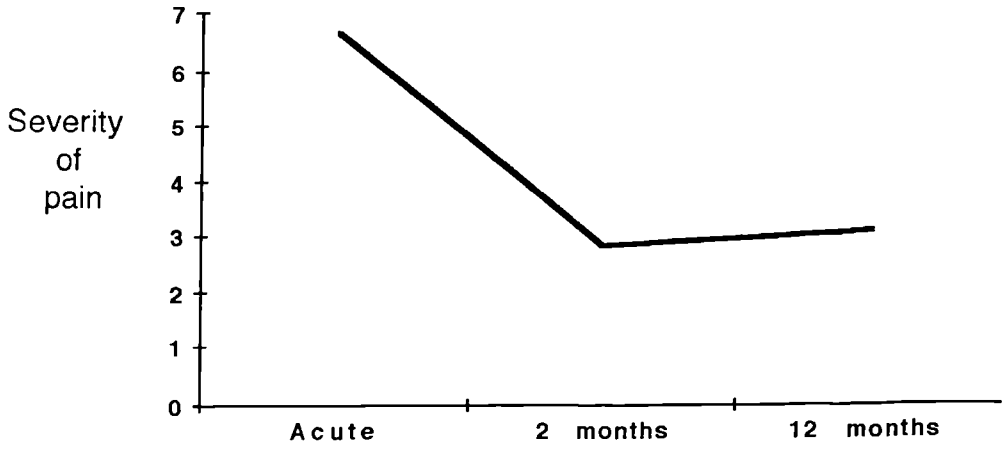


Figure 6.2

Mean severity of disability (1-24) at acute stage, 2 months and 12 months follow-ups (N = 123)

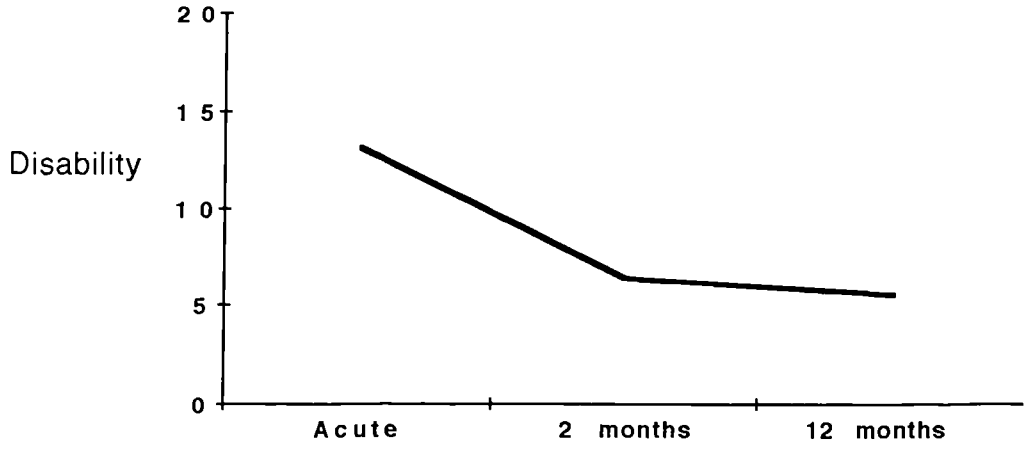


Figure 6.3

Subjects on sick leave at acute stage, two months and twelve months

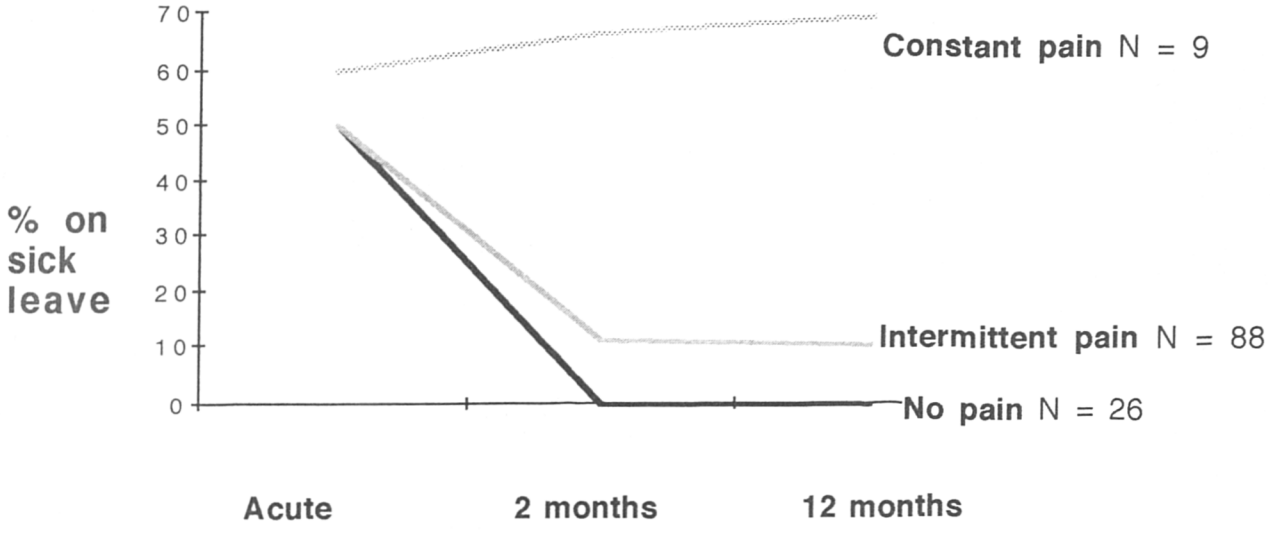


Figure 6.4

Mean severity of pain (1-10) at acute stage, 2 months follow up and 12 months follow-up for subjects with no pain, intermittent pain and constant pain (Total N = 123)

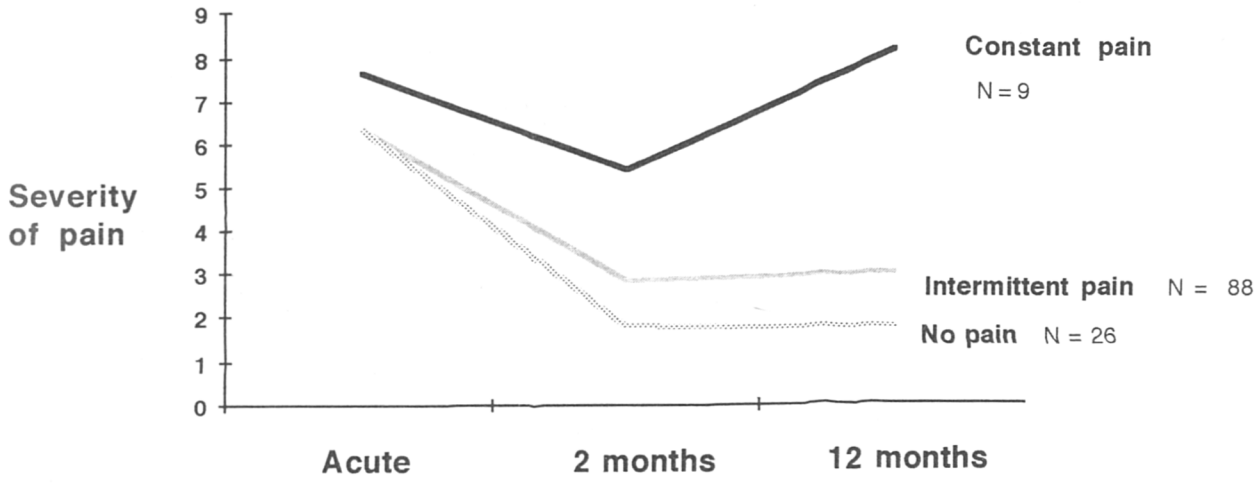
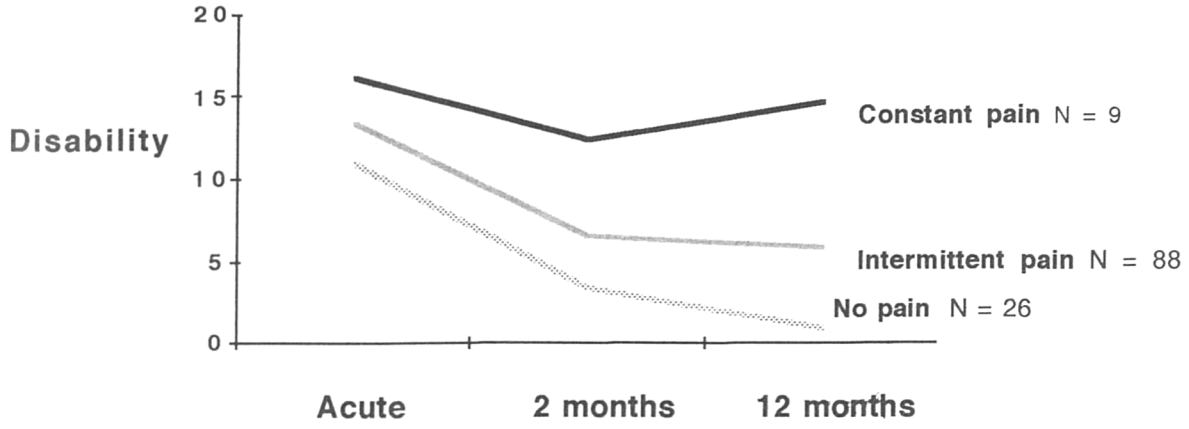


Figure 6.5

Mean disability (1-24) at acute stage, 2 months and 12 months follow-up for subjects with no pain, intermittent pain and constant pain (Total N = 123)



At two months follow-up, ANOVA demonstrated significant differences between the groups in terms of pain ($F = 8.58$; $df = 118,2$; $P = 0.0003$). Scheffe's post-hoc test revealed the significant difference (at the 0.05 level) to be between the 'pain stayed away' group and the 'constant pain' group and between the 'pain on and off' group and the 'constant pain' group.

At two months follow-up, ANOVA demonstrated significant differences between the groups in terms of disability ($F = 7.88$; $df = 120,2$; $P = 0.0006$). Sceffe's post-hoc test revealed the significant differences (at the 0.05 level) to be between the 'pain stayed away' group and the 'constant pain' group and between the 'pain on and off' group and the 'constant pain' group.

At twelve months follow-up, ANOVA demonstrated significant differences between the groups in terms of pain score ($F = 38.97$; $df = 120,2$; $P = 0.0001$). Scheffe's post-hoc test revealed the significant differences (at the 0.05 level) to be between each pair of the outcome groups.

At twelve months follow-up, ANOVA demonstrated significant differences between the groups in terms of disability ($F = 30.29$; $df = 120,2$; $P = 0.0001$). Scheffe's post-hoc test revealed the significant differences (at the 0.05 level) to be between each pair of the outcome groups.

Table 6.1

Reported course of low back pain since two months follow-up - response to postal questionnaire

History of pain since 2 months follow-up	N
No pain	26 (21.1%)
Intermittent pain	88 (71.5%)
Constant pain	9 (7.3%)
Total =	123

Table 6.2

Sick leave at acute stage, two months follow-up and twelve months follow-up

	Acute stage	Two months	Twelve months
Not on sick leave	37 (39.4%)	74 (89.2%)	93 (86.1%)
On sick leave	57 (60.0%)	9 (10.8%)	15 (13.9%)
	94	83	108

6.3 THE DEVELOPMENT OF OUTCOME MEASURES

It was decided that history of back pain since two months follow-up or screening (if subjects had not attended at two months), severity of present pain, disability (Roland and Morris, 1982) and sick leave status were meaningful variables which represented quantitative outcome.

The history of pain variable identified subjects who recovered from their acute attack of low back pain (either to remain pain free or experience further intermittent episodes) and those who experienced constant pain. This latter group were, in terms of the usual definitions of chronicity, the group who had become chronic low back pain patients.

This group represented 8.3% of subjects who provided data at the twelve months follow-up stage of the study (N = 196).

Self reported pain and functional disability have been shown to correlate (Roland and Morris, 1982, Deyo, 1986, Waddell, 1987, Waddell et al, 1993). Therefore, in order to avoid repetition and to facilitate interpretation of the predictive results (Chapter 8) severity of pain and disability were combined (see below) into a single measure of outcome.

Therefore, different aspects of twelve months outcome are expressed in the following ways:

i) in terms of self-reported history since the acute or two months follow-up stage. (This resulted in a dichotomous variable. The first group consisted of subjects who recovered and the second group consisted of those who had become chronic low back pain sufferers).

ii) in terms of combined severity of pain and disability. In order to combine these variables, they were entered into a principal components analysis. The factor extracted (which accounted for 84.3% of the variance) represented a new pain-disability variable (Table 6.5).

iii) in terms of sick leave status.

It was also important to develop a means of predicting outcome at two months. A continuous two months outcome measure was identified. This was:

i) combined severity of pain and disability. This variable was developed in the same way as the twelve months outcome variable

Table 6.1

Combination of severity of pain and disability at twelve months

Factor	Eigenvalue	Percentage of variance
1	1.686	84.3
2	0.313	15.7

Table 6.2

Combination of severity of pain and disability at two months

Factor	Eigenvalue	Percentage of variance
1	1.599	80.0
2	0.401	20.0

Principal components analysis extracted one factor which accounted for 80% of the variance (Table 6.6).

6.4 DISCUSSION

The results presented in this chapter support Hypothesis 5 of the study which proposes that individuals who are going to recover (in terms of pain, disability and return to work) from an attack of low back pain do so in large measure by two months after onset of pain. The subjects of this study who were categorised as 'recovered' were those who reported 'no pain' or 'pain on and off' at twelve months follow-up.

These findings reflect those of Roland and Morris (1982). In their study of 237 subjects with back pain of not more than 28 days duration, they demonstrated that 92% of subjects were able to return to work at approximately two months after onset (89% in this study). They also demonstrated a significant reduction in pain and disability at approximately two months after onset. By two months, 84% of their subjects had a score of 14 or less on the Roland and Morris disability questionnaire (85% in this study) and 85% reported "moderate or little pain" (88.5% had pain scale scores of 5 or less in this study at the same stage).

Philips and Grant (1991) also found that 'the largest shift in the pain problem occurs between the acute and subchronic (three months) points' in a study of 117 acute back pain sufferers. They demonstrated that at the acute stage (< 15 days) the mean severity of pain was 4.7 (6.5 in this study), at three months the pain score was 3.6 (2.9 at two months in this study and at six months the mean pain score was 3.1 (3.1 in this study). Philips and Grant also demonstrated that disability, although measured differently, halved between the acute stage and three months and increased slightly between three months and twelve months. These results are similar to those of this study (Figure 9.2).

However, it is worthy of note that the mean pain and disability scores of the sample are greater than zero twelve months after onset and that a small percentage of subjects had not returned to work at that stage. Examination of the results demonstrates that subjects who were not categorised as being 'chronic' still reported a level of pain and disability at twelve months follow-up. (The fact that the 'pain stayed away' group had a mean score of severity of pain greater than one may be explained either by a misunderstanding of the postal questionnaire by some subjects or by a semantic issue concerning the meaning of the word 'pain'.) These results suggest that, rather than the majority of acute low back pain subjects recovering completely, they recover to the extent that

they are able to return to normal function without the need for further treatment.

However, despite the fact that most subjects reported some residual pain and disability long after their initial attack, it is apparent that subjects who reported that their pain was 'constant' over the study period also reported severe pain and disability which was unremitting and significantly more severe than that of the 'recovered' groups and had not returned to work. Change in pain and disability were not only functions of time but of the outcome group into which subjects were categorised. The statistical analysis also reveals that in terms of pain and disability, the chronic group were not significantly different from the recovered groups at the acute stage. However, by two months a significant difference was demonstrated between recovered and chronic groups. This was not due to an increase in the severity of pain and disability of the chronic group but by their failure to recover.

A feature of the natural history of the chronic group is an increase in pain and disability between two and twelve months after onset. This may be explained by the effects of providing data through the post rather than at interview. This issue was discussed in Chapter 6.

At two months, significant differences were demonstrated between the chronic group and the 'intermittent' and 'no pain

groups' (recovered group). However, by twelve months significant differences were demonstrated between each of the three groups. In terms of disability, this may be due to a further decrease by the 'no pain' group between two months and twelve months.

The definition of chronic pain as that which has lasted for more than seven weeks (Larocca, 1989) rather than the traditional six months is supported by the results of this study. At two months the pain and disability of low back pain subjects who will 'recover' are already significantly less than those who will not. This may have implications for those involved in back pain research, in that the follow-up period of longitudinal studies of acute low back pain patients might, logically, be reduced. This may facilitate the gathering of larger data sets over the same period of time or a reduction in the length of time needed to collect longitudinal data. For those involved in the management of low back pain patients the results suggest that if patients do not report a substantial reduction in pain and disability by two months after onset, the chances that they will recover are reduced.

The results presented in this chapter demonstrate that over eight percent of subjects reported a twelve months history of constant pain following onset and that these subjects differed at an early stage in terms of pain, disability and return to work from subjects who reported that their symptoms were

alleviated. The results of this chapter do not explain why a subgroup of the sample did not recover to the same extent as the rest of the sample. The outcome variables discussed in this chapter will form the focus of the predictive analyses designed to explore this issue. The results of these analyses will be reported in Chapter 8.

CHAPTER 7

THE RELATIONSHIP BETWEEN PSYCHOLOGICAL, PHYSIOLOGICAL, FUNCTIONAL AND HISTORICAL VARIABLES.

7.1 INTRODUCTION

This chapter focuses on Hypotheses 3 and 4 of the thesis which state that 'low back pain is mediated in the long term by psychosocial variables rather than by physical pathology or impairment' and 'the degree of limitation of physiological movement is mediated in large measure by psychological variables rather than by physical pathology'.

Exploratory analyses were conducted in order to identify underlying factors which represent the multi-faceted nature of low back pain experience. The aim of the final analysis reported in this chapter was to identify an underlying factor which integrates the data in theoretical terms in order to form a contrived 'model'. It was intended that this model would be compared with the Fear Avoidance Model in terms of prediction of outcome in later analyses.

It was decided that factor analytic techniques were the most appropriate means of exploring the relationships between the variables included in the study. Principal components factor analysis was used because the investigation was exploratory

with few assumptions about underlying structure. This is a method of forming linear composites (factors) based on the correlations between the variables. The correlation of each variable with each composite yields a set of loadings which may then be transformed (rotated) to facilitate interpretation. High loadings indicate the variables which are most strongly associated with a particular factor. Each factor is derived to explain as much of the variance in the data as possible; the first factor will always explain the largest percentage of the variance, the second and subsequent factors accounting for additional and independent variance. Factor analytic techniques are flexible tools, which can be used to simplify and render interpretable data sets containing large numbers of variables. Principal components analysis has been used in this study to identify the different facets of low back pain experience and identify a contrived 'model' which will be used in comparison with Fear Avoidance Model.

This chapter reports the results of a series of principal components analyses and presents interpretations of the results.

7.3 RESULTS OF PRINCIPAL COMPONENTS ANALYSIS

Three principal components analyses were conducted.

In the first analysis, variables representing the Fear Avoidance Model, historical model and measures of pain, disablity and sick leave status at the acute stage were entered. (The variables concerned with time since first attack and number of previous attacks are not suitable for inclusion in factor analysis. These variables are neither ordinal nor interval variables as they include the category 'never had back pain previously'. They were therefore excluded.)

Five factors were extracted using Kaiser's criterion which extracts factors with latent roots (Eigenvalues) greater than one. The factors were not rotated and accounted for 65.5% of the total variance of the data. The first factor contains the perceived severity of the highest ever externally, internally and accidentally produced pain and is called the 'general pain memory factor'. The second factor contains severity of present low back pain, whether on sick leave and disability score. These variables all represent the perceived severity of the acute attack of low back pain. This factor is called the 'perceived present severity factor'. The third factor contains the perceived severity of the first attack of low back pain and the perceived severity of the worst attack of low back pain. This factor is called the 'back pain memory factor'. The fourth

factor contains MSPQ and weighted life events. This factor is called the 'stress factor'. The fifth factor contains only one variable, percent active coping strategies which does not load significantly on any other factor. These results are presented in Tables 7.1 and 7.2.

The second analysis was concerned with identifying the relationship between physical variables measured at two months follow-up. The factors were rotated in order to facilitate interpretation. Four factors, which accounted for 61.6% of the total variance, were extracted using Kaiser's criterion. The first factor contains the inappropriate signs and symptoms score, mean straight leg raise and passive knee bend. In view of the nature of the inappropriate signs and symptoms variable in terms of its correlation with psychological variables (Waddell, 1989) this factor is called the 'psycho-physical factor'. The second and third factors can be considered together. The second factor represents a 'spinal mobility factor'. The third factor includes sagittal flexion, which also loads on the spinal mobility factor. The negative loading of Body Mass Index with the third factor (which also includes sagittal flexion) suggests, reasonably, that spinal flexion is inhibited by abdominal fat. The third factor is called the 'spinal flexion' factor. The fourth factor contains neurological deficit and nerve root tethering. This factor is called the 'neurological factor'. These results are presented in Tables 7.3 and 7.4

The third analysis included Fear Avoidance Model variables, other psychological variables, outcome variables, historical variables and physical variables measured at two months follow-up. The analysis had two aims. The first was to explore the relationship between psychological, Fear Avoidance and physical variables measured at two months in order to test hypotheses 3 (low back pain is mediated in the long term by psychosocial variables rather than by physical pathology or impairment) and hypothesis 4 (the degree of limitation of physiological movement is mediated in large measure by psychological variables rather than by physical pathology) of the study.

The second aim of this analysis was to identify *an underlying* factor which included psychological and physical variables and could be interpreted theoretically. It was intended that this factor would represent an alternative model which could be tested in terms of its utility as a predictor of twelve months outcome in comparison with the Fear Avoidance Model.

Two different methods of determining the number of factors to extract were used in order to fulfill both aims. The first method, designed to explore the relationship between variables and test hypotheses 3 and 4, used Kaiser's criterion to determine the number of factors to be extracted. The second method was designed to identify a contrived model against which the Fear Avoidance Model could be further tested in terms of prediction of outcome. The analysis was 'forced' in terms of

number of factors extracted until a factor (or factors) were identified which a) accounted for a significant proportion of variance and b) could be interpreted.

The first method, using Keiser's criterion, extracted Eight factors which accounted for 63.5% of the variance. These results are presented in Tables 7.5 and 7.6.

The first factor contains measures of self-reported disability, depression, somatic awareness, pain drawing, inappropriate signs and symtoms and sick leave absence from work. This factor is called the 'psycho-physical factor'. Straight leg raise and passive knee bend also load on this factor (in a negative direction). In addition, neither variable is associated with neurological deficit and nerve root tethering which are objective physical measures. This suggests straight leg raise and passive knee bend correlate negatively with the 'distress' associated with low back pain and are functions of distress rather than physical pathology. This view is supported by the results of the second analysis (reported above) which demonstrates that straight leg raise and passive knee bend load on a factor structure which includes inappropriate signs and symtpoms rather than objective physical signs. The results also suggest that side flexion is negatively associated with distress rather than physical pathology, although this variable also loads with nerve root tethering.

Factor 2 contains variables associated with memory of the severity of previous episodes of low back pain or memory of the severity of previous painful events. This factor is called the 'pain memory factor'. Severity of the worst attack of low back pain also loads on the 'low back pain distress' factor.

Factor 3 contains the nature of onset of present pain, severity of first attack and neurological deficit. These variables are correlate positively with the factor structure. An interpretation of this factor suggests that if the first ever episode of back pain is severe, neurological deficit may result. In addition, neurological deficit may be more likely following injury rather than insidious onset of pain. This factor is therefore called the 'physical pathology factor'.

Factors 4,5 and 6 can not be interpreted.

Factor 7 contains only one variable, nerve root tethering. However, neurological deficit has a factor loading of .317 on this factor which is therefore called the 'neurological involvement factor'.

Factor 8, which contains ability to sit up and sagittal flexion is called the 'sagittal flexion' factor.

These results are presented in Tables 7.5 and 7.6.

Following the second method, concerned with developing a competing 'model' in order to further test the Fear Avoidance Model, it was found that the extraction of 17 factors following varimax rotation produced two factors (consisting of three variables each) which accounted for 30.9% of the total variance. Factor 1 contains measures of self reported disability and severity of pain. These variables have been described in terms of a form of behaviour, dependent upon social and psychological influences as well as physical disease (Waddell, 1987, 1989). They can be interpreted in the context of the communication of the perceived severity of underlying pathology rather than being an expression of actual physical impairment. Factor 2 contains variables which represent somatic awareness, affect and pain behaviour. These variables in combination have been shown by Main et al (1991) to predict the outcome of chronic low back pain management programmes and categorise acute patients in terms of their risk of future 'distress'.

Factors 1 and 2, although statistically independent in terms of the principal components analysis, contain variables which collectively represent what can best be described as low back pain 'distress'. The contrived 'model' which they represent will therefore be called the 'distress model' of low back pain. These results are presented in Tables 7.7 and 7.8.

Table 7.1

Principal components factor analysis of Fear Avoidance Model variables, pain history variables, severity of present pain and disability measured at the acute stage.

Factor items	Factor loadings				
	1	2	3	4	5
Highest ever internally produced pain	**.767**	.224	-.097	-.016	-.046
Highest ever externally produced pain	**.746**	-.166	.053	-.055	-.048
Highest ever accidentally produced pain	**.623**	.001	.127	.110	-.031
Severity of present pain	.233	**.701**	-.102	.216	.248
Whether on sick leave	-.156	**.687**	.079	-.182	-.070
Disability	.012	**.629**	.114	.219	-.502
Severity of first attack	-.005	-.078	**.939**	-.069	-.092
Severity of worst attack	.230	.372	**.637**	.142	.356
Weighted life events	-.107	-.133	-.091	**.789**	-.136
MSPQ	.178	.239	.108	**.707**	-.136
Percent active coping strategies	-.123	.011	.044	-.027	**.825**

Table 7.2

Eigenvalues and percentage variance explained from factor analysis of Fear Avoidance Model variables, pain history variables, severity of present pain and disability measured at the acute stage.

Factor	Eigenvalue	% Variance	Cumulative variance
1	2.189	19.9	19.9
2	1.463	13.3	33.2
3	1.366	12.4	45.6
4	1.11	10.2	55.3
5	1.072	9.7	65.5

Table 7.3

Principal components factor analysis of physical variables measured at two months follow-up

Factor items	Factor loadings			
	1	2	3	4
Inappropriate signs and symptoms	**-.825**	-.009	.018	.063
Mean straight leg raise	**.758**	.027	.077	-.214
Passive knee bend	**.656**	.219	.394	-.013
Sagittal extension	-.326	**.662**	-.062	.051
Ability to sit up	.294	**.599**	.068	-.084
Side flexion	.516	**.577**	.007	-.025
Body mass index	-.052	.092	**-.896**	-.066
Sagittal flexion	.161	.467	**.530**	-.172
Neurological deficit	.011	.041	-.131	**.857**
Nerve root tethering	-.220	-.113	.129	**.627**

Table 7.4

Eigenvalues and percentage variance explained from factor analysis of physical variables measured at two months follow-up.

Factor	Eigenvalue	% Variance	Cumulative variance
1	2.809	28.1	28.1
2	1.204	12.0	40.1
3	1.125	11.3	51.4
4	1.021	10.2	61.6

Table 7.5 Principal components factor analysis of variables measured at two months follow-up using Kaiser's criterion for extraction of factors.

	Factor loadings	
FACTOR 1		
Oswestry disability	.832	
Roland and Morris disability	.804	
Inappropriate signs and symptoms	.736	
Severity of present attack	.695	
Zung Depression Inventory	.668	
Whether on sick leave	.657	
Pain drawing score	.651	
Mean straight leg raise	-.609	
Passive knee bend	-.594	
Side flexion	-.561	(.348 factor 7)
MSPQ	.549	(-.430 factor 7)
FACTOR 2		
Highest ever accidentally produced pain	.739	
Highest ever externally produced pain	.613	
Highest ever internally produced pain	.588	
Severity of worst attack	.435	(.408 factor 1)
FACTOR 3		
Insidious or accidental onset	.671	
Severity of first attack	.618	
Neurological deficit	.317	(.316 factor 7)
FACTOR 4		
Whether this is first episode of LBP	.517	
FACTOR 5		
Weighted life events	.545	
Percent active coping strategies	.437	
FACTOR 6		
Body Mass Index	-.662	
Sagittal extension	-.469	
FACTOR 7		
Nerve root tethering	.392	
FACTOR 8		
Ability to sit up	.543	
Sagittal flexion	.455	

Table 7.6

Eigenvalues and percentage variance explained from factor analysis of variables measured at two months follow-up using Kaiser's criterion for extraction of factors.

Factor	Eigenvalue	% Variance	Cumulative variance
1	5.723	22.9	22.9
2	2.010	8.0	30.9
3	1.735	6.9	37.9
4	1.502	6.0	43.9
5	1.435	5.7	49.6
6	1.258	5.0	54.7
7	1.149	4.6	59.3
8	1.052	4.2	63.5

Table 7.7 Principal components factor analysis of variables measured at two months follow-up. Seventeen factors extracted

FACTOR 1	Factor loading
Oswestry disability	.835
Severity of present attack	.820
Roland and Morris disability	.798
FACTOR 2	
MSPQ	.864
Zung depression inventory	.738
Inappropriate signs and symtoms	.433
FACTOR 3	
Passive knee bend	-.779
Pain drawing	.678
Side flexion	-.448
FACTOR 4	
Highest accidentally produced pain	.828
Highest externally produced pain	.762
FACTOR 5	
Whether this is first episode of LBP	.925
Severity of worst attack	.644

Remaining factors consisted of single variables

Table 7.8

Eigenvalues and percentage variance explained by first five factors from factor analysis of variables measured at two months follow-up. Seventeen factors extracted

Factor	Eigenvalue	% Variance	Cumulative variance
1	5.723	22.9	22.9
2	2.010	8.0	30.9
3	1.735	6.9	37.9
4	1.502	6.0	43.9
5	1.435	5.7	49.6

7.4 DISCUSSION

The results presented in this chapter support, in large measure, Hypotheses 3 and 4 of the thesis which state that 'low back pain is mediated in the long term by psychosocial variables rather than by physical pathology or impairment' and 'the degree of limitation of physiological movement is mediated in large measure by psychological variables rather than by physical pathology'.

Analyses 2 (Table 7.3) and 3 (table 7.5) demonstrate that straight leg raise and side flexion, both of which are held to represent severity of physical impairment, are more closely associated with psychological distress than with objective physical signs. Furthermore, these variables were consistent in terms of loading with the same psychological variables. However, interpretation of analyses in terms of other physical variables suggests that a phyiological component of low back pain experience does exist, particularly in terms of neurological dysfunction. In the view of the author, these results suggest that some physical variables (straight leg raise and side flexion) are more likely to represent distress than others.

The results of the analyses which include physical and psychological variables can be interpteted within the context of the fear avoidance construct. Straight leg raise, passive

knee bend and side flexion are negatively correlated with the variables which represent psychological distress. This 'distress' may be the result of fear of pain which, in turn, leads to avoidance behaviour, characterised in this instance by limited range of physical movement.

Several groups of variables which, in the view of the author, represent different but related mediators of low back pain experience have been identified by principal components analyses. These are:

1) Memory of previous general pain
2) 'Distress' (fear)
3) Behavioural (avoidance characterised by limitation of straight leg raise etc)
4) Memory of previous back pain
5) Spinal mobility
6) Neurological factor

The first three *factors collectively represent the fear* avoidance construct. The fourth represents the 'historical' model described in Chapter 1 and the fifth and sixth factors represent the physical model also described in Chapter 1. In addition, an imposed model of 'low back pain distress' has been developed from the data. These models will be compared with the Fear Avoidance Model of Exaggerated Pain Perception in terms of

prediction of outcome of an acute episode of low back pain in Chapter 8.

CHAPTER 8

THE PREDICTION OF OUTCOME AT TWO MONTHS AND TWELVE MONTHS AFTER ONSET OF PAIN, USING DATA COLLECTED AT THE ACUTE STAGE AND AT TWO MONTHS FOLLOW-UP

8.1 INTRODUCTION

This chapter fulfills the first aim of the study: to test the validity of the Fear Avoidance Model of Exaggerated Pain Perception. Parts a and c of the fourth objective of the study are addressed in this chapter. They are: to use inferential statistics in order to a) test the validity of the Fear Avoidance Model as a predictor of outcome and c) examine the relationship between other models and the development of chronicity. Hypotheses 1 and 2 of the study are tested. These are:

1) The Fear Avoidance Model has utility in terms of predicting the outcome of an acute episode of low back pain and is therefore valid in terms of explaining chronic low back pain.
2) Low back pain is mediated in the long term by psychosocial variables rather than by physical pathology or impairment'.

Testing hypothesis 1 The predictive utility of the Fear Avoidance Model was assessed directly in terms of the outcome measures identified (see below). In addition, the predictive

utility of competing models was also assessed using the same outcome measures. A comparison of the competing models was undertaken using two approaches. Initially each model was tested individually using standard multiple regression and discriminant function analyses. A direct comparison was then made of the unique outcome variance attributable to the Fear Avoidance Model and that of the competing models using a heirarchical approach (see below).

Testing hypothesis 2 This hypothesis was tested by comparing the predictive utility of psychosocial models (Fear Avoidance and 'low back pain distress') with the physical model using standard and heirarchical regression and discriminat function analyses.

This chapter describes the the variables included in the analyses, the statistical methods used, the results of the analyses and discusses the results.

8.2 VARIABLES INCLUDED IN THE ANALYSES

Outcome (dependent) variables

The development of continuous and dichotomous outcome variables (see Chapter 6) presented an opportunity to address the issue of prediction of outcome in two ways. An exploration of the

validity of a group of variables as predictors of a continuous outcome variable is an appropriate method of testing the potential of those variables to form a theoretical explanatory model. However, the primary concern of the clinician may be the identification of the individuals who fail to recover. It is usual therefore, in clinical practice, to dichotomise individuals into those who do well and those who do not do well (in whatever terms are appropriate to the clinical discipline). The continuum of outcomes of those who are identified as having a 'successful outcome' is, in the latter situation, ignored.

The outcome variables used in the predictive analyses were:

1) Combined (by factor analysis) severity of pain and Roland and Morris disability score at <u>two</u> months after onset (see Table 6.6 on page 186).

2) Combined (by factor analysis) severity of pain and Roland and Morris disability score at <u>twelve</u> months after onset (see Table 6.7 on page 188).

3) Work status (whether on sick leave) at twelve months after onset.

4) History of pain since onset: 'no pain' and 'pain on and off' were combined into a 'good outcome group': and

patients reporting 'continuous pain' were labelled 'chronic low back pain patients' at twelve months after onset.

Predictor (independent) variables

Independent variables entered into the analyses represented the Fear Avoidance Model and the competing models described in Chapter 1. In addition, the contrived 'low back pain distress model' (the development of which was described in the previous chapter) was also used in comparison with the Fear Avoidance Model. Predictive analyses were carried out between three stages; acute to two months, acute to twelve months and two months to twelve months. The independent variables are as follows:

1) Fear Avoidance Model variables measured at the acute stage.
2) Demographic model variables measured at the acute stage.
3) Historical model variables measured at the acute stage
4) Physiological model variables measured at two months follow-up. Straight leg raise and passive knee bend were excluded from the physiological model as the results of principal components analyses presented in Chapter 7 suggest that these variables represent a psychological, rather than physical, construct. This issue has been discussed in Chapter 7.
5) Fear Avoidance Model variables measured at two months follow-up

6) 'Low back pain distress' model variables measured at two months follow-up (see Chapter 7)

8.3 STATISTICAL METHODS

Multiple regression analysis.

Multiple regression analysis is a technique which allows more than one potential predictor variable to be used in the prediction of outcome at the same time. The technique provides an equation which best describes the relationship between a group of predictor variables and a continuous outcome variable and is based on a linear model. The equation may be described thus:-

$V = W0 + W1\ V1 + W2\ V2 + ...Wi\ Vi + ...+ Wn\ Vn$

(Where V = Outcome, W0 = constant, W,Wi,Wn = weighting and V1.....Vi to Vn are predictor variables.)

In this technique the difference between the true outcome and the outcome predicted from the equation is called the residual. The smaller the residual, the more accurately the predictor variables predicted the outcome. A summary of the closeness of the predictions to the actual value (goodness of fit) is given in the analysis in the form of R^2 which varies between 0 and 1 (0 - 100%). This is the proportion of variability which can be

accounted for by the predictor variables, or, in other words, how much variance is in the fit of the equation and how much is in the residual. R is the correlation between predicted and actual outcome.

In order to assess the statistical significance of the contribution made by each independent variable to the prediction, a Students t - test is applied to each variable. A large value of t implies that that predictor variable has accounted for variability over and above the other variables in the equation if differences in the other variables are allowed for. This does not imply that a low significance of t signifies that the predictor involved does not play a part in the prediction of outcome, as that variable may well influence the'behaviour'of the other variables in the equation. Significance tests are sensitive only to the unique variance a variable adds to R^2. A very important variable that shares variance with another in the analysis may be nonsignificant although the two variables in combination are responsible in large part for the size of R^2. Snedecor's F test is applied to the model as a whole and the significance of F implies the significance of the prediction equation.

Outliers and influential points may affect the 'line of best fit' and the resulting regression equation. Outliers are points which lie some distance away from the plotted line of predicted variable against outcome variables. Influential

points exert a disproportionate influence or leverage on the plane of predictor variables and will alter the equation for prediction. A measure which is sensitive to both outliers and influential points is known as Cook's Deleted Residual (Cook, 1977). This measures the extent to which the regression coefficients would have changed if that case were excluded. If an influential point is identified and the data has been checked for error, the case can be excluded from the analysis. However this is only appropriate if the case turns out to be fundamentally different in some way from the other cases in the population (Dewey, 1988). In the case of the following analyses, plots of standardised residuals and Cook's deleted residuals were examined and no unusual phenomena were identified.

