Cognitive-Behavioural Therapy for persistent pain: does adherence affect outcome at one-month follow-up?

Charlotte Curran

D.Clin.Psy. thesis (Volume 1), 2007

University College London

UMI Number: U591902

UMI U591902

Overview

This volume of the thesis comprises the major research project, and it is presented in three parts. The first part consists of the review paper, which is a focused review of the literature on outcome in psychological therapies for persistent pain. The second part is the empirical paper. The empirical paper will report on my study, which examines the extent to which cognitive and behavioural adherence predicts better outcome of cognitive-behavioural therapy for persistent pain. The final part of the thesis is formed of the critical appraisal. This section provides the opportunity to critically reflect on the whole process of conducting this piece of research.

Table of Contents

List of Tables

List of Figures

Acknowledgements

I am so grateful to INPUT for providing the data set for analysis in this project. I would like to thank my supervisor, Amanda Williams, for her constant support throughout the period of conducting this research. Without her vast knowledge, both of the data set and of the field of persistent pain, it would have been impossible to get this study off the ground. Thanks also to Henry Potts for his help and reassurance when it felt like structural equation modelling was going to get the better of me. I would also like to thank Geoff and Mary-Claire for their patience and thoroughness when proof reading my never ending drafts. And finally, I would like to say a big thank you to my mum for her continuous support and for the constant reminder that you can only do your best.

Part 1: Literature Review

Psychological therapies for persistent pain - a review of predictors of outcome

Abstract

Background: The varied effectiveness of Cognitive-Behavioural Therapy (CBT) and other psychological therapies in the treatment of persistent pain has led researchers to examine a range of factors that may predict treatment response. Studies have identified a diverse set of personal (i.e., age, gender, and marital status), specific (i.e., level of depression, treatment expectancy, and catastrophising), and non-specific (i.e., adherence to treatment) variables that appear to be associated with treatment outcome, although few with any consistency. This review summarises the available literature on predictors of outcome for persistent pain and discusses limitations of such studies.

Data Sources and Review Method: The persistent pain literature was examined to identify proposed variables that predict response to multicomponent treatment for persistent pain. PsycINFO and Medline databases were searched spanning articles published from 1990 to 2006 of treatment studies and review articles.

Results: Very few studies that focused on predictors of outcome following CBT for persistent pain in the adult population were found through the database searches. Therefore, in addition, a hand-search of key journals, of reference lists from searched articles, and of relevant books was completed and articles prior to 1990 were consequently included in the review at the author's discretion. All types of persistent pain were included except headache.

Conclusions: Main results: The review showed that of the many personal variables investigated to influence outcome, only socioeconomic status (SES), social support, and applying for compensation or disability pension consistently predicted outcome. Of the

specific variables, changes in catastrophising, self-efficacy, pain-related fear, pain tolerance, pain helplessness and perceived disability have each been shown to consistently predict outcome. Few studies were found that had investigated the role of adherence during or after treatment on treatment outcome. Finally, suggestions for future research are discussed.

Keywords: Predictor(s), factor(s), cognitive-behavio(u)ral therapy, chronic pain, persistent pain, treatment outcome.

Introduction

Pain is defined as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' (International Association for the Study of Pain, 1979). Chronic or persistent pain is defined as continuous, long-term pain that has continued beyond three months, or beyond the expected time for healing (Elliott, Smith, Penny, Smith & Chambers, 2000). One in seven people in the UK live in persistent pain and one third of UK households are affected by persistent pain (Elliott, Smith, Penny, Smith & Chambers, 1999). Persistent pain is the most common reason for patients to enter healthcare settings and the most common reason given for self-medication (Eccleston, 2001). Persistent pain patients who present for treatment are often disabled and report other associated problems, such as sleep difficulties and fatigue. It has widespread destructive consequences often resulting in a range of emotional problems, such as depression and pain-related fear, and it significantly lowers quality of life (Eccleston, 2001).