There are three major types of multiple regression analysis: standard multiple regression, hierarchical regression and statistical (stepwise and setwise) regression. In the standard model, all independent variables enter into the regression equation at once; each one is assessed as if it had entered the regression after all other variables had entered. Each variable is evaluated in terms of what it adds to the prediction of the dependant variable that is different from the predictability afforded by all the other variables. In heirarchical regression, variables enter the equation in an order specified by the researcher. Each variable is assessed in terms of what it adds to the equation at its own point of entry. Assigned

order of entry is determined by logical or theoretical considerations. The apparent 'importance' of a variable is determined in large measure by the order of entry of it and other variables. Statistical regression is a controversial procedure in which order of entry of variables is based solely on statistical criteria. This procedure is a model building rather than model testing procedure. To assess relationships among variables and answer the basic questions of multiple correlation, standard multiple regression is recommended (Tabachnick and Fidell, 1989).

In the context of this study, no assumptions have been made regarding the relative importance of individual variables which constitute the Fear Avoidance and other models. Therefore, standard multiple regression analyses are used.

Later analyses directly compare models in terms of the unique outcome variance they account for by using a 'repeated heirarchical procedure'. Each group of variables (models) is entered into the regression equation and assessed as if it had entered after all other groups. The increase in R^2 accounted for by the addition of the model is evaluated. The procedure is repeated for each group of variables. This enables the models to be compared in terms of the unique variance of outcome accounted for by each one. (This technique is described by Tabachnick and Fidell, 1989, page 157, and is available through the SPSS^X 'Regression/test' command).

Discriminant function analysis

Discriminant function analysis also involves the concept that linear combinations of the predictor variables are formed, which serve to classify cases into one of a number of groups (in this case outcome is measured in terms of history and sick leave status at twelve months). An equation is derived from the cases whose outcome is known (ie two and twelve months outcome) and applied retrospectively to the sample at the acute and two months stages. The validity of the equation as a predictor of outcome is expressed in terms of the percentage of cases correctly classified at the acute or two months stage. The percentage of 'poor outcomes' (individuals on sick leave at twelve months and individuals who report having been in constant pain) correctly classified is known as the sensitivity of the combination of predictor variables. Chi-square test is applied to the model as a whole and the significance of chi-square reflects the significance of the prediction equation. It is thought possible, by looking at the size and signs of standardised classification coefficients to determine the general influence that individual variables will have on the discriminant score (and hence classification) (Klecka WR, 1980, Discriminant Analysis, Sage University Paper).

8.4

STATISTICAL AND METHODOLOGICAL CONSIDERATIONS

Sex differences

Several differences in terms of potential predictor variables were demonstrated between males and females at the acute stage of the study (see Chapter 5). Therefore, the option of reporting the separate analyses of male and female data was considered. However, it was decided not to do this for the following reasons:

i) Separate analyses would have divided the sample in two, thereby reducing the number of subjects within each sample. This would inevitably lead to further instability of statistical methods, particularly discriminant function analysis, of which the assumption of similar outcome group sizes had already been violated (see below).

ii) Separate analyses may have led to difficulties in the interpretation of results and consequently interfered with the testing of the validity of the Fear Avoidance Model.

Missing data

The default options in multiple regression analysis and discriminant function analysis performed by SPSS^X in terms of method of entry of variables is 'listwise'. This results in the inclusion in the analysis of subjects who have a complete data set. Subjects with one or more missing variables are not included in the analysis and this leads to a reduction in the number of subjects included in the analysis.

The method of entry can be modified to include all subjects, regardless of missing data. However, this may lead to mathematical problems with the fitting process and also means that different parts of the model are being estimated from different subsamples.

The prediction of dichotomous outcome variables using discriminant function analysis

An assumption of discriminant function analysis concerns the approximate equality of outcome group size. A failure to meet this requirement can lead to instability of the analysis and its results. However, chronic low back pain has been reported to result from an acute attack in only ten percent of cases, and if discriminant function analysis is to be used to test a theoretical model as a predictor of chronicity outcome group sizes will inevitably be unequal. In order to minimise the

effect of unequal outcome group sizes, the analysis was designed to keep the number of subjects within each group as large as possible. Therefore, predictive analyses involving dichotomous outcome variables were carried out between the acute stage and twelve months follow-up only.

8.5 RESULTS

PREDICTION OF TWO MONTHS OUTCOME - COMBINED PAIN AND DISABILITY

Fear Avoidance Model variables measured at the acute stage

Multiple regression analysis demonstrated that the six component variables of the Fear Avoidance Model accounted for 25% of the variance of the combined severity of pain and disability outcome measure at two months after onset of pain ($F = 7.96$, $P = 0.0001$). The MSPQ score, highest ever internally experienced pain and percent active coping strategies had statistically significant effects over and above the other variables in the model ($P = 0.001$, $P = 0.016$ and $P = 0.031$ respectively). These results are presented in Table 8.1.

Historical model variables measured at the acute stage

Multiple regression analysis demonstrated that the historical variables accounted for 15% of the variance of the combined

severity of pain and disability outcome measure at two months after onset of pain (F = 5.04, P = 0.0003). Nature of onset of pain and severity of worst attack had statistically significant effects over and above the other variables in the model (P = 0.001, P = 0.002 respectively). These results are presented in Table 8.2.

Demographic model variables measured at the acute stage

Multiple regression analysis demonstrated that demographic variables accounted for 5% of the variance of the combined severity of pain and disability outcome measure at two months after onset of pain (F = 1.65, P = 0.151). These results are presented in Table 8.3.

Combined Fear Avoidance Model, historical and demographic models variables measured at the acute stage

Multiple regression analysis demonstrated that the Fear Avoidance Model, historical and demographic variables combined accounted for 34% of the variance of the combined severity of pain and disability outcome measure at two months after onset of pain (F = 3.881, P = 0.0001). Nature of onset of pain and MSPQ had statistically significant effects over and above the other variables in the model (P = 0.034, P = 0.002 respectively). These results are presented in Table 8.4. The

results of the analysis of the relationship between the three groups of variables is presented below.

PREDICTION OF TWELVE MONTHS OUTCOME

Fear Avoidance Model Variables measured at the acute stage

COMBINED PAIN AND DISABILITY

Multiple regression analysis demonstrated that the six component variables of the Fear Avoidance Model accounted for 14% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain ($F = 4.67$, $P = 0.0002$). The MSPQ score had a statistically significant effect on the predicted outcome score over and above the other variables in the model ($P = 0.001$). These results are presented in Table 8.5.

SICK LEAVE STATUS

Discriminant function analysis demonstrated that a combination of the six component variables of the Fear Avoidance Model correctly classified 70% of subjects in terms of sick leave status at twelve months after onset of pain (Chi square = 14.37, $P = 0.026$). These results are presented in Table 8.6.

CHRONICITY

Discriminant function analysis demonstrated that a combination of the six component variables of the Fear Avoidance Model correctly classified 66% of subjects in terms of chronicity (Chi square = 7.27, P = 0.296). These results are presented in Table 8.7.

Historical model variables measured at the acute stage

COMBINED PAIN AND DISABILITY

Multiple regression analysis demonstrated that historical variables accounted for 15% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain (F = 3.72, P = 0.004). Severity of the worst attack of low back pain had a statistically significant effect on the predicted outcome score over and above the other variables in the model (P = 0.003). These results are presented in Table 8.8.

SICK LEAVE STATUS

Discriminant function analysis demonstrated that a linear combination of historical variables correctly classified 68% of subjects in terms of sick leave status at twelve months

after onset of pain (Chi square = 12.49, P = 0.029). These results are presented in Table 8.9.

CHRONICITY

Discriminant function analysis demonstrated that a linear combination of historical variables correctly classified 79% of subjects in terms of chronicity (Chi square = 16.164, P = 0.006). These results are presented in Table 8.10.

Demographic model variables measured at the acute stage

COMBINED PAIN AND DISABILITY

Multiple regression analysis demonstrated that demographic variables accounted for 12% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain (F = 5.13, P = 0.0002). Smoking status and referring practice had statistically significant effects on the predicted outcome score over and above the other variables in the model (P = 0.021 and P = 0.002). These results are presented in Table 8.11.

SICK LEAVE STATUS

Discriminant function analysis demonstrated that a linear combination of demographic variables correctly classified 70% of subjects in terms of sick leave status at twelve months after onset of pain (Chi square = 14.34, P = 0.014). These results are presented in Table 8.12.

CHRONICITY

Discriminant function analysis demonstrated that a linear combination of demographic variables correctly classified 78% of subjects in terms of chronicity (Chi square = 18.044, P = 0.003). These results are presented in Table 8.10.

Combined Fear Avoidance Model, historical and demographic variables measured at the acute stage

COMBINED PAIN AND DISABILITY

Multiple regression analysis demonstrated that the three groups of variables combined accounted for 32% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain (F = 2.667, P = 0.002). Severity of the worst attack of low back pain, referring practice and MSPQ had statistically significant effects on

the predicted outcome score over and above the other variables in the model (P = 0.02, P = 0.034 and P = 0.006 respectively). These results are presented in Table 8.14. The results of the analysis of the relationship between these three groups of variables are presented below.

SICK LEAVE STATUS

Discriminant function analysis demonstrated that a linear combination of the combined variables correctly classified 85% of subjects in terms of sick leave status at twelve months after onset of pain (Chi square = 29.65, P = 0.02). These results are presented in Table 8.15.

CHRONICITY

Discriminant function analysis demonstrated that a linear combination of the combined variables correctly classified 86% of subjects in terms of chronicity (Chi square = 33.54, P = 0.006). These results are presented in Table 8.16.

PREDICTION OF TWELVE MONTHS OUTCOME - COMBINED PAIN AND DISABILITY

Fear Avoidance Model variables measured at two months

Multiple regression analysis demonstrated that the six

component variables of the Fear Avoidance Model accounted for 23% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain (F = 5.32, P = 0.0001). The MSPQ score had a statistically significant effect over and above the other variables in the model (P = 0.001). These results are presented in Table 8.17.

Physical model variables measured at two months

Multiple regression analysis demonstrated that the physical variables accounted for 21% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain (F = 4.89, P = 0.0003). Side flexion and nerve *root tethering had statistically significant effects* over and above the other variables in the model (P = 0.002, P = 0.012 respectively). These results are presented in Table 8.18.

Low back pain distress model variables measured at two months

Multiple regression analysis demonstrated that psycho-physical variables accounted for 37% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain (F = 10.53, P = 0.0001). These results are presented in Table 11.19. MSPQ had a statistically significant effect over and above the other variables in the model (P = 0.041). These results are presented in Table 8.19.

Combined Fear Avoidance Model, physical and low back pain distress models measured at two months

Multiple regression analysis demonstrated that the Fear Avoidance Model, physical and psycho-physical variables combined accounted for 49% of the variance of the combined severity of pain and disability outcome measure at twelve months after onset of pain ($F = 4.94$, $P = 0.0001$). Highest accidental pain had a statistically significant effect over and above the other variables in the model ($P = 0.039$,). These results are presented in Table 8.20. The results of the analysis of the relationship between the three groups of variables is presented below.

A summary of these results is presented in Table 8.24.

8.6 A COMPARISON OF THE FEAR AVOIDANCE MODEL WITH OTHER MODELS IN TERMS OF TWO AND TWELVE MONTHS OUTCOME VARIANCE

Examination of the results of separate standard regression analyses suggest that, in terms of variance predicted, the Fear Avoidance Model is comparable with historical, demographic and physical models and weaker than a model consisting of low back pain distress variables. In addition, eight out of nine regression equations have statistically significant values of F.

However, examination of the results of regression analyses which include Fear Avoidance Model, historical and demographic variables or Fear Avoidance Model (Tables 8.4 and 8.14), physical and low back pain distress variables (Table 8.20) demonstrates that the variance accounted for by combined models is greater than that accounted for by individual models. This suggests that the individual models account for discrete variance although some overlap occurs.

In order to compare the Fear Avoidance Model with the other models in terms of prediction of outcome, the effects of adding separate groups of variables to an overall combined model and thereby the contributions made by each group of variables to the overall model were identified. The method used is described above (section 8.3).

Examination of Tables 8.21 to 8.22 shows that in terms of two months prediction of combined pain and disability the Fear Avoidance Model adds explanatory power to the overall model. In terms of twelve months prediction of combined pain and disability this is not so. Table 8.23 shows that neither the Fear Avoidance Model nor the physical model (measured at two months) add explanatory power to the combined model in terms of prediction of twelve months combined pain and disability. It is the 'distress' model which adds statistically significant explanatory power to the overall model.

In view of the results of earlier regression analyses in which the t values of MSPQ were statistically significant, a similar procedure was used to examine the relationship between MSPQ and the other Fear Avoidance Model variables (pain history, coping strategies and life events). Examination of Table 8.24 demonstrates that MSPQ and the other Fear Avoidance Model variables add separate explanatory power to the Model in terms of prediction of two months combined pain and disability. Examination of Table 8.25 demonstrates that MSPQ adds significantly to the predictive power of the Fear Avoidance Model in terms of prediction of combined pain and disability at twelve months. The remaining variables do not add significantly to the predictive power of MSPQ.

A summary of the results of the predictive analyses is presented in Table 8.26.

Table 8.1

Prediction of two months outcome (combined pain and disability) using Fear Avoidance Model variables.

Multiple R	R Square	DF	F-Value	Probability of F
.500	.250	143	7.967	0.0001

Variables in the equation	B	SE of B	t	Probability of t
Weighted life events	.00036	.00079	.45	.653
Highest external pain	.051	.036	1.40	.162
Percent active coping	-.005	.002	-2.18	.031
MSPQ	.091	.018	4.81	.001
Highest accidental pain	-.068	.039	-1.73	.085
Highest internal pain	.087	.035	2.43	.016
Constant	-.786	.352	-2.23	.027

Table 8.2

Prediction of two months outcome (combined pain and disability) using historical variables.

Multiple R	R Square	DF	F-Value	Probability of F
.389	.151	141	5.037	0.0003

Variables in the equation	B	SE of B	t	Probability of t
Whether this is first episode	-.362	.266	-1.36	.175
Number of previous attacks	.025	.026	0.97	.331
Severity of first attack	.009	.036	0.25	.802
Insidious or accidental onset	.066	.019	3.41	.001
Severity of worst attack	.162	.053	3.07	.002
Constant	-.664	.412	-1.61	.109

Table 8.3

Prediction of two months outcome (combined pain and disability) using demographic variables.

Multiple R	R Square	DF	F-Value	Probability of F
.227	.051	151	1.648	0.1505

Variables in the equation	B	SE of B	t	Probability of t
Smoking	.066	.177	.373	0.709
Maghull or others	.199	.163	.100	0.244
Occupational group	.142	.107	.114	0.187
Married or single	.120	.085	1.407	0.161
Age	-.159	.123	-1.294	0.197
Constant	-.526	.417	-1.261	0.209

Table 8.4

Prediction of two months outcome (combined pain and disability) using Fear Avoidance Model, demographic and historical variables measured at the acute stage.

Multiple R	R Square	DF	F-Value	Probability of F
.582	.339	121	3.881	0.0001

Variables in the equation	B	SE of B	t	Probability of t
Whether this is first episode	-.320	.264	-1.20	.228
Number of previous attacks	.021	.026	.08	.418
MSPQ	.075	.019	3.79	.002
Severity of first attack	.031	.037	.85	.396
Occupational group	.020	.102	.19	.843
Smoking	-.003	.163	-.02	.981
Marital status	.109	.081	1.35	.178
Percent active coping	-.004	.003	-1.95	.054
Highest accidental pain	-.047	.002	-1.22	.226
Insidious or accidental onset	-.242	.112	-2.15	.034

Table 8.4 (cont.)

Variables in the equation	B	SE of B	t	Probability of t
Maghull or others	.103	.156	.65	.513
Weighted life events	.0006	.0007	.82	.412
Highest external pain	.018	.037	.49	.624
Age	-.022	.122	-.18	.857
Severity of worst attack	.098	.055	1.79	.075
Highest internal pain	.057	.037	1.55	.124
Constant	-1.272	.583	-2.18	.031

Table 8.5

Prediction of twelve months outcome (combined pain and disability) using Fear Avoidance Model variables measured at the acute stage.

Multiple R	R Square	DF	F-Value	Probability of F
.370	.137	176	4.673	0.0002

Variables in the equation	B	SE of B	t	Probability of t
Weighted life events	.00026	.00065	.39	.696
Highest external pain	.035	.032	1.08	.281
Percent active coping	-.00017	.002	-0.06	.945
MSPQ	.079	.018	4.30	.001
Highest accidental pain	.036	.037	.94	.341
Highest internal pain	.014	.037	.38	.702
Constant	-1.086	.369	-2.94	.003

Table 8.6

Prediction of twelve months outcome (sick leave status) using Fear Avoidance Model variables measured at the acute stage.

	Predicted	
Actual	Not on sick leave	On sick leave
Not on sick leave	106	41
On sick leave	10	12

Percent correctly classified = 70%

Sensitivity = 55% Specificity = 72%

Chi square = 14.37 DF = 6 Probability = 0.0258

Standardized canonical discriminant function coefficients

Weighted life events	-0.262
Highest external pain	0.423
Percent active coping	0.003
MSPQ	0.987
Highest accidental pain	0.087
Highest internal pain	0.423

Table 8.7

Prediction of twelve months outcome (chronicity) using Fear Avoidance Model variables measured at the acute stage.

	Predicted	
Actual	Not chronic	Chronic
Not chronic	112	55
Chronic	6	9

Percent correctly classified = 66%

Sensitivity = 60% **Specificity** = 67%

Chi square = 7.273 **DF** = 6 **Probability** = 0.296

Standardized canonical discriminant function coefficients

Weighted life events	0.141
Highest external pain	-0.289
Percent active coping	0.026
MSPQ	0.810
Highest accidental pain	0.046
Highest internal pain	0.368

Table 8.8

Prediction of twelve months outcome (combined disability and pain) using historical variables.

Multiple R	R Square	DF	F-Value	Probability of F
.383	.147	108	3.722	0.0038

Variables in the equation	B	SE of B	t	Probability of t
Whether this is first episode	-.239	.315	-0.75	.449
Number of previous attacks	.027	.029	0.91	.362
Severity of first attack	.035	.044	0.78	.439
Insidious or accidental onset	-.228	.127	-1.79	.075
Severity of worst attack	.186	.062	3.01	.003
Constant	-1.377	.493	-2.79	.006

Table 8.9

Prediction of twelve months outcome (sick leave status) using historical variables.

	Predicted	
Actual	Not on sick leave	On sick leave
Not on sick leave	57	30
On sick leave	2	11

Percent correctly classified = 68.0%

Sensitivity = 85% Specificity = 66%

Chi square = 12.49 DF = 5 Probability = 0.0286

Standardized canonical discriminant function coefficient

Severity of first attack	0.183
Severity of worst attack	0.803
Insidious onset	-0.593
Number of previous attacks	0.246
Whether this was first attack	-0.363

Table 8.10

Prediction of twelve months outcome (chronicity) using historical variables.

	Predicted	
Actual	Not chronic	Chronic
Not chronic	82	24
Chronic	0	8

Percent correctly classified = 79%

Sensitivity = 100% **Specificity** = 77%

Chi square = 16.164 **DF** = 5 **Probability** = 0.0064

Standardized canonical discriminant function coefficients

Severity of first attack	0.487
Severity of worst attack	0.681
Insidious onset	-0.495
Number of previous attacks	0.065
Whether this was first attack	-0.058

Table 8.11

Prediction of twelve months outcome (combined pain and disability) using demographic variables.

Multiple R	R Square	DF	F-Value	Probability of F
.349	.122	184	5.131	0.0002

Variables in the equation	B	SE of B	t	Probability of t
Smoking	.345	.148	2.322	0.021
Maghull or others	.461	.147	.229	0.002
Occupational group	.158	.095	1.659	0.098
Married or single	.069	.066	1.049	0.295
Age	.026	.102	.260	0.795
Constant	-1.623	.365	-4.443	0.000

Table 8.12

Prediction of twelve months outcome (sick leave status) using demographic variables.

	Predicted	
Actual	Not on sick leave	On sick leave
Not on sick leave	109	43
On sick leave	10	14

Percent correctly classified = 70%

Sensitivity = 58% Specificity = 72%

Chi square = 14.347 DF = 5 Probability = 0.014

Standardized canonical discriminant function coefficient

Smoking	.252
Maghull or others	.572
Occupational group	.584
Married or single	.468
Age	.413

Table 8.13

Prediction of twelve months outcome (chronicity) using demographic variables.

	Predicted	
Actual	Not chronic	Chronic
Not chronic	137	37
Chronic	4	12

Percent correctly classified = 78%

Sensitivity = 75.00% **Specificity** = 79%

Chi square = 18.044 **DF** = 5 ***Probability*** = *0.0029*

Standardized canonical discriminant function coefficients

Smoking	-.205
Maghull or others	.631
Occupational group	.413
Married or single	.554
Age	.332

Table 8.14

Prediction of twelve months outcome (combined pain and disability) using Fear Avoidance Model, demographic and historical variables measured at the acute stage.

Multiple R	R Square	Df	F-Value	Probability of F
.565	.319	91	2.667	0.0017

Variables in the equation	B	SE of B	t	Probability of t
Whether this is first episode	-.271	.324	-.84	.404
Number of previous attacks	.029	.032	.11	.373
MSPQ	.069	.025	2.79	.006
Severity of first attack	.028	.046	.62	.538
Occupational group	.111	.128	.87	.386
Smoking	.349	.213	1.64	.103
Marital status	-.00002	.096	-.01	.997
Percent active coping	-.00006	.003	-.18	.852
Highest accidental pain	.015	.048	.33	.746
Insidious or accidental onset	-.130	.135	-.97	.336

Table 8.14 (cont.)

Variables in the equation	B	SE of B	t	Probability of t
Maghull or others	.412	.192	2.15	.034
Weighted life events	.0001	.0009	.15	.885
Highest internal pain	.009	.051	.19	.854
Age	.052	.150	.35	.728
Severity of worst attack	.157	.066	2.36	.020
Highest external pain	-.012	.045	-.27	.788
Constant	-2.969	.760	-3.90	.001

Table 8.15.

Prediction of twelve months outcome (sick leave status) using Fear Avoidance Model, historical and demographic variables measured at the acute stage.

	Predicted	
	Not on sick leave	**On sick leave**
Actual		
Not on sick leave	71	12
On sick leave	2	9

Percent correctly classified = 85%

Sensitivity = 81% **Specificity** = 86%

Chi square = 29.652 **DF** = 16 **Probability** = 0.0199

Standardized canonical discriminant function coefficients

Whether this is first episode	-.349
Number of previous attacks	.331
MSPQ	.359
Severity of first attack	-.026
Occupational group	.365
Smoking	.114
Marital status	.105

Table 8.15 (cont.)

Standardized canonical discriminant function coefficients

Percent active coping	-.087
Highest accidental pain	.035
Insidious or accidental onset	-.186
Maghull or others	.582
Weighted life events	-.167
Highest internal pain	-.479
Age	.379
Severity of worst attack	.547
Highest external pain	-.012

Table 8.16.

Prediction of twelve months outcome (chronicity) using Fear Avoidance Model, historical and demographic variables measured at the acute stage.

	Predicted	
	Not chronic	Chronic
Actual		
Not chronic	86	13
Chronic	0	8

Percent correctly classified = 88%

Sensitivity = 100% Specificity = 86%

Chi square = 33.544 DF = 16 Probability = 0.0063

Standardized canonical discriminant function coefficients

Whether this is first episode	-.001
Number of previous attacks	.004
MSPQ	.096
Severity of first attack	.455
Occupational group	.373
Smoking	.058
Marital status	-.093

Table 8.16 (cont.)

Standardized canonical discriminant function coefficients

Percent active coping	-.043
Highest accidental pain	-.165
Insidious or accidental onset	-.338
Maghull or others	.556
Weighted life events	.357
Highest internal pain	.108
Age	.293
Severity of worst attack	.346
Highest external pain	-.061

Table 8.17

Prediction of twelve months outcome (combined pain and disability) using Fear Avoidance Model variables measured at two months.

Multiple R	R Square	DF	F-Value	Probability of F
.475	.226	109	5.317	0.0001

Variables in the equation	B	SE of B	t	Probability of t
Weighted life events	.0006	.0008	.720	.473
Percent active coping	-.0005	.003	-.188	.850
Highest external pain	-.018	.041	-.455	.650
MSPQ	.113	.024	4.683	.001
Highest accidental pain	.069	.045	1.528	.129
Highest internal pain	.027	.044	.626	.532
Constant	-1.135	.412	-2.751	.007

Table 8.18

Prediction of twelve months outcome (combined pain and disability) using physical variables measured at two months.

Multiple R	R Square	DF	F-Value	Probability of F
.453	.206	113	4.889	0.0002

Variables in the equation	B	SE of B	t	Probability of t
Side flexion	-.034	.010	-3.155	.002
Neurological deficit	.202	.216	.936	.351
Sagittal extension	.016	.008	1.890	.061
Nerve root tethering	.466	.183	2.55	.012
Sagittal flexion	.009	.009	1.01	.315
Ability to sit up	-.205	.164	-1.24	.214
Constant	-.043	.552	-.08	.936

Table 8.19

Prediction of twelve months outcome (combined pain and disability) using 6 distress variables identified by discriminant function analysis of two months data.

Multiple R	R Square	DF	F-Value	Probability of F
.607	.369	108	10.53	0.0001

Variables in the equation	B	SE of B	t	Probability of t
Inappropriate signs and symptoms	.070	.044	1.59	.115
MSPQ	.050	.024	2.06	.041
Roland and Morris disability	.016	.022	.70	.483
Zung	.006	.009	.62	.530
Severity of present pain	.032	.048	.67	.505
Oswestry disability	.011	.010	1.16	.248
Constant	-1.930	.630	-3.06	.002

Table 8.20

Prediction of twelve months outcome (combined pain and disability) using Fear Avoidance Model variables, physical variables and distress variables measured at two months.

Multiple R	R Square	DF	F-Value	Probability of F
.698	.488	98	4.94	0.0001

Variables in the equation	B	SE of B	t	Probability of t
Passive knee bend	-.008	.005	-1.65	.101
Sagittal extension	.012	.008	1.50	.136
Neurological deficit	.103	.198	.52	.603
Life events	.0004	.0008	.41	.681
Highest external pain	-.042	.040	-1.03	.305
MSPQ	.048	.026	1.83	.071
Ability to sit up	-.092	.152	-.61	.545
Percent active coping	.004	.003	1.16	.251
Nerve root tethering	.280	.182	1.54	.126
Sagittal flexion	.015	.009	1.60	.113
Highest accidental pain	.089	.043	2.08	.039
Highest internal pain	-.004	.043	-.10	.919

Table 8.20 (cont)

Variables in the equation	B	SE of B	t	Probability of t
Side flexion	-.005	.012	-.52	.602
Straight leg raise	-.011	.006	-1.70	.092
Pain drawing	-.003	.014	-.27	.788
Zung	.015	.010	1.55	.124
Inappropriate signs and symptoms	.047	.048	.96	.340
Constant	-.802	1.179	-.68	.498

Table 8.21

A comparison of the Fear Avoidance Model, historical model and demographic model in terms of explained 2 months outcome variance

Variables	DF	Sum of squares	R Squared change	F	Probability of F
Fear Avoidance Model	6	19.294	.153	4.649	.0003
Historical	5	7.591	.059	2.195	.059
Demographic	5	2.025	.015	.586	.711

Table 8.22

A comparison of the Fear Avoidance Model, historical model and demographic model in terms of explained 12 months outcome variance

Variables	DF	Sum of squares	R Squared change	F	Probability of F
Fear Avoidance Model	6	7.518	.070	1.563	.1668
Historical	5	8.926	.083	2.227	.058
Demographic	5	7.338	.068	1.832	.114

Table 8.23

A comparison of the Fear Avoidance Model, physical model and 'distress' model in terms of explained 12 months outcome variance

Variables	DF	Sum of squares	R Squared change	F	Probability of F
Fear Avoidance Model	6	6.403	.060	1.723	.125
Physical	5	4.615	.043	1.242	.293
Distress	6	13.214	.124	4.268	.002

Table 8.24

A comparison of MSPQ and other Fear Avoidance Model variables in terms of outcome variance explained - 2 months

Variables	DF	Sum of squares	R Squared change	F	Probability of F
Fear Avoidance Model without MSPQ	5	12.884	.084	3.213	.009
MSPQ	1	18.554	.121	23.137	.001

Table 8.25

A comparison of MSPQ and other Fear Avoidance Model variables in terms of outcome variance explained - 12 months

Variables	DF	Sum of squares	R Squared change	F	Probability of F
Fear Avoidance Model without MSPQ	5	3.651	.020	.826	.533
MSPQ	1	16.361	.091	18.501	.001

Table 8.26

Summary of predictive analyses

Variables	Two months Outcome variables	Results
Acute stage		
FAM	Combined pain/disability (at 2 months)	R Square = .250 F = 7.967 P = .0001
Historical		R Square = .151 F = 5.037 P = .0003
Demographic		R Square = .051 F = 1.648 P = 0.151
FAM and Historical and Demographic		R Square = .339 F = 3.881 P = .0001

Table 8.26 (cont.)

Summary of predictive analyses

Variables	Twelve months Outcome variables	Results
Acute stage		
FAM	Combined pain/disability (at 12 months)	R Square = .137 F = 4.673 P = .0002
Historical		R Square = .147 F = 3.722 P = .004
Demographic		R Square = .122 F = 5.131 P = 0.0002
FAM and Historical and Demographic		R Square = .319 F = 2.667 P = .0017

Table 8.26 (cont.)

Summary of predictive analyses

Variables	Twelve months Outcome variables	Results
Acute stage		
FAM	Sick leave status (at 12 months)	% classified = 70%
		Sensitivity = 55%
		Specificity = 72%
		Chi square = 14.37
		P = .0258
	Chronicity (at 12 months)	% classified = 66%
		Sensitivity = 60%
		Specificity = 67%
		Chi square = 7.273
		P = .296
Historical	Sick leave status (at 12 months)	% classified = 68%
		Sensitivity = 85%
		Specificity = 66%
		Chi square = 12.49
		P = .0286
	Chronicity (at 12 months)	% classified = 79%
		Sensitivity = 100%
		Specificity = 77%
		Chi square = 16.16
		P = .0064

Table 8.26 (cont.)

Summary of predictive analyses

Variables	Twelve months Outcome variables	Results
Acute stage		
Demographic	Sick leave status (at 12 months)	% classified = 70% Sensitivity = 58% Specificity = 72% Chi square = 14.34 P = .014
	Chronicity (at 12 months)	% classified = 78% Sensitivity = 75% Specificity = 79% Chi square = 18.04 P = .0029
FAM and Historical and Demographic	Sick leave status (at 12 months)	% classified = 85% Sensitivity = 81% Specificity = 86% Chi square = 29.65 P = .0199
	Chronicity (at 12 months)	% classified = 88% Sensitivity = 100% Specificity = 86% Chi square = 33.54 P = .0063

Table 8.26 (cont.)

Summary of predictive analyses

Variables	Twelve months Outcome variables	Results
Two months follow-up		
FAM	Combined pain/disability (at 12 months)	R Square = .226 F = 5.317 P = .0001
Physical		R Square = .206 F = 4.889 P = .0002
Distress		R Square = .369 F = 10.53 P = 0.0001
FAM and Physical and Distress		R Square = .488 F = 4.94 P = .0001

8.7 DISCUSSION

The results presented in this chapter support Hypothesis 1 of the study which states that 'the Fear Avoidance Model has utility in terms of predicting the outcome of an acute episode of low back pain and is therefore valid in terms of explaining chronic low back pain'.

In addition, hypothesis 2, which states that 'low back pain is mediated in the long term by psychosocial variables rather than by physical pathology or impairment' is supported in part by the results of this study.

In terms of the prediction of combined pain and disability at two and twelve months, and the prediction of sick leave status at twelve months, the validity of the Fear Avoidance Model of Exaggerated Pain Perception is supported. In terms of the prediction of'chronicity' (defined in terms of constant pain) at twelve months after onset the Model has not been shown to have statistically significant predictive utility.

The physical model has been shown to have significant utility in terms of predicting twelve months combined pain and disability. However, when tested against the Fear Avoidance Model and 'Low Back Pain distress' model (derived from principal components analysis reported in Chapter 7) it does not account for significant outcome variance over and above the

other models. The author interprets this finding as being in support of Hypothesis 2 and proposes that the outcome variance explained by the physical model is an expression of psychological reaction to pain rather than physical pathology. The apparent correlation between the Fear Avoidance and physical models at two months supports the fear avoidance construct described in Chapter 1. The finding suggests that spinal weakness and stiffness are positively associated with fear and avoidance. This suggests that fear (which leads to an unwillingness to rehabilitate) results in actual physical deterioration. Viewed this way, the author suggests that the physical model simply represents an alternative Fear Avoidance Model.

The best predictor of the course of low back pain over the first two months appears to be the Fear Avoidance Model. From then on, the competing acute stage models overlap in terms of outcome variance accounted for (Tables 8.21 and 8.22). This suggests that Fear Avoidance may become less important as a mediator of outcome as the natural history lengthens and other variables become increasingly more important. However, this finding may also be a consequence of the drop out rate which characterised the study. The two and twelve months sample differed in terms of numbers and in the way in which data were collected at each stage. It is possible that the characteristics of the twelve months sample was such that the

outcome of their back pain was less easily mediated by fear-avoidance than that of the two months sample.

The historical, demographic, physical and 'distress' models have also been demonstrated to have significant predictive utility. When the separate models are combined at the acute and two months stages, the overall model accounts for a larger proportion of outcome variance than any of the individual models. This suggests that each model accounts for a measure of unique outcome variance, although some overlap exists. This finding supports the notion that low back pain experience is complex and multi-faceted. The author proposes that, although appealing, simple, single construct models of chronic low back pain have limited use as theoretical foundations for clinical intervention. This view is discussed further in the following chapter.

In terms of the prediction of sick leave status and chronicity, historical and demographic models are more 'successful' than the Fear Avoidance Model. In large measure, this may be accounted for by the nature of the component parts of the models. In terms of the demographic model, occupational group carries the largest standardized weighting on the sick leave discriminant function. In terms of the historical model, the coefficients suggest that outcome may be a function of the remembered severity of previous attacks. If severity of previous back pain represents severity of pathology, these

findings suggest that working class, industrial workers with advanced degenerative pathology are more likely to suffer a severe episode of low back pain from which they are unlikely to recover. However, an alternative view of the 'severity of previous low back pain' variables is presented below.

The apparent difference in performance between the Models in the prediction of different measures of outcome may also be related to the nature of the outcome measures themselves. Combined pain and disability is based upon an individual's self reported level of dysfunction and pain perception. Psychological variables may play a major part in the perception of these outcome measures. On the other hand, it can be argued that sick leave status and the experience of constant pain (without assigning a numerical value of intensity of experience) may be less likely to be influenced by psychological variables alone but may also be a function of physiological pathology. If this is so, a psychological model such as the Fear Avoidance Model may be more sensitive in its prediction of a psychological phenomenon (self reported pain and disability) than models which may have a large physiological component such as the historical and demographic models which in turn have been demonstrated to be superior predictors of sick leave status and chronicity. In addition, it can be argued that the large difference in outcome group size makes the results of discriminant function analysis unreliable. However, taking this into account, the author believes that the

results of discriminant function analysis are valid, especially in terms of prediction of chronicity by the historical and combined models.

Predictive analyses between two months and twelve months demonstrates the superiority (in terms of variance accounted for) of the low back pain distress model as a predictor of outcome. This is perhaps not surprising as the model contains severity of pain and Roland and Morris disability. These variables, when combined, represent the twelve months outcome which the model predicts. It has been demonstrated in Chapter 6 that, in large measure, twelve months outcome in terms of pain and disability is a reflection of two months outcome.

In terms of the contribution made to the prediction of outcome by individual variables, the coefficients assigned to variables by regression analysis do not enable the author to comment on the relative importance of each variable in the same way as univariate analyses (see section 8.3). However, the direction of the relationship between predictor and outcome variables can be identified, as can variables which account for outcome variance over and above that of the other variables in the equation. The coefficients which are presented in the results of discriminant function analysis allow the author to comment on the direction of the relationship between predictor variables and outcome variables, and the relative importance of one predictor variable compared with another. In general, no

consistent pattern can be identified in terms of the 'behaviour' of individual variables which form the Fear Avoidance and competing models. In addition, it has been recognised earlier in this chapter that the absence of a statistically significant t value associated with a regression coefficient does not necessarily indicate that the variable is unimportant in terms of the overall model. Therefore, the component variables of the Fear Avoidance Model only will be discussed. However, variables which represent memory for previous back pain made a consistent contribution to the prediction of outcome as part of the historical model. Because of this, and an interpretation of the data which incorporates these variables into the fear-avoidance construct (Chapter 9), memory for pain variables are also discussed below.

Life events - In the context of the Fear Avoidance Model or combined models, weighted life events score makes very little contribution to the prediction of outcome of an acute attack of low back pain. This finding is in accord with the view of Rabkin and Struening (1976) who stated that "in practical terms, then, life events scores have not been shown to be predictors of the probability of future illness". However, the generally low significance of the t value associated with life events score throughout the analyses does not necessarily imply that life events do not play a part in the genesis of poor outcome, as that variable may well influence the 'behaviour' of other variables in the equation. The interactive nature of

'stress' emphasises the need to consider life events in this context as part of an integrated model that accounts for the importance of cognitive and behavioural variables such as context, appraisal and coping strategies.

Coping strategies - The concept of avoidance or confrontation in response to pain experience is central to the Fear Avoidance Model of Exaggerated Pain Perception. The results of the predictive analyses support the hypothesis that pain avoiders are less likely to recover from an acute attack of low back pain. The coping strategies variable accounted for a significant proportion of combined pain and disability variance at two months after onset and was negatively related to outcome. This finding supports the view that individuals who have a poor outcome are those who adopt passive coping strategies in response to pain experience (Lethem et al., 1983). However, in terms of twelve months outcome, coping strategies do not play a significant part in its prediction, although the majority of analyses demonstrated that the relationship between active coping and poor outcome was in a negative direction. Several other studies have identified a relationship between passive coping strategies and physical and psychological dysfunction resulting from pain experience (Rosenstiel and Keefe, 1983, Turner and Clancy, 1986, Brown and Nicassio, 1987, Keefe et al., 1990). However, these studies have included the use of validated coping strategy questionnaires such as the Coping Strategies Questionnaire

(CSQ) (Rosenstiel and Keefe, 1983) which consists of 42 items including different types of cognitive strategies and behavioural strategies. In the view of the author, the inclusion in this study of a comprehensive coping strategies instrument such as the CSQ, which contains cognitive elements in addition to behavioural elements, would have led to the identification of a clearer relationship between coping strategies and outcome.

Pain history - Viewed separately, the contributions made by the three measures of pain history (highest ever internally, externally and accidentally produced pain), or memory for pain, are difficult to interpret. However, the use of highest ever pain rather than the mean value for each type of pain was shown at the preliminary analysis stage to increase the predictive power of the regression analyses. This supports in part the hypothesis of Lethem et al (1983) who proposed that "previous attacks of acutely crippling pain might sensitize the individual to fear of pain and increase the probability of an avoidance response whenever more pain is threatened". The results of a subsequent cross-sectional study by the same authors (Slade et al., 1983) demonstrated a positive correlation between internally and externally produced pain and severity of previous episodes of low back pain. The results of this study demonstrate both positive and negative relationships between outcome and severity of previous pain and are not in accord with the results of Slade et al. However,

these findings may be statistical artefacts which are a result of the small values of the associated coefficients. These results suggest that pain history may not play a significant role in development of confrontational or avoidance behaviour in response to acute back pain. However, the effect of pain history on behaviour in response to a new episode of pain may be pain specific, ie memory of severe abdominal pain may lead to avoidance of abdominal pain and not affect behaviour in response to toothache. This view is supported by the part played by 'severity of worst ever attack of low back pain' in the prediction of outcome in this study (see below) and the findings of Rose et al., (1992) who demonstrated that the influence of memory for internal, accidental and external pain is related to the nature of the original physical pathology (see Chapter 1).

Modified Somatic Perception Questionnaire (MSPQ) - The MSPQ is consistent in the contribution it makes to the prediction of outcome throughout this study and its use as a predictor of outcome within the framework of fear of pain and avoidance behaviour has been justified. In the prediction of twelve months combined pain and disability its removal from the Fear Avoidance Model results in a significant reduction in variance explained whereas removal of the other five variables leaving only MSPQ does not. In all cases, MSPQ is positively related to poor outcome. It was included in the study in order to represent the 'personality' component of the Fear Avoidance

Model because it has been shown to correlate with several scales on the MMPI. In addition, the MSPQ may represent the somatic component of the fear construct and is therefore central to the Fear Avoidance Model (see Chapter 1). The results of this study support the view of Lethem et al. (1983) who proposed that individuals who were hypochondriacal, hysterical and depressed (MMPI scales with which the MSPQ has been shown to correlate) were less likely to confront pain and, as a consequence, were less likely to recover from an attack of low back pain. In addition, the success of the MSPQ in explaining outcome variance supports in large measure the notion that fear and avoidance mediate the natural history of low back pain. However, these findings are in contrast with those of Deyo et al. (1989) who, in a cross-sectional study of chronic low back pain patients did not identify a relationship between MSPQ and functional outcome measures and identified only a weak relationship between MSPQ and pain outcome. Despite this, the results of this and other studies (Main et al, 1983, Greenhough et al, 1989) support the inclusion of the MSPQ in a any theoretical model of chronic low back pain which is underpinned by fear-avoidance theory.

Severity of worst attack of low back pain - Memory for the severity of the worst ever attack of low back pain consistently contributes to the prediction of outcome reported in this study. This is in contrast to the apparent contribution made by severity of the first attack or present attack of low back pain

and in large measure that made by the severity of the worst internal, external and accidental pains (see above). The relationship between severity of worst attack and poor outcome is in a positive direction.

Memory for pain is implied in the nature of the visual analogue scales used throughout this study as they are anchored by 'no pain' and 'worst pain imaginable' (Erskine et al., 1990) and it is likely that the 'worst pain imaginable' was interpreted by subjects as meaning the worst pain they had experienced. The issues of the prediction of the severity of a future episode of pain and pain memory may account for the apparent importance of memory for the severity of the worst ever episode of low back pain in the prediction of outcome demonstrated by this study. Rachman and Arntz (1991) state that "the overprediction of aversive events is linked to avoidance behaviour, and in cases of chronic pain, excessive/persistent overpredictions of expected pain can promote disabling avoidance and invalid behaviour". The same authors make the observation that fearful people may overpredict fear and panic (or pain) and that a back pain patient may remember an intensely painful acute pain vividly and forget thousands of pain free (or minor) episodes. It is also possible that the issue of whether memory of pain is recalled from either semantic or episodic memory (Erskine et al., 1990) may be in part responsible for the apparent importance of 'severity of worst ever back pain' compared with other pain memories. It is likely that the experience of what

will become the worst ever back pain is associated with fear and includes the mood, lifestyle consequences and other 'cognitive referents of the pain problem'. Other minor episodes of back pain (such as the first) may be less likely to 'tap into' the affective and cognitive dimensions of back pain experience and consequently be less likely to predict the future course of a condition which may be mediated by affective and cognitive variables.

8.8 CONCLUSIONS

The results presented in this chapter have demonstrated that the Fear Avoidance Model of Exaggerated Pain Perception has predictive utility in terms of the outcome of an episode of low back pain and is therefore a valid model of chronic low back pain, particularly at an early stage.

Competing models of chronic low back pain have also been shown to have predictive utility. The results have also shown that low back pain is a multi-faceted and complex phenomenon and can be explained in part by several, apparently different, models. However, these models can be integrated within the framework of the fear avoidance construct.

The chapter presents evidence which suggests that a physical model of back pain experience is less valid than those which incorporate psychosocial constructs.

Finally, individual variables have been identified which make a significant contribution to explaining the outcome of acute low back pain. The following chapter will include an interpretation of these findings within the context of the overall study and discuss the importance of the individual variables in the development of a reformulated Fear Avoidance Model.

CHAPTER 9

GENERAL DISCUSSION

9.1 INTRODUCTION

The following chapter is concerned with the identification of the main findings of the study and their wider implications. In addition, the shortcomings of the study are discussed as are the recommendations for future research. The key element of this chapter concerns the reformulation of the Fear Avoidance Model which results in part from the findings of the present study and from a theoretical analysis based upon recent literature and clinical experience. The above areas of discussion are drawn together in the 'implications for future research' section. The final part of the chapter consists of the overall conclusions which can be drawn from the study.

9.2 MAIN FINDINGS

The results of this study provides support for the predictive utility and consequent validity of the Fear Avoidance Model of Exaggerated Pain Perception (Lethem et al, 1983). This support has been provided within the context of what, to the author's knowledge, is the largest longitudinal study of acute low back

pain patients in a general practice setting to date. The component of the 'psychosocial context' of the Model which contributed most in terms of prediction of outcome is the MSPQ. In the author's view, this reflects first, that the MSPQ has sound psychometric properties and second, is a measure of the somatic component of anxiety and is therefore an appropriate instrument to include in a model which is concerned with the fear reaction to pain experience. In terms of prediction of two months outcome, the other variables which Lethem et al (1983) use to define the 'psychosocial context' (ie life events, pain memory and coping strategies) collectively make a significant contribution in addition to the MSPQ. However, individually, these variables do not add to the prediction of outcome over and above other variables in the regression analyses. Between two and twelve months, pain memory, life events and coping strategies combined fail to make a significant contribution to the prediction of outcome. These findings are interpreted as representing the weaknesses of the variables concerned, rather than the fear avoidance concept in two ways. First in psychometric terms and second in theoretical terms. Although statistically significant, the test-retest repeatability of the memory for pain variables and 'percent active coping strategies' is low (Chapter 3). In addition, from a theoretical standpoint, it can be argued that 'previous stressful life events' and 'general pain history' do not fall within the framework of fear-avoidance theory in the context of low back pain and consequently will not contribute to the prediction of

outcome of an acute attack. This view is expanded in section 9.2. A reformulated Fear Avoidance Model will be presented below.

The results of this study demonstrate the complex nature of low back pain experience. Cross-sectional and longitudinal correlational analyses of the variables included in the study undermine the traditional model of pain experience which attaches particular importance to the role of pathophysiology in determining the natural history of acute low back pain. However, in the view of the author, the true complexity of back pain experience is not fully elucidated by the results of this study because of the limited nature of the variables included, particularly those which measure the cognitive aspects of pain experience. The focus of the study concerned the fear avoidance concept represented by the Lethem's Fear Avoidance Model. However, the effects of low back trouble can be mediated by variables derived from all physical, psychological, sociological and economic aspects of human experience. Physical impairment, illness behaviour, attributional style regarding disability, affect and 'distress' all shape individual response to low back pain. At a more macro level, there is no doubt that the social infrastructure within which disability operates acts as a reinforcer of disability, particularly as time persists and social, economic and family relationships change (Slade, 1984).

The identification of these aspects of back pain experience in the literature and the clinical experience of the author has led to the formulation of a 'rehabilitation drive' construct which may be a powerful factor in shaping adjustment and disability. This construct is characterised by fear avoidance (including fear avoidance cognitions) but also recognises socioeconomic variables outside the individual's own control which shape the drive, or desire, to rehabilitate. This view of back pain distress and dysfunction is based upon the assumption that psychological factors are either secondary to the experience of low back pain or are only operative from its inception. However, a recent study by Polatin et al (1993) demonstrates that chronic low back pain patients are more likely to have experienced psychiatric disturbance as defined by DSM III before onset than the non-back population. This finding adds a further possible dimension to an already multi-faceted phenomenon and needs to be considered in the evaluation and management of low back pain.

A possible explanation which helps to integrate the findings of Polatin with those of this and other studies suggests that there may be at least two 'types' of chronic low back pain patient. The first (and more common) 'type' is characterised by fear avoidance which is often a consequence of misinformation concerning pathology and prognosis (Rose, 1993). These individuals appear to benefit from therapeutic intervention which focuses on reducing fear and consequent avoidance via

cognitive and behavioural therapy (Reilly, 1993). The second 'type' often disclose other pathologies, notably 'post-viral fatigue syndrome', recount a series of stressful life events, appear to be disadvantaged in terms of social and family circumstances and on occasion suggest a history of childhood abuse. This view may suggest that the fear-avoidance and rehabilitation drive concepts are applicable to the majority of chronic low back pain sufferers and may form the theoretical focus of therapy, yet there remain a minority of individuals whose needs should be met in the context of highly tailored and focussed psychotherapy.

The results of the predictive analyses of physiological variables in this study, which demonstrate that 'physical' variables can predict outcome, appear to contradict the view that psychological variables, particularly those which represent fear avoidance, determine the outcome of acute low back pain. This is supported by recent pathophysiological studies which have identified neurophysiological mechanisms which may be responsible for the experience of chronic low back pain (Fields, 1988). If this is so, psychological variables may either serve to mediate the effects of aberrant neurophysiological mechanisms rather than determine outcome of the acute pain state or merely be a secondary response to what is fundamentally a physiological problem. If this is so, the failure to prevent chronic back pain reflects medicine's limited understanding of neurophysiology rather than its

failure to appreciate the complex relationship between physical, psychological and sociological factors.

However, the results of this study demonstrate that the addition of psychological variables to physical variables in predictive analyses enhances their predictive power. In addition, principal component analysis reported in Chapter 7 demonstrated that variables such as straight leg raise correlated with psychological variables rather than with objective physiological signs such as neurological deficit. In addition, Pope et al (1980) showed that restricted spinal mobility, reduced flexion-extension torque ratio and reduced straight leg raising due to low back pain were all associated with lower pain threshold and tolerance rather than any objective pathological sign.

Interpreted conservatively, these findings suggest that back pain is a complex phenomenon which involves an interaction between physical and psychological variables. In the view of the author, physical measurements of patients with back pain measure the degree of movement which is acceptable to them within the context of their fear of pain rather than representing the mobility of anatomical structures. Within this theoretical framework, the success of the 'physical model' at predicting outcome tends to support the fear avoidance concept.

The results of this study support the notion that members of certain social and occupational groups are affected more by an episode of low back pain than others. However, in view of the methodological problems (discussed below) which may have affected the results it is difficult to draw firm conclusions regarding this issue.

In terms of the natural history of an acute attack of low back pain, the results of this study suggest that the majority of sufferers recover in large measure by two months after onset. However, many of these go on to experience further episodes and most of those who recover still experience symptoms. In terms of pain and disability, subjects who become chronic low back pain patients are identifiably different from those who will recover, by two months after onset. This has implications for those concerned with the primary care of back pain and also for researchers interested in the design of longitudinal studies of acute back pain patients. If a patient with acute low back pain has not improved or recovered by two months in terms of pain complaint and disability the clinician needs to consider appropriate referral. Chronic pain has traditionally been defined as that which has lasted for six or more months. The findings of this study suggest that two months is a more appropriate cut off. This enables researchers concerned with the prediction of chronicity to either shorten the overall time needed to collect follow-up data or to increase the number of subjects who can be followed up.

The description of a sample of acute low back pain patients presenting to their General Practitioner was comprehensive. The data suggests that the majority of individuals who present with this condition are free of psychological pathology and physiological signs. The main feature of the analysis of sub group differences concerned the differences between males and females, especially in terms of the effect of the present attack, affect and memory for pain. Possible reasons for these differences are presented in Chapter 5 and below.

9.3 METHODOLOGICAL ISSUES

In the view of the author, the primary flaw in the study design concerns the stage at which the physical examination of subjects and the collection of some psychological data occurred. Fear Avoidance Model, historical and demographic data were collected within two weeks of onset of low back pain. However, the physical examination data, pain drawing, Oswestry Disability Index and Zung Depression Inventory data were not collected until the two months follow-up stage. This prevented the direct comparison of physical data with acute psychological data in terms of prediction of two months and twelve months outcome, the description of the physical characteristics of acute low back pain patients attending general practice and a description of the natural history of acute low back pain in terms of physical data. In view of the apparently complex nature of low back pain experience and the limited nature of

the literature concerning acute low back pain, the collection of physiological data at the acute stage may have made a significant contribution to the debate concerning outcome and the results of the study.

The results of the study may also have been affected by the drop out rate at each stage. Although analyses of differences between attenders and non-attenders revealed demographic differences only, a reduction in the numbers of subjects who failed to attend at follow-up would have increased the power of the statistical tests used throughout the study. In view of the results of the study of the natural history of acute low back pain presented in Chapter 6, it may be appropriate for future longitudinal studies to follow subjects up earlier than twelve months after onset. One effect of this may be a reduction in the attrition rate of subjects included in the study.

The outcome measures used in the study were chosen to represent several aspects of low back pain experience. It was originally intended that other measures of outcome would be used at twelve months follow-up in order to further represent the experience of low back pain. However, the decision to collect limited twelve months outcome data by post as a result of the drop out rate prevented this. The decision to combine severity of present pain and Roland and Morris disability was based upon the close correlation of these variables. This decision was validated by the results of principal components analysis of

the measures and the results of multiple regression analyses in which combined pain and disability was the dependent variable. The choice of appropriate outcome measures in a study of physical (or psychological) pathology depends, in large measure, on the framework from which the researcher views the issue. In this study, for example, pain, disability and return to work were chosen because they represented apparently different aspects of pain experience and for pragmatic reasons. History of pain over twelve months was chosen because of the way in which chronic pain has been traditionally defined (pain which has lasted for more than six months). Future predictive studies may benefit from the use of a more comprehensive range of outcome measures which represent the different aspects of low back pain experience. These may include scales which measure the affective, cognitive, behavioural and functional dimensions of back pain experience. In addition, further research into the relative importance of different measures of outcome from the view of sufferers may be appropriate.

9.4 A REFORMULATION OF THE FEAR AVOIDANCE MODEL OF EXAGGERATED PAIN PERCEPTION

The Fear Avoidance Model can be reformulated within the context of the discussion concerning the close relationship between the concepts of fear of pain and anxiety in general is

presented in Chapter 1. Speilberger (1972) states that "there is nothing to be gained in the conceptual distinction between anxiety and fear unless the pattern of response in fear reactions differs from the response pattern in anxiety reaction". Lang (1968) identifies three sources of data from which anxiety (and fear) may be inferred: physiological, behavioural and verbal indices. McCracken et al (1992) stated that fear is best construed as a set of loosely coupled components, the three most important of which are avoidance behaviour, physiological reactivity and cognitive reports of subjective fear. The Fear Avoidance Model represents two of these three components, the behavioural and physiologically reactive components in terms of two of the four component variables of the 'psycho-social context' (see Chapter 1). These variables are MSPQ (physiological reactivity) and 'coping strategies' (avoidance behaviour). It can be argued that the failure of 'stressful life events' and 'general pain history' to make a significant contribution to the prediction of outcome was inevitable as these variables do not fall within the framework of anxiety/fear theory.

This partially complete model of fear of pain has been shown to account for a significant proportion of outcome variance, albeit in large measure as a result of the contribution made by the MSPQ. However, in order to represent a conceptually complete model of fear-avoidance the cognitive component of fear/anxiety theory needs to be included. This view is

supported by the growing interest in, and recognition of the importance of, cognitive variables in the area of low back pain.

Hugdahl (1981) has stated that anxiety (fear) is a complex emotional reaction which cannot be characterized as a unitary phenomenon and the role of patients beliefs, cognitions and appraisals about pain need to be examined. Clinical assessment of pain and disability ultimately depends on the patients own subjective report. Such an appraisal is considerably influenced by patients attitudes and beliefs as well as psychological distress and illness behaviour (Main et al 1992, Jensen et al 1991, Melzack 1965). Such ideas are supported by research which has shown how rehabilitation and adaptation to chronic pain are significantly associated with an individuals locus of control (Rudy et al, 1988), perceived ability to control pain (Jensen et al, 1990), attributional style (Cheatle et al 1990) and self efficacy beliefs (Nicholas and Wilson, 1989). Cognitive errors defined as a negatively distorted belief about oneself or one's situation have also been found to predict long term adjustment to low back disability as well as mediate disease severity and rehabilitation and contribute to the prediction of adjustment to disability (Jensen et al, 1991).

An increasing focus on cognitive dimensions of pain experience in both the assessment and treatment of low back pain illustrates recognition of a need to assess and identify

dysfunctional cognitions of pain and disability. Recent research has stressed the role of cognitive variables as instrumental in shaping the response to acute low back pain (Rose et al, 1993). In the clinical experience of the author, there are recurrent themes in terms of dysfunctional cognitions of individuals who have become chronic back pain patients. Cognitive errors include the belief that the spine is degenerating or crumbling, that the condition is deteriorating and that movement or exercise will worsen the pathology and consequent pain experience. These observations support the notion that cognitions represent an important component of fear in the context of back pain. In addition, patients often demonstrate an external locus of control, endorsing a disease model of pain experience and a belief that their own responsibility for alleviation of suffering is minimal. Many patients catastrophise their pain experience in terms of their future deterioration and often believe that they are destined to become increasingly dependent on artificial aids such as wheelchairs, their family and the State.

Evidence suggests that patients' perception of physical activity and its relation to pain and also their perception of their physical capabilities are often quite erroneous. Patients with low back pain do generally show lower physical performance levels than normal asymptomatic subjects but their perception of their physical capacity is reduced more than actual performance (Linton, 1985, Schmidt, 1985). Council et al (1988)

found that chronic low back pain patients' expectations of the pain associated with certain physical activities correlated 0.40-0.74 with their subsequent performance. They concluded that actual performance was best predicted by self-efficacy ratings, which in turn appeared to be determined by pain response expectancies.

The Pain and Impairment Relationship Scale (PAIRS) Beliefs Questionnaire developed by Riley et al (1988) specifically attempted to measure patients' beliefs about the relationship between pain and functional impairment. They demonstrated a strong relationship between PAIRS beliefs and functional restriction, even after allowing for severity of pain. More recently, Waddell et al (1993) developed the Fear Avoidance Beliefs Questionnaire (FABQ) focussing upon patients' perceptions of physical activity and work in the context of their chronic back pain (eg 'physical activity might harm my back' or 'I cannot do my normal work until my pain is treated'). Regression analysis demonstrated that, after controlling for pain severity, fear avoidance beliefs about work accounted for 23% of the variance in disability and 26% of the variance in work loss. There was little direct relationship between fear avoidance beliefs and the mechanical characteristics of low back pain. They were not related to the pathological severity but rather to increasing uncertainty of diagnosis. This offers further support for the view of the author that physical measurements based upon symptom report

represent the extent of avoidance behaviour rather than biological dysfunction.

Therefore, the reformulated Fear Avoidance Model should be underpinned by a psycho-social context of injury which represents the physiological, behavioural and cognitive components of fear. In order that the reformulated Model can be of use in terms of future research and the management of back pain, these components should be represented by measures which have been shown to be valid and reliable.

The author suggests that the cognitive component of the psychosocial context may be represented by three measures. These are '*memory for worst ever episode of low back pain*' (1-10), the PAIRS (Riley et al, 1988) and the FABQ (Waddell et al, 1993). The development of the psychometric properties of the last two measures has been described above. In terms of memory for previous back pain, a relationship between cognitions and outcome has been supported in this study by the statistically significant outcome variance explained by memory of the 'severity of the worst ever attack of low back pain'. In the author's view, this variable may represent the semantic component of pain memory which shapes cognitions about the future. A result of the cognitive associations with an episode of severe low back pain may be the overprediction of the likely severity of the present attack. This overprediction in turn may be responsible for fear and consequent avoidance behaviour

which leads to reduction in activity and concommitant illness behaviour. The end result of this process is chronic low back pain syndrome. In addition, previous work concerning the Fear Avoidance Model (Rose et al, 1992) suggests that memory for pain may be specific in terms of the relationship between the pathology leading to the pain remembered and the pathological basis for the present pain. These findings may be interpreted within the theoretical framework of Tulving and Donaldson (1972) who conceptualise semantic memory as registering the cognitive referents of input signals, not the properties of the input itself. However, Tulving and Donaldson implicitly suggest that semantic memory is a consequence of chronic pain. The author proposes that the findings of this and other studies (Rose et al, 1992) suggest that the cognitive referents are not secondary to repeated pain experience (chronicity) but rather are causal factors of chronic pain. In conclusion, the inclusion of memory for previous back pain in the reformulated model and the exclusion of memory for general pain is supported by the findings of this study and by theoretical considerations.

The central element of the Fear Avoidance Model, the behavioural component of the psychosocial context, was represented in this study by a crude and unvalidated measure of 'coping strategies' developed by Lethem et al (1983). This is evidenced by poor reliability of the measure reported in Chapter 3. In the view of the author this accounts for the

failure of this variable to make a significant contribution to the prediction of outcome reported in Chapter Eight rather than mistaken theoretical reasoning. Several valid and reliable measures of of coping strategies have been developed. One such measure, the Coping Strategies Questionnaire (CSQ) developed by Rosenstiel and Keefe (1983) contains 44 items and yields scores on cognitive coping strategies, behavioural coping strategies and overall effectiveness of coping strategies.

The 'physiological reaction' component of the psychosocial context of the reformulated model should be represented by the MSPQ. This variable has been shown by the results of this and other studies (Main, 1983, Greenhough, 1992) to be a valid and reliable measure and have predictive utility in terms of chronic low back pain. In addition, it contains items which reflect the autonomic nervous system's reactions to 'stress' anxiety or fear. The nature of the MSPQ has been discussed in Chapters 3, 7 and 8.

9.5 IMPLICATIONS FOR MANAGEMENT

The results of this study reinforce the notion that fear-avoidance is an important mediator in the outcome of acute low back pain. In addition, the strength of fear avoidance beliefs and their powerful relationship to disability demonstrated by others (eg Waddell, 1993) has implications for medical management. It may be postulated that current medical advice

and treatment for low back pain, and in particular unjustified restriction of activity, the prescription of rest and sick certification by rote (Waddell, 1987b) would appear likely to cause or re-inforce fear avoidance beliefs and hence iatrogenic disability. Medical management of this kind may even be responsible for the present epidemic of low back disability (Allen and Waddell, 1989). Clinicians should be aware of the possibly central role of fear-avoidance in the development of chronic disability. To prevent chronicity, inappropriate fear avoidance beliefs need to be recognised at the acute stage, tackled directly and changed early before they become fixed.

9.6 FUTURE RESEARCH AND APPLICATIONS

9.6.1 Outcome measures

Assessment of the severity of low back pain is fundamental to decisions about treatment and monitoring progress. The issue of outcome of an episode of low back pain, the outcome of intervention and the outcome of disease processes generally remains a source of future research. The recognition that outcome is defined in the context of the framework of the observer suggests the need for the identification of outcomes which are relevant and important to the patient. This may lead to the development of global measures of outcome which will increase the relevance and meaning of studies designed to

develop theoretical explanations for failure to recover. There remains the need for an objective assessment of physical impairment to compare with the patient's subjective report (of pain severity, symptoms and disability). This is particularly important given that there appears to be no particular relationship between severity of pathology evidenced by radiographs and pain complaint (see Chapter 1) and that for as many as 85% of back pain episodes the cause of pain is unclear (White and Gordon, 1985). Future research must enhance the specificity of clinical assessment in low back pain. In addition, assessment must include a comprehensive evaluation of the patient's pain, cognitions about the pain, the affective dimension of pain and the patterns of illness behaviour and disability that result.

9.6.2 The reformulated Fear Avoidance Model

A further longitudinal study of acute low back pain patients should be undertaken to test the validity of the Reformulated Fear Avoidance Model of Exaggerated Pain Perception. In addition, a physical examination of the subjects should be performed at the acute stage in order to determine the role, if any, played by physical variables in the development of chronicity. In view of the results of this study, the follow-up period of future longitudinal studies could be reduced in order to either increase the number of subjects studied or decrease the funding necessary to conduct the study. Such a

study may lead to the development of an easily administered predictive algorithm which could be used in a general practice setting in order to identify those who may be at risk of chronicity.

The issue of chronic low back pain 'type' discussed above represents a rich potential source of future research. Analysis of data which represent previous experience of psychopathology in addition to the reformulated model may identify 'clusters' of individuals suffering from chronic back pain with different causal mechanisms. Furthermore, the issues surrounding the definition of 'chronicity' are worthy of further consideration. In the view of the author, definitions of chronicity which fail to include the psychological and socio-economic (for the individual and the Exchequer) consequences of long term pain experience are inadequate. Research designed to identify variables which closely represent the 'suffering' component of pain experience should be developed. In addition, the implications for the National Health Service and State should be considered when defining chronic low back pain. For example, is the individual who actively copes with daily pain yet continues to work and avoid his or her GP, more or less 'chronic' than the individual who experiences bi-annual pain which is disabling and leads to time off work and inclusion on orthopaedic waiting lists of ever increasing length?

There has been an increasing interest in computer-based interview systems for patients with low back pain (Burton and Tillotson, 1991) with the aim of syndrome identification. If these programmes incorporated predictive models based upon the variables shown to be predictive of outcome in this study, they could conceivably become an aid in the reduction of unnecessary treatment. In addition, early intervention, with the aim of reducing fear of pain and based upon the multi-modal low back pain management programme approach, could be targeted at individuals who were identified as being at risk of developing chronic low back pain in order to prevent the onset of the syndrome.

9.6.3 Gender differences in pain experience

The differences between males and females in terms of memory for pain, affect, pain severity and disability demonstrated by the results of this study remain unexplained. The findings of recent research suggest an important role for hormonal variations in the neuronal mechanisms of analgesia (Mogil et al, 1993) and socialization practices which create gender appropriate pain behaviours (Ruda, 1993). Further exploration of gender differences may result in a contribution to the debate concerning the development and maintenance of chronic back pain and of pain in general.

9.6.4 Therapy and management

The implications for future research concerning therapy resulting from this study are considerable. If, as the author believes, fear is a central component in the genesis of chronic low back pain, treatment strategies need to include ways of alleviating this fear. Research may be directed at determining the relative effectiveness of cognitive or behavioural therapy in fear reduction, whether patients are more effectively treated as part of a group or individually and research designs may be constructed in order to determine which aspects of therapy reduce fear or, conversely increase it. At the acute stage, studies may be designed to compare the relative effectiveness, in terms of preventing chronicity, of 'conventional' management and early cognitive-behavioural therapy. This may lead to further research and debate concerning current issues in health care such as 'skills mix', professional boundaries and areas of competence in terms of early management of acute sufferers.

9.6.5 Towards a better understanding of other conditions

The concept of fear-avoidance may be equally applicable to other physical conditions. The results of a recent study by Rose et al (1992) suggests that the construct may be valid in terms of explaining post herpetic neuralgia and reflex sympathetic dystrophy. However, as this study was cross-

sectional a longitudinal study of acute shingles and fracture patients needs to be conducted in order to test the fear avoidance hypothesis within the context of these pathologies. The fear avoidance construct may also be useful in explaining conditions such as 'post viral distress syndrome' for which, in common with much of the disability associated with benign chronic back pain, no physiological basis has been identified.

9.7 CONCLUSIONS OF THE THESIS

To the author's knowledge, this study is the largest of its kind conducted to date. In addition, descriptive analyses of the sample suggest that the results are, in large measure, generalizable to the population of patients who present to their general practitioner with acute low back pain.

The results of this study support the utility of the Fear Avoidance Model of Exaggerated Pain Perception developed by Lethem et al. (1983) in terms of the prediction of outcome of an acute episode of low back pain.

Low back pain experience has been shown to be multi-faceted and the outcome of an acute attack of low back pain is determined by a range of variables representing different explanatory 'models'. However, interpretation of the results of predictive analyses using these other 'models' lends further support for

the fear avoidance concept. These findings suggest that clinical management of acute low back pain patients should be concerned with reducing fear of pain in order to prevent chronicity. In particular, the view that rest is the appropriate management for back pain is challenged.

The importance of cognitive factors in determining fear of pain has been recognised and described. This, along with an analysis of the utility of other variables used in the study, has led to a reformulation of the Fear Avoidance Model of Exaggerated Pain Perception. It is predicted that this new Model will enable individuals at risk of chronicity due to excessive fear of pain to be identified at an early stage of the natural history of their pain. In addition, the reformulated model has already provided the author with a theoretical focus for his clinical work with acute and chronic low back pain patients.

It is suggested that the reformulated model is relevant to the study and clinical management of other physical conditions for which the rehabilitation process can be hindered by fear.

The longitudinal study demonstrated that the large majority of acute low back pain patients who present to their general practitioner have neither physical signs nor significant psychopathology.

This study throws clear light on the natural history of acute episodes of low back pain. It demonstrates that the majority of individuals who present with an acute attack of low back pain will either improve significantly or recover within a two months period, while those who do not will become chronic sufferers. Therefore the first two months appear to be a critical period in the natural history of low back pain.

At the acute stage of low back pain, analysis of sub-group differences demonstrates a difference in response between males and females. Females have higher scores on variables which represent their pain experience in terms of the perception of the severity of pain and consequent disability and emotional reaction.

The results of this study support the notion that fear of pain and consequent avoidance behaviour are fundamentally important mediators of outcome of acute low back pain. In the view of the author, much of the suffering associated with chronic low back pain is a result of failure by clinicians to recognise the importance of reducing fear at the acute stage of the condition. The view that chronic low back pain is, in large measure, an iatrogenic phenomenon (Waddell, 1993) suggests the need for a therapeutic and preventative strategy which recognises, and is responsive to, the complexity of low back pain experience.

REFERENCES

Absi MA and Rokke PD. (1991) Can anxiety help us tolerate pain? Pain 46: 43-51

Allan DB and Waddell G. (1989) An historical perspective on low back pain and disability. Acta Orthopaedica Scandanavica Supplementum, 234: 60.

Ahern DK, Hannon DJ, Goreczny AK, Follick MJ, Parziale JR. (1990) Correlaton of chronic low back pain, behaviour and muscle function examination of flexion-relaxation response. Spine 15: 2. 92-95

Andersson GBJ. (1981) Epidemiological aspects of low back pain in industry. Spine 6: 53

Arntz A, van Eck M, De Jong P (1991) Avoidance of pain of unpredictable intensity. Beh. Res. Ther. 29: 197-201

Atkinson JH, Slater MA, Grant IG, Patterson TL, Garfin SR. (1988) Depressed mood in chronic low back pain : relationship with stressful life events. Pain 35: 47-55

Atkinson JH, Slater MA, Patterson TL, Grant I, Garfin SR. (1991) Prevalence, onset and risk of psychiatric disorders in men with chronic low back pain: a controlled study. Pain 45: 111-121

Auchincloss S. (1980) The painful back. Practical aspects of management. MEDICINE Publishing foundation. Oxford. In: Wells N. (1985) Back Pain. Office of Health Ecconomics.

Barnes D, Smith D, Gatchel RJ, Mayer TG. (1989) Psychosocioeconomic predictors of treatment success/failure in chronic low back pain. Spine 14: 4. 427-430

Barrett J and Goulding DN (1984) The practical treatment of backache and sciatica. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone.

Barron WM (1980) Failed appointments. Who misses them, why they are missed and what can be done. Primary Care 7: 4. 563-574

Basmajian JV (1978) "Muscles alive; Their function revealed by electromyography". Williams and Wilkins, Baltimore, MD

Battie MC, Bigos SJ, Fisher LD, Spengler DM, Hansson TH, Nachemson AL, Wortley MD. (1990) Anthropometric and clinical measures as predictors of back pain complaints in industry: A prospective trial J. Spinal Disorders 3: 3. 195-204

Beals RK, Hickman NW. (1972) Industrial injuries of the back and extremities. J Bone and Joint Surgery 54A: 8. 1593-1611

Beardwell A. (1969) Familial ankylosing vertebral hyperostosis and tylosis. Ann. Rheum. Dis. 28: 518. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone.

Beecher HK. (1956) Relationship of significance of wounds to pain experience. J.A.M.A. 161: 17. 1609-1613 Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Penguin, London

Beecher HK. (1959) Measurement of subjective reponses. Oxford University Press. New York. Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Penguin, London.

Bendix T, Sorenson SS, Klausen K. (1984) Lumbar curve, trunk muscles and line of gravity with different heel heights. Spine 9: 223

Benson H, Epstein MD. (1975) The placebo effect: a neglected asset in the care of patients. J Amer Med Assn 232: 1225-7. Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Penguin, London.

Bergner M, Bobbitt RA, Carter WB, Gilson BS. (1981) The Sickness Impact Profile: Development and final revision of a health status measure. Med. Care 19: 787-805

Bergenudd H, Nilsson B. (1988) Back pain in middle age; Occupational and social factors: An epidemiological survey. Spine 13: 1. 58-60

Berwick DM, Budman S, felstein M (1989) No clinical effect of back schools in an HMO. A randomised trial. Spine 14: 3. 338-344

Bickler CB (1985) Defaulted appointments in general practice. J.Roy. Col. Gen. Pract. January 1985

Biering-Sorenson F.(1984) Physical measurements as risk indicators for low back trouble over a one year period. Spine 9: 106.

Biering-Sorenson F, Thomsen C. (1986) Medical ,social and occupational history as risk indicators for low-back trouble in a general population. Spine 11: 7. 720-725

Billings A, Moos R. (1981) The role of coping responses and social resources in attenuating the stress of life events. J. of Behav. Med. 4: 2. 139-157

Bigos SJ, Spengler DM, Martin NA (1986) Back injury in industry: A retrospective study. Employee related factors. Spine 11: 252-256

Bland MJ, Altman. (1986) Statistical methods for assessing agreement between two methods of clinical measurement. Lancet, Feb. 8: 307-310.

Block AR. (1980) Behavioural treatment of chronic pain: the spouse as a descriminative cue for pain behaviour. Pain 9: 2-12

Bogduk N. (1987) Chapter 3. Physical Therapy of the Low Back. Churchill Livingstone.

Bogduk N, Jull G. (1985) The theoretical pathology of acute locked back: a basis for manipulative therapy. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Bokan JA, Ries RK, Katon WJ. (1981) Tertiary gain and chronic pain. Pain 10: 331-335

Bond MR. (1971) Cited by Sternbach RA (Ed.) (1978) The psychology of pain. Raven Press

Bond MR. (1978) Psychological and psychiatric aspects of pain Anaesthesia 33: 355-361

Bortz WM (1984) The disuse syndrome. West.J. Med. 691-694

Bowsher D. (1991) Neurogenic pain syndromes and their management. British Medical Bulletin. 47: 3. 644-666

Brodie DA and Slade PDS (1988) The relationship between body image and body fat in adult women. Psych. Med. 18: 623-621.

Brown GK, Nicassio PM. (1987) Development of a questionnaire for the assessment of active and passive coping strategies in chronic pain patients. Pain 31: 53-64.

Burton AK. (1986) Regional lumbar sagittal mobility; measurement by flexicurves. Clin. Biomech 1: 20-26.

Burton AK, Tillotson KM. (1989) Is recurrent low back trouble associated with increased lumbar sagittal mobility? J. Biomed. Eng. 11

Burton AK and Tillotson KM. (1991) Prediction of the clinical course of low-back trouble using multivariable models. Spine 16:1. 7-14

Bush FM (1993) Analysis of gender effects on pain perception and symptom presentation in temporomandibular pain Pain 53. 73-80

Cairns D, Pasino J. (1977) Comparison of verbal reinforcement and bio-feedback in the operant treatment of disability due to low back pain. Behavior Therapy 8: 621-630

Caldwell AB, Chase C. (1977) Diagnosis and treatment of personality factors in chronic low back pain. Clin. Orth. and Related Res. 129: 141-149

Cats-Baril WL and Frymower JW (1991) Identifying patients at risk of becoming disabled because of low back pain. Spine 16: 6. 605-607

Chapman SL. (1986) A review and clinical perspective on the use of EMG and thermal biofeedback for chronic headache. Pain 27: 1-43

Cheatle MD, Brady JP, Ruland T (1990) Chronic low back pain, depression and attributional style. Clin.J.Pain. 6: 114-117

Clark WC, Clark SB (1980) Pain responses in Nepalese porters. Science 209: 410-412. Cited by Melzack R, Wall P. (1988) "The Challenge of Pain". Penguin, London.