Treatment options for the management of persistent pain range from invasive surgical procedures to use of medications such as opioids and other analgesic agents, alternative therapies such as needle acupuncture, and psychological therapies. The costs for treating persistent pain are huge, yet we are still a long way from eliminating or adequately controlling pain for large numbers of patients in pain (McCracken & Turk, 2002). Most treatment methods for persistent pain aim to reduce pain, whereas when patients are referred to psychological treatment it is often for the purpose of learning

behavioural and cognitive methods for reducing the experience and impact of pain (McCracken, Vowles & Eccleston, 2004). A variety of psychologically based treatment approaches have been developed and empirically validated for helping people to manage their persistent pain (Osborne, Raichle & Jensen, 2006). For example, treatment approaches include operant behavioural therapy and cognitive-behavioural therapy (CBT). These approaches aim to enhance quality of life and pain-related coping, and to reduce disability and pain-related difficulties with functioning. They are therefore rehabilitative approaches and the emphasis is placed on improvement in function despite residual pain.

The theory informing a behavioural approach is that pain and pain disability are influenced not only by somatic pathology, but also by psychological and social factors. The theory informing a cognitive-behavioural approach to persistent pain is that a person's belief about their pain has an impact on both their experience of the pain and on the psychological and functional consequences of the pain. Cognitive-behavioural theory states that psychological factors such as pain catastrophising, pain-related anxiety/fear, and helplessness are associated with increased pain, psychological distress, and physical disability. The theory also holds that factors such as self-efficacy and acceptance would be associated with decreased pain, psychological distress, and physical disability (Keefe, Rumble, Scipio, Giordano & Perri, 2004). A cognitive-behavioural formulation of persistent pain therefore implies that a person's subjective experience of pain and their behavioural and emotional state can be altered through the manipulation of key cognitive and behavioural elements (Morley & Williams, 2002).

The treatment of choice for persistent pain is now considered to be CBT (Koes, van

Tulder & Thomas, 2006; Williams, Nicholas, Richardson, Pither, Justins, Chamberlain,

Harding, Ralphs, Jones, Dieudonne, Featherstone, Hodgson, Ridout & Shannon, 1993).

CBT's principles of active participation, education and guided personal experimentation

make it relatively easy to incorporate into multidisciplinary pain programmes as inputs

from other disciplines can be framed as following the principles of CBT (Morley, 2004).

The focus of treatment should be on the detrimental effects of pain and treatment should

target the domains of pain experience, mood/affect, existing coping methods, pain

behaviour, physical fitness, social role functioning, and healthcare behaviour (Eccleston,

2001). The strategies employed in CBT concentrate on emotional, behavioural, and

social responses to pain, and aim to help patients to increase their feelings of self-

efficacy regarding control of pain (Dalton & Coyne, 2003). CBT for persistent pain

incorporates components of education, relaxation, use of cognitive coping strategies,

pacing, exercise, promotion of a self-management perspective, and reduction of

analgesic and/or psychotropic drugs that have not proved helpful. In addition,

behavioural techniques, such as activity scheduling to gradually increase activity levels,

which has previously been shown to be helpful in the treatment of depression, have been

applied in the persistent pain field (Winterowd, Beck & Gruener, 2003).

This rehabilitative treatment therefore endeavours to restore optimal physical function,

mood, and self-management of pain, and to give an individual a more helpful

understanding of and beliefs about pain and its implications (Williams, Morley & Black,

2002). Its aim is to help patients improve their quality of life by coping better with pain

rather than by decreasing pain intensity (Williams, 1993). Successful cognitive-

behavioural treatment of persistent pain is said to 'produce a patient who describes the

pain in mild terms, engages in productive and satisfying activity on a daily basis, is free

of excess emotional suffering, and uses medical resources at a prudent level'

(McCracken & Turk, 2002).

There is good evidence that CBT is generally effective when conducted in group formats

for diverse patients with persistent pain. For example, the 2004 European Guidelines for

the management of chronic non-specific lower back pain state that the most promising

approach seems to be cognitive-behavioural interventions encouraging activity and

exercise. Similarly, the evidence on treatment of persistent pain from Cochrane and

other systematic reviews shows that CBT delivered in a multidisciplinary treatment

programme is beneficial and recommended (Koes, van Tulder, & Thomas, 2006).