Cohen MJ, Collins GA, Bonebakker AD. (1983) Group outpatient physical and behavioural therapy for chronic low back pain. J. of Clinical Psychology 39: 3. 326-333

Cohen J, (1960) Educational and Psychological Measurement 20: 37-46

Cook RD, (1977) Detection of influential observations in linear regression Technometrics 19: 15-18

Council JR, Ahern DK, Follick MJ, Kline CL. (1988) Expectancies and functional impairment in chronic low back pain. Pain 33: 323-331

Crauford DIO, Creed F, Jayson MIV. (1990) Life events and psychological disturbances in patients with low back pain. Spine 15: 6. 490-494

Cyron BM, Hutton WC (1980) Articular tropism and stability of the lumbar spine. Spine 5: 168 Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Dales JL, McDonald EB, Porter RW. (1986) Back pain; the risk factors and its prediction in work people. Clinical Biomechanics 1: 216-221

Daniels L and Worthingham C (1986) Muscle testing - techniques of manual examination (5th edn) WB Saunders, Philadelphia

Davis PR and Troup JDG. (1966) Effects on the trunk of erecting pit props at different heights. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Descartes R. (1664) L'Homme Paris. E. Angot Cited by Melzack R and Wall P (1988) The Challenge of Pain. Penguin, London

Dewey ME, (1988) The prediction of outcome. Unpublished.

Dewey ME, Dickinson CE, Foreman TK, Troup JDG. (1989) Back pain, ventilatory function, chest symptoms and smoking. Journal of Spinal Disorders 2: 4. 241-248

Deyo RA, Diehl AK, Rosenthal M. (1986) How many days of bed rest for acute back pain. New Eng. J. Med. 315: 17. 1064-1070

Deyo RA, Diehl AK. (1988) Psychosocial predictors of disability in patients with low back pain. J. Rheum. 15: 10.

Deyo RA, Walsh NE, Schoenfeld LS, Ramamurthy S. (1989) Studies of the Modified Somatic Perception Questionnaire (MSPQ) in patients with back pain.Psychometric and predictive properties. Spine, 14: 5.

Dillain JB. (1966) Acute back syndrome - A study from general practice BMJ. 2: 9th July, 82-84

Dixon ASJ (1980) Diagnosis of low back pain: sorting the complainers. In: Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Doxey NCS., Mitson GL., Dzioba RB., Lacroix JM., (1988) Predictors of outcome in back surgery candidates. J. Clin.Psych. 44: 4.

Dunnell K, Cartwright A (1972) Medicine takers, prescribers and hoaders, Routledge and Keegan Paul, London. Cited by Humphrey M 'Back Pain', 1988.

Dwyer AP, (1987) Back ache and its prevention. Clin. Orth. Rel. Res. 222: 35-43

Eastrand N. (1987) Medical, psychological and social factors associated with back abnormalities and self reported back pain: a cross sectional study of male employees in a Swedish pulp and paper industry. British Journal of Industrial Medicine 44: 327-336

Editorial (anonymous) (1970) Stay young by good posture. New Scientist 82: 544. Cited by Grieve GP,(1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Egbert LD, Battit GE, Welsh CD, Bartlett MK (1964) Reduction of post-operative pain by encouragement and instruction of patients. New Eng, J. Med 270: 825-7. Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Penguin, London.

Eich E, Reeves JL, Jaeger B (1985) Memory for pain: relation between past and present pain intensity. Pain 23: 375-379

Engel GL, (1959) "Psychogenic" pain and the pain-prone patient. American Journal of Medicine. June 899-918 Cited by Sternbach RA (Ed.) (1978) The psychology of pain. Raven Press

Erskine A, Morley S and Pearce S (1990) Memory for pain: a review Pain 41: 255-265

Evans FJ. Expectancy, therapeutic instructions and the placebo response. Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Pub. Penguin.

Fairbank JC, Couper J, Davies JB, O'Brien JP. (1980) The Oswestry low back pain disability questionnaire. Physiotherapy 66: 8. 271-273

Farfan HF, (1973) Mechanical disorders of the low back. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Farfan HF, Huberdeau RM, Dubow HI. (1972) Lumbar intervertebral disc degeneration. J.Bone Jt. Surg. 54A: 492 Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Feine JS, Bushnell MC, Miron D and Duncan GH. (1991) Sex differences in the perception of noxious heat stimuli Pain 44: 255-262

Felton BJ, Revenson TA. (1984) Coping with chronic illness; A study of illness controlability and the influence of coping strategies on psychological adjustment. Jounal of Consulting and Clinical Psychlogy 52: 3. 343-353

Feuerstein M, Sult S, Houle M. (1985) Environmental stressors and chronic low back pain: life events, family and work environment. Pain, 22: 295-307.

Feuerstein M, Carter RL, Papciak AS. (1987) A prospective analysis of stress and fatigue in recurrent low back pain. Pain 31: 333-344

Fielding JM, (1980) Psychiatric aspects of pain. The Medical Journal of Australia Dec, 27: 705-707

Field HL (1988) Sources of variability in the sensation of pain. Pain 33: 195-200

Finlay-Jones RA (1981) Types of stressful life-event and the onset of anxiety and depression Psych.Med. 11: 803-815

Fitzpatrick RM, Bury M, Frank AO, Donnelly T. (1987) Problems of assessing outcome in a back pain clinic. Int. Disabil. Studies 9: 4.

Fordyce WE, Fowler RS, Leynmann JF, DeLateur BJ. (1968) Some implications of learning in problems of chronic pain. J. Chron. Dis. 21: 179-190

Fordyce WE, Fowler RS, Leynmann JF, DeLateur BJ, Sand PL, Trieschmann RB. (1973) Operant conditioning in the treatment of chronic pain. Arch. Phys. Med. and Rehabil. 54: 399-408

Fordyce WE (1976) Behavioural methods for chronic pain and illness. Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Penguin, London.

Fordyce WE. (1978) Learning processes in pain. In: "The psychology of pain." Sternbach RA (Ed) Raven Press. New York

Fordyce WE. (1981) Pain complaint-exercise peformance relationship in chronic pain. Pain 10: 311-321

Fordyce WE, (1988) Letter to the editor. Pain 33: 385-387

Fordyce WE, Shelton JL, Dundore D E. (1982) The modification of avoidance learning behaviours. J. Behav. Med. 5: 4. 405-414

Foreman TK, Baxter CE, Troup JDG. (1984) Ratings of acceptable load and maximal isometric lifting strengths: the effects of repetition. Ergonomics 27: 12. 1283-1288

Freeman W, Watts JW. (1948) Ann. Internal. Med. 28: 747-754

Freeman W, Watts JW (1950) Psychosurgery in the treatment of mental disorders and intractable pain. Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Penguin, London.

Freud S. (1952) Studien uber Hysterie. In: Gesammelte Werte, London Cited by Ghadiali EJ (1987) Dissertation, University of Liverpool

Fry J, Sandler G, Brooks D. (1986) Disease Data Book. MTP Press Limited.

Frymower JW, Pope MH, Clements JH, Wilder DG MacPherson B, Ashigaka T. (1983) Risk factors in low back pain. J Bone Jt Surg 65A: 213 Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Gallagher RM, Rauh V, Haugh LD. (1989) Determinants of return to work among low back pain patients. Pain 39: 55-67.

Gallon RL. (1989) Perception of disability in chronic back pain patients: a long term follow-up. Pain 37: 67-75

Gamsa A, Vikis-Friebergs V. (1991) Psychological events are both risk factors in, and consequences of, chronic pain Pain 44: 271-277

Gentry WD, Shows WD, Thomas M. (1974) Chronic low back pain: a psychological profile. Psychosomatics 15: 174-177

Gentry WD, Bernal GAA (1977) Chronic pain. In: "behavioural approaches to Medical Treatment". Eds Williams RB and Gentry WD, Ballinger, Cambridge, MA. 173-182

Ghadiali EJ (1987) The development, standardisation and validiatiorn of an instrument designed to measure coping with chronic pain. PhD dissertation, University of Liverpool, England.

Giles LGF,Taylor JR. (1981) Low back pain associated with leg length equality. Spine, 6: 5. 510.

Glass JB. (1975) The Practitioner. 222: 821-825. Cited by Wells N. (1985) Back Pain. Office of Health Ecconomics.

Glover JR (1971) Occupational health research and the problem of back pain Trans Soc Occup Med 21: 2 Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Grabel JA (1973) Electromyographic study of low back muscle tension in subjects with and without chronic back pain. Cited by Ghadiali EJ (1987) Dissertation, Liverpool University.

Greenough CG and Fraser RD. (1989) The effects of compensation on recovery from low back pain. Spine, 14: 9.

Greenough CG and Frazer RD. (1991) Comparison of eight psychometric instruments in unselected patients with back pain. Spine 16: 9. 1068-1073

Greenhough CG and Frazer RD. (1992) Assessment of outcome in patients with low back pain Spine 17:136-41

Grieve GP. (1987) Some psychological aspects of benign spinal pain. Physiotherapy 73: 9. 489-501

Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Guest GH and Drummond PD. (1992) Effect of compensation on emeotional state and disability in chronic back pain. Pain 48: 125-130

Guten G. (1981) Herniated lumbar disc associated with running. Am.J.Sports Med. 9: 155. Cited by Grieve GP, (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Hanvik LJ (1951) MMPI profiles in patients with low back pain. J. of Cons. Clin. Psychol. 15: 350-353

Harms-Ringdahl K, Carlsson AM, Ekholm J, Raustorp A, Svensson T, Torreson HG. (1986) Pain assessment with different intensity scales in response to loading of joint structures. Pain 17: 45-56

Hapidou EG and DeCatanzaro D. (1992) Responsiveness to laboratory pain in women as a function of age and childbirth experience. Pain 48: 177-181

Harkapaa K, Jarvikoski A, Mellin G, Hurri H and Luoma J. (1991) Health locus of control beliefs and psychological distress as predictors for treatment outcome in low back pain patients: results of a 3-month follow-up of a controlled intervention study. Pain 46: 35-41

Harris CM. (1984) Seasonal variations in depression and osteoarthritis. J.Coll.Gen.Prac. 34: 436. In: Grieve GP,(1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Harris RI. (1954) Structural Changes in lumbar intervertebral discs. J.Bone.Jt.Surg. 36B:304. In: Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Hay MC (1974) The incidence of low back pain in Busselton. In: Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Hazard RG, Fenwick JW, Kalisch SM, REdmond J, Reeves V, Reid S, Frymower JW. (1989) Functional restoration with behavioural support. A one year prospective study of patients with chronic low back pain. Spine 14: 2. 157-161

Heaton RK, Getto CJ, Lehman RAW, Fordyce WE, Brauer E, Groban SE. (1982) A standardized evaluation of psychosocial factors in chronic pain. Pain 12: 165-174

Hilgard ER, Hilgard J. (1986) Hypnosis in the relief of pain. Cited by Melzack R, Wall P (1988) "The Challenge of Pain". Penguin, London.

Hippocrates, Corpus Hippocrateum. Peri Arthron. Ch.47. (400 BC). Cited by Grieve GP (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Hokanson JE, DeGood DE (1971) Availability of avoidance behaviours in modulating vascular stress response. J. Personality. Soc. Psychol. 19: 60-68

Holmes TH, Rahe TH. (1967) The Social Readjustment Rating Scale. J. Psychosom. Res. **11**: 213-218

Horral J. (1969) The clinical appearance of low back disorders in the city of Gothenburg, Sweden. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Hosobuchi Y. (1980) The majority of umyelinated afferent axons in human ventral roots probably conduct pain. Pain 8: 167

Howe JF,Loeser JD,Calvin WH. (1977) Mechano-sensitivity of dorsal root ganglia and chronically injured axons; A physiological basis for the radicular pain of nerve root compression. Pain, 3: 25.

Howell DS, Woessner JF. (1978) A view of the pathogenesis of osteoarthritis. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Hoyt WH (1982) Electromyographic assessment of chronic low back pain syndrome. J. Amer. Osteopath. Ass. 80: 722-730 Cited by Ghadiali EJ (1987) Dissertation Liverpool University

Humphrey M (1989) Back Pain. Routledge, London.

Hunt CC, McIntyre AK. (1960) An analysis of fibre diameter and receptor characteristics of myelinated cutaneous afferent fibres in the cat. J. Physiol. (Lond.) 153: 99.

Ingham JG, Miller P. (1979) J. Epid. Comm. Hlth. Cited by: Wells N. (1985) Back Pain. Office of Health Ecconomics.

Jackson CP, Brown MD. (1983) Analysis if current approaches and a practical guide to prescription of exercise. Clin. Orth. Rel. Res. 179: October.

Janda V. Schmid HJA. (1980) Muscles as a pathogenic factor in back pain. In: Proc. 4th IFOMT Conf. Christchurch p.1.

Jenson J. (1988) Life events in neurological patients with headache and low back pain (in relation to diagnosis and persistance of pain). Pain, 32: 47-53.

Jenson MP, Turner JA and Romano JM. (1991) Self-efficacy and outcome expectancies: relationship to chronic pain coping strategies and adjustment. Pain 44: 263-269

Johnson JE. (1973) effects of accurate expectations about sensation on the sensory and distress components of pain J. Personality Soc. Psychol. 27: 261-275

Keefe FJ, Dolan E. (1986) Pain behaviour and pain coping strategies in low back pain and myofascial pain dysfunction syndrome patients. Pain, 24: 49-56.

Keefe FJ. (1982) Mini-series on behavioural analysis of chronic pain. Behaviour Therapy 13: 363-375

Keefe FJ, Crisson J, Urban BJ, Williams DA. (1990) Analysing chronic low back pain: the relative contribution of pain coping strategies. Pain 40: 293-301

Kelsey JL,Hardy RJ.(1975) Driving of motor vehicles as a risk factor for acute herniated lumbar intervertebral disc. Am.J.Epediom, 102: 63. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Kores RC, Murphy WD, Rosenthal TD, Elias DB, North WC. (1990) Prdicting treatment outcome of chronic pain treatment via a modified self-efficacy scale. Behav. Res Ther. 28: 2. 165-169

Krusen EM, Ford DE. (1958) Compensation factors in low back injuries. JAMA 166: 1128-1133

Lacroix JM, Powell J, LLoyd GF, Doxey NCS, Mitson GL, Aldman CF. (1990) Low back pain: Factors in predicting outcome. Pain 15: 6. 495-499

Lambert WE, Libman E, Poser EG (1960) Effect of increased salience of membership group on pain tolerance. J. Personality 28: 350-357. Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London

Lander J (1989) Gender effects in pain perception. Percept. Mot. Skills. 63. 3 1088-90

Lang PJ (1968) Fear reduction and fear behaviour: Problems in treating a construct. In: Shlien JM (ed) Research in psychotherapy III 90-103 APA, Washington DC

Lang PJ (1985) The cognitive psychophysiology of emotion: fear and anxiety. In: Tuma AH (ed) Anxiety and the anxiety disorders. Lawrence Erlbaum, NJ

Langer E, Janis IL, Wolfer JA (1975) Reduction of psychological stress in surgical patients. J. Exper. Soc. Psychol. 11: 155-156 Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London

Larocca H (1987) Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for clinicians. Report of the Quebec Task Force on spinal disorders. Pain 12: 7. (supplement).

Lawrence JS. (1977) Rheumatism in populations. Heineman, London, p.146. Cited by Grieve GP (1988) Common vertebral joint problems, 2nd Edition. Churchill Livingstone, Glasgow.

Leavitt F, Garron DC. (1979) The detection of psychological disturbance in patients with low back pain. J. Psychosom Res. 23: 591

Leavitt F, Garron DC, Bieliauskas LA. (1979) Stressing life events and the experience of low back pain. J. Psychosom. Res. 23: 49-55.

Leavitt F, Garron DC. (1979) Validity of a back pain classification scale among patients with low back pain not associated with demonstrable organic disease. J. Psychosom. Res. 23: 301-306

Leavitt F, Garron DC. (1979) Psychological disturbance and pain report differences in in both organic and non-organic low back patients. Pain 7: 187-195

Leavitt F, Garron DC. (1982) Patterns of psychological disturbances and pain report in patients with low back pain. J. Psychosom. Res. 26: 301-307

Lee PC. (1986) Principles and practice of muscle energy and functional techniques. Cited by Grieve GP (1988) Common vertebral joint problems. Churchill Livingstone, Glasgow.

Lee PWH, Chow SP, Lieh-Mak F, Chan KC, Wong S. (1989) Psychological factors influencing outcome in patients with low back pain. Spine 14: 8.

Lethem J, Slade PD, Troup JDG, Bentley G. (1983) Outline of a fear-avoidance model of exaggerated pain perception-1. Behav. Res. Ther. 21: 4. 401-408.

Linton SJ, Gotestam KG. (1984) A controlled study of the effects of applied relaxation and applied relaxation plus operant procedures in the regulation of chronic pain. British Journal of Clinical Psychology 23: 291-299

Linton SJ. (1974) Behavioural remediation of chronic pain: a status report. Pain 24: 125-141

Livingston WK (1943) "Pain Mechanisms" MacMillan, New York. Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London

LLoyd DCEF, Troup JDG. (1983) Recurrent back pain and its prediction J.Soc.Occup.Med. 33: 66-74.

Lund EL. Psychological factors in back pain: Self related back pain.

McKeon J (1984) Life events and personality traits in obsessive-compulsive neurosis. Br.J.Psych. 144: 185-189

MacGibbon B, Farfan HF. (1979) A radiological survey of various configurations of the lumbar spine. In: Grieve GP (1988) Common vertebral joint problems. Churchill Livingstone, Glasgow.

Magora A.(1975) Investigation of the relation between low back pain and occupation. Scand.J.Rehab.Med. 7: 146. Cited by Grieve GP (1988) Common vertebral joint problems. Churchill Livingstone, Glasgow.

Magora A, Schartz A.(1978) Relation between low back pain and X-ray findings. Scand.J.Rehab.Med. 10: 135. Cited by Grieve GP (1988) Common vertebral joint problems. Churchill Livingstone, Glasgow.

Magora A. (1973) Investigation in the relationship between low back pain and occupation. V. Psychological aspects. Scan. J. Rehab. Med. 5: 191

Main CJ. (1983) The Modified Somatic Perception Questionnaire (MSPQ) J. Psychosom. Res. 27: 6. 503-514.

Main CJ, Waddell G. (1984) The identification of psychological abnormality in patients with low back pain. (Manual).

Main CJ, Waddell G. (1984) The detection of psychological abnormality in chronic low back pain using four simple scales. Current Concepts in Pain. 2: 1.

Main CJ, Waddell G. (1987) Personality assessment in the management of low back pain. Clinical Rehabilitation 1: 139-142

Main CJ, Wood PLR, Hollis S, Spanswick CC, Waddell G (1992) The distress and risk assessment method. A simple patient classification to identify distress and evaluate the risk of poor outcome. Spine 17: 1

Mathews A and McLeod C (1986) Discrimination of threat cues without awareness in anxiety states. J.Ab.Psychol. 95: 131-138

Mayer TG, Gatchel RJ, Kishino N. (1985) Objective assessment of spine function following industrial injury. Spine, 10: 6.

McDuffie FC. (1981) Arthritis and nutrition EULAR Bull. 10: 3. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

MacGlashan TH, Evans FJ, Orne MT. (1969) The nature of hypnotic analgesia and placebo response to experimental pain Psychosom. Med. 31: 227-46 Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London

McKenzie RA.(1987) Physical therapy of the low back. Churchill Livingstone, Glasgow. Chapter 6.

McNeil TW, Sinkora G, Leavitt F. (1986) Psychological classification of low-back pain patients: a prognostic tool. Spine, 11: 9.

McQuade KJ, Turner JA, Buchner DM. (1988) Physical fitness and low back pain. Clin. Orth. Rel. Res 233: 198-204

Mechanic D. (1962) The concept of illness behaviour. J. Chron. Dis. 15: 189-194

Meichembaum DH, Turk DC. (1976) The cognitive-behavioural management of anxiety, depression and pain. In: "The Behavioural management of anxiety, depression and pain". Davidson PD (Ed). Brunner/Mazel, New York. 1-34

Mellin G. (1986) Chronic low back pain in men 54-63 years of age. Correlatons of physical measurements with the degree of trouble and progress after treatment. Spine, 11: 5.

Mellin G. (1990) Decreased joint and spinal mobility associated with low back pain. J. of Spinal Disorders. 3: 3. 238-243

Mendelson G. (1984) Compensation, pain complaints and psychological disturbance. Pain 20: 169-177

Merriam WF,Burwell RG. (1983) A study revealing a tall pelvis in subjects with low back pain. J.Bone Jt.Surg. 65B: 153. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Melzack R, Wall PD. (1965) Pain mechanisms: a new theory. Science, 150: 971.

Melzak R, Casey KL. (1968) Sensory, motivational and central control determinants of pain: a new conceptual model. In: "The Skin Senses". Kenshalo D (Ed). Thomas, Springfield, IL.

Melzack R, Perry C. (1980) Self regulation of pain: the use of alpha-feedback and hypnotic training for the control of chronic pain. Exper. Neurol. 46: 452-69. Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London

Melzack R, Scott TH (1957) The effects of early experience on the response to pain. J. Comp. Physiol. Psychol. 50: 155-61 Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London

Merskey H (1983) The psychological treatment of pain. Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London

Meyers JE, Lyon HP. (1979) Stress and distress. Proc. Am. Occup. Med. Assoc.

Miller H. (1961) Accident neurosis. Br. Med. J. i 919-925, 992-998

Milligan KA and Atkinson RE. (1991) The "two week syndrome" associated with injection treatment for chronic pain - fact or fiction? Pain 44: 165-166

Million R, Hall W, Nilson H, Baker RD, Jayson MIV. (1981) Assessment of the progress of the back pain patient. Spine, 7: 3

Mitchell KR, Wallis BJ. (1987) Patterns of illness behaviour and perceived coping in chronic low back and chronic neck pain patients. Psychiatric Medicine, 5: 2.

Mogil (1993) Sex differences in the antagonism of swim stress-induced analgesia: effects of gonadectomy and estrogen replacement. Pain 55.

Morley SJ. (1991) Memory and Pain. Talk given at the British Psychological Society. Unpublished

Morrell DC, Wale CJ. (1976) J. Roy. Coll. Gen. Practit. 26. 329-403

Muller J. (1987) Cited by Nolan MF, Chpt.1. Pain, Clinics in Physiotherapy. Echternach JL.(Ed). Churchill Livingstone, Glasgow.

Murphy KA., Cornish RD. (1984) Prediction of chronicity in acute low back pain. Arch. Phys. Med. Rehabil. 65: 334-337.

Murray RO, Duncan C.(1971) Athletic activity in adolescence as an aetiological factor in degenerative hip disease. J.Bone Jt.Surg. 53B: 406. Cited by Grieve GP (1988) Common vertebral joint problems, 2nd Edition. Churchill Livingstone, Glasgow.

Nachemson A. (1987) Lumbar intradiscal pressure. Cited by Grieve GP (1988) Common vertebral joint problems, 2nd Edition. Churchill Livingstone, Glasgow.

Nachemson A. (1983) Work for all. For those with low back pain as well. Clin. Orth. Rel. Research. 179: 77-85

Naliboff BD, Cohen MJ, Yellen AN. (1982) Does the MMPI differentiate chronic illness from chronic pain? Pain 13: 333-341

Naliboff BD, Cohen MJ, Swanson GA, Bonebakker AD, McArthur DL. (1985) Comprehensive assessment of chronic low back pain patients and controls: physical abilities, level of activity, psychological adjustment and pain perception. Pain 23: 121-134

Nehemkis AM, Carver DW, Evanski PM. (1979) The predictive utility of the orthopaedic examination in identifying the low back pain patient with the hysterical personality features. Clin. Orth. Rel. Res. 145.

Newton M, Waddell G (1991) Reliability and validity of clinical measurement of the lumbar spine in patients with chronic low back pain. Physiotherapy 77: 12 796-800

Nichols PJR. (1960) Short leg syndrome. Br.Med.J.Clin.Res. 1863. Cited by Grieve GP.(1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Nicholas MK and Wilson PH (1989) The pain self-efficacy questionnaire: Self efficacy in relation to chronic pain. Abstracts from the BPS annual conference The BPS, 1989, 1-15

Nolan FM. (1987) Clinics in physical therapy, 12, Pain. Chapter 1. Echternach JL (Ed). Churchill Livingstone, Glasgow

Ohlen G, Spangfort E, Tingvall C. (1989) Measurement of spinal sagittal configuraton and mobility with Debrunner's Kyphometer. Spine, 14: 6. 580-583.

Oostdam EMM, Duivenvoorden HJ. (1983) Predictability of the result of surgical intervention in patients with low back pain. J. Psychosom. Res. 27: 4. 273-281.

Oostdam EMW, Duivenvoorden HJ. (1987) Description of pain and the relationship with psychological factors in patients with low back pain. Pain 28: 357-364

Parker N. (1977) Accident litigants with neurotic sympoms. Med. J. Aust. 2: 318-322

Parsons T (1951) The social system. Free Press. New York. Cited by Allan and Waddell in 'An historical perspective on low back pain. Acta Orth. Scand 1989 60: 234

Payne B, Norfleet MA. (1986) Chronic pain and the family: A review. Pain 26: 1-22

Pearcy M, Portek I, Shepherd J. (1985) The effects of low back pain on lumbar spinal movements measured by three-dimensional X-Ray analysis. Spine, 10: 2.

Perl ER. (1968) Myelinated afferent fibres innervating the primate skin and their reponse to noxious stimuli. J. Physiol.

(Lond) 197: 593 Cited by Grieve GP (1988) Common vertebral joint problems. Churchill Livingstone, Glasgow.

Philips HC. (1974) A psychological analysis of tension headache. In Rachman S (Ed.) Contributions to Medical Psychology. Pergamon Press, Oxford

Philips (1988) Changing chronic pain experience. Pain 32: 165-172

Philips HC, Hunter M. (1981) Pain behaviour in headache sufferers. Behaviour Analysis and Modification. 4: 257-266

Philips HC, Grant L. (1991a) Acute back pain: A psychological analysis. Behav. Res. Ther. 29: 5. 429-434

Philips HC, Grant L. (1991b) The evolution of chronic back pain problems: a longitudinal study. Behav. Res. Ther. 29: 5. 435-441

Philips HC, Grant L, Berkowitz J. (1991c) The prevention of chronic pain and disability: a preliminary investigation. Behav. Res. Ther. 29: 5. 443-450

Pilowsky I. (1967) Dimensions of hypochondriasis, Brit. J. Psych. 113: 89-93

Pilowsky I, Spence ND. (1976) Is illness behaviour related to chronicity in patients with intractable pain? Pain 2: 176-173

Pilowsky I, Chapman CR, Bonica JJ. (1977) Pain, depression and illness behaviour in a pain clinic population. Pain 4: 183-192

Pilowsky I. "Psychodynamic aspects of pain experience" In: The psychology of pain (1978) Echternach J (Ed) Raven press, New York

Polatin PB, Gatchel RJ, Barnes D, Mayer H, Arens C, Mayer TG. (1989) A psychosociomedical prediction model of response to treatment by chronically disabled workers with low-back pain. Spine. 14: 9.

Pope MH, Wilder DG, Frymoyer JW. (1980) Vibration as an aetiological factor in low back pain. Cited by Grieve GP, (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Pope MH, Bevins T, Wilder DG, Frymoyer JW. (1985) The relationship between anthropometric, postural, muscular and mobility characteristics of males aged 18-55. Spine, 10: 644.

Porter RW, Hibbert C, Wellman P. (1989) Backache and the lumbar spinal canal. Spine, 5: 99.

Price DJ, McGrath PA, Rafii A, Buckingham B. (1983) The validation of a visual analogue scale as a ratio measure for chronic pain and experimental pain. Pain 17: 45-56

Rabkin G, Struening EL. (1976) Life events, stress and illness. Science, 194: 1013-1020.

Rachman S, Arntz A. (1991) The overprediction and underprediction of pain. Clin. Psych. Rev. 11: 339-355

Rachman SJ, Hodgson RI (1974) Synchrony and desynchrony in fear and avoidance. Beh. Res. Ther. 311-318

Radin EL, Orr RB, Kelmson JL, Paul IL, Rose RM. (1982) Effects of prolonged walking on concrete on the knees of sheep. J. of Biomech. 15: 487. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Ransford AO, Cairns D, Mooney V. (1976) The pain drawing as an aid to the psychological evaluation of patients with low back pain. Spine 1: 127

Reesor KA, Craig KD. (1988) Medically incongruent chronic back pain: physical limitations, suffering and ineffective coping. Pain 32: 35-45

Repko GR, Coper R. (1983) A study of the avereage workers' compensation case. J. Clin.Psych. 39: 287-295

Reilly JP (1993) The efficacy of a pain management programme for people with chronic low back pain. PhD Thesis. At Liverpool University

Reynolds PMG (1975) Measurement of spinal mobility: A comparison of three methods. Rheumatology and Rehabilitation 14: 180-185

Riley JF, Ahern DK, Follick MJ. (1988) Chronic pain and functional impairement: assessing beliefs about their relationship Arch. Phys. Med. Rehabil. 69(B): 579-82

Robin O, Vinhard H, Vernet E, Saumet JL. (1987) Influence of sex and anxiety on pain threshold and tolerance. Funct. Neurol. 2: 2. 173-9

Roland M, Morris R. (1982) A study of the natural history of low back pain. Spine, 8: 2. 141-144

Romano JM, Turner JA. (1985) Chronic pain and depression: Does the evidence support a relationship? Psychological Bulletin 97: 1. 18-34

Rose HJ. (1975) The lives of patients before presentation with pain in the neck or back. J. of the Royal Coll. Of Gen .Pract. 25: 771-772

Rose MJ (1991) The statistical analysis of the intra-observer repeatability of four clinical measures. Physiotherapy 77: 2. 89-91

Rose MJ, Klenerman L, Atchison L, Slade PD (1992) A comparison of three chronic pain conditions using the Fear Avoidance Model of Exaggerated Pain Perception Beh. Res. Ther. 30: 4 359-365

Rose MJ, Reilly JP, Slade PD, Pennie B (1993) Chronic low back pain: a consequence of misinformation? Employee councelling today 5: 1.

Rosenstiel AK, Keefe FJ. (1983) The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. Pain 17: 33-44

Royal College of General Practitioners (1979) Morbidity Statistics from General Practice 1971-72, second national study. Studies on medical and population subjects. No 36.HMSO.

Rubins JL, Friedman AD. (1948) Asymbolia for pain. Arch. Neurol. Psychiat. 60: 554-573 Cited by Melzack R and Wall P (1988) "The Challenge of Pain" Penguin, London.

Rudy TE, Verns RD, Turk DC (1988) Chronic pain and depression: towards a cognitive behavioural mediation model Pain 35: 129-140

Ryden O, Lindal A, Hansson B. (1985) Diffentiation of back pain patients using a pain questionnaire. Scand. J. Rehab. Med. 17: 155-161

Sandstrom J, Esbjornsson E. (1986) Return to work after rehabilitation. The significance of the patients own prediction. Scand. J. Rehab. Med. 18: 29-33

Scambler A, Scambler G, Craig D. (1981) J. Roy. Coll. Gen. Practit. 31: 746-50

Shea DD, Ohnmeiss DD, Stith WJ, Guyer RD, Rashbaum RF, Hochschuller SH and Regan JJ. (1991) Spine 16: 8. 560-561

Sibley JT. (1985) Weather and arthritis symptoms J. Rheum 12: 707 Cited by Grieve GP (1988) Common vertebral joint problems. Churchill Livingstone, Glasgow.

Sicuranza BJ,Richards J,Tisdall LH. (1970) The short leg syndrome in obstetrics and gynaecology. Am. J. Obstet. Gynaecol. 107: 217. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Siegerist HE. (1944) Civilization and disease. Cornell University Press. Chicago. Cited by Allan and Waddell in 'An historical perspective on low back pain. Acta Orth. Scand 1989 60: 234

Slade PD. (1984) Theories of Chronic Pain Phenomena. In: Current Issues in Clinical Psychology" Vol.2 Karas E (Ed). Plenum Publications.

Slade PD, Troup JDG, Lethem J, Bentley G. (1983) the Fear Avoidance Model of Exaggerated Pain Perception-II, preliminary studies of coping strategies for pain. Behav. Res. Ther. 21: 4. 409-416.

Slater MA, Hall HF, Hampton J, Garfin SR. (1991) Pain and impairment beliefs in chronic low back pain: validation of the pain and impairment relationship scale (PAIRS) Pain 44; 51-56

Smith TW, Follick MJ, Ahern DK. (1985) Life-events and psychological disturbance in chronic low back pain. Br. J. Clin. Psychol. 24: 207-208.

Smyth MJ, Wright V. (1958) Sciatica and the intervertebral disc: an experimental study. J.Bone Joint Surg. 40A: 1401. Cited by Grieve GP,(1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Spengler DM. (1983) Chronic low back pain. The team approach. Clin. Orth. Rel. Res. 179: 71-76

Spinehoven P, Kuile MM, Linssen ACG, Gazendam B. (1989) Pain coping strategies in a Dutch population of chronic low back patients. Pain 37: 77-83

Spratt KF, Lehman TR, Weinstein JN, Sayre HA. (1990) A new approach to the low-back physical examination. Behavioural assessment of mechanical signs. Spine 15: 2. 96-102

Sriwatanakul K. (1983) Studies with different types of visual analogue scales for measurment of pain. Clin. Pharmacol. Ther. 14: 2. 234-239

Sternbach R. (1974) Pain patients: traits and treatments. Academic Press, New York.

Sternbach R. (1975) Psychophysiology of pain International J. Psychiatry In Medicine 6(1/2): 63-73

Sternbach R, Timmermans G, (1975) "Personality Changes Associated with reduction of Pain". Pain 1: 171-179

Stoddard A (1983) Manual of osteopathic practice, 2nd edition. Hutchinson, London.

Svensson H, Andersson GBJ. (1989) The relationship of low-back pain, work history, work environment and stress. A retrospective cross sectional study of 38 to 64 year old women. Spine 14: 5. 517-522

Szasz T (1957) Pain and pleasure Tavistock Press, London. In: "The psychology of pain" Ecternach J (Ed.) 1978 Raven Press, New York.

Tan SY (1982) Cognitive and cognitive behavioural methods: a selective review. Pain 12: 201-218

Tillotson KM, Burton AK. (1989) Non-invasive measurement of lumbar sagittal mobility; an assessment of the flexicurve technique. Spine.

Trief PM. (1983) Chronic back pain: a tripartite model of outcome. Arch. Phys. Med. Rehabil. 64: 53-56.

Trief PM, Stein N. (1985) Pending litigation and rehabilitation outcome of chronic back pain. Arch. Phys. Med . Rehabil 66: 2. 95-9

Troup JDG. (1979) Straight leg raising (SLR) and the qualifying tests for increased root tension. Their predictive value after back and sciatic pain. Physiotherapy 65: 238

Troup JDG, Foreman TK, Baxter CE, Brown D. (1987) The perception of back pain and the role of psychophysical tests of lifting capacity. Spine, 12: 7.

Turk DC, Flor H. (1984) Etiological theories and treatments for chronic back pain. II. Psychological models and interventions. Pain 19: 209-233

Turk DCM Meuchenbaum D, Genest M. (1983) "Pain and Behavioural Medicine: A Cognitive Behavioural Perspective". Guildford Press, New York.

Turner JA, Robinson J, McReary CP. (1983) Cronic low back pain: Predicting response to treatment. Arch. Phys. Med. Rehabil. Vol. 64.

Turner JA, Clancy S. (1986) Strategies for coping with chronic low back pain: Relationship to pain and disability. Pain: 24. 355-364.

Twomey LT, Taylor JR (1987) Physical Therapy of the Low Back. Churchill Livingstone, London.

Uden A, Astrom M, Bergenudd H. (1988) Pain drawings in chronic back pain. Spine 13: 4. 389-392

Venn EK, Wakefield KA, Thompson PR. (1983) A comparitive study of leg length checks. Eur. J. Chiro. 31: 68. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

Versloot JM, Rozeman A, van Son AM and van Akkerveeken PF. (1992) The cost-effectiveness of a back school program in industry. A longitudinal controlled field study. Spine 17: 1. 22-27

Von Frey YM. (1894) In; Nolan MF. (1987) Pain, Clinics in Physiotherapy. Chapter 1: Echternach JL. (Ed) Churchill Livingstone, Glasgow.

Waddell G, McCulloch JA, Kummel ED, Venner RM. (1979) Nonorganic physical signs in low-back pain. Spine 5: 2.

Waddell G. (1987). Clinical assessment of lumbar inpairment. Clin. Orth. Rel. Res. 221.

Waddell G. (1987). A new clinical model for the treatment of low back pain. Spine, 12: 7.

Waddell G, Bircher M, Finlayson D, Main CJ. (1984) Symptoms and signs: physical disease or illness behaviour. BMJ, 289.

Waddell G, Main CJ. (1984). Assessment of severity in low-back disorders. Spine, 9: 2.

Waddell G, Morris EW, Di Paola MP, Bircher M, Finlayson D. (1986) A concept of illness tested as an improved basis for surgical decisions in low-back disorders. Spine, 11: 7.

Waddell G, Newton M, Henderson I, Somerville D, Main CJ. (1993) A fear-avoidance beliefs questionnaire (FABQ) and the role of fear-avoidnace beliefs in chronic low back pain and disability. Pain 52: 157-168

Wade JB, Dougherty LM, Hart RP and Cook DB. (1992) Patterns of normal personality structure among chronic pain patients. Pain 48: 37-43

Walsh NE, Dumitru D. (1988) The influence of compensation on recovery from low back pain. Occupational medicine: State of the art reviews 3: 1. 109-121

Weddell G (1955) Somesthesis and the chemical senses Ann. Rev. Psychol. 6. 119-36 Cited by Melzack and Wall (1988) The challenge of pain. Penguin.

Wells JA, Zipp JF, Schuette PT, McEleney J. (1983) Musculoskeletal disorders among letter carriers. J. Occup. Med. 25: 814. Cited by Grieve GP. (1988) Common Vertebral Joint Problems, 2nd Edition. Churchill Livingstone, Glasgow.

White AW. (1966) The compensation back. Applied Therapy 8. 871-874

Wickstrom G, Pentti J, Hyytianen K, Uutela A. (1989) Type A behaviour and back pain. Work and Stress 3: 2. 203-207

Williams DA and Keefe FJ. (1991) Pain beliefs and the use of cognitive-behavioural coping styles. Pain 46: 185-190

Wood PHN (1980) Undergraduate education in rheumatology in Great Britain. EULAR Bulletin (Eng edn) 9: 61 Cited by Grieve GP (1988) Common vertebral joint problems, 2nd Edition. Churchill Livingstone, Glasgow.

Wood PHN, Badley EM (1980) Epidemiology of back pain. In: Jayson MIV (Ed.) The lumbar spine and back pain, 2nd ed. Pitman Medical, Tunbridge Wells, p 47

Wood PHN. (1983) "Towards an appreciation of the social impact of back pain." Proceedings of a symposium organised by the back Pain Association. Cited by Wells N. (1985) Back Pain. Office of Health Economics.

Wyke BD (1981) The neurology of low back pain. In: Jayson MIV (ed) The lumbar spine and back pain, 3rd edn. Churchill Livingstone, Edinburgh, p 56

Zigmond AS, Snaith RP (1983) The Hospital Anxiety and Depression Scale. Acta Psych. Scand. 67: 361-370

Zitman FG, Linssen ACG, Rien H. (1992) Chronic pain beyond patienthood. J. Nervous and Mental Disease 180: 2. 97-100

Supplement to References

Block AR (1981) Investigation of the response of the spouse to chronic pain behaviour. Psychosom. Med. 43: 415-422

Denny RM (ed) (1991) Fear, avoidance and phobias: a fundamental analysis. Laurence Erlbaum, Hillsdale.

Dodge O and Martin W (1970) Social stress and chronic illness (Univ. of Notre Dame Press, Notre dame, Ind.)

Findlay-Jones RA and Brown GW (1981) Types of stressful life event and the onset of anxiety and depressive disorder. Psychological Medicine 11; 803-815

Hugdahl K (1981) The three systems model of fear and emotion - a critical examination. BRAT 19: 77-85

Jenson MP, Turner JA, Romano JM and Karoly P (1991) Coping with chronic pain: a critical review of the literature. Pain 47 249-283

Keefe FJ and Williams DA. (1989) Pain management programmes. Clinical and research perspectives. N.C. Med. J. 49(10) 526-9

Kent G (1989) Memory of dental experiences as related to naturally occurring changes in state anxiety. Cognitive Emotion. 1: 3-28

Klecker WR (1980) Discriminant analysis. Sage Publications, London.

Linton SJ (1985) The relationship between activity and chronic pain. Pain 21, 289-294

Main CJ and Parker H. (1989) Pain management programmes. In 'Back pain: new approaches to rehabilitation and education'. (Eds) Roland M and Jenner J, Manchester University Press, Manchester, UK, 1989, 128-153

McGracken LM, Zayfert C and Gross RT (1992) The Pain Anxiety Symptoms Scale. Development and validation of a scale to measure fear of pain. Pain 50, 67-73

Polatin PB, Kinney RK, Gatchel RJ, Erin L, Mayer TG (1993) Psychiatric illness and chronic low back pain: The mind and the spine - which goes first? Spine 18(1) 66-71.

Roche PA and Gijsbers KA (1986) A comparison of memory for induced ischaemic pain and chronic rheumatoid pain. Pain 25: 337-343

Ruda MA. (1993) Gender and pain. Pain 53 (1) 1-2

Schmidt AJ (1985) Cognitive factors in the performance level of chronic low back pain patients. J. Psychosom. Res. 29, 183-189

Tabachnick BG and Fidell LS. (1989) Using multivariate statistics, 2nd edition. Harper and Row, New York.

Tulving E and Donaldson W. (1972) Organisation of memory. Academic Press, New York.

BACK PAIN RESEARCH

Thankyou for taking part in the research we are doing about peoples' back ache.

We are trying to find out why some people with back ache get better quickly but some others take a bit longer.

To do this we need PEOPLE LIKE YOU to help us.

We would like you to come back to the Doctor's soon and answer a few simple questions about your back ache. PLEASE SEE THE RECEPTIONIST AT THE DESK BEFORE YOU LEAVE AND GIVE HER YOUR TELEPHONE NUMBER AND ADDRESS. She will arrange a meeting for you to meet one of us.

After about 2 months we would like you to come again and have your back examined by a physio and a specialist from the hospital. You will also be asked a few more simple questions. We will send for you when the time comes.

Then we will do the same in a year, so that you can tell us how you have been over that time.

The visits to see us will NOT TAKE VERY LONG,WILL NOT HURT AND WILL BE IN PRIVATE

ANYTHING YOU TELL US WILL ALSO BE KEPT PRIVATE

The people you will be seeing are:-

Mr. Penny - A doctor and a Back Specialist

Mr. Rose - Physio

Mr. Reilly - Research Worker

We are very grateful for your help and look forward to meeting you.

YOU WILL BE HELPING A LOT OF PEOPLE IN THE FUTURE WHO HAVE A BAD BACK

The University of Liverpool

RESEARCH INTO BACK PAIN

Warmest thanks from the University of Liverpool for your cooperation. The research is about how back pain should be treated: how to improve the quality of treatment, not only in terms of pain-relief but also in terms of preventing recurrence and chronic disability.

One of the factors we believe to be important is the personal element. No two people are exactly alike in their sensitivity or reaction to pain and we think that treatment should take account of this.

One question we have to answer is how much personal information we need. Hence the need for cooperation. There are quite a lot of questions and we hope you will do your best to answer them because that is vital to the research.

First of all, we promise that ALL PERSONAL INFORMATION YOU GIVE US WILL BE HELD IN STRICT CONFIDENCE. It will not be shown, even to your G.P., unless you ask us to. SO CONFIDENTIALITY IS GUARANTEED.

The results of the research are potentially of benefit to the millions of people who suffer back pain every year. It is an important project.

We may contact you again in the near future to ask some further questions.

It is funded by the ARTHRITIS AND RHEUMATISM COUNCIL and is conducted by the University Departments of Orthopaedic & Accident Surgery, General Practice and Clinical Psychology, with the active cooperation of General Practitioners and Health Centre Staff and, of course, yourself.

WE ARE MOST GRATEFUL

NO. OF PAST EPISODES OF BACK PAIN .....

PLEASE RATE PRESENT ATTACK

1	2	3	4	5	6	7	8	9	10

NAME OF PATIENT .....

NAME OF DOCTOR .....

SECTION A

NAME ..... DATE OF BIRTH ..... MALE / FEMALE *

ADDRESS .....

.....

PRESENT EMPLOYMENT ..... IF UNEMPLOYED, PREVIOUS EMPLOYMENT.....

MARRIED: SINGLE: DIVORCED: WIDOWED: SEPARATED: OTHER *

DO YOU SMOKE? YES: NO: EX: * If YES, HOW MANY?

HAVE YOU EVER HAD BACK PAIN BEFORE? YES / NO *

WHEN WAS THE FIRST ATTACK OF BACK PAIN YOU CAN REMEMBER? ..... (Month) ..... (Year)

WAS IT CAUSED BY AN INJURY? YES / NO *

HOW BAD WAS THE FIRST ATTACK? PLEASE RATE BY TICKING THE APPROPRIATE BOX BELOW:

NO PAIN AT ALL | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | WORST PAIN IMAGINABLE

WHEN WAS THE WORST ATTACK OF BACK PAIN YOU CAN REMEMBER? ..... (Month) ..... (Year)

HOW BAD WAS THE WORST ATTACK? PLEASE RATE BY TICKING THE APPROPRIATE BOX BELOW:

NO PAIN AT ALL | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | WORST PAIN IMAGINABLE

SECTION B

HAVE YOU EVER EXPERIENCED PAIN FROM ANY OF THE ACTIVITIES DESCRIBED IN THE FIRST COLUMN? IF SO, RATE THE WORST PAIN YOU CAN REMEMBER BY TICKING THE APPROPRIATE BOX OPPOSITE. IF YOU HAVE NOT EXPERIENCED THE PAIN SPECIFIED, TICK THE N.A. BOX.

1. FRACTURES (BROKEN BONES)	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
2. CHILDBIRTH	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
3. *JOINT SPRAINS*	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
4. DENTISTRY	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
5. OPERATIONS	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
6. SPORTS TRAINING	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
7. INJECTIONS	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
8. OTHER (SPECIFY)	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.

SECTION C

HAVE YOU EVER HAD ANY OF THE FOLLOWING ACHES OR PAINS? IF SO, RATE THE WORST PAIN YOU CAN REMEMBER BY TICKING ONE OF THE TEN BOXES FROM 'NO PAIN AT ALL' TO 'WORST PAIN IMAGINABLE'. IF YOU HAVE NOT EXPERIENCED THE PAIN SPECIFIED, TICK THE N.A. BOX.

1.	HEADACHES	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.
2.	MIGRAINE	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.
3.	MENSTRUAL PAIN	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.
4.	CHEST PAIN	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.
5.	SORE THROAT	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.
6.	STOMACH ACHE	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.
7.	TOOTHACHE	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.
8.	OTHER (SPECIFY)	NO PAIN AT ALL	1	2	3	4	5	6	7	8	9	10	WORST PAIN IMAGINABLE	N.A.

SECTION D

If you have had any of the pains listed below please indicate what you did in response to the worst attack you remember by putting a tick (✓) or a cross (X) under each of the five headings.
(N.B.: for each symptom more than one heading may apply.

	NO EXPERIENCE	(a) TOOK PAIN KILLERS	(b) TOOK PHYSICAL EXERCISE	(c) WENT TO DOCTOR	(d) IGNORED IT AND CARRIED ON	(e) RESTED
1. HEADACHES						
2. MIGRAINE						
3. MENSTRUAL PAIN						
4. CHEST PAIN						
5. SORE THROAT						
6. STOMACH ACHE						
7. TOOTHACHE						
8. OTHER (SPECIFY)						

ANY RELEVANT COMMENTS:

SECTION E

In this section we would like you to estimate the amount of pain which 'common pain situations' can produce by ticking the appropriate box on the scale from 'no pain atall' to 'worst pain imaginable'. If you have not experienced the pain specified, tick the N.A. box.

1.	*BANG THUMB* WITH HAMMER	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.
2.	*BANG ELBOW* (FUNNY BONE) ON DOOR	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.
3.	BANG HEAD ON BOOKSHELF	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.
4.	BANG SHIN ON TABLE LEG	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.
5.	CUT FINGER WITH SHARP KNIFE	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.
6.	GRATE THUMB ON CHEESE GRATER	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.
7.	PRICK FINGER WITH PIN	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.
8.	KNEEL ON DRAWING PIN	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE N.A.

SECTION E (CONTINUED)

9. SCALD (BURN) HANDS IN BOILING WATER	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A
10. BURN HANDS ON LIGHTED CIGARETTE	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A
11. *BURN HANDS ON* HOT IRON	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
12. BURN TONGUE ON BOILING SOUP	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
13. CRUSH LITTLE FINGER ON CAR DOOR	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
14. DROP BRICK ON BIG TOE	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
15. GET STUNG ON FACE BY WASP	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.
16. GET ELECTRIC SHOCK FROM FAULTY PLUG	NO PAIN AT ALL	1 2 3 4 5 6 7 8 9 10	WORST PAIN IMAGINABLE	N.A.

SECTION F

Please describe how you have felt during the PAST WEEK by making a check mark (✓) in the appropriate box.
Please answer ALL questions.

SENSATION	NOT AT ALL	A LITTLE/SLIGHTLY	A GREAT DEAL / QUITE A BIT	EXTREMELY / COULD NOT HAVE BEEN WORSE
1. FEELING HOT ALL OVER				
2. SWEATING ALL OVER				
3. DIZZINESS				
4. BLURRING OF VISION				
5. FEELING FAINT				
6. NAUSEA				
7. PAIN IN STOMACH				
8. CHURNING IN STOMACH				
9. MOUTH BECOMING DRY				
10. NECK MUSCLES ACHING				
11. LEGS FEELING WEAK				
12. MUSCLES TWITCHING OR JUMPING				
13. TENSE FEELING ACROSS FOREHEAD				

SECTION G

LIFE CHANGE QUESTIONNAIRE

Did any of the following occur to you in the YEAR PRECEDING your pain?

EVENT	YES
1. Change in responsibilities at work	...
2. Gain of new family member	...
3. Change in financial state	...
4. Outstanding personal achievement	...
5. Death of spouse	...
6. Marriage	...
7. Taking on a mortgage or loan over £10,000	...
8. Change in sleeping habits	...
9. Business readjustment	...
10. Divorce	...
11. Change to different line of work	...
12. Vacation	...
13. Trouble with boss	...
14. Marital separation	...
15. Son or daughter leaving home	...
16. Change in residence (over 50 miles)	...
17. Change in recreation	...
18. Pregnancy	...
19. Personal injury or illness	...
20. Taking on a mortgage or loan less than £10,000	...
21. Trouble with in-laws	...
22. Spouse got a new job or lost one	...
23. Death of a close family member	...

LIFE CHANGE (CONT)

EVENT	YES
24. Fired at work	...
25. Change in religious beliefs	...
26. Retirement	...
27. Foreclosure of mortgage or loan	...
28. Change in number of family get-togethers	...
29. Change in residence (same area)	...
30. Sex difficulties	...
31. Change in living conditions	...
32. Traffic ticket	...
33. Marital reconciliation	...
34. Change in eating habits	...
35. Change in hours or conditions	...
36. Revision of personal habits	...
37. Change in health of family member	...
38. Death of close friend	...
39. Change in schools	...
40. Change in number of arguments with spouse	...
41. Change in social activities	...
42. Finding a breast lump	...
43. Other	...

SECTION H

DISABILITY QUESTIONNAIRE

When your back hurts, you may find it difficult to do some of the things you normally do.

This list contains some sentences that people have used to describe themselves when they have back pain. When you read them, you may find that some stand out because they describe you today. When you read a sentence that describes you today, put a tick against it. If the sentence does not describe you, then leave the space blank and go on to the next one. Remember, only tick the sentence if you are sure that it describes you today.

		Tick here
1.	I stay at home most of the time because of my back.	....
2.	I change position frequently to try and get my back comfortable.	....
3.	I walk more slowly than usual because of my back.	....
4.	Because of my back I am not doing any of the jobs that I usually do around the house.	....
5.	Because of my back, I use a handrail to get upstairs.	....
6.	Because of my back, I lie down to rest more often.	....
7.	Because of my back, I have to hold on to something to get out of an easy chair.	....
8.	Because of my back, I try to get other people to do things for me.	....
9.	I get dressed more slowly than usual because of my back.	....
10.	I only stand up for short periods of time because of my back.	....
11.	Because of my back, I try not to bend or kneel down.	....
12.	I find it difficult to get out of a chair because of my back.	....
13.	My back is painful almost all the time.	....
14.	I find it difficult to turn over in bed because of my back.	....
15.	My appetite is not very good because of my back pain.	....
16.	I have trouble putting on my socks (or stockings) because of the pain in my back.	....
17.	I only walk short distances because of my back pain.	....

SECTION H (CONTINUED)

18. I sleep less well because of my back. ....

19. Because of my back pain, I get dressed with help from someone else. ....

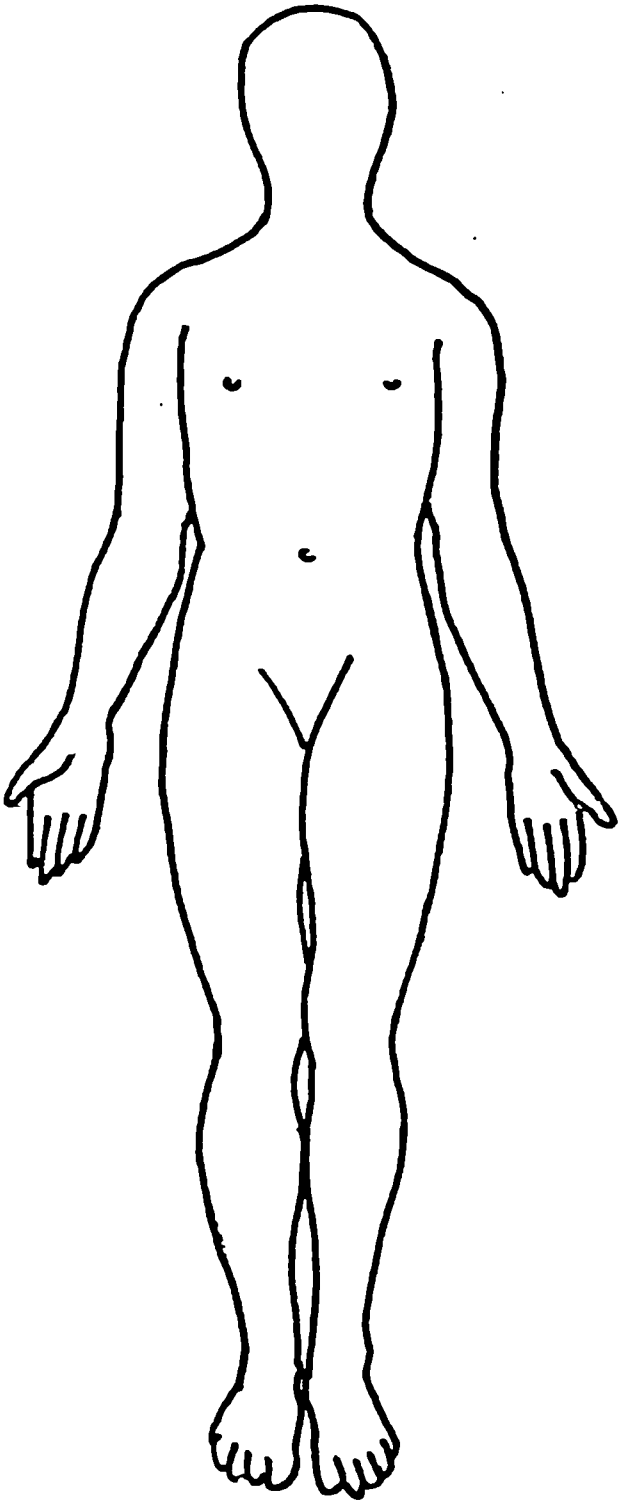
20. I sit down for most of the day because of my back. ....

21. I avoid heavy jobs around the house because of my back. ....

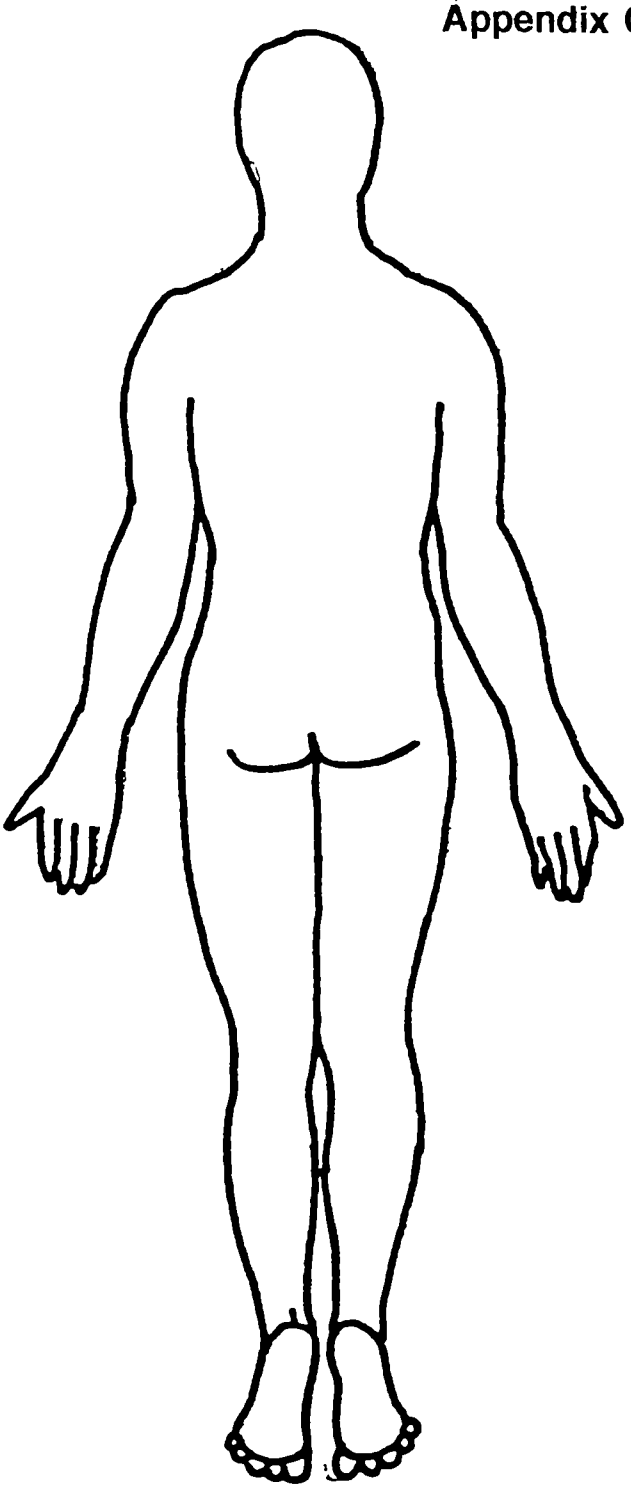
22. Because of my back pain, I am more irritable and bad tempered with people than usual. ....

23. Because of my back, I go upstairs more slowly than usual. ....

24. I stay in bed most of the time because of my back. ....



Right Left
FRONT



Left Right
BACK

MOD.-IED ZUNG

Please indicate for each of these questions which answer best describes how you have been feeling recently.

	Rarely or none of the time (less than 1 day per week	Some or little of the time (1-2 days per week	A moderate amount of time (3-4 per week)	Most of the time [5-7days per week)
1. I feel downhearted and sad				
2. Morning is when I feel best				
3. I have crying spells or feel like it				
4. I have trouble getting to sleep at night				
5. I feel that nobody cares				
6. I eat as much as I used to				
7. I still enjoy sex				
8. I noticed I am losing weight				
9. I have trouble with constipation				
10 My heart beats faster than usual				
11 I get tired for no reason				
12 My mind is as clear as it used to be				
13 I tend to wake up too early				
14 I find it easy to do the things I used to				
15 I am restless and can't keep still				
16 I feel hopeful about the future				
17 I am more irritable than usual				
18 I find it easy to make a decision				
19 I feel quite guilty				
20 I feel that I am useful and needed				
21 My life is pretty full				
22 I feel that others would be better off if I were dead				
23 I am still able to enjoy the things I used to				

OSWESTRY DISABILITY QUESTIONNAIRE

THIS PART OF THE QUESTIONNAIRE LOOKS AT HOW YOUR PAIN IS AFFECTING YOUR LIFE.
IT IS DIVIDED INTO SECTIONS. ONLY TICK ONE BOX IN EACH SECTION. THIS SHOULD BE THE ONE WHICH MOST CLOSELY APPLIES TO YOU.

Section 1 — Pain Intensity
- ☐ I can tolerate the pain I have without having to use pain killers
- ☐ The pain is bad but I manage without taking pain killers
- ☐ Pain killers give complete relief from pain
- ☐ Pain killers give moderate relief from pain.
- ☐ Pain killers give very little relief from pain.
- ☐ Pain killers have no effect on the pain and I do not use them.

Section 2 — Personal Care (Washing, Dressing, etc)
- ☐ I can look after myself normally without causing extra pain.
- ☐ I can look after myself normally but it causes extra pain
- ☐ It is painful to look after myself and I am slow and careful
- ☐ I need some help but manage most of my personal care
- ☐ I need help every day in most aspects of self care
- ☐ I do not get dressed, wash with difficulty and stay in bed

Section 3 — Lifting
- ☐ I can lift heavy weights without extra pain
- ☐ I can lift heavy weights but it gives extra pain.
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, eg on a table.
- ☐ Pain prevents me from lifting heavy weights but I can manage light to medium weights if they are conveniently positioned.
- ☐ I can lift only very light weights.
- ☐ I cannot lift or carry anything at all

Section 4 — Walking
- ☐ Pain does not prevent me walking any distance.
- ☐ Pain prevents me walking more than 1 mile.
- ☐ Pain prevents me walking more than ½ mile.
- ☐ Pain prevents me walking more than ¼ mile.
- ☐ I can only walk using a stick or crutches.
- ☐ I am in bed most of the time and have to crawl to the toilet.

Section 5 — Sitting
- ☐ I can sit in any chair as long as I like.
- ☐ I can only sit in my favourite chair as long as I like.
- ☐ Pain prevents me sitting more than 1 hour.
- ☐ Pain prevents me from sitting more than ½ hour.
- ☐ Pain prevents me from sitting more than 10 mins.
- ☐ Pain prevents me from sitting at all.

Section 6 — Standing
- ☐ I can stand as long as I want without extra pain.
- ☐ I can stand as long as I want but it gives me extra pain.
- ☐ Pain prevents me from standing for more than 1 hour.
- ☐ Pain prevents me from standing for more than 30 mins.
- ☐ Pain prevents me from standing for more than 10 mins.
- ☐ Pain prevents me from standing at all.

Section 7 — Sleeping
- ☐ Pain does not prevent me from sleeping well.
- ☐ I can sleep well only by using tablets.
- ☐ Even when I take tablets I have less than six hours sleep.
- ☐ Even when I take tablets I have less than four hours sleep.
- ☐ Even when I take tablets I have less than two hours sleep.
- ☐ Pain prevents me from sleeping at all.

Section 8 — Sex Life
- ☐ My sex life is normal and causes no extra pain.
- ☐ My sex life is normal but causes some extra pain.
- ☐ My sex life is nearly normal but is very painful.
- ☐ My sex life is severely restricted by pain.
- ☐ My sex life is nearly absent because of pain.
- ☐ Pain prevents any sex life at all.

Section 9 — Social Life
- ☐ My social life is normal and gives me no extra pain.
- ☐ My social life is normal but increases the degree of pain.
- ☐ Pain has no significant effect on my social life apart from limiting my more energetic interests, eg dancing, etc.
- ☐ Pain has restricted my social life and I do not go out as often.
- ☐ Pain has restricted my social life to my home.
- ☐ I have no social life because of pain.

Section 10 — Travelling
- ☐ I can travel anywhere without extra pain.
- ☐ I can travel anywhere but it gives me extra pain.
- ☐ Pain is bad but I manage journeys over two hours.
- ☐ Pain restricts me to journeys of less than one hour.
- ☐ Pain restricts me to short necessary journeys under 30 minutes.
- ☐ Pain prevents me from travelling except to the doctor or hospital.

NAME .....

Appendix D

Back pain stayed away
bothered on and off
painful all the time

you feel any PERSISTENT OR ACHING
AIN": cross-hatch the site

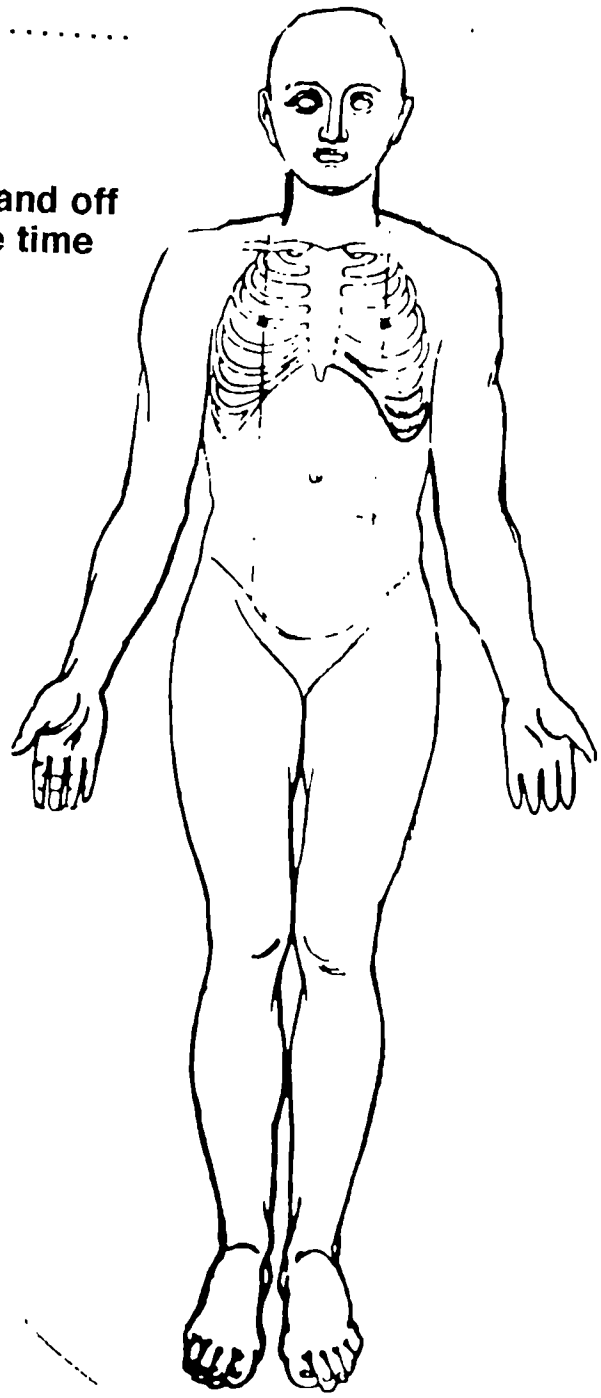
you feel any SHOOTING OR
TABBING PAIN": put a point where it
egins and an arrow to show how far
t goes

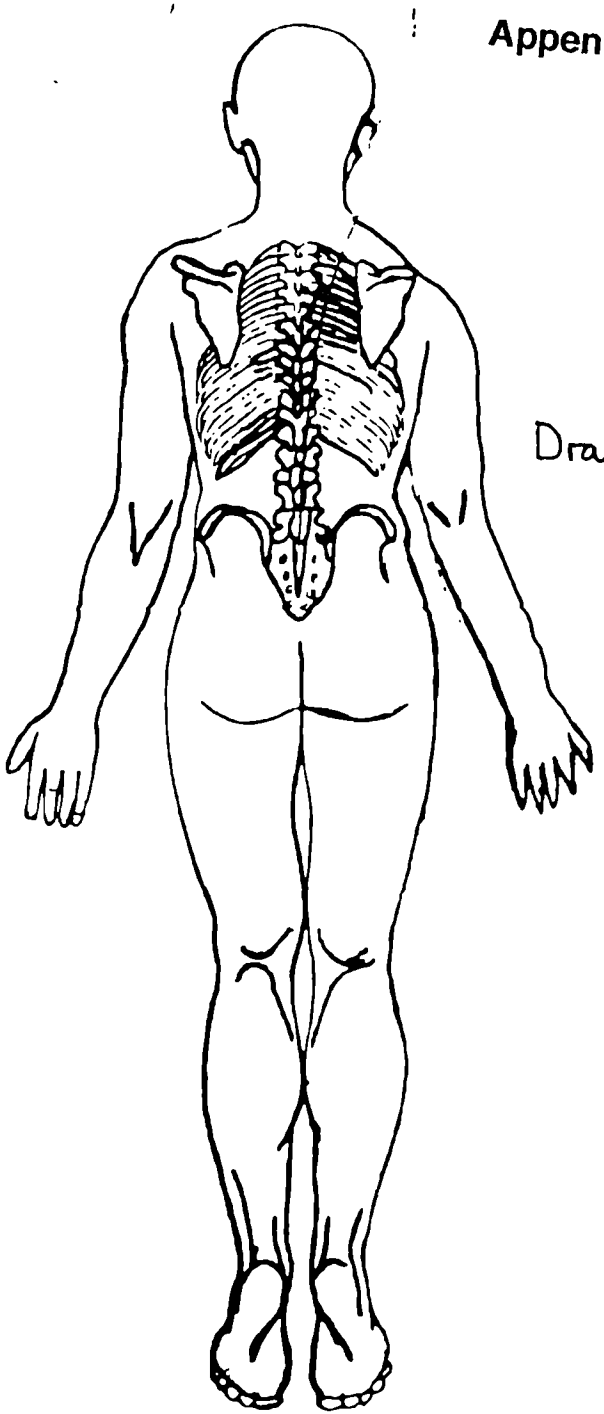
ou have CRAMPS (muscular spasm):
rk site with zigzags

feel any SHARP PRICKLING (PINS &
ES): mark site with crosses

el a general TINGLING sensa-
mark site with dots

el any NUMBNESS (the skin
dead to the touch): mark
h small circles





Draw shift

Do you get pain at the tip of your tail bone?

Does your whole leg ever become painful?

Does your whole leg ever go numb?

Does your whole leg ever give way?

In the past year have you ever had any spells of very little pain?

Have you ever been made worse by treatment?

Have you ever been admitted into hospital because of your LBP (emergency)?

Onset..... Insidious/non-accidental/trully accidental

Weight height

Buttock wasting y/n

Heel walking

Toe walking

RSF

LSF

Axial loading

Rotation

Extension

PKB L

PKB R

Ankle jerk L clonus
brisk
present
reduced
only with reinforcement
absent

Ankle jerk R clonus
brisk
present
reduced
only with reinforcement
absent

Tenderness light pinch, wide area y/n

deep pressure, wide area y/n

Overreaction to examination y/n

SLR R
L

SLR distraction

Situp y/n

Muscle strength

quads
hams
tib ant
EHL
FHL

Cogwheel giving way? y/n

Sensation deficit? y/n

Sensory regional disturbance? y/n

Flexion

Knee jerk L clonus
brisk
present
reduced
only with reinforcement
absent

Knee jerk R clonus
brisk
present
reduced
only with reinforcement
absent

Slump

Appendix E

ID No .....

Please answer the following question by ticking the box which describes how you are now

No pain at all **1 2 3 4 5 6 7 8 9 10** **Worst pain imaginable**

Are you off work now because of your back pain? **Yes/no**

Since we saw you last has your back pain:

Stayed away completely

Bothered you on and off

been painful all of the time

(please tick appropriate answer)

DISABILITY QUESTIONNAIRE

When your back hurts, you may find it difficult to do some of the things you normally do.

This list contains some sentences that people have used to describe themselves when they have back pain. When you read them, you may find that some stand out because they describe you today. When you read a sentence that describes you today, put a tick against it. If the sentence does not describe you, then leave the space blank and go on to the next one. Remember, only tick the sentence if you are sure that it describes you today.

		Tick here
1.	I stay at home most of the time because of my back.	....
2.	I change position frequently to try and get my back comfortable.	....
3.	I walk more slowly than usual because of my back.	....
4.	Because of my back I am not doing any of the jobs that I usually do around the house.	....
5.	Because of my back, I use a handrail to get upstairs.	....
6.	Because of my back, I lie down to rest more often.	....
7.	Because of my back, I have to hold on to something to get out of an easy chair.	....
8.	Because of my back, I try to get other people to do things for me.	....
9.	I get dressed more slowly than usual because of my back.	....
10.	I only stand up for short periods of time because of my back.	....
11.	Because of my back, I try not to bend or kneel down.	....
12.	I find it difficult to get out of a chair because of my back.	....
13.	My back is painful almost all the time.	....
14.	I find it difficult to turn over in bed because of my back.	....
15.	My appetite is not very good because of my back pain.	....
16.	I have trouble putting on my socks (or stockings) because of the pain in my back.	....
17.	I only walk short distances because of my back pain.	....

18. I sleep less well because of my back. ....

19. Because of my back pain, I get dressed with help from someone else. ....

20. I sit down for most of the day because of my back. ....

21. I avoid heavy jobs around the house because of my back. ....

22. Because of my back pain, I am more irritable and bad tempered with people than usual. ....

23. Because of my back, I go upstairs more slowly than usual. ....

24. I stay in bed most of the time because of my back. ....

Appendix F

Subgroup differences

Table F1

Sex differences at screening and two months follow-up (M = male, F = female) (Continuous variables)

Variable	N	Mean	SD	t value	P
Per cent active coping	M 146 F 144	41.8 34.8	30.6 27.8	2.04	0.042
Weighted life events	M 150 F 148	144 161	96 113	-1.41	0.159
Highest external pain	M 150 F 146	7.38 8.17	2.26 2.03	-3.14	0.002
Highest internal pain	M 146 F 147	6.59 7.28	2.33 2.23	-2.61	0.010
Highest accidental pain	M 151 F 144	7.48 7.17	1.90 2.25	1.28	0.204
Severity of present attack	M 148 F 145	5.93 6.65	2.39 2.53	-2.51	0.013
Severity of worst attack LBP	M 147 F 144	8.02 8.68	1.75 1.46	-3.52	0.001
Severity of first attack LBP	M 146 F 143	6.39 6.67	2.39 2.19	-1.01	0.311
MSPQ	M 150 F 148	4.06 6.56	3.35 4.35	-5.56	0.001
Disability	M 150 F 148	12.13 13.23	5.46 5.97	-1.66	0.097

Table F1 (cont)

Variable (not in MANOVA)*	N	Mean	SD	t value	P
Pain drawing	M 80	7.33	8.97	1.00	0.323
	F 72	6.10	5.71		
Zung	M 85	14.47	10.30	-8.37	0.001
	F 73	20.81	10.19		
Oswestry	M 87	12.53	14.28	-3.68	0.001
	F 72	21.07	14.91		

* In view of the limited number of psychological variables measured at two months follow-up, only univariate analyses of sex differences of these variables were performed.