Morley, Eccleston & Williams (1999) conducted a systematic review and meta-analysis

of all randomized controlled trials (RCTs) of CBT for persistent pain for adults. They

compared the effectiveness of cognitive-behavioural treatments with waiting list and

alternative treatment control conditions and all domains of persistent pain problems with

sufficient data were coded for effect sizes. Outcome domains were characterised as pain

experience, mood/affect, positive cognitive coping and appraisal, negative cognitive

coping and appraisal, behaviour/expression, behaviour/activity, and social role

performance. They revealed that compared with a no-treatment control, CBT produced

significant effect sizes with a median of 0.50 across all domains (effect sizes across

domains ranged from 0.36 to 0.6). CBT also produced significantly greater changes for

domains of pain experience, cognitive coping and appraisal, and pain behaviour, when

compared to alternative active treatments such as physiotherapy and medical management. There was insufficient data on health care use and work status. They concluded that active psychological treatments based on the principle of CBT are effective for persistent pain in adults.

However, not all patients respond identically to treatment and unfortunately not all patients reap the benefits of treatment to the same degree. The observed differential effectiveness of CBT has led to studies attempting to identify patient and psychosocial variables that influence treatment outcome, in an attempt to understand the impact of sources of heterogeneity on treatment outcome. Researchers in the field of persistent pain can learn much from research in general mental health. For example, in depression research, increasing theoretical model sophistication and design manipulation has generated a wealth of information regarding the specific and non-specific factors associated with treatment outcome (Hamilton & Dobson, 2002). Such information is of concern to treatment providers, to patients on long waiting lists, and to treatment funders, and it would facilitate treatment planning, and ultimately increase treatment efficiency.

The purpose of this article is to review the available literature on demographic, non-specific and specific predictors of outcome following CBT for persistent pain. Although other sources of variability in therapeutic outcome exist, for example therapist variables, such as level of training and adherence to treatment protocol, it is not within the scope of this review to address these. The review instead focuses on the variability explained by differences between pain patients, differences in process variables, and where possible

the interaction between these factors. Finally, limitations of the research and directions

for further studies of predictors of outcome in CBT for persistent pain are provided.

A literature search was conducted for all published studies from 1990 to 2006 on

variables that influence treatment response to CBT for persistent pain. A computer

search on PsycINFO and Medline databases was performed to look for articles and

reviews published using the following keywords: persistent pain, chronic pain,

cognitive-behavio(u)ral therapy, predictor(s), factor(s), and treatment outcome. In

addition, reference lists from empirical studies and review articles were perused for

other relevant information, and a hand search for articles in the journals PAIN, European

Journal of Pain, and Clinical Journal of Pain was conducted for the years 2000 to 2006.

Literature was included for review if it was published in the English language, and if it

focused on predictors of persistent pain outcome in the adult population. All types of

persistent pain populations were included, except headaches, as the treatment of

headaches has a different focus (See Grazzi, Usai, & Bussone (2006) for further

information). All relevant articles from the search are presented. For clarity of

presentation, the variables examined as potential predictors of treatment response have

been separated into three categories: personal variables, specific (psychological) factors

e.g. cognitive change, and non-specific (process) variables e.g. adherence to treatment.

Many of the studies reviewed examined variables across multiple categories.

Predictors of CBT Outcomes for Persistent Pain

It is important to have a note of caution before proceeding, and to keep it in mind when reading the review. My concern is that little of the research findings in this area of predictors of outcome appear to be derived from theory based hypotheses. Instead, it seems that much of the evidence has been found as a result of data mining for findings of interest and from post-hoc analyses. Although this method can be valuable to highlight areas for further research, results are likely to be specific only to a given population and/or treatment and therefore findings may be un-replicable in many cases.

In addition, in carrying out this review in an area where research is sparse, many of the studies reviewed have methodological weaknesses. Due to limitations of space, such deficiencies will not be discussed for each study individually, but the discussion section will comment on issues likely to effect the interpretations that can be drawn from studies.