Table F2

Sex differences at screening (M = male, F = female) (Nominal variables)

	Male	Female
Site		
Maghull Health Centre	71	54
Other practices	80	94
		Exact P = 0.001
Onset		
Insidious onset	85	103
Onset due to injury	61	44
		Exact P = 0.039
Smoking		
Non-smokers	102	86
Smokers	49	62
		Exact P = 0.095
Sick leave status		
Not on sick	40	46
Onsick	67	53
		Exact P = 0.205
Previous history		
No history of back pain	31	26
History of back pain	120	122
		Exact P = 0.528
Occupational status		
Occupational groups I to III	69	50
" " IV and V	46	54
Not at work	36	44
		Exact P = 0.112

Table F2 (cont)

	Male	Female	
Time since onset of first attack			
No previous	30	23	
1-5 years	54	45	
More than 5 years	67	80	
			Exact P = 0.244
Number of previous attacks			
No previous	30	22	
1-9 attacks	75	61	
More than 9	42	62	
			Exact P = 0.038

Table F3

Work status differences at screening (N = not on sick, Y = on sick) (continuous variables)

Variable		N	Mean	SD	t value	P
Per cent active coping	N	82	37.09	28.37	-0.33	0.738
	Y	119	38.42	27.26		
Weighted life events	N	86	159	107	0.40	0.691
	Y	121	153	102		
Highest external pain	N	85	7.83	2.33	0.04	0.971
	Y	119	7.82	2.25		
Highest internal pain	N	83	6.92	2.14	-0.14	0.840
	Y	120	6.97	2.42		
Highest accidental pain	N	86	7.30	1.97	-0.06	0.947
	Y	118	7.32	2.26		
Severity of present attack	N	86	5.65	2.48	-2.34	0.021
	Y	121	6.46	2.44		
Severity of worst attack LBP	N	84	8.01	1.72	-1.67	0.100
	Y	110	8.40	1.59		
Severity of first attack LBP	N	84	6.21	2.18	-0.70	0.478
	Y	116	6.44	2.44		
MSPQ	N	85	4.92	3.69	-0.44	0.652
	Y	121	5.18	4.28		
Disability	N	86	10.65	5.56	-4.34	0.001
	Y	119	14.04	5.49		

Table F4

Work status differences at screening (nominal variables)

	Not on sick leave	On sick leave	
Practice			
Maghull Health Centre	33	62	
Other practices	53	59	
			Exact P = 0.089
Onset			
Insidious onset	56	74	
Onset due to injury	28	46	
			Exact P = 0.554
Smoking			
Non-smokers	55	72	
Smokers	31	49	
			Exact P = 0.564
Previous history			
No history of back pain	14	71	
History of back pain	12	96	
			Exact P = 0.026
Social group			
Social Groups I to III	42	54	
" " IV and V	30	61	
Not at work	14	6	
			Exact P = 0.009
Marital status			
Married	55	91	
Others	31	30	
			Exact P = 0.090

Table F4 (cont)

	Not on sick leave	On sick leave
Time since onset of first attack		
No previous	15	21
1-5 years	26	41
More than 5 years	45	59
		Exact P = 0.861
Number of previous attacks		
No previous	14	21
1-9 attacks	36	63
More than 9	35	37
		Exact P = 0.284

Table F5

Differences at screening between smokers and non-smokers (N = non-smokers, S = smokers) (Continuous variables)

Variable	N	Mean	SD	t value	P
Per cent active coping	N 182 S 109	40.41 38.66	28.56 27.80	0.49	0.625
Weighted life events	N 188 S 111	155 163	104 111	-0.58	0.568
Highest external pain	N 186 S 111	7.59 7.98	2.12 2.22	-1.43	0.157
Highest internal pain	N 185 S 109	7.01 6.93	2.28 2.35	0.24	0.810
Highest accidental pain	N 188 S 108	7.26 7.51	2.09 2.06	-0.98	0.328
Severity of present attack	N 185 S 109	6.59 6.33	2.33 2.54	*0.84*	*0.408*
Severity of worst attack LBP	N 183 S 109	8.19 8.55	1.73 1.56	-1.73	0.081
Severity of first attack LBP	N 182 S 108	6.49 6.55	2.30 2.40	-0.21	0.836
MSPQ	N 189 S 110	5.23 5.70	4.29 3.98	-0.91	0.361
Disability	N 189 S 109	12.45 13.00	5.96 5.70	-0.73	0.463

Table F6

Differences at screening between smokers and non-smokers (N = non-smokers, S = smokers) (Nominal variables)

	Non-Smokers	Smokers
Onset		
Insidious onset	116	72
Onset due to injury	68	38
		Exact P = 0.707
Social group		
Social groups I to III	81	38
" " IV and V	58	43
Not at work	50	30
		Exact P = 0.267
Previous history		
No history of back pain	40	12
History of back pain	145	96
		Exact P = 0.026
Site		
Maghull Health Centre	86	40
Others	103	71
		Exact P = 0.117
Sex		
Males	102	49
Females	86	62
		Exact P = 0.095

Table F6 (cont)

	Non-smokers	Smokers	
Time since onset of first attack			
No previous	41	12	
1-5 years	62	38	
More than 5 years	86	61	
			Exact P = 0.048
Number of previous attacks			
No previous	40	12	
1-9 attacks	85	51	
More than 9	60	45	
			Exact P = 0.052

Table F7

Age group differences of variables measured at the acute stage and two months follow-up (continuous variables)

Group 1 = 13-40 years
Group 2 = 41-59 years
Group 3 = 60-78 years

Variable	Group	N	Mean	SD	F-Ratio	P
Highest ever internal pain	1	138	6.57	2.31	3.27	0.039
	2	110	7.30	2.29		
	3	43	7.06	2.20		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 2)

Variable	Group	N	Mean	SD	F-Ratio	P
Weighted life events	1	139	175	111	12.15	0.001
	2	112	147	96		
	3	45	90	72		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 3 and between groups 2 and 3)

Variable	Group	N	Mean	SD	F-Ratio	P
Severity of first attack	1	135	6.24	2.25	2.51	0.083
	2	108	6.90	2.38		
	3	44	6.52	2.18		

Variable	Group	N	Mean	SD	F-Ratio	P
Severity of worst attack	1	137	8.18	1.68	2.30	0.101
	2	108	8.61	1.50		
	3	44	8.18	1.83		

Variable	Group	N	Mean	SD	F-Ratio	P
Severity of present attack	1	137	6.15	2.44	0.48	0.619
	2	110	6.46	2.41		
	3	44	6.25	2.76		

Table F7 (continued)

Variable	Group	N	Mean	SD	F-Ratio	P
Highest ever external pain	1	139	7.58	2.20	0.75	0.470
	2	110	7.90	2.24		
	3	45	7.91	1.92		
Variable	Group	N	Mean	SD	F-Ratio	P
Highest ever accidental pain	1	137	7.29	1.96	0.70	0.495
	2	112	7.22	2.23		
	3	44	7.65	2.10		
Variable	Group	N	Mean	SD	F-Ratio	P
Disability	1	138	12.24	5.64	1.96	0.142
	2	112	13.47	5.62		
	3	45	11.82	6.23		
Variable	Group	N	Mean	SD	F-Ratio	P
MSPQ	1	140	5.41	4.07	1.95	0.144
	2	112	5.57	4.26		
	3	44	4.18	3.58		
Variable	Group	N	Mean	SD	F-Ratio	P
Percent active coping strategies	1	139	36.43	27.54	2.26	0.106
	2	106	43.15	29.94		
	3	43	33.68	33.18		
Variable	Group	N	Mean	SD	F-Ratio	P
Pain drawing	1	57	7.26	5.63	0.38	0.687
	2	70	6.34	5.54		
	3	24	5.85	5.59		
Variable	Group	N	Mean	SD	F-Ratio	P
Zung	1	59	16.45	10.85	0.86	0.424
	2	72	18.35	10.86		
	3	26	15.54	10.06		
Variable	Group	N	Mean	SD	F-Ratio	P
Oswestry	1	60	15.00	15.70	0.81	0.446
	2	72	17.65	14.78		
	3	26	13.96	13.81		

Table F8

Age group differences (nominal variables)

Group 1 = 13-40 years
Group 2 = 41-59 years
Group 3 = 60-78 years

	1	2	3
Site			
Maghull Health Centre	43	61	21
Other practices	97	51	24
			Exact P = 0.001
Onset			
Insidious onset	101	61	23
Onset due to injury	37	50	19
			Exact P = 0.004
Smoking			
Non-smokers	88	68	32
Smokers	52	44	13
			Exact P = 0.477
Sick leave status			
Not on sick leave	43	32	10
On sick leave	54	60	6
			Exact P = 0.087
Previous history			
No history of back pain	28	20	7
History of back pain	111	91	38
			Exact P = 0.807

Table F8 (cont)

	1	2	3
Time since onset of first attack			
No previous	27	19	7
1-5 years	64	26	8
More than 5 years	49	67	30
			Exact P = 0.001
Number of previous attacks			
No previous	25	19	8
1-9 attacks	73	36	25
More than 9	38	55	11
			Exact P = 0.002

Table F9

Time since onset group differences (continuous variables)

Group 1 = No previous back pain
Group 2 = First attack up to five years previously
Group 3 = First attack more than five years previously

Variable	Group	N	Mean	SD	F-Ratio	P
Highest ever internal pain	1	52	6.32	2.34	6.05	0.002
	2	97	6.58	2.40		
	3	145	7.40	2.14		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 3 and 2 and 3)

Variable	Group	N	Mean	SD	F-Ratio	P
Highest ever external pain	1	52	7.48	2.46	3.57	0.029
	2	100	7.43	2.28		
	3	145	8.11	1.94		

(No two groups are significantly different at the 0.05 level)

Variable	Group	N	Mean	SD	F-Ratio	P
Severity of worst attack	1	45	6.95	2.02	22.23	0.001
	2	100	8.50	1.50		
	3	147	8.67	1.38		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 2 and 3)

Variable	Group	N	Mean	SD	F-Ratio	P
Weighted life events	1	53	127	84	4.11	0.017
	2	100	175	106		
	3	146	146	108		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 2)

Table F9 (continued)

Time since onset group differences

Variable	Group	N	Mean	SD	F-Ratio	P
Severity of first attack	1	44	6.90	2.03	1.69	0.185
	2	100	6.21	2.36		
	3	146	6.62	2.31		

Variable	Group	N	Mean	SD	F-Ratio	P
Severity of present attack	1	51	6.94	2.23	2.17	0.115
	2	99	6.12	2.43		
	3	144	6.16	2.57		

Variable	Group	N	Mean	SD	F-Ratio	P
Highest ever accidental pain	1	53	7.09	2.16	1.12	0.327
	2	100	7.58	1.98		
	3	145	7.26	2.12		

Variable	Group	N	Mean	SD	F-Ratio	P
Disability	1	53	12.73	6.86	0.039	0.961
	2	100	12.78	5.01		
	3	145	12.57	5.77		

Variable	Group	N	Mean	SD	F-Ratio	P
MSPQ	1	53	4.77	3.39	0.704	0.495
	2	100	5.21	4.23		
	3	146	4.18	3.58		

Variable	Group	N	Mean	SD	F-Ratio	P
Percent active coping strategies	1	52	32.40	24.92	1.30	0.270
	2	98	39.55	28.69		
	3	141	39.75	31.27		

Table F10

Time since onset group differences (nominal variables)

Group 1 = No previous back pain
Group 2 = First attack up to five years previously
Group 3 = First attack more than five years previously

	1	2	3
Site			
Maghull Health Centre	21	41	64
Other practices	32	59	83
			Exact P = 0.881
Smoking			
Non-smokers	41	62	86
Smokers	12	38	61
			Exact P = 0.047
Sick leave status			
Not on sick	51	26	45
Onsick	21	41	59
			Exact P = 0.861
Number of previous attacks			
No previous	51	0	1
1-9 attacks	1	74	61
More than 9	0	25	80
			Exact P = 0.001

Table F11

Occupational group differences

Group 1 = Social groups I to III
Group 2 = Social groups IV and V
Group 3 = Not in paid employment

Variable	Group	N	Mean	SD	F-Ratio	p
Highest ever internal pain	1	118	6.54	2.35	3.77	0.024
	2	100	7.40	2.14		
	3	79	6.94	2.35		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 2)

Variable	Group	N	Mean	SD	F-Ratio	p
Weighted life events	1	118	170	111	4.08	0.017
	2	101	152	100		
	3	80	127	95		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 3)

Variable	Group	N	Mean	SD	F-Ratio	p
Highest ever external pain	1	118	7.57	2.13	0.88	0.415
	2	100	7.85	2.37		
	3	79	7.97	1.98		

Variable	Group	N	Mean	SD	F-Ratio	p
Severity of worst attack	1	116	8.22	1.60	0.756	0.470
	2	97	8.36	1.56		
	3	79	8.51	1.79		

Variable	Group	N	Mean	SD	F-Ratio	p
Severity of first attack	1	115	6.56	2.28	0.383	0.682
	2	96	6.36	2.34		
	3	79	6.66	2.291		

Table F11 (continued)

Occupational group differences

Variable	Group	N	Mean	SD	F-Ratio	P
Severity of present attack	1	115	6.16	2.52	2.87	0.750
	2	101	6.32	2.37		
	3	78	6.42	2.58		
Highest ever accidental pain	1	119	7.07	2.08	2.32	0.099
	2	99	7.68	2.14		
	3	78	7.33	1.97		
Disability	1	118	12.66	5.68	0.868	0.420
	2	100	12.18	5.68		
	3	80	12.67	5.73		
MSPQ	1	119	5.32	3.97	0.834	0.435
	2	101	4.92	4.07		
	3	79	5.70	4.24		
Percent active coping strategies	1	118	42.00	28.32	2.29	0.102
	2	97	38.39	27.78		
	3	78	32.85	32.30		

Table F12

Occupational group differences (nominal variables)

	Social groups I to III	Social groups IV and V	Not at work
Sick leave status			
Not on sick	42	30	14
On sick	54	61	6
			Exact P = 0.009
Site			
Maghull Health Centre	58	40	28
Others	61	61	52
			Exact P = 0.132
Time since onset of first attack			
No previous	24	16	13
1-5 years	40	35	25
More than 5 years	55	50	42
			Exact P = 0.877
Number of previous attacks			
No previous	22	16	14
1-9 attacks	54	48	34
More than 9	39	37	29
			Exact P = 0.956

Table F13

Physical variables: sex differences (continuous variables)

M = Male, F = Female

Variable	Group	N	Mean	SD	t value	P
"Inappropriate" signs and symptoms	M	86	0.95	1.9	-3.42	0.001
	F	72	2.13	2.40		
Body Mass Index	M	86	25.70	3.18	0.76	0.449
	F	71	25.25	4.46		
Sagittal Movement	M	86	58.19	13.10	-1.35	0.178
	F	72	61.45	17.17		
Passive Knee Bend	M	86	133.96	23.46	1.11	0.270
	F	72	130.09	19.68		
Straight leg raise	M	86	64.36	14.53	0.75	0.452
	F	72	62.50	16.47		
Side flexion	M	86	35.01	9.88	1.23	0.222
	F	72	33.27	7.45		

Table F14

Physical variables: sex differences (nominal variables)

	Male	Female	
Situp			
No situp	27	28	
Sit-up	59	43	
			Exact P = 0.402
Neurological involvement			
No neurological involvement	69	57	
Neurological involvement	17	15	
			Exact P = 1.00
Slump test			
-ve Slump Test	56	48	
+ve Slump Test	30	24	
			Exact P = 0.868

Table F15

Physical variables: age differences (continuous variables)

Group 1 = 13-40 years
Group 2 = 41-59 years
Group 3 = 60-78 years

Variable	Group	N	Mean	SD	F-ratio	P
"Ina-ppropriate" signs and symptoms	1	59	63.04	16.15	0.49	0.610
	2	73	63.24	15.12		
	3	26	66.48	15.73		

Variable	Group	N	Mean	SD	F-ratio	P
Body Mass Index	1	59	24.19	3.40	6.14	0.002
	2	72	26.43	3.97		
	3	26	26.01	3.53		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 2)

Variable	Group	N	Mean	SD	F-ratio	P
Sagittal Movement	1	59	64.67	15.92	8.02	0.001
	2	72	58.56	13.83		
	3	25	51.34	12.77		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 3)

Variable	Group	N	Mean	SD	F-ratio	P
Passive Knee Bend	1	59	139.48	22.96	5.53	0.006
	2	73	128.22	20.06		
	3	26	127.59	20.79		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 2)

Table F15 (continued)

Physical variables: age differences

Group 1 = 13-40 years
Group 2 = 41-59 years
Group 3 = 60-78 years

Variable	Group	N	Mean	SD	F-ratio	P
Straight leg raise	1	59	63.04	16.16	0.49	0.610
	2	73	63.24	15.13		
	3	26	66.48	15.73		

Variable	Group	N	Mean	SD	F-ratio	P
Side flexion	1	59	37.49	8.59	7.15	0.001
	2	73	33.03	8.93		
	3	26	30.77	8.87		

(Scheffe post-hoc test shows significant (at 0.05 level) differences between groups 1 and 2 and 1 and 3)

Table F16

Physical variables: age differences (nominal variables)

Group 1 = 13-40 years
Group 2 = 41-59 years
Group 3 = 60-78 years

	Group 1	Group 2	Group 3
Situp			
No sit-up	11	29	15
Sit-up	48	44	11

Exact P = 0.001

Table F16 (continued)

Neurological involvement			
No neurological involvement	46	59	21
Neurological involvement	13	14	5
			Exact P = 0.963
Slump test			
-ve Slump Test	34	50	21
+ve Slump Test	13	14	5
			Exact P = 0.109

Table F17

Differences in terms of screening variables between patients referred by doctors in Maghull Health Centre and patients referred by doctors in other practices. (M = Maghull Health Centre, O = Others).

Variable	Group	N	Mean	SD	t value	P
MSPQ	M	125	4.19	3.53	-4.05	0.001
	O	174	6.08	4.27		
Highest ever accidentally produced pain	M	125	6.96	2.10	-2.73	0.007
	O	169	7.62	2.03		
Severity of worst attack	M	125	8.12	1.67	-2.00	0.047
	O	167	8.51	1.61		
Weighted life events	M	125	137	102	-2.14	0.033
	O	174	163	105		
Severity of present pain	M	122	6.18	2.30	-0.56	0.573
	O	172	6.35	2.60		
Severity of first attack	M	125	6.44	2.28	-0.49	0.624
	O	165	6.58	2.31		

Table F17 (continued)

Variable	Group	N	Mean	SD	t value	P
Highest ever externally produced pain	M	126	7.80	2.04	0.24	0.812
	O	171	7.74	2.28		
Highest ever internally produced pain	M	126	6.89	2.29	-0.29	0.768
	O	170	6.97	2.32		
Per cent active coping strategies	M	124	39.03	27.04	0.33	0.742
	O	167	37.88	31.11		
Disability	M	125	12.30	6.14	-0.95	0.343
	O	173	12.94	5.41		

Appendix G

Differences between attenders and non-attenders at each stage of the study

Table G1

Comparison of two months follow-up attenders with non-attenders in terms of screening variables (continuous variables)

N = non-attenders, A = attenders

Variable	Group	N	Mean	SD	t value	P
Age	N	138	40.24	13.77	-2.85	0.005
	A	162	44.84	13.89		
Disability	N	136	12.30	5.47	-1.03	0.304
	A	162	12.98	5.93		
Severity of present pain	N	136	6.11	2.62	-1.12	0.262
	A	158	6.43	2.53		
Severity of worst attack	N	133	8.39	1.59	0.40	0.693
	A	159	8.31	1.69		
Severity of first attack	N	132	6.63	2.33	0.76	0.448
	A	158	6.43	2.27		
MSPQ	N	138	5.49	4.14	0.79	0.429
	A	161	5.11	4.02		
Per cent active coping strategies	N	134	37.43	29.25	-0.49	0.624
	A	157	39.15	29.59		
Weighted life events	N	137	156	115	0.62	0.534
	A	162	149	96		
Highest externally produced pain	N	137	7.92	2.12	1.12	0.265
	A	160	7.64	2.22		
Highest internally produced pain	N	136	7.07	2.21	0.91	0.366
	A	158	6.82	2.38		

Table G1 (continued)

Variable	Group	N	Mean	SD	t value	P
Highest accidentally produced pain	N	135	7.47	2.01	0.95	0.344
	A	161	7.23	2.15		

Table G2

Comparison of two months follow-up attenders with non-attenders in terms of screening variables (nominal variables)

	Non-attenders	Attenders	
Site			
Maghull HC	38	88	
Others	100	74	
			Exact P = 0.001
Smoking			
Non-smokers	70	119	
Smokers	68	43	
			Exact P = 0.001
Sex			
Males	63	88	
Females	75	73	
			Exact P = 0.132
Social group			
Social groups I to III and students	47	73	
Others	90	89	
			Exact P = 0.075
Sick leave			
Not on sick leave	40	46	
On sick leave	48	73	
			Exact P = 0.392

Table G2 (continued)

	Non-attenders	Attenders
History		
No previous history of LBP	27	28
History of LBP	111	134
		Exact P = 0.654
Time since onset of first attack		
No previous	25	28
1-5 years	52	48
More than 5 years	61	86
		Exact P = 0.265
Number of previous attacks		
No previous	26	26
1-9 attacks	68	68
More than 9	41	64
		Exact P = 0.205

Table G3

Comparison of patients who provided twelve months follow-up data (either at interview or by post) with those who did not.

N = non-providers, P = providers

Variable	Group	N	Mean	SD	t value	P
Age	N	104	41.20	13.99	-1.38	0.170
	P	196	43.54	13.98		
Disability	N	103	12.42	5.22	-0.56	0.574
	P	195	12.81	6.98		
Severity of present pain	N	101	6.20	2.55	-0.39	0.698
	P	193	6.32	2.45		
Severity of worst attack	N	103	8.33	1.68	-.070	0.942
	P	189	8.35	1.63		
Severity of first attack	N	104	6.47	2.28	-.027	0.791
	P	187	6.55	2.31		
MSPQ	N	104	5.64	4.24	1.09	0.275
	P	195	5.10	3.98		
Per cent active coping strategies	N	104	40.06	30.16	0.73	0.466
	P	187	37.43	29.01		
Weighted life events	N	103	144	91	-0.98	0.327
	P	196	157	111		
Highest externally produced pain	N	102	7.91	2.07	0.80	0.427
	P	192	7.70	2.23		
Highest internally produced pain	N	103	6.71	2.49	-1.23	0.220
	P	194	7.06	2.19		
Highest accidentally produced pain	N	101	7.11	2.07	-1.32	0.187
	P	195	7.45	2.09		

Table G3 (cont) (N = non-providers, P = providers)

Variable at 2 months	Group	N	Mean	SD	T-value	P
Zung	N	37	17.92	10.89	0.39	0.694
	P	122	17.12	10.72		
Pain Drawing	N	38	6.56	6.63	-0.14	0.889
	P	122	6.76	7.88		
Oswestry	N	38	16.65	15.01	0.15	0.879
	P	122	16.22	15.20		
Inappropriate signs and symptoms	N	38	16.34	1.51	-0.43	0.666
	P	121	16.52	2.39		
Mean straight leg raise	N	38	64.70	15.29	-0.43	0.644
	P	121	63.35	15.64		
Sagittal movement	N	38	65.52	15.88	2.79	0.006
	P	121	57.85	14.41		
Prone knee bend	N	38	134.34	18.63	0.66	0.512
	P	121	131.67	22.75		
BMI	N	38	25.36	4.54	-0.28	0.770
	P	119	25.56	3.56		
Side flexion	N	38	35.53	8.26	1.00	0.318
	P	121	33.88	9.05		

Table G4

Comparison of patients who provided twelve months follow-up data (either at interview or by post) with those who did not (nominal variables).

	Non-providers	Providers	
Site			
Maghull HC	41	85	
Others	63	111	
			Exact P = 0.540
Smoking			
Non-smokers	58	131	
Smokers	46	65	
			Exact P = 0.061
Sex			
Males	52	99	
Females	52	97	
			Exact P = 1.00
Social Group			
Social groups I to III	37	82	
Social groups IV and V	29	72	
Others	38	42	
			Exact P = 0.019
Sick leave			
Not on sick leave	26	60	
On sick leave	40	81	
			Exact P = 0.729

Table G4 (cont.)	Non-providers	Providers
History		
No previous history of LBP	21	32
History of LBP	83	164
		Exact P = 0.701
Time since onset of first attack		
No previous	21	32
1-5 years	34	66
More than 5 years	49	98
		Exact P = 0.661
Number of previous attacks		
No previous	20	32
1-9 attacks	42	94
More than 9	39	66
		Exact P = 0.498
Onset		
Insidious onset	65	123
Accidental onset	36	70
		Exact P = 1.00
Marital status		
Married	64	143
Not married	40	51
		Exact P = 0.035

Table G4 (cont.)

	Non-providers	Providers
Neurological deficit		
No neurological deficit	31	96
Neurological deficit	7	25
		Exact P = 0.900
Situp		
Can sit up	9	46
Can not sit up	29	74
		Exact P = 0.119
Nerve root tethering		
No nerve root tethering	27	78
Nerve root tethering	11	43
		Exact P = 0.557

Table G5

Comparison of twelve months postal respondents with twelve months attenders in terms of screening data and physical data (collected at two months)

A= attenders, P = postal repondents

Variable	Group	N	Mean	SD	t value	P
Age	A	58	47.29	14.16	2.47	0.017
	P	138	41.93	13.64		
Disability	A	58	11.93	6.60	-1.34	0.183
	P	137	13.18	5.68		
Severity of present pain	A	58	6.48	2.08	0.58	0.563
	P	135	6.25	2.59		
Severity of worst attack	A	56	7.94	1.72	-2.26	0.025
	P	133	8.52	1.56		
Severity of first attack	A	55	6.18	1.72	-1.41	0.159
	P	132	6.70	2.24		
MSPQ	A	57	4.59	3.69	-1.14	0.255
	P	138	5.31	4.09		
Per cent active coping strategies	A	57	42.14	29.36	1.47	0.142
	P	130	35.36	28.72		
Weighted life events	A	58	149.59	107.24	-0.61	0.545
	P	138	160.18	113.46		
Highest externally produced pain	A	58	7.51	2.21	-0.75	0.456
	P	136	7.77	2.24		
Highest internally produced pain	A	58	6.87	2.39	-0.76	0.448
	P	134	7.14	2.11		
Highest accidentally produced pain	A	58	7.15	2.19	-1.31	0.191
	P	137	7.58	2.04		

Table G5

Comparison of twelve months postal respondents with twelve months attenders in terms of screening data and physical data (collected at two months)

A= attenders, P = postal repondents

Variable	Group	N	Mean	SD	t value	P
Age	A	58	47.29	14.16	2.47	0.017
	P	138	41.93	13.64		
Disability	A	58	11.93	6.60	-1.34	0.183
	P	137	13.18	5.68		
Severity of present pain	A	58	6.48	2.08	0.58	0.563
	P	135	6.25	2.59		
Severity of worst attack	A	56	7.94	1.72	-2.26	0.025
	P	133	8.52	1.56		
Severity of first attack	A	55	6.18	1.72	-1.41	0.159
	P	132	6.70	2.24		
MSPQ	A	57	4.59	3.69	-1.14	0.255
	P	138	5.31	4.09		
Per cent active coping strategies	A	57	42.14	29.36	1.47	0.142
	P	130	35.36	28.72		
Weighted life events	A	58	149.59	107.24	-0.61	0.545
	P	138	160.18	113.46		
Highest externally produced pain	A	58	7.51	2.21	-0.75	0.456
	P	136	7.77	2.24		
Highest internally produced pain	A	58	6.87	2.39	-0.76	0.448
	P	134	7.14	2.11		
Highest accidentally produced pain	A	58	7.15	2.19	-1.31	0.191
	P	137	7.58	2.04		

Table G5 (cont)

A= attenders, P = postal respondents

Variable	Group	N	Mean	SD	t value	P
Zung	A	55	17.20	10.46	0.07	0.943
	P	65	17.06	11.00		
Pain drawing	A	55	6.66	6.50	-0.12	0.902
	P	66	6.84	8.89		
Oswestry	A	55	14.45	13.99	-1.17	0.244
	P	67	17.68	16.09		
Inappropriate signs and symptoms	A	55	16.50	2.48	-0.05	0.962
	P	66	16.53	2.33		
Mean straight leg raise	A	55	63.90	13.41	0.35	0.726
	P	66	62.90	17.36		
Sagittal	A	55	52.43	11.49	-4.10	0.001
	P	66	62.37	15.10		
" knee bend	A	55	132.10	19.21	0.19	0.852
	P	66	131.31	15.10		
BMI	A	55	25.75	3.48	0.53	0.597
	P	64	25.40	3.65		
Side flexion	A	55	33.05	9.72	-0.91	0.364
	P	66	34.56	8.45		

Table G6

Comparison of twelve months postal respondents with twelve months attenders in terms of screening data and physical data (collected at two months) (nominal variables)

	Attenders	Postal	
Site			
Maghull HC	38	47	
Others	20	91	
			Exact P = 0.001
Smoking			
Non-smokers	41	90	
Smokers	17	48	
			Exact P = 0.509
Sex			
Males	33	66	
Females	25	72	
			Exact P = 0.275
Social group			
Social groups I to III	26	56	
Social groups IV and V	20	52	
Others	12	30	
			Exact P = 0.881

Table G6 (cont)

History	Attenders	Postal
No previous history of LBP	11	21
History of LBP	47	117
		Exact P = 0.674
Time since onset of first attack		
No previous	11	21
1-5 years	17	49
More than 5 years	30	68
		Exact P = 0.658
Number of previous attacks		
No previous	11	21
1-9 attacks	26	68
More than 9	21	4
		Exact P = 0.001
Insidious onset	36	87
Accidental onset	19	51
		Exact P = 0.868
Marital status		
Married	49	94
Not married	8	40
		Exact P = 0.028

Table G6 (cont)

	Attenders	Postal
Neurological deficit		
No neurological deficit	45	51
Neurological deficit	10	15
		Exact P = 0.653
Situp		
Can sit up	26	20
Can not sit up	29	45
		Exact P = 0.089
Nerve root tethering		
No nerve root tethering	38	40
Nerve root tethering	17	26
		Exact P = 0.348

The University of Liverpool

DEPARTMENT OF ORTHOPAEDIC & ACCIDENT SURGERY
DEPARTMENT OF GENERAL PRACTICE
SUB-DEPARTMENT OF CLINICAL PSYCHOLOGY

Please reply to: Sub-Department of Clinical Psychology, New Medical School, Ashton Street, Liverpool, L69 3BX.
Tel: 051 794 5535

APPENDIX H

2nd July 1990

Dear

BACK PAIN RESEARCH - UNIVERSITY OF LIVERPOOL

We are planning to hold an informal evening to meet doctors from the practices involved in the above study and most warmly invite you to attend. On this occasion we will present a report on our progress to date and discuss our plans for the next two years.

The date will be 18th September 1990, 7.30pm for 8.00 pm to be held in the University Department of Orthopaedic & Accident Surgery, Royal Liverpool Hospital. A buffet and wine will be provided.

We hope you are able to attend and will write to you again nearer the time.

In the meantime, may we take this opportunity to thank you and your staff for your continued support with the project.

With best wishes,

Yours sincerely,

Carol Foreman
on behalf of the
RESEARCH TEAM

FROM THE DEPARTMENT OF GENERAL PRACTICE

HEAD OF DEPARTMENT: PROFESSOR I.M. STANLEY

THE WHELAN BUILDING
P.O BOX 147 LIVERPOOL L69 3BX

TEL NOS. DIRECT: 051-794 -
051-794-5597/98
UNIVERSITY: 051-794-2000
TELEX NO: 627095 UNILPL G
FAX: 051-708-6502
DEPT. FAX: 051-794-5604

The University of Liverpool

Dr M Smith
Princes Park Health Centre
Bentley Road
Liverpool L8 OSY

Dear Dr Smith

Low Back Pain Project

As you are aware, I have been involved with the University of Liverpool Low Back Pain Project for the last eighteen months.

The aims of this project are to develop a screening instrument which is capable of predicting outcome of an acute attack of low back pain and to establish and evaluate a pain management programme directed at patients with chronic low back pain.

The first part of the project is well under way, the second part is about to commence.

As our researchers, Mike Rose and James Reilly, have explained, we still need to see all the new or first ever episodes of acute low back pain which present to you as well as the long term, chronic low back pain patients you may have on your lists. I believe the machinery for this to happen is now well established within your practice.

I would like to express my gratitude and that of the team for your cooperation so far in providing the subjects for the project. However, there has been a steady fall-off in recruitment over the last few months and we are now in danger of failing to fulfil our obligation to the body who funded the project.

This is particularly unfortunate as the researchers are almost at the point where a useful predictive instrument could be developed to help in the management of acute low back pain.

I understand the difficulties you are facing in the current climate and once again express my gratitude for the help you have given us so far. However, I am sure you would agree that it would be sad if a project which addresses such a common and difficult clinical problem such as low back pain should fail at the last hurdle for want of sufficient subjects.

May I therefore ask you to try to increase the referral rate of new acute or long standing chronic low back pain patients to the project at least until the summer of this year.

Yours sincerely

Ian Stanley

The Statistical Analysis of the Intra-observer Repeatability of Four Clinical Measurement Techniques

M J ROSE MCSP
Research Physiotherapist, Department of Clinical Psychology, Liverpool University

Key words: Clinical measurement, repeatability, statistical tests, lumbar spine.

Summary: Four clinical measurements, namely the extent of straight leg raise, prone knee bend, lateral lumbar flexion, and lumbar saggital mobility, were assessed for intra-observer repeatability on 18 physiotherapy students. Statistical tests used, and compared, were Pearson's correlation coefficient, and least significant difference. Test retest correlations were high for most measurement techniques. However, using the more appropriate statistical method, least significant difference, the actual repeatability of the tests was found to be low.

The results suggest that little confidence can be placed in these measurement techniques (as carried out in this study), when used to record changes in mobility

Biography: Mr Rose qualified at Leeds School of Physiotherapy in 1986. He worked at Burnley General Hospital, Fazakerly District General Hospital, and for St Helens and Knowlsey Health Authority before taking up his present post as research physiotherapist in Liverpool, and working part-time in private practice His main clinical interest is in orthopaedic out-patients.

Introduction

THE purpose of this study was to estimate the intra-observer repeatability of four clinical measures that are to be used to examine patients in a large, prospective study of various aspects of low back pain*.

The opportunity was taken to explore the clinical performance of the tests, and compare and contrast two statistical techniques which have been described by other workers (Bland and Altman, 1986; Burton, 1986; Tillotson and Burton, 1989) for assessing the repeatability of clinical measurement methods.

The results were expected to show that common clinical measurement techniques were highly repeatable and therefore valuable tools for assessing the physical impairment of patients with low back pain; and that least significant difference, a statistical technique for assessing the repeatability of matched pairs of readings, is more useful than the more commonly used technique, Pearson's correlation technique, in assessing the repeatability of clinical measurement techniques.

The statistical tests involved are:

Pearson's correlation coefficient (r): This test produces a number between −1 and 1 which represents a measure of association between two sets of scores. The nearer to 1 or −1 the correlation coefficient is, the closer the positive or negative association between the sets of scores: +0.9 indicates strong positive association and −0.9 indicates strong negative association; ±0.1 indicates a weak association.

Least significant difference (LSD) (Bland and Altman, 1986): The results of this test are in the units of clinical measurement and provide an estimate of the difference between matched pairs of readings which is sufficient to conclude that the values dif.. :r at the 5% significance level. The higher the LSD the lower the repeatability of the measurement. As LSD is expressed in the units of clinical measurement it is particularly useful to clinicians. If the LSD for a particular clinical measurement is known, it is possible to decide immediately whether the alteration in value of a clinical measurement is due to a change in mobility of a patient or merely to the lack of sensitivity of the clinical measurement used.

Three of the clinical measures used for this analysis are in routine use in orthopaedic and physiotherapy departments — straight leg raising, prone knee bending, and lateral lumbar flexion. The other is saggital mobility of the lumbar spine measured using a flexible rule, a test developed by workers in the field of low back pain and subjected to one or more of the statistical tests used in this study (Burton, 1986; Tillotson and Burton, 1989).

Measurement Techniques

Lateral Lumbar Flexion

This test was carried out with the subjects standing upright with feet together. Marks were drawn level with the end of the subjects' middle fingers on the lateral aspects of both thighs. The subjects were asked to slide their left hand as far down their left leg as they could, without flexing or extending their lumbar spine, or lifting their foot off the floor. Another mark was made and the distance between the two marks measured with a tape measure and recorded. This was then repeated on the right.

Straight Leg Raising

The subjects' leg was held straight at the knee, and a hydrogoniometer (Medesign, UK) placed at the mid length of the shaft of the tibia (the hydrogoniometer was zeroed when placed flat on the examination couch). The leg was then flexed at the hip until the operator felt resistance or the subjects felt tension behind the leg. A reading was made from the hydrogoniometer scale and the result recorded. This was repeated on the other leg.

Prone Knee Bending

The prone subjects' knee was passively flexed until resistance was felt by the operator or anterior thigh tension felt by the subjects. The knee angle was measured using a goniometer with arms two feet in length, one arm being parallel with the examination couch, the other along the midline of the leg. This was repeated on the other leg.

Sagittal Mobility of the Lumbar Spine

The method has been fully described by Burton (1986). Briefly, a draughtsman's flexible curve is moulded over the maximally flexed or maximally extended lumbar spine, the location of the spinous processes of the 12th thoracic,

*Prediction of chronicity and the value of active intervention in patients presenting with low back pain (ARC granted study based in the Department of Clinical Psychology, Liverpool University).

4th lumbar and 2nd sacral segments are recorded, the curve transferred to paper and a line drawn round the curve making a permanent trace. Tangents are then drawn to the curve at the level of S2, L4 and T12, and the angles between S2 and L4, and L4 and T12 are measured, giving angular values for upper and lower lumbar mobility in extension and flexion.

The only modification of the original measurement technique of Burton was that the surface markings of the spinous processes were found with the subjects positioned in prone lying, rather than seated with the lumbar spine in slight flexion.

Method

Eighteen physiotherapy students (15 female, 3 male, mean age 19.5 years, SD 4.617) underwent the tests as described in three groups over three consecutive days.

None of the subjects reported a history of low back pain.

The tester was a physiotherapist with three years post-qualification experience.

The tests were repeated three weeks later in the same order, again over three days, and at the same time of day as the first tests. The subjects had been instructed to try to reproduce their 24-hour pre-test activity level so as to avoid bias resulting from exercise-induced stiffness. However, the subjects from the second day's follow-up had taken part in an exercise class the day before.

All the measurements had 18 scores entered into the analysis except left straight leg raising which had 17 owing to a hamstring injury in one subject. The missing value was given the mean for that measurement.

Statistical analysis was via the SPSS^X Statistical Package for the Social Sciences; LSD was calculated using a pocket calculator.

Statistics

Descriptive statistics were calculated for all measures.

Pearson's correlation coefficient (r) was calculated for each pair of measurements.

The least significant difference (LSD) was calculated using the following formula: LSD = t * SD where t is derived from two-tailed t-test tables at, in this case, the 5% significance level with degrees of freedom equal to number of subjects minus one. For 18 subjects t = 2.110. SD = standard deviation of test/retest differences. LSD is expressed in the units of measurement. The value for LSD is the extent to which repeated measures must differ for this to be statistically significant; conversely, test/retest variations less than the value of LSD cannot be considered different (at the 5% significance level).