Personal Variables

Gender

It has been suggested by some researchers that gender differences in response to pain management interventions exist, although no theory has been offered to suggest why this may be the case. The idea was investigated by Keogh, McCracken and Eccleston (2005) using a sample of 98 persistent pain patients (33 males, 65 females), who attended a 3- or 4-week residential pain management program. Measures of pain and distress were

completed pre-treatment, post-treatment and at 3-month follow-up. The study revealed that both men and women exhibited significant improvements in ratings of pain post-treatment. However, at 3-months follow-up it was shown that only men maintained the treatment gains, while women showed no significant differences from their pre-treatment scores. This indicates that men respond better to treatment, but other studies have revealed the opposite effect or no significant difference.

Jensen, Bergstrom, Ljungquist, Bodin & Nygren (2001) conducted an RCT using 214 patients (97 men, 117 women) suffering with long-term, non-specific spinal pain in Sweden. They compared treatment as usual, CBT, behavioural orientated physical therapy, and combined physical therapy and CBT treatment groups on outcome measures of early retirement, sick leave and health-related quality of life (as measured by the SF-36, Ware & Sherbourne, 1992). Their controlled component analysis found that women showed more consistent improvements in health-related quality of life than men did.

Williams et al's (2002) conducted a study of the influences on outcome of pre-treatment patient characteristics, treatment characteristics, and their interactions, and looked in part at the influence of gender on treatment outcome. The study will be introduced in detail here and findings from the study will be recalled here and in various other sections throughout this review as necessary.

Williams et al. (2002) utilised data from 1977 persistent pain patients who attended a Nation Health Service (NHS) inpatient pain management unit. Patients attended either

19

an inpatient programme of 4-weeks or 2-weeks, or a combined inpatient-outpatient programme. The age range of patients was 18 years to 84 years old and 63% of the sample were female (1245 female, 732 male). The main sites of pain for patients were back and/or leg pain (66.2%), upper body pain (23.5%), and pelvic, perineal, genital and/or anal pain (10.3%). The mean duration of pain was nine years. Measures of depression (measured using the Beck Depression Inventory (BDI); Beck, Ward, Mendelson, Mock & Erbaugh (1961)), catastrophising (measured using the catastrophising subscale of the Coping Strategies Questionnaire (CSQ); Rosenstiel and Keefe (1983), self-efficacy (measured using the Pain Self-Efficacy Questionnaire (PSEQ); Nicholas (1989), distance walked in five minutes, and use of drugs were taken pre-treatment, post-treatment, at one-month follow-up, and at 6-9 month follow-up. They used hierarchical (stepwise) regression techniques to analyse each dependent variable separately. Regarding the influence of gender on treatment outcome, Williams et al. (2002) revealed that being female predicted greater improvement in catastrophising at 6-12 months post-treatment, but not on other outcome measures.

Age

In general, older adults benefit from the same psychological interventions and treatments have comparable outcomes as with younger adults (Pawlick & Middaugh, 2002). However, persistent pain studies have identified differences in treatment outcome which could be attributed to age. One hypothesis may be that older people do not make as good treatment gains as younger individuals with persistent pain as they are generally more likely to have co-morbid conditions, such as having persistent pain and arthritis.

People over the age of 65 years old are often excluded from studies conducted in America as they are less likely to have health insurance, but the impact of age on outcome has been investigated in studies in the UK.

The study by Williams et al. (2002) looked at the impact of age on treatment outcome. They showed that younger age was a predictor of better depression scores at one-month post-treatment. However, other outcome variables, for example measures of catastrophising and walk distance, were not shown to be significantly predicted by age.

Elliott, Smith, Hannaford, Smith & Chambers (2002) later undertook a 4-year follow-up study of 2184 individuals living in Grampian, UK, and aimed to describe patterns and predictors of change in persistent pain in the community. Participants were asked to complete and return by post the chronic pain grade (CPG) questionnaire (Von Korff, Ormel, Keefe & Dworkin, 1992), the SF-36 general health questionnaire (Ware, 1992) and socio-demographic questions in 1996 and again in 2000. It was therefore a survey of self-reported persistent pain in the community. The response rate was 83% at follow-up in 2000. Their study revealed that among those with persistent pain at baseline, individuals between the ages of 45-74 years were less likely to recover from their persistent pain than those aged 25-34 years. Although this is not a treatment study, it is included as it provides an insight into how age may be a factor in recovery from persistent pain, and highlights the need to investigate age further as a possible predictor of treatment outcome. It is worth noting also that the influence of age as a predictor of outcome from persistent pain is likely to be confounded by other important factors such as duration of pain and acceptance of pain, and these also require investigation.