Results

Correlation was high and statistically significant (r = 0.61) for all measures except upper lumbar flexion (r = 0.13) which was not statistically significant (table 1).

LSD was high for most measures, ranging between 3.0 and 19.2 (table 1).

The results were similar for both left and right sides for straight leg raise, lateral flexion and passive prone knee bending (tables 1 and 2).

It was confirmed that a high value for LSD does not mean that a low level of correlation exists between the test/retest of that measure (Bland and Altman, 1986). A high r can conceal serious differences between repeat measures.

Table 1: Intra-observer repeatability of four clinical measurement techniques using Pearson's correlation coefficient (r), and least significant difference (LSD)

Measurement	*r(P)*	*LSD*
Lateral flexion		
Right	0.89 (0.000)	3.0 cm
Left	0.78 (0.000)	4.0 cm
Straight leg raise		
Right	0.86 (0.000)	17.4°
Left	0.83 (0.000)	18.9°
Prone knee bend		
Left	0.61 (0.004)	18.9°
Right	0.69 (0.001)	16.5°
Lumbar flexion		
Upper	0.80 (0.000)	5.2°
Lower	0.13 (0.302)	9.9°
Lumbar extension		
Upper	0.64 (0.002)	19.2°
Lower	0.69 (0.001)	11.1°

Table 2: Descriptive statistics of test (1) and retest (2) results of four clinical measurements

Measurement	*Mean*	*SD*
Lateral flexion		
Right (1)	22.94 cm	3.07
Right (2)	22.72 cm	3.06
Left (1)	22.72 cm	2.54
Left (2)	22.44 cm	3.01
Straight leg raise		
Right (1)	74.05°	16.10
Right (2)	74.44°	12.78
Left (1)	73.70°	15.87
Left (2)	72.58°	12.58
Prone knee bend		
Right (1)	141.61°	9.81
Right (2)	140.88°	10.47
Left (1)	141.33°	9.18
Left (2)	140.00°	10.38
Lumbar flexion		
Upper (1)	14.27°	3.83
Upper (2)	15.94°	3.97
Lower (1)	7.88°	3.83
Lower (2)	8.83°	3.24
Lumbar extension		
Upper (1)	28.27°	8.51
Upper (2)	29.22°	11.75
Lower (1)	15.66°	6.94
Lower (2)	16.61°	6.40

Discussion

Clinical measurements are often used to determine the severity of a patient's low back problem, and then to evaluate the results of treatment. This evaluation can take place over a period of weeks or months, or immediately after a treatment technique has been carried out. The outcome often determines the course that the treatment will take. It is therefore important that, if the patient's range of mobility apparently alters, the clinician can feel confident that any alteration is not a result of unreliable measurements.

The implication of a high value for LSD can be explained using as an example a measure used in this study. It can be seen that the correlation coefficient for straight leg raise (left) is 0.8285 (p = 000). This might suggest that repeatability is high. However, the LSD for this measurement is 18.9° (table 1). This means that if the test is used clinically the patient's straight leg raise (left) must have altered by at least 19° in one direction before it can

confidently be concluded that the patient's range of straight leg raising has altered. Any less than 19° could be a result of inaccuracy of the test method. Similar results were found for straight leg raising (right).

Lateral flexion (left and right) have correlation coefficients of 0.78 and 0.89, again high. However, their LSD values indicate that retest measurements need to alter by 4 cm and 3 cm before it can be concluded that lateral flexion has altered significantly (table 1).

There is no apparent relationship between correlation coefficients and LSD for angles of saggital mobility but it is evident that the most repeatable measurement as defined by LSD is upper lumbar flexion at 5°. The second highest LSD is that for lower lumbar flexion, yet the r value for this measure is very low — another example of how correlation coefficients can mask the true repeatability of a clinical measurement.

The decision whether a given value for LSD in respect of a particular clinical test technique is acceptable depends in part on the possible range of the clinical measurement. An LSD of 10 units for a clinical measurement of a joint which has a range of movement between 0 and 180 units suggests greater repeatability than the same LSD for a joint which has a range of movement between 0 and 40 units.

One reason for the low repeatability found here for these clinical measures may be the error introduced following the exercise class in which several of the subjects took part during the study. Nevertheless, it is interesting that such low repeatability (shown by LSD) can be demonstrated for standard clinical tests which are used to assess the efficacy of treatment techniques.

As for saggital movement of the lumbar spine, the LSDs in this study are considerably higher and the r values lower than those found by Tillotson and Burton (1989). This may be due to a practice effect where accuracy of measurement, tangent drawing, and surface marking are improved with experience. Furthermore, the surface markings of the spinous processes were found here in prone lying rather than slight flexion in sitting, as described by Burton (1986). It seems likely that strict adherence to the described clinical method of a clinical measurement technique is important if similar performance from the method is to be achieved. The subjects who took part in an exercise class may also have affected the results. The insignificant correlation coefficient for lower lumbar flexion may be due to the generally small range of movement found in this study at this level, a small test/retest difference therefore becoming amplified.

The fact that low levels of reproducibility as measured by LSD can coexist with high correlation coefficients can be explained by the fact that the two statistical techniques do not necessarily measure the same thing. The correlation coefficient measures the strength of an association between two variables. On a graph where the axes are test/retest then r will be high if the points lie close to any straight line. LSD will be low only if the points lie close to a line of equality (Bland and Altman, 1986).

Conclusion

LSD is an easily calculated measure of reproducibility which is expressed in the units of measurement, and can be used to express the meaning of a clinical test, so that the relevance of that test regarding the patient's management can be considered.

It has been shown that a high correlation coefficient does not necessarily indicate that a clinical test of the type described is an accurate tool in clinical examination.

Despite the possible errors in the present study, widely used clinical tests such as straight leg raise, prone knee bend and lateral flexion have been shown to have poor reproducibility.

It therefore appears advisable that clinicians exercise caution in their interpretation of these clinical measurements when using them to measure the level of severity or change in severity of their patients' impairment.

Further work is needed to define and develop truly useful clinical measurements and objective assessment protocols.

ACKNOWLEDGMENTS

This study was funded by the Arthritis and Rheumatism Council. The author would like to thank the students and staff of Liverpool School of Physiotherapy; Dr K Burton, visiting research Fellow, Huddersfield Polytechnic; Mr M Dewey, lecturer in statistics, Department of Psychiatry, Liverpool University; and Professor P Slade, head of department of clinical psychology, Liverpool University.

REFERENCES

Bland, M J and Altman, D G (1986). 'Statistical methods for assessing agreement between two methods of clinical measurement', *Lancet*, February 8, 307–310.

Burton, A K (1986). 'Regional lumbar saggital mobility: Measurement by flexicurves', *Clinical Biomechanics*, **1**, 20–26.

Ohlen, G, Spangfort, E and Tingvall, C (1989). 'Measurement of spinal saggital configuration and mobility with Debrunner's kyphometer', *Spine*, **14**, 6, 580–583.

Tillotson, K M and Burton, A K (1991). 'Non-invasive measurement of lumbar saggital mobility: An assessment of the Flexicurve technique', *Spine* (in press).

Behav Res Ther Vol 30, No 4 pp 359 365, 1992
Printed in Great Britain

0005-7967/92 $5.00 + 0.00

AN APPLICATION OF THE FEAR AVOIDANCE MODEL TO THREE CHRONIC PAIN PROBLEMS

MICHAEL J ROSE,* LESLIE KLENERMAN,[2] LESLEY ATCHISON[1] and PETER D. SLADE[1]

Department of Clinical Psychology and [2]Department of Orthopaedic and Accident Surgery, University of Liverpool, P O Box 147, Liverpool L69 3BX, England

(Received 9 October 1991)

Summary The Fear Avoidance Model of Exaggerated Pain Perception was developed in an attempt to explain how, and why, some individuals develop a more substantial psychological overlay to their low back pain problem than do others

The present paper describes a study in which three chronic pain groups, consisting of Post-Herpetic neuralgia patients, Reflex Sympathetic Dystrophy patients and chronic low back pain patients were compared with three pain-free comparison groups using the Fear Avoidance Model of Exaggerated Pain Perception

The results show statistically significant differences between the chronic groups and the recovered comparison groups

These results demonstrate the usefulness of the Fear Avoidance Model as an explanation of psychological overlay in chronic pain conditions regardless of pathology

INTRODUCTION

In many cases, the presence of chronic pain can be adequately explained by the nature and severity of the underlying pathology. In others, however, this is not the case and a significant proportion of chronic pain patients exhibit a common set of behavioural patterns.

These behavioural patterns include: (1) a tendency towards reduced consistency between organic pathology and pain behaviour, (2) very high levels of self-reported pain maintained over long time periods, (3) a tendency towards persistent high levels of self-reported pain despite medical intervention, (4) withdrawal from virtually all occupational, social and family responsibilities.

The Fear Avoidance Model of Exaggerated Pain Perception

Lethem, Slade, Troup and Bentley (1983) and Slade, Troup, Lethem and Bentley (1983) proposed a model which incorporates several psychological theories in an attempt to explain why some individuals with acute back pain develop a more substantial psychological overlay than others and go on to become chronic low back pain sufferers.

The model followed on from the work of Philips (1974) and Philips and Hunter (1981) who suggested that the subjective, physiological and behavioural aspects of pain might be subject to desynchrony under a range of conditions.

Lethem *et al.* (1983) defined exaggerated pain perception as:

> "Pain experience and or pain behaviour (and or physiological responses to pain stimulation) which is out of all proportion to demonstrable organic pathology or current levels of nociceptive stimulation".

Lethem *et al.* (1983) identified four distinct courses that the natural history of an attack of low back pain can take. These are:

(1) Natural remission, in which the organic basis of the pain resolves, accompanied by a reduction of the sensory and emotional (pain behaviour) components.
(2) Progressive organic, in which the organic basis gets worse with a corresponding increase in the sensory and emotional components.

*Author for correspondence

(3) Static organic, in which the organic basis remains static yet the emotional component increases.
(4) Organic resolving, in which the organic and sensory components resolve although the emotional component continues to increase.

The latter two cases are examples of exaggerated pain perception in which sensory and emotional components are wholly 'desynchronous'.

The Fear Avoidance Model is concerned with the contrast between these desynchronous cases and natural remission.

The central concept of the model is fear of pain and consequent pain avoidance or confrontation. Both are seen as influencing the behavioural response of the individual to acute pain.

Confrontation of pain is seen as adaptive. It is associated with behaviours such as increasing the range of physical and social activities as healing occurs, thereby calibrating pain experience against the nature of the sensory-discriminative stimulus. The adaptive pain confronter maintains synchrony between pain sensation and pain behaviour.

By contrast, the non-adaptive pain avoider is considered to be motivated to avoid any fresh exposure to pain. The pain avoider will avoid pain experience and painful activities. Physically, this may lead to loss of mobility, loss of muscular strength, and weight gain. Psychologically, the consequences of avoiding physical activity include fewer opportunities for calibrating pain sensation against pain experience. This leads to desynchrony between pain sensation and pain behaviour and may lead to invalid status.

Lethem *et al* (1983) proposed a continuum between avoidance and confrontation of pain and where an individual is placed on this continuum is determined by their fear of pain.

In turn, fear of, and the tendency to avoid pain is determined by the 'psychosocial context' in which initial injury or disease takes place. Four factors have been listed which influence the psychosocial context. These are stressful life events, personal pain history, personal pain coping strategies and personality characteristics.

It has been proposed that stressful life events serve to undermine the way in which an individual copes with pain at the crucial time so that avoidance of pain is more likely than confrontation (Sternbach, 1974).

Another aspect of stressful life events concerns the Legitimization Motivation Theory (Meyers & Lyon, 1979) which proposes that chronic illness, especially if associated with pain provides an individual with a means of avoiding stressful life events.

The previous experience of severe pain may sensitize the individual to fear of pain and lead to an avoidance response whenever more pain is threatened (Lethem *et al.*, 1983).

Personal pain coping strategies will reflect a combination of personal experience, imitation and modelling of peers and conditioning by society, health professionals, family and friends.

The personality characteristics of chronic back pain patients have been the subject of much research A number of studies have shown that patients with chronic back pain tend to score higher on scales which measure hypochondriasis, hysteria and depression (Sternbach, 1974).

Although the Fear Avoidance Model was developed to explain chronicity of low back pain, the authors hypothesised that the model may be equally applicable to other chronic pain conditions.

To test this hypothesis, it was decided to compare three groups of chronic pain patients with three associated comparison groups.

METHOD

The recovered comparison groups consisted of recovered shingles patients, recovered acute low back pain patients and recovered fracture patients.

The chronic pain groups consisted of Post Herpetic Neuralgia (PHN) patients, chronic low back pain patients, and Reflex Sympathetic Dystrophy (RSD) patients. The recovered shingles patients and recovered acute low back pain patients were selected from the records of a large general practice.

The recovered fracture patients, chronic low back pain patients and RSD patients were supplied by the University of Liverpool Department of Orthopaedic and Accident Surgery. The *S*s with recovered fractures were selected at the time of their discharge.

Table 1 Description of *Ss*

	Number of males	Number of females	Total	Mean age
Recovered groups				
Recovered shingles	6	5	11	52.8 (17.7)
Recovered back pain	8	4	12	40.2 (12.7)
Recovered fractures	9	2	11	46.8 (19.0)
Total recovered	23	11	34	46.4 (17.0)
Chronic groups				
PHN	4	7	11	68.1 (13.0)
Chronic back pain	2	9	11	43.5 (13.2)
RSD	4	8	12	59.7 (10.1)
Total chronic	10	24	34	57.1 (15.5)

Values of standard deviations are given in parentheses.

The PHN patients were selected from a group of patients receiving treatment at a chronic pain management clinic attached to a general hospital.

The Fear Avoidance Model questionnaire was administered by a research assistant, in the order described below.

The Fear Avoidance Model of Exaggerated Pain Perception Questionnaire

The questionnaire consists of four sections. These measure the component parts of the Model.

Pain history

Patients were asked to rate on a linear scale between 1 and 10 (from 'no pain' to 'the worst pain imaginable') the severity of the most painful 'internal', 'external' and 'accidental' pains they have experienced in the past.

The pain history section has three sub-scales. The first sub-scale consists of eight externally produced pains. Examples of these are joint sprains, operations and injections. The second sub-scale consists of eight examples of internally produced pains such as headaches and toothache. The third sub-scale consists of 16 pains which arise as a result of a minor accident, for example banging one's thumb with a hammer or cutting one's finger with a knife.

The highest score from each of the three sections is recorded, giving three pain experience variables: highest ever external pain, highest ever internal pain, and highest ever accidental pain.

Pain coping strategies

This section was developed in order to derive a measure of the behavioural response to pain of an individual and consequently a measure of confrontation or avoidance of pain.

It consists of seven examples of painful experiences such as migraine or sore throat and *Ss* are asked what they did in response to the worst experience of each pain in the past. Four options are given. These are: took physical exercise, ignored it and carried on, rested, and took pain killers. The first and second options are considered to be 'passive' coping strategies and the third and fourth are considered to be 'active' coping strategies.

A derived measure of pain coping style is obtained by computing the overall percentage of coping strategies which are 'active'.

Personality

The personality component of the model is assessed by the use of The Modified Somatic Perception Questionnaire (MSPQ). This scale was developed to measure the autonomic and somatic perception of chronic low back pain patients.

The MSPQ has been shown to correlate with the MMPI scales for hypochondriasis, depression, hysteria, psychopathic deviation, paranoia, psychasthenia, schizophrenia and social introversion (Main, 1983).

Correlations between the MSPQ and the Sickness Impact Profile and the MSPQ and the Zung Depression Scale have also been demonstrated (Deyo, Walsh, Schoenfield & Ramamurthy, 1989).

The MSPQ consists of 13 autonomic sensations such as sweating all over, blurring of vision and feeling faint. The patient is asked to recount the frequency with which he she experienced each of the sensations in the previous week.

Table 2 Mean scores of Fear Avoidance Model of Exaggerated Pain Perception component variables for recovered backs and chronic backs

	Highest external pain	Highest internal pain	Highest accidental pain	MSPQ*	Percent active coping strategies	Life* events
Recovered	6 4 (2 7)	6 2 (2 4)	7 6 (2 1)	0 6 (2 0)	43 9 (40.2)	185.3 (128.2)
Chronic	8 2 (1 5)	8 1 (2 5)	7 2 (2 2)	10 8 (6 4)	43 2 (32.1)	56.0 (26.1)

Values of standard deviations are given in parentheses
*$P < 0.05$

Previous life events

The Weighted Life Events Questionnaire lists 43 possible stressful life events which the *S* may have experienced in the previous year.

Each life event carries a score which is weighted according to the severity of the stress induced by that event. The relative severity of different stressful events and their corresponding scores were evaluated by Holmes and Rahe (1967) in the development of their Social Readjustment Scale.

Statistical analysis

Two-tailed *t*-tests were used to compare the pain history, pain coping strategies, personality and life events of recovered and chronic pain patients who had shared the same pathology.

A two-way analysis of variance (ANOVA) was carried out on pain history, coping strategies, personality and life events data for all six groups in order to determine whether the Fear Avoidance Model explains chronic low back pain only or explains chronic pain regardless of pathology.

Discriminant function analysis was performed using pain history, coping strategies, personality and life events as independent variables. Recovery or chronicity were used as dependent variables to assess the predictive validity of the Fear Avoidance Model in relation to chronicity over a range of pathologies.

RESULTS

The discriminant function analysis was significant ($\chi^2 = 26.777$, d.f. = 6, $P = < 0.001$). Recovery or chronicity was correctly predicted in 82% of the sample. Statistically significant differences (in this case $P < 0.05$) in the scores of individual component parts of the Fear Avoidance Model were demonstrated between recovered and chronic low back pain and between recovered and chronic shingles patients (Tables 2 and 3, Figs 1 and 2). No significant differences in mean Fear Avoidance Model scores were demonstrated between fracture and RSD patients although highest ever accidental pain approached significance (Table 4 and Figs 1 and 2). A two-way ANOVA on data from the combined group revealed a significant main effect of condition (recovered or chronic) for highest externally produced pain ($F = 4.09$, d.f. = 1.63, $P < 0.05$), MSPQ ($F = 23.065$, d.f. = 1, $P < 0.001$) and weighted life events ($F = 5.385$, d.f. = 1, $P < 0.025$) (Fig. 1). No significant main effect of pathology was demonstrated.

DISCUSSION

One criticism which may be levelled at the methodology of this study involves the absence of homogeneity of the *S*s regarding age, and it may be argued that *S*s should have been matched for this variable.

Table 3 Mean scores of Fear Avoidance Model of Exaggerated Pain Perception component variables for recovered shingles and Post-Herpetic neuralgia

	Highest* external pain	Highest* internal pain	Highest accidental pain	MSPQ*	Percent active coping strategies	Life* events
Recovered	8 5 (1 2)	7 0 (1 4)	7.1 (2 2)	2 2 (2 4)	44 2 (36 6)	130 8 (115.6)
PHN	9 5 (1 0)	8 8 (1 9)	6 5 (2 5)	8 8 (6 6)	34 0 (33 6)	92.5 (87 1)

Values of standard deviations are given in parentheses
*$P < 0.05$

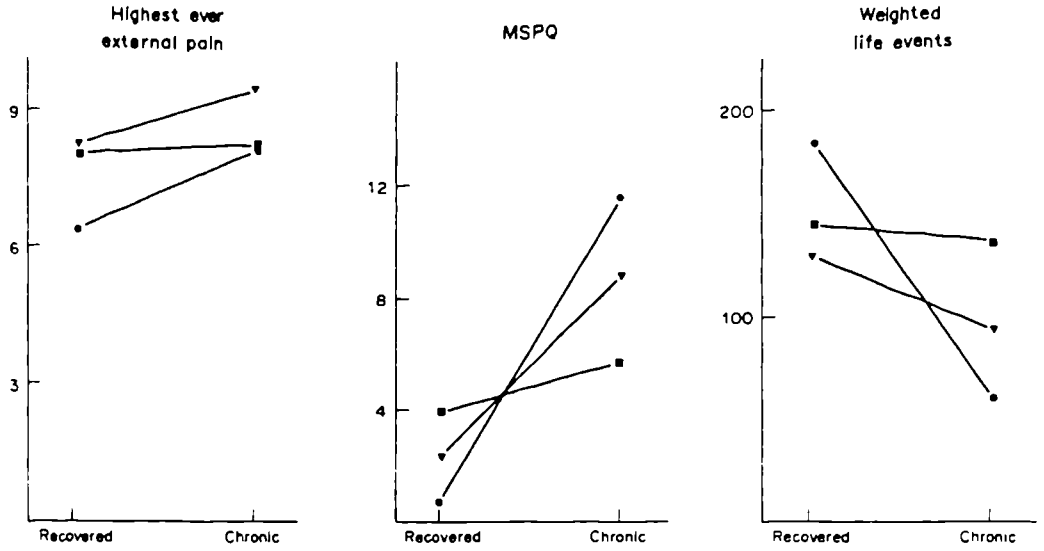


Fig 1 Mean highest ever external pain, MSPQ and weighted life events for the three recovered groups and the three chronic groups ▼, Backs, ■, fractures, ●, shingles

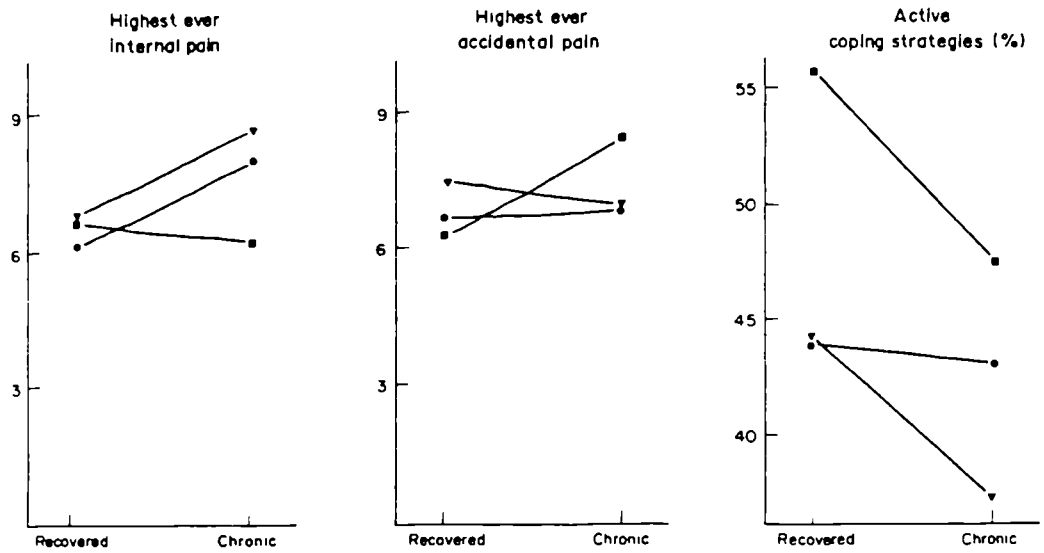


Fig 2 Mean highest ever internal pain, mean highest ever accidental pain and percent active coping strategies for the three recovered groups and the three chronic groups ▼, Backs, ■, fractures, ●, shingles.

However, the authors felt that this may have led to the collection of data from recovered and chronic pain groups who were in fact less representative of the populations from which they were derived, given the positive correlation between age and chronic pain (Bowsher, 1991). (This positive correlation is also demonstrated by the differences in the mean age of randomly selected *S*s in this study.) Age and chronicity may be inextricably inter-related and age matching may have had the effect of 'diluting' the effect of chronicity, thus undermining the inferences which have been made.

Table 4 Mean scores of Fear Avoidance Model of Exaggerated Pain Perception component variables for recovered fractures and Reflex Sympathetic Dystrophy

	Highest* external pain	Highest* internal pain	Highest accidental pain	MSPQ*	Percent active coping strategies	Life* events
Recovered	8 1 (2 2)	6 6 (2 0)	6 4 (2 7)	3 9 (4 2)	56 5 (28 0)	145 1 (100 3)
PHN	8 3 (2 5)	6 3 (2 6)	8 4 (2 8)	4 3 (1 4)	47 6 (32 5)	134 8 (88 6)

Values of standard deviations are given in parentheses
*$P < 0.05$

The authors hypothesised that the Fear Avoidance Model provides a unified psychological theory which explains the development of chronic pain resulting from a benign, acute pain experience, across a range of pathologies.

The results of this study support this hypothesis.

82% of the study's population were correctly predicted as being either chronic or recovered using the Fear Avoidance Model regardless of pathology. Variables which represent three of the four component parts of the Model (pain history, personality and incidence of stressful life events) differed significantly between recovered and chronic pain *S*s, once again, regardless of pathology.

No significant difference was demonstrated between the coping style of chronic and recovered *S*s (percent active coping strategies). This may be explained by the lack of sensitivity of the instrument used to measure this variable or by the small sample size. However, Fig. 2 demonstrates a downward trend between the percent active coping strategies of recovered pain patients and the percent active coping strategies of the chronic group.

The findings of Main (1983) in his work with the MSPQ and low back pain are supported by the results of this study. MSPQ scores were higher for each of the chronic pain groups than their recovered controls, yet not significantly different between recovered fractures and RSD. However, the difference between MSPQ scores of recovered and chronic *S*s was highly significant across pathology and was accounted for by chronicity, not pathology.

The results of this study also support the stressful life events theory of chronic pain (Meyers & Lyon, 1979) which proposes that the desire to escape from stress can be a reinforcer of chronic pain behaviour

The weighted life events score of the chronic *S*s was less than that of the recovered *S*s, chronicity being the significant main effect, not pathology (Fig. 1).

A serendipitous finding of this study suggests that chronic pain patients may be sensitized to previous pain experience which has the same nature of onset as their present pathology yet remain relatively untouched by previous pain experience with a different aetiology. Reference to Figs 1 and 2 reveals that only the highest ever externally produced pain was significantly different between chronic and recovered *S*s across pathology. However, mean accidental pain was higher for RSD patients than recovered fractures yet remained stable for shingles and backs. The mean internal pain was higher for PHN and chronic backs than their recovered controls yet remained stable for the fracture group

The findings of this study reinforce the concept of psychological phenomena which influence the onset of chronic pain regardless of physical signs and symptoms associated with specifically 'labelled' pathology

In order to expand this study, further research is necessary using a larger, longitudinal study of several pathological groups. This may lead to further support for the Fear Avoidance Model of Exaggerated Pain Perception and an expression of the serendipitous demonstration of the specificity of the effects of past experience of pain and their relationship to present chronic pain experience

A possible outcome of such a study may also be the development of an instrument which, when applied to the acute pain patient, will enable a prediction to be made regarding the course their condition will take.

Acknowledgements This study was funded by the Arthritis and Rheumatism Council. We thank Ms S. Kaney, Research Clinical Psychologist, Department of Clinical Psychology, University of Liverpool and Mr M. Dewey, Lecturer in Psychological Statistics, Department of Psychiatry, University of Liverpool for their advice and Mr J. Reilly, Research Assistant, Department of Clinical Psychology, University of Liverpool for assistance with data collection. We are also grateful to Dr D Bowsher at the Pain Research Institute, University Department of Neurological Science, Walton Hospital, Merseyside for facilitating access to his chronic pain patients.

REFERENCES

Bowsher, D (1991) Neurogenic pain syndromes and their management. *British Medical Bulletin, 47*, 644–666.

Deyo, R A, Walsh, N E, Schoenfeld, L S. & Ramamurthy, S. (1989). Studies of the Modified Somatic Perception Questionnaire (MSPQ) in patient with back pain. Psychometric and predictive studies. *Spine, 14*, 5–10.

Holmes, T H & Rahe, T H (1967). The Social Readjustment Rating Scale. *Journal of Psychosomatic Research, 11*, 213 218

Lethem, J., Slade, P. D., Troup, J. D. G. & Bentley, G. (1983) Outline of a fear-avoidance model of exaggerated pain perception—I. *Behaviour Research and Therapy*, *21*, 401–408.
Main, C. J. (1983) The modified somatic perception questionnaire (MSPQ). *Journal of Psychosomatic Research*, *27*, 503–514.
Meyers, J. W. & Lyon, H. P. (1979) Stress and distress. Proceedings of the American Occupational Medicine Association.
Philips, C. (1974) A psychological analysis of tension headache. In Rachman, S. (Ed.), *Contributions of medical psychology*. Oxford: Pergamon Press.
Philips, C. & Hunter, M. (1981) Pain behaviour in headache sufferers. *Behaviour Analysis and Modification*, *4*, 257–266.
Slade, P. D., Troup, J. D. G., Lethem, J. & Bentley, G. (1983) The fear-avoidance model of exaggerated pain perception—2, preliminary studies of coping strategies for pain. *Behaviour Research and Therapy*, *21*, 409–416.
Sternback, R. (1974) *Pain patients: Traits and treatments*. New York: Academic Press.

CHRONIC LOW BACK PAIN: A CONSEQUENCE OF MISINFORMATION?

Rose MJ [1]
Reilly JP[1]
Pennie B[2]
Slade PD[1]

[1] Department of Clinical Psychology, University of Liverpool.
[2] Department of Orthopaedics and Accident Surgery, University of Liverpool

Correspondence: Mr MJ Rose, Department of Clinical Psychology
University of Liverpool, Whelan Building
PO Box 147, Liverpool L69 3BX

Abstract

The overall effects of chronic low back pain can lead to profound psychological and physical distress for the individual and a waste of human resources for society.

In the course of their work as facilitators of a chronic low back pain management programme at the University of Liverpool, the authors have identified inappropriate belief systems and dysfunctional behaviour which act as powerful inhibitors to rehabilitation.

This paper is concerned with the proposed genesis of these factors and describes how they may be responsible for the development of chronic low back pain.

Key words

Chronic low back pain

Psychology

Counselling

Misinformation

Biography

Michael Rose is a research physiotherapist and co-therapist on the University of Liverpool pain management programme.

James Reilly is research psychologist and co-therapist on the University of Liverpool pain management programme.

Peter Slade is Professor of clinical psychology at the University of Liverpool.

Bruce Pennie is a consultant orthopaedic surgeon at Aintree Hospitals Trust, Liverpool.

Although low back pain is a common symptom it is, In the main, self limiting. It is thought that 90% of individuals with an acute attack of low back pain recover within 8 to 10 weeks with conventional management which usually involves advice to rest and analgesic or antiinflammatory medication [1].

However, a small but significant proportion of patients fail to recover and are given the label "chronic low back pain" patients. Traditionally, pain which has lasted for 6 months or more has been labelled chronic but more recently the definition has been modified to include pain of more than eight weeks duration [2]).

Patients who fail to recover enter the therapeutic milieu. Typically, such individuals will be referred by their GP to a physiotherapy department or to an orthopaedic or rheumatology department and will have undergone a battery of non invasive and invasive tests. Many will have undergone one or more surgical procedures. Some patients may also resort to the private sector and seek relief of symptoms from practitioners ranging from osteopaths to faith healers. Most patients will be encouraged to persist in the consumption of large amounts of potentially toxic medication throughout what has euphemistically been called their 'pain career.

The psychosocial consequences of the experience of chronic pain and the unsuccessful search for relief are often profound. Withdrawal from family, employer and society in general is common [3]) as is depression In the experience of the authors suicidal thoughts are often alluded to and attempted suicide is not uncommon. In addition to the consequences to sufferers, the costs to industry and the exchequer are considerable [4].

The overall effects of unremitting low back pain lead to physical and emotional distress for the individual and a waste of human resources for society.

It has long been recognised that psychological variables can be powerful mediators in the process which transforms nociception into pain experience. The gate control theory of pain [5] explains how psychological factors can influence the perception of pain and consequent behaviour. In recent years the multi-modal pain management programme approach to the rehabilitation of chronic low back pain patients has been developed. These programmes usually consist of educational elements, often in the

form of anatomy and physiology tuition, target setting, challenging of dysfunctional cognitions and additional elements concerned with the modification of behaviour, often involving structured exercise programmes [6].

The purpose of these programmes is not curative. Rather, they are designed to allow individuals to maximise their potential, given the restraints of their physical pathology. The approach is based in part upon the belief that fear of pain is a central and key element to dysfunctional behaviour and a reduction of inappropriate fear of pain will lead to rehabilitation. A model of chronic low back pain, which has as its key element fear of pain, has been proposed by Lethem et al [7].

In the course of their work as facilitators of a multi-modal low back pain management programme, the authors have become aware of several psychological factors which, when combined, act as powerful inhibitors of the sufferers desire for, or belief in the possibility of, rehabilitation. It is the recognition of these inhibitory factors which forms the theoretical basis of the pain management programme at the University of Liverpool and the content of the programme is aimed at enabling clients to recognise for themselves the powerful effect of these variables on pain experience. The authors believe that this recognition allows clients to attain their emotional and functional potential within the context of their physical pathology .

This paper is concerned with a description of the proposed genesis of these psychological factors which consist of inappropriate belief systems and dysfunctional behaviours which the authors believe are, in large measure, responsible for the suffering associated with chronic low back pain.

Inappropriate belief systems

Of major importance to all clients is the search for permanent and complete pain relief. A large number of individuals spend many years and considerable sums of money in pursuit of this unattainable goal, never letting go of the notion that it is within the power of the health services to fulfil the curative role assigned to it by the expectations of the public and inexperienced health professionals. This expectation by chronic back pain sufferers of a cure is damaging in several respects. First, the repeated failure of one health

professional after another to relieve symptoms leads to increasing levels of disappointment, frustration and ultimately desperation which may in themselves constitute profound psychological distress. Second, repeated failure to cure symptoms, in the context of the belief of the client that benign back pain is curable, often leads to the mistaken view that pain is a symptom of some serious condition, usually cancer. Not unsurprisingly, such individuals also experience psychological distress. Why many individuals who attend the pain management programme have never been told that their condition is unlikely to respond to medical intervention is open to interpretation and may involve issues such as difficulties faced by clinicians in giving bad news and the feelings of failure engendered by 'patients' who fail to improve. Nevertheless, the authors have noted that the giving of permission to clients to get off the therapeutic merry-go-round results in the expression of two kinds of feeling. The first is relief that the search for the Holy Grail of pain relief is now over and the second is anger that so much emotional energy has been allowed to have been wasted on such a search. The acceptance by the client that further medical intervention is pointless and will not be offered is considered by the authors to be of primary importance and a necessary prerequisite to rehabilitation. This is reflected in the reluctance of the authors to accept in to the programme clients who have not been discharged from the care of the referring clinician.

Fear of the implications of spinal pain, and the associated unwillingness to remain functionally active is as much a result of misinformation concerning the nature of the underlying pathology which causes the pain than an aversion to the pain itself. The sources of this misinformation are numerous and range from various kinds of health professional to family members and friends. One particular case which the authors often use as an example of the effect of misinformation is a young, fit and healthy woman who had been told that she could think of herself as being like "a good apple with a rotten core". This information had had a profound effect upon the woman's life and her willingness to remain physically active. She had given up her role as a wife, mother and employee in order to escape the consequences of irreparably damaging her 'rotten core', which she felt would lead to a life of paralysis in a wheelchair. Although this may be an extreme example, most clients have been given explanatory models which usually involve such adjectives as 'crumbling' or 'degenerative'. The result of the use of these models is inevitably a reduction in physical activity which in turn leads to a reduction in the strength of spinal and other muscle groups and local spinal and generalised joint stiffness. The consequences of this reduced physical ability lead to an increased vulnerability of the spine to re-injury and pain as a result of joint stiffness. This 'stiffness pain' can

be misinterpreted by clients as pain of spinal origin which, given their beliefs concerning their pathology leads to an even greater reduction in activity. The effects of inappropriate beliefs concerning pathology are compounded by the widespread , but generally unjustified use of rest as the mainstay of therapy. Although bed rest may be appropriate in the management of acute low back pain , it has been demonstrated to have an adverse effect if continued for longer than two days [8]. The advice given by many health professionals to chronic low back pain patients is to rest when they feel discomfort and to 'be guided by their pain'. In the view of the authors this advice reinforces the belief of clients that they are suffering from a serious pathology . This in turn leads to a reduction in physical activity and confrontation of pain which has been described by Lethem et al [7] as 'avoidance behaviour'.

Dysfunctional behaviours

In this context, behaviour consists of abnormal illness behaviour and avoidance behaviour. Abnormal illness behaviour has been defined as a 'persistence of inappropriate or maladaptive mode of experiencing, perceiving, evaluating and acting in relation to one's own state of health....despite accurate reassurance after thorough examination [9]. If illness behaviour is viewed as a means of communicating the distress which results from pain experience, inappropriate illness behaviour can be interpreted as communication of distress which is disproportionate to the true nature of underlying pathology. An example of inappropriate illness behaviour in the context of chronic low back pain is the use of and dependence on walking sticks, crutches and spinal corsets. The use of aids such as these has physiological and psychological consequences which, the authors believe, contribute in large measure to the maintenance and worsening of disability. Physically, 'normal' movement is prevented by the wearing of corsets and the reliance on crutches and can, theoretically, increase muscle weakness and spinal stiffness. General fitness may be reduced and the spine may become more vulnerable to injury. Re-injury or an increase in pain due to stiffness will reinforce the use of such aids and the process will continue. Psychologically, the use of such aids serves to remind the wearer that normal movement is impossible in their disabled condition. The use of such aids is constantly reinforced by the attention focused upon the wearer by others. The use of a walking stick changes the standing of the individual in society to the extent that doors will be held open, objects will be picked up and, perhaps most importantly, a measure of sympathy never previously experienced will be freely given. Corsets and walking aids serve to reduce the

physical ability of the chronic back pain sufferer and act as constant reminders that the users of these aids are severely disabled and unable to compete with others in society on equal terms.

Other forms of illness behaviour witnessed by the authors include facial expressions, descriptions of the nature and severity of pain, gasping, crying out in pain, limping, falling over due to the 'severity of the pain', rubbing and so on. All these behaviours can be reinforced by the environment to the extent that they become habitual.

The issue of financial reinforcement of inappropriate illness behaviour is complex. In the opinion of the authors, the uncharitable view held by some that illness behaviour is is an overtly manipulative attempt at gaining maximum financial reward from employer or exchequer is not supported by clinical observation. However, it has become apparent that the 'system' which dictates that chronic low back pain patients remain inactive in order to receive either sick pay or disability allowances is wasteful in terms of human resource. In addition, as described previously, inactivity begets inactivity and the physical and psychological consequences of enforced unemployment are significant contributory factors in the distress associated with chronic low back pain. Involvement in the tortuous processes associated with negotiating compensation or disability allowance forces the individual with low back pain to maximise their symptoms, be prepared to enact the role required of them at all times and ultimately risk a change in self-concept in which they come to see themselves as disabled. This view is supported by the evidence which suggests that low back pain litigants fail to recover to the same extent as non-litigants, even after payment of compensation has been made [10].

The component parts of inappropriate belief systems and inappropriate behaviours do not act in isolation. Rather, the system is dynamic. Beliefs (cognitions), behaviour and emotions (affect) are related and form a model which, in part, serves to explain chronic low back pain experience and disability (Fig 1). In addition, the authors recognise the limitations imposed upon the potential levels of rehabilitation by physical pathology and accept that many clients are unlikely to resume their previous employment. However, clinical experience has demonstrated that few clients are fulfilling their functional or psychological potential and it is a reduction in the gap between this potential and existing levels of activity which is the aim of the programme.

Figure 1 about here please

It is important to note that this description of the role of psychological and sociological aspects of low back pain experience is not exhaustive but merely represents the key elements which are addressed by the authors in an attempt to counsel and rehabilitate clients on the University of Liverpool low back pain management programme.

It is apparent that the seeds of psychological and physical dysfunction are sown at an early stage in the development of chronic low back pain. It is a firmly held belief of the authors that appropriate advice and counselling at this stage, maybe in the workplace, and an alteration in the way in which society manages litigation and disability benefits issues could either prevent or significantly reduce the suffering involved in chronic low back pain experience.

References

1 Waddell G (1987) A new clinical model for the treatment of low back pain Spine 12, 7

2 Philips HC and Grant L (1991) Acute low back pain: a psychological analysis Beh. Res. Ther. 29,5

3 Slade PD (1984) Theories of chronic pain phenomena. In: "Current issues in clinical psychology" Vol.2 Karas E (Ed). Plenum Publications

4 Department of Health and Social Security (1989). Report on low back pain. DHSS, London

5 Melzack R and Wall PD (1965) Pain mechanisms: a new theory. Science 150, 971-9

6 Main CJ and Parker H (1989) Pain management programmes. In: Roland M and Jenner J (Eds), Back pain: New Approaches to Rehabilitation and Education, Manchester University Press, Manchester

7 Lethem J, Slade PD, Troup JDG, Bentley G. (1983) Outline of a fear-avoidance model of exaggerated pain perception - 1. Beh. Res. Ther. 21, 4 401- 408

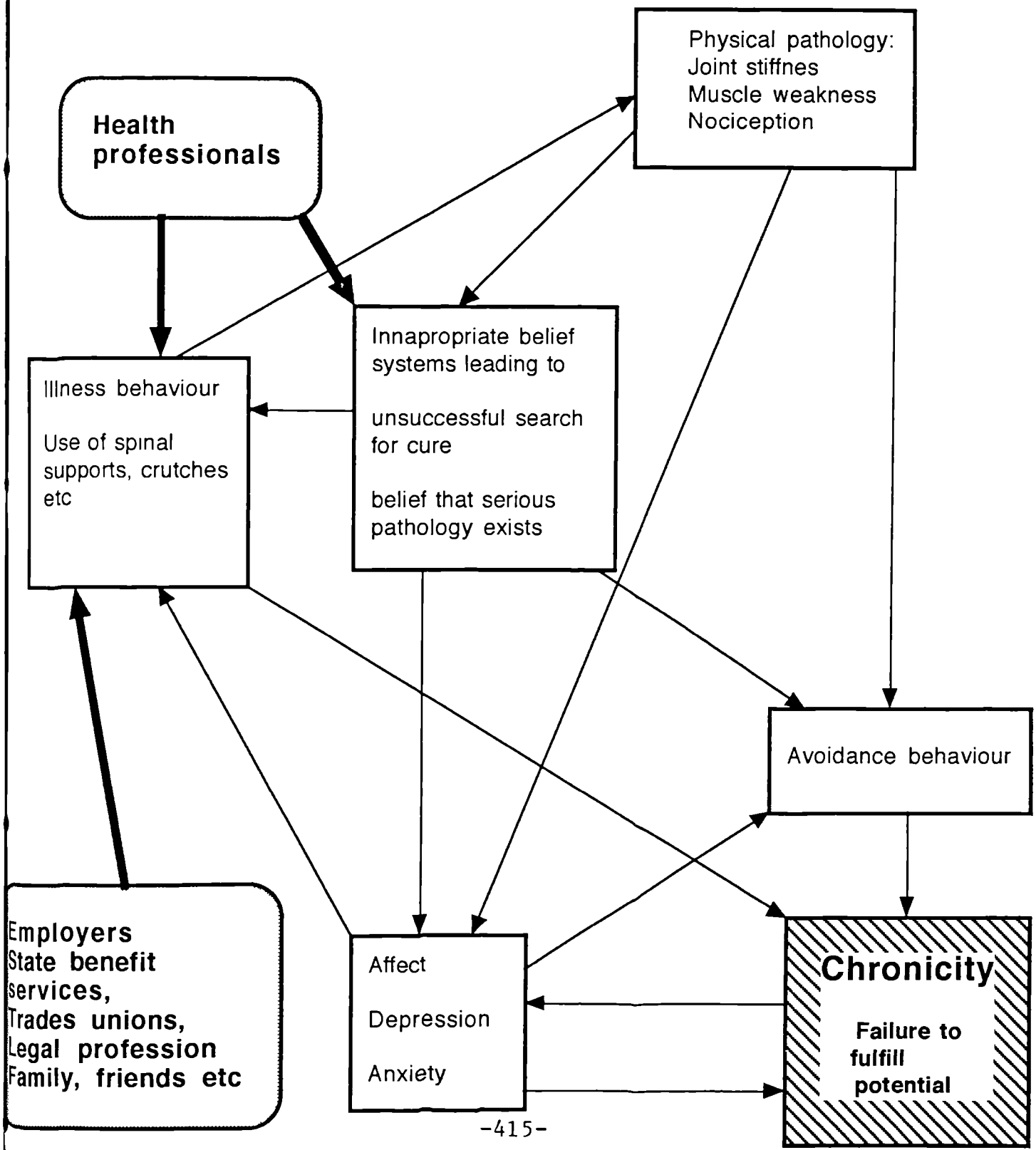
8 Deyo RA, Diehl AK, Rosenthal M (1986) How many days of bed rest for acute low back pain? New England J. Med.

9 Pilowsky I (1969) Abnormal illness behaviour Br J. Med. Psychol. 347-351

10 Greenough CG, Fraser RD (1989) The effects of compensation on recovery from low back pain Spine 14 9

Figure 1

The relationship between inappropriate belief systems and dysfunctional behaviour in the development and maintenance of chronic low back pain



THE PREDICTION OF CHRONICITY IN PATIENTS PRESENTING WITH AN ACUTE ATTACK OF LOW BACK PAIN IN A GENERAL PRACTICE SETTING.

L. KLENERMAN, ChM (1) P.D. SLADE, PhD (2) I.M. STANLEY, FRCGP (3)
B. PENNIE, MCH (Orth) (4) J.P. REILLY, PhD (5) L.E. ATCHISON, MSc (6)
J.D.G. TROUP,DSc (Med) (7) M.J. ROSE, PhD (8)

(1) Professor of Orthopaedic and Accident Surgery,
The University of Liverpool, England.

(2) Professor of Clinical Psychology,
The University of Liverpool, England.

(3) Professor of General Practice,
The University of Liverpool, England.

(4) Consultant Orthopaedic Surgeon,
Aintree Hospitals, Liverpool,England.

(5) Research Psychologist,
The University of Liverpool, England.

(6) Research Psychologist,
The University of Liverpool, England.

(7) Honorary Senior Research Fellow, Department of Orthopaedic Surgery,
The University of Aberdeen, Scotland.

(8) Back Pain Rehabilitation Programme Co-ordinator,
Wirral Hospital Trust, Wirral, England

CORRESPONDENCE TO:
Professor Peter Slade,
Department of Clinical Psychology,
The University of Liverpool,
Whelan Building,
P.O.Box 147,
Liverpool L69 3BX.
U.K.

ACKNOWLEDGEMENT:
This work was supported by a generous grant from the Arthritis and Rheumatism Council, UK, to four of the authors.

THE PREDICTION OF CHRONICITY IN PATIENTS PRESENTING WITH AN ACUTE ATTACK OF LOW BACK PAIN IN A GENERAL PRACTICE SETTING.

STRUCTURED ABSTRACT

Study Design

Three hundred patients, attending their general practitioners with attacks of acute low back pain (LBP), formed the subject population for a study of fear avoidance and other variables in the prediction of chronicity. Follow-up was at two and twelve months.

Objective

The hypothesis to be tested was that evidence of psychological morbidity , particularly fear-avoidance behaviour, would be manifest from the outset of the presenting attack in susceptible subjects.

Summary of Background

While back pain is an almost universal human experience, only about five percent of sufferers seek medical advice. Most of these respond to conservative treatment. However, approximately ten percent of those who experience an acute attack of low back pain go on to become chronic sufferers.

Data/Methods

Psychosocial and physiological data (including fear-avoidance measures) were collected from a sample of 300 acute low back pain patients within one week of presentation and at two months, in order to try to predict 12 month outcome

Results

Data analysis showed that subjects who had not recovered by two months were those who went on to become chronic LBP patients (7.3%). Using multiple regression analyses, fear-avoidance variables were the most successful in predicting outcome. Using multiple discriminant function analyses , the results suggest that the outcome in terms of the future course of LBP can be correctly classified in 66% from fear-avoidance variables alone and in 88% of patients from all variables.

Conclusions

The results suggest that, at the earliest stage of LBP, *fear of pain* should be identified by clinicians and, where this is severe, pain confrontation should arguably form part of the approach to treatment.

KEY WORDS Low back pain; Chronicity; Fear-avoidance behaviour; Prediction of outcome; Psychosocial screening

THE PREDICTION OF CHRONICITY IN PATIENTS PRESENTING WITH AN ACUTE ATTACK OF LOW BACK PAIN IN A GENERAL PRACTICE SETTING.

MINI ABSTRACT

Three hundred patients presenting with attacks of acute low back pain to general practitioners were administered a psychosocial screening instrument and followed up at two and twelve months. Subjects who had not recovered by two months went on to become chronic sufferers and were best identified by fear-avoidance measures.

PREDICTION OF CHRONICITY IN LOW BACK PAIN

INTRODUCTION

While back pain is an almost universal human experience, particularly with advancing age, Roland [13] suggests that only about five percent of sufferers seek medical advice . Nevertheless, as a presenting complaint, back pain is a common problem in the workload of general practitioners in the UK. The Third National Morbidity Survey[217] reported a total consultation rate across age groups of 31.1 per thousand patients per annum. Other studies have suggested rates between 24.2 and 42.9 [3,14]. While some of this variability in reported incidence may be accounted for by differing rates and patterns of employment in the populations studied, other factors such as the level of undetected psychological problems are likely to be involved[5].

Over the last 50 years, explanatory models of back pain adopted by the medical profession have exhibited swings in fashion between the mechanical/postural and the behavioural/psychological. Thus Cyriax[2] suggested that 90 percent of symptoms could be explained on the basis of disc pathology; Connolly and Jardon[1] report the related and striking finding that in the USA 60,000 discs are removed surgically every year. Others[6,11,24,25] have reported increased levels of

psychological distress such as depression and anxiety among back pain sufferers, reports that are consistent with a multifactorial aetiology for LBP[19] in which psychological factors are seen to play a significant role[21]. The question then arises, are psychological factors secondary to the experience of LBP or may they be operative from its inception?.

Basing their conclusions on one of the few studies of the natural history of back pain conducted in a general practice setting, Roland and Morris[3] are unequivocal in their conclusions : " psychological factors are not of great importance in the majority of new presentations of back pain in general practice - the increased incidence of psychological abnormalities found in patients attending hospital is a result of long-standing pain". Thus, Roland and Morris suggest that psychological morbidity is a consequence of back pain rather than a contributory factor to the development of the condition . Clearly there is a need for a longitudinal survey of a cohort of new back pain sufferers to elucidate this issue. This is what the authors set out to accomplish in the present study.

Ten years ago Lethem et al [8], Slade et al[18] and Troup and Slade[20] proposed a Fear-avoidance Model of Exaggerated Pain Perception. This theoretical model was developed in the context of an interdisciplinary back pain clinic and attempted

to explain why the majority of individuals who experience an acute episode of LBP recover spontaneously while a small minority go on to become chronic LBP sufferers . As it underpins the present investigation, we shall now briefly describe this model.

THE FEAR-AVOIDANCE MODEL OF CHRONIC PAIN (8,18 and 20)

The basis of the Fear-Avoidance Model (FAM) is that there are individual differences in response to a painful experience which have their primary manifestation in terms of a continuum of fear of pain. At the low end of this continuum individuals experience severe discomfort but only minimal fear of pain : they therefore remain motivated and able to confront their pain and gradually increase their exposure to painful activities and to the experience of pain itself. At the high end of the continuum individuals experience, in addition to severe discomfort, a strong fear of pain : the latter leads them to avoid painful activities and indeed the experience of pain itself. It is further proposed that 'confronters' calibrate their pain realistically and thereby succeed in rehabilitating themselves once the organic basis for their pain recedes; while 'avoiders' become progressively less able to consider their pain objectively and end up as chronic low back invalids. The model also suggests that there are somatic consequences (i.e. loss of mobility and muscular strength, weight gain, etc.) which follow directly from

the avoidance of physical and other activities. It is suggested that these consequences operate to reinforce avoidance behaviour and the sick role.

Four "Psychosocial Factors" are hypothesised to determine whether or not an individual person is likely to be a 'confronter' or an 'avoider' following an acute attack of LBP. These are as follows :

Stressful Life Events - people who experience stressful life events prior to the experience of back pain are hypothesised to be more likely to become 'avoiders' and therefore to become chronic low back pain sufferers.

Personality - people who exhibit a particular personality profile involving concern with bodily/physical symptoms are likely to become 'avoiders'.

Previous Pain History - people who have had a history of very severe pain experience previously are more likely to opt for 'avoidance' than their counterparts.

Normal Pain Coping Strategies - individuals who normally respond to pain in a passive manner are more likely to exhibit 'avoidance' following an acute attack of low back pain.

Recent studies by Rose et al[15] and Waddell et al[23] have established general support for the validity of the fear-avoidance concept. However, it should be noted that the first of these studies was cross-sectional in nature, using direct comparisons between groups of acute and chronic pain patients: while the latter was carried out on a population of patients attending an orthopaedic clinic, with an average LBP history of 7 years and an average duration of 14 months for the present attack. In contrast, our study was concerned to identify and follow - up a group of patients presenting with an acute attack of LBP in order to determine the natural history of the condition and to predict their outcome at two months and twelve months.

Literature review identifies other factors which have also been used to explain benign chronic LBP: these consist of 'physical', 'demographic' and 'historical' variables. In the present study the FAM was used in conjunction with, and in comparison to, physical, demographic and historical variables as the basis for an attempt to predict who would develop a chronic low back condition at an earlier stage in its natural history.

METHOD

Subjects

Three hundred patients (151 males and 149 females) suffering from a first or new episode of acute low back pain were recruited into the study. Two essential inclusion criteria were used : first, that in the opinion of the general practitioner, the patient was suffering from 'benign, musculosketal low back pain'; and, second that the patients' pain had begun not more than one week before presentation to their general practitioner.

The general practices were selected in such a way as to provide a catchment sample similar to the population of the Merseyside Region as a whole. The general practices were approached by one of the authors (I.S.) and asked if they would be willing to cooperate in the study. If they agreed, our research team met the GP's concerned to discuss the rationale and the procedure of the investigation. In each participating practice an administrator was identified to take responsibility for communicating with the research team; this was usually the practice manager.

When a patient who fulfilled the inclusion criteria was identified by a GP, he or she gave the patient a letter which explained the aims and method of the project. If the patient then agreed to participate in the study they were given an appointment to

see a research worker on the practice premises within one week. If the patient was house-bound because of pain an appointment was made for a home visit by a research worker.

Assessment Methods

Subjects were assessed at the acute stage, 2 months follow-up and 12 months follow-up.

Acute Stage (see table 1).

Subjects were asked to complete a screening questionnaire which combined information on demographic characteristics (age, sex, referring doctor, marital status, employment status and smoking habits); information about previous and present history and severity of low back pain ; and ratings on measures of the four fear-avoidance contextual variables, as follows:

1. Stressful Life Events . These were evaluated using the well established rating scale of Holmes and Rahe[7] which has been extensively used in studies of this kind. It involves a list of 43 life events which the respondent has to endorse as either having occurred or not during the previous year. Two measures are derived, the Total No. of Life Events experienced and a

Weighted Life Events score, the latter taking into account the severity of the events experienced.

2. Personality. The Modified Somatic Perception Questionnaire (MSPQ) of Main[9] was selected for this purpose. It consists of 13 somatic symptoms which the respondent has to report as having occurred 'not at all', 'a little/slightly', 'a great deal/quite a bit', 'extremely/could not have been worse'.

3. Previous Pain History. This was measured using scales previously developed by Slade et al[18]. Subjects were asked to rate their previous pain history on a scale from 1 (no pain at all) to 10 (worst pain imaginable) in terms of three categories of pain experience, as follows: externally produced pain (e.g. factures, dentistry, etc.); internally produced pain (e.g. headaches,toothache,etc.); and accidental pain (e.g. bang thumb with hammer, cut finger, etc.). In each case the highest rating was used as the index of the most severe pain previously experienced.

4. Pain Coping Strategies. This was measured using a scale previously developed by Slade et al [18]. For each of the 8 categories of internally

produced pains the subject was asked to indicate what they normally did in terms of the following five strategies i.e. took pain killers , rested, went to doctor, took physical exercise, ignored it and carried on. The first two responses are considered to be passive , the third to be neutral , and the last two to be examples of active strategies. Three indices are derived from this scale - the 'no. of active' and the 'no. of passive' strategies reported, and the percent of the total which are 'active'.

Two Months Follow up (see table 1).

The fear-avoidance model measures were readministered together with a number of other questionnaire measures and a physical examination was undertaken, as follows:

1. Pain Drawing. This comprises outline drawings of the anterior and posterior views of the human body on which the individual is required to mark the areas where they are experiencing pain. The scoring procedures recommended by Ransford et al[12] were used.
2. Modified Zung Depression Inventory. Main and Waddell[10] developed this 23 item self-report scale which measures depressive symptoms in back pain sufferers. The range of possible scores is zero to sixty-nine.

3. Oswestry Disability Scale. This questionnaire was developed by Fairbank et al. (4) to assess the impact of back pain on daily functioning. There are ten sections, each involving six statements. Each section is scored on a 0 to 5 basis, 5 representing the greatest disability. Fairbank et al. demonstrated that the instrument was a valid measure of disability, with a high test-retest reliability.

4. Inappropriate signs and symptoms. Waddell and Main(22) have described seven inappropriate symptoms and eight inappropriate signs which, in combination, identify low back sufferers who have a significant psychological overlay to their pain and disability. These signs and symptoms were assessed according to the Waddell and Main schedule during a physical and interview examination undertaken by one of us (MR).

5. Physical Examination. A standard physical examination was undertaken which included the following: Neurological tests, Straight leg raising, Prone knee bend, Hip flexion, a Sit up test, Lateral flexion and Sagittal movement of the lumbar spine. This examination was carried out by the research physiotherapist (MR). In addition, a formal interview and examination was carried out by another of the authors (BP), an orthopaedic surgeon, on 110

of the subjects in order to ensure that inappropriate patients were not included in the study.

An attempt was made to follow up all 300 of the original sample. Unfortunately, despite repeated and persistent attempts, only 162 (54%) made themselves available for the two-month follow up interview and 3 of these refused to be physically examined.

Twelve Months Follow up (see table 1).

All measures (questionnaire, interview and physical examination), which had been administered at two months, were repeated together with a further question concerning the Course of Back Pain over the twelve months since recruitment to the study. The latter enabled subjects to be allocated to one of three groups, namely: No Pain, Intermittent Pain and Constant Pain. The original design called for all 12 months follow-up subjects to attend their health centres so that they could be re-interviewed and examined. However, it became apparent that the attrition rate was such that insufficient 12 month data could be generated in this way to make a meaningful statistical analysis possible. We therefore decided to follow up as many as possible of the remainder using a postal questionnaire. Subjects

were asked to provide four sets of information : a rating of the severity of their present pain, to complete the Roland and Morris Disability Questionnaire[14], whether their pain was preventing them from attending work (Work Status) and the Course of their Back Pain over the past twelve months (as previously described). A further 138 (46%) of the original sample provided this information postally. Thus, a minimum twelve month follow up data set was obtained from 196 (65%) of the original sample of three hundred subjects .

STATISTICAL METHODS

Because of the inevitably incomplete follow up rates achieved, comparisons were first made between attenders (54%) and non attenders (46%) at two months ; between respondents (65°o) and non respondents (35%) at twelve months ; and between attenders (19%) and postal respondents (46%) at twelve months. Statistical comparisons were carried out between all three subgroups using all variables collected at the acute stage to determine whether any systematic differences existed.

Where variables were continuous and normally distributed, anovas were used; where vaiables were discrete or not normally distributed, chi-square analyses were used.

Two statistical procedures were used in order to determine the individual and combined predictive power of the predictive models/variables. First, in the case of a continuous and normally distributed outcome variable, 'Present Pain and Disability', a series of multiple regression analyses were undertaken. And, secondly, in the case of two discrete variables, 'Sick Leave' and 'Course of LBP', a series of multiple discriminant function analyses were conducted.

RESULTS

Natural History of Low Back Pain

A core group of 123 subjects (41%) were assessed at all three data collection points, enabling comparisons to be made between different subgroups over time. For this purpose the sample of 123 subjects were subdivided on the basis of reported 'Course of Back Pain' over the twelve month period into : No Pain (N=26) Intermittent Pain (N=88) and Constant Pain (N=9). These three subgroups were then compared at all three data points in terms of Severity of Pain, Disability and Sick Leave (see Figure 1).

It can be seen from Figure 1 that a similar pattern emerges for all three variables:

namely, that the No Pain and the Intermittent Pain groups show improvement/recovery during the acute to two month follow up period, with no significant further improvement during the two month to twelve month period. Thus the improvement shown by these subgroups occurs during the first two months following an acute episode of low back pain.

By contrast, the Constant Pain Subjects (7.3% of the core group) report no real improvement at two months or twelve months and, in the case of pain, they report increased severity at twelve months. Thus the constant pain subgroup appears to have become fixed in their "pain, disability and sickness "mould by two months following the acute attack of back pain.

Follow-up Attenders versus Non Attenders

In a study of this kind it is invariably impossible to achieve follow up data on all subjects initially recruited to the study. It is therefore important to establish whether the subgroup followed up is representative of the group as a whole and, if not, in what respects there is a bias present. We therefore carried out statistical comparisons on all variables obtained at the acute stage (using either anovas for continuous and normally distributed variables; or chi-square analysis for discrete or non normal data) between : (a) attenders (n=162) and non-attenders (n=138) at

two months , (b) respondents (n=196) and non-respondents (n=104) at twelve months follow-up, and (c) attenders (n=58) and postal recipients (n=138) at twelve months follow-up.

At two months the attenders differed from non-attenders on only one out of eleven of the continuous variables (i.e. they were significantly older) and on only two out of six of the discrete variables (i.e. they were more likely to come from a particular middle class practice and to be non-smokers). The bias, if it exists, is therefore towards individuals who are likely to be concerned with their health to attend for follow up.

At twelve months the respondents differed from non-respondents on only one out of twenty of the continuous variables (i.e. they had significantly more saggital movement) and on only two out of the thirteen discrete variables (i.e respondents were more likely to come from social classes 1 and 2 and to be married). It is not clear whether these differences are chance or meaningful ones.

At twelve months the attenders differed from the postal respondents on only three out of twenty one continuous variables (i.e. they were significantly older, they rated their worst attack as significantly less severe and they had significantly less sagittal

movement) and on only three out of thirteen discrete variables (i.e. they were more likely to come from a particular middle class practice, they were more likely to have had fewer previous attacks and they were more likely to be married). As with the two month follow-ups, the only clear bias, if it exists, is for the attenders to be a subgroup who are particularly concerned about their health.

For predictive purposes the potential biases reflected in the above comparisons are only likely to be a problem in so far as they reduce the heterogeneity of the follow-up samples and therefore the magnitude of the predictive variance.

Prediction of Outcome

Following on from a principal component analysis of outcome variables a combined measure of 'Present Pain and Disability' was used as the dependent variable in a series of Multiple Regression Analyses, which are presented in table 2. In the first series (see table 2A), the data derived from the 'demographic','historical'and 'fear-avoidance' variables obtained at the acute stage were used to predict the dependent variable at two months, both singly and in combination. It can be seen from table 2A that the demographic variables were unsuccessful in this respect, while the fear-avoidance variables were the most successful, accounting for 25% of the variance (R-Sqare = .25). However, adding

the demographic and historical variables increased the amount of variance predicted to 34%.

In the second series, the data derived from all three sets of variables obtained at the acute stage were used to predict the dependent variable at twelve months (see table 2B). All the sets of variables now predict outcome significantly and to a similar extent. However, once again the combination of all three increases the predictive outcome to over 30%.

In the third series of multiple regression analyses, the dependent variable at twelve months was predicted from data collected at two months (see table 2C). Three sets of predictor variables were used: (1) physical variables obtained from the physical examination (i.e. side flexion, neurological deficit, sagittal extension, nerve root tethering, sagittal flexion and ability to sit up); (2) the fear-avoidance model variables previously described; and (3) six psycho-social variables identified through discriminant function analysis (i.e. Inappropriate Signs and Symptoms, MSPQ, Roland and Morris Disability, Zung Depression, Severity of Present Pain and Oswestry Disability). As can be seen from table 2C, all three sets of variables significantly predict the dependent variable, with the six psycho-social variables proving to be the best predictor at this stage. Once again, however, it is the

combination of all variables which provides the best prediction of outcome, in this case accounting for 49% of the total variance.

Finally, an attempt to predict the discrete outcomes of 'sick leave' and 'course of back pain' at twelve months was made using multiple discriminant analyses, the results of which are presented in table 3. It can be seen that a combination of all three sets of acute variables provide the best prediction .

DISCUSSION and CONCLUSIONS

As far as the authors are aware, the study described here is the largest undertaken to date in a General Practice setting and there are some useful conclusions to be drawn.

The first concerns the completeness of follow-up and the demands that such research may make on the subject-population. In previous epidemiological studies on Merseyside in which the follow-up has been limited to postal questionnaires, responses have reached levels of 85%. The present study required re-attendance for further interview and tests and thus made considerable demands on the subjects' time. The authors believe that this component of the research design may

explain the attrition rate between each of the follow-up stages. However, careful comparison of data collected at the acute stage from subjects who were followed up with those of subjects who dropped out of the study failed to demonstrate important differences between the groups. This allows the authors to make inferences from subsequent analyses which are *not* substantially weakened by the incomplete two- and twelve-month follow-up data sets.

Secondly, this study throws clear light on the natural history of acute episodes of low back pain . It demonstrates that the majority of individuals who present with an acute attack will either improve significantly or recover within a two month period, while those who do not will become chronic sufferers. Therefore, the first two months appears to be a critical period in the natural history of low back pain. If the patient has not improved during this period the general practitioner needs to consider an appropriate referral.

Thirdly, the best predictor of the course of low back pain during the first two months appears to be the Fear-Avoidance model, which incorporates both stress and personality variables, although demographic and pain history variables also make a contribution to the prediction.

Fourthly, the prediction of outcome from then on is best made using a combination of physical, psychosocial and physical variables. Indeed, when such a combination of variables is used at two months, the prediction of outcome at twelve months using multiple regression analysis can account for approximately 50%. And using multiple discriminant analysis, 85% to 88% of individuals can be correctly classified.

Waddell et al[18] have demonstrated the predictive value of the fear-avoidance concept in patients with chronic back pain. Data from this study reveal the value of the fear-avoidance concept at an earlier stage in the natural history of LBP.

On this basis, fear of pain and its avoidance need to be taken into account in both the assessment and the management of musculo-sketal disorders such as LBP. The authors have applied these principles to the management of chronic LBP within the context of an interdisciplinary treatment programme which is proving very successful with patients for whom all other treatments have failed. Moreover, the authors believe that chronic LBP which involves discomfort, disability and deterioration, can be identified at an early stage and treated : and that this project makes an important contribution to this process.

REFERENCES

1. Connolly J. and Jardin O. : Orthopaedics in Family Practice. Ch. In R. Rakel (Ed) Textbook of Family Practice (3rd edition). Philadelphia, Saunders Co, 1984.

2 Cyriax J. Textbook of Orthopaedic Medicine. 11th Edition, Volume 2, Bailliere Tindall, London; 1986.

3 Dillane JB, Fry J and Kalton G. : Acute back pain - A study from General Practice. Brit. Med. J., 2; 1966;82-84.

4 Fairbank JC, Couper J, Davies JB and O'Brien JB : The Oswestry low back pain disability questionnaire. Physiotherapy; 66; 1980; 271-273.

5. Goldberg DP Recognition of psychological illness by general practitioners. Ch. In G Edwards (Ed) Textbook of Family Practice (3rd edition). Philadelphia, Saunders Co 1981

6 Hanvik LJ : MMPI profiles in patients with low-back pain. J. Consult. Clin. Psychol. 1951; 15, 350-353.

7 Holmes TH.and Rahe TH : The social readjustment rating scale. J. Psychosom Res 11; 1967; 213-218.

8 Lethem J, Slade PD. Troup JDG and Bentley G : Outline of a fear-avoidance model of exaggerated pain perception - 1. Behaviour Research and Therapy;1983; 21; 401-408.

9 Main CJ: The Modified Somatic Perception Questionnaire (MSPQ). J Psychosom Res 27; 1983; 503-514.

10 Main CJ and Waddell G : The identification of psychological abnormality in patients with low back pain. (Manual) 1984

11 Polatin PB, Kinney RK, Gatchel RJ, Erin L. and Mayer TG. : Psychiatric illness chronic low back pain : The mind and the spine - which goes first? Spine, 1993;18(1), 66-71.

12 Ransford AO, Cairns D and Mooney V : The pain drawing as an aid to the psychological evaluation of patients with low back pain. Spine, I, 1976; 127.

13 Roland M. The management of back pain. RCGP Members' Reference Book. London, Royal College of General Practitioners, 1983.

14 Roland M. and Morris R. : A Study of the Natural History of Low-Back Pain. Spine, 8 (2): 1983; 145-150.

15 Rose M., Klenerman L., Atchison L. and Slade PD : An application of the fear-avoidance model to three chronic pain problems. Behaviour Research and Therapy, 30, 1992; 359-365.

16 Rose M, Reilly J, Pennie B and Slade PD : Chronic Low Back Pain : a consequence of misinformation? Employee Counselling Today, 1993

17 Royal College of General Practitioners, Office of Population Censuses and Surveys 1982 : Morbidity statistics from general practice : Third national study. London, HMSO.

18 Slade PD,Troup JDG, Lethem J. : The fear-avoidance model of exaggerated pain perception - 2, preliminary studies of coping strategies for pain. Behaviour Research and Therapy, 1983; 21, 409-416.

19 Troup JDG: Causes, prediction and prevention of back pain at work. Scand. J. Work. Environ. Health, 10, 1984; 419-428.

20 Troup, JDG and Slade, PD : Fear-avoidance and chronic musculoskeletal pain. Stress Medicine, 1, 1985; 217-220.

21 Troup JDG and Videman T. : Inactivity and the aetiogenesis of musculoskeletal disorders. Clinical Biomechanics, 4, 1989; 173-178.

22 Waddell G, McCulloch JA, Kummel ED and Venner RM Non-organic physical signs in low back pain. Spine, 5,2; 1979.

23. Waddell G., Newton M., Henderson I., Somerville D. and Main CJ : A fear-avoidance beliefs questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back and disability. PAIN, 52, 1993; 157-168.

24 Wiltse LL and Rocchio PD : Preoperative psychological tests as predictors of success of chemonucleolysis in the treatment of low-back pain. J. Bone and Joint Surg. 1975; 57-A; 478-483.

25 Wolkind SN and Forrest AJ : Low back Pain: a psychiatric investigation. Postgraduate Medical Journal 1972; 48, 76-79.

TABLE 1:
INFORMATION/MEASURES COLLECTED ON PATIENTS AT THE THREE DATA COLLECTION POINTS

SCREENING QUESTIONNAIRE	TWO MONTHS FOLLOW-UP	TWELVE MONTHS FOLLOW-UP INTERVIEW	POSTAL QUESTIONNAIRE
Variables Demographic Age Sex Doctor Employment Marital status Smoking habits Historical History and severity of previous LBP Severity of present LBP Onset Fear-Avoidance Worst accidental pain Worst internal pain Worst external pain Coping strategies Stressful life events MSPQ Disability Whether of work because of pain	*Variables* As for Screening Questionnaire Plus Pain drawing Modified Zung Disability (Oswestry) Non-organic symptoms Non-organic signs Body Mass Index Neurological tests Straight leg raise Prone knee bend Hip flexion Sit up Lateral flexion Sagittal movement Area affected Clinical diagnosis by Doctor	*Variables* As for Two Months Follow-Up Plus Course of LBP	*Variables* Level of pain Disability Whether off work Course of LBP
No of patients: 300	**No of patients:** 162 interviewed (159 examined)	**No of patients** 58 plus 138 Total = 196	

TABLE 2:
PREDICTION OF OUTCOME (COMBINED PAIN AND DISABILITY)
REGRESSION ANALYSES WITH CONTINUOUS OUTCOME VARIABLE

(A) ACUTE TO 2 MONTHS (N = 162)

VARIABLES	MULTIPLE R	R-SQUARE	'F'	SIGNIF
DEPENDENT = "PAIN & DISABILITY"				
DEMOGRAPHIC	.227	.051	1.65	NS
HISTORICAL	.389	.151	5.04	0.0003
FEAR-AVOIDANCE	.500	.250	7.97	0.0001
ALL	.582	.339	3.88	0.0001

(B) ACUTE TO 12 MONTHS (N = 196)

VARIABLES	MULTIPLE R	R-SQUARE	'F'	SIGNIF
DEPENDENT "PAIN & DISABILITY"				
DEMOGRAPHIC	.349	.122	5.13	0.0002
HISTORICAL	.383	.147	3.72	0.0038
FEAR-AVOIDANCE	.370	.137	4.67	0.0002
ALL	.565	.319	2.67	0.0017

(C) TWO MONTHS TO TWELVE MONTHS (N = 162)

VARIABLES	MULTIPLE R	R-SQUARE	'F'	SIGNIF
DEPENDENT - "PAIN & DISABILITY"				
PHYSICAL	.453	.206	4.89	0.0002
PSYCHO-SOCIAL	.369	.369	10.53	0.0001
FEAR-AVOIDANCE	.475	.226	5.32	0.0001
ALL	.698	.488	4.94	0.0001

TABLE 3:
PREDICTION OF 12 MONTHS OUTCOME USING MULTIPLE DISCRIMINANT FUNCTION ANALYSES WITH DISCRETE OUTCOME VARIABLES

VARIABLES	CHI-SQ	SIG	SENSITIVITY	SPECIFICITY	% CORRECTLY CLASSIFIED
DEPENDENT = "SICK LEAVE"					
DEMOGRAPHIC	14.35	0.01	58%	72%	70%
HISTORICAL	12.49	0.02	85%	66%	68%
FEAR-AVOIDANCE	14.37	0.02	55%	72%	70%
ALL	29.65	0.02	81%	86%	85%
DEPENDENT "COURSE OF LBP"					
DEMOGRAPHIC	18.04	0.00	75%	79%	78%
HISTORICAL	16.16	0.01	100%	77%	79%
FEAR AVOIDANCE	7.27	NS	60%	67%	66%
ALL	33.54	0.01	100%	86%	88%

Figure 1(a)
Mean disability (1-24) at acute stage, two months and twelve months follow-up

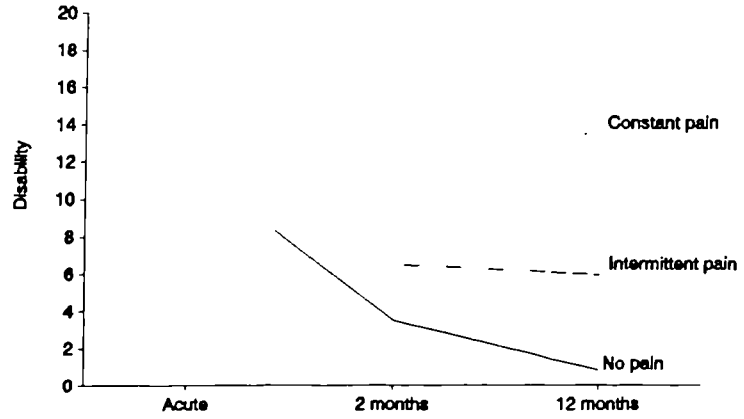


Figure 1(b)
Mean severity of pain (1-10) at acute stage, two months and twelve months follow-up

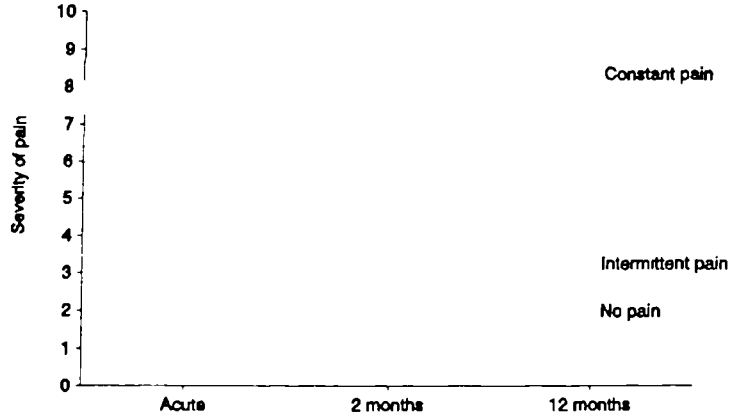


Figure 1(c)
Subjects on sick leave at acute stage, two months and twelve months