Marital Status

It has been suggested that single working mothers differ in their response to health treatment compared to married females or to male counterparts (Gatchel, Mayer, Kidner & McGeary, 2005). One hypothesis to explain this association is that single working mothers have less social support and less time to focus on their own needs than married females or male counterparts. One study was found to have investigated this hypothesis.

Gatchel et al. (2005) investigated this hypothesis in their study of 1679 chronically disabled work related spinal disorder patients participating in a 5-7 week functional restoration program for persistent pain. Psychosocial variables were measured pre- and post-treatment, and a structured clinical interview was conducted at 1-year follow-up to evaluate long-term outcome. In contrast to what was expected, their study revealed that no differences in levels of depression or pain severity were shown at follow-up between single working mothers and other patients. This indicates that single parent patients can make similar treatment gains to married females or to males.

Minority Group Status

No research investigating the potential role of ethnicity to treatment outcome was found in the area of persistent pain. Research in this area is potentially complicated by issues of diversity and small numbers, as well as by lack of theory about what factors to do with ethnicity may be important. For this reason it would be difficult to interpret any

findings. For example, it would be unknown whether treatment success may be affected by factors such as first language spoken, health beliefs, or access to health care, all of which may vary among clients of different minority groups. Research in this area is necessary to begin to answer these questions.

Socioeconomic Status (SES)

Education level, occupation and income are most frequently used as measures of SES. It is hypothesised that socioeconomic disadvantage may have a negative impact on persistent pain treatment outcome as motivation levels to return to work may be reduced in this population as they have less rewarding jobs and poorer incomes. A number of studies have investigated the role of SES on treatment outcome.

Williams et al. (2002) found that longer education was a variable that predicted better scores on catastrophising short-term, and better scores on self-efficacy and walk distance at long-term follow-up. They also found that being at work at the start of treatment predicted better scores on a measure of self-efficacy short-term, and on a measure of depression at long-term follow-up. However, these results were found through data mining and post-hoc analyses, rather than via theory-driven hypothesis testing.

More recently, Hankin and Killian (2004) researched the extent to which demographic, psychological, and readiness to change indicators predicted functional outcomes in patients with persistent pain. Their prospective study used data from 26 persistent pain patients. The Pain Disability Index (PDI; Tait, Pollard, Margolis, Duckro & Krause,

1987) and the Pain Patient Profile (P-3; Tollison, 1993) were completed pre-treatment

and at 3-months into treatment. Regression analyses indicated that work status and

being in the contemplation stage of readiness to change at baseline accounted for 49% of

the variance in functional outcome at 3-month follow-up. Results from this study must

however be interpreted cautiously due to the very small sample size used, as small scale

studies tend to over-estimate effects.

Social Support

It has been hypothesised that social resources in terms of amount of social support may

have lasting effects on functional disability and levels of pain in persistent pain patients

and a number of studies have demonstrated an association between higher levels of

social support and lower levels of chronic pain. For example, in a study by Jamison and

Virts (1990), patients with chronic pain reported their perceptions of family support

prior to entering an outpatient pain programme. At follow-up, one year following the

completion of the programme, it was discovered that patients who initially reported

having supportive families reported significantly less pain intensity, less reliance on

medication and greater activity levels.

Another study by Tota-Faucette, Gil, Williams, Keefe & Goli (1993) aimed to identify

that family factors, including amount of social support, may influence treatment

outcome. They showed that patients whose families were less supportive, more

controlling and less cohesive made fewer treatment gains than others. Also, Radojevic,

Nicassio & Weisman (1992) conducted a RCT study on rheumatoid arthritis patients

experiencing chronic pain. Patients were assigned to four treatment groups: CBT; CBT with family support; education with family support group; and a no treatment group. Measures of level of pain, disease activity and psychological status were taken during treatment and at 2-months follow-up. The study revealed that the intervention with family support produced improved outcome at post-treatment compared to all other conditions. They reported that spousal participation in treatment tended to enhance outcome in rheumatoid arthritis patients and concluded that it is useful to include family members in pain treatment.

Another later study by Evers, Kraaimaat, Geenen, Jacobs & Johannes (2003) also investigated this hypothesis using 78 patients with rheumatoid arthritis. They examined the role of pain coping and social support at the time of diagnosis in relation to outcome after three and five years. Their study revealed that low levels of social support at the time of diagnosis consistently predicted both more functional disability and increased pain at follow-up. Therefore they again revealed social support to have a positive impact on outcome. They believed that their results supported the proposition that early interventions should target the social resources of patients in an effort to influence long-term outcome in rheumatoid arthritis.

Each of the above studies indicates that social support is beneficial to persistent pain sufferers in terms of their overall health. It is worth considering that according to behavioural theory social support would instead be viewed as disabling in relation to pain. One hypothesis based on this theory may suggest that an individual with good social support may not be motivated to continue using self-management coping skills or

to build on their physical progress, and would therefore have a poorer outcome. However, the reviewed studies do not support this hypothesis.

Compensation Claim and Disability Pension

Persistent pain patients in the process of applying for a compensation claim or those receiving a disability pension are believed to be less likely to show improvements on outcome measures post-treatment. Such a hypothesis is based on the assumption that secondary gain factors may play a role in maintaining a patient's difficulties. A number of studies have been conducted to investigate such a claim.

Confirming the earlier work of Mendelson (1992), who was interested in the relationship between compensation and chronic pain, Vaccaro, Ring, Scuderi, Cohen & Garfin (1997) reported that treatment outcome from surgical management of chronic low back pain was negatively affected if compensation or litigation proceedings were being pursued. A further study by Rainville, Sobel, Hartigan & Wright (1997) investigated the effect of compensation involvement on the reporting of pain and disability by 192 patients referred to a spine rehabilitation programme for chronic low back pain. They also showed that compensation involvement had an adverse effect on self-reported pain, depression, and disability, both before and after treatment.

In addition, Becker, Hojsted, Sjogren & Eriksen (1998) conducted a study in Denmark to investigate whether non-malignant persistent pain patients receiving or applying for disability pension benefit from multidisciplinary treatment. They focussed on the impact

that receiving or applying for disability pension benefit may have on outcome in 286

patients. They found that retirement pension and disability pension status significantly

predicted outcome. For example, patients on retirement pension, who were over the age

of 68 years, showed a significantly larger reduction in pain than those not receiving

pensions, who were younger. They also showed that patients receiving a disability

pension improved as much as those not receiving a disability pension. By contrast,

patients applying for disability pension were shown not to improve when compared to

patients already receiving disability pension. In fact they were found to show a decline

in functioning.

Diagnostic Status

Diagnostic status in this case refers to what pain diagnosis patients have, for example

rheumatoid arthritis or non-specific lower back pain. No theory was found in the

literature from which hypotheses on the expected influence of diagnostic status on

treatment outcome could be based. In addition, diagnostic status is not generally

described or investigated in patients with persistent pain as no satisfactory or reliable

system of diagnoses exists (Wall (p 1-7), In Wall & Melzack, 1994). However, one

study was found to have investigated the possible association.

Hildebrandt, Pfingsten, Saur & Jensen (1997) studied potential factors associated with

success from a multidisciplinary treatment programme for chronic back pain patients.

Ninety chronic low back pain patients who were admitted to an 8-week programme of

functional restoration and behavioural support were recruited for the study, although

only 82 were available at follow-up. Outcome measures were taken pre- and post-treatment, and at 6 and 12-months follow-up and included an assessment of pain intensity and pain disability, return to work, and measurement of strength and general endurance. From analysis they concluded that medical background (duration of pain, surgical procedures), medical diagnosis, and physical impairment variables appeared to have little influence on treatment outcome.

Previous Treatment

The mean duration of pain reported by patients treated at pain clinics exceeds 7 years (Eccleston, 2001), and so patients have commonly undergone a large number of previous treatments at the point where they enter a programme for psychological treatment. The number and type of previous treatments for persistent pain that a patient has engaged in may be one of the most important factors influencing treatment outcome. This is because the success or failure of previous treatments may have a powerful effect on a patient's beliefs and self-efficacy. For example, if a patient has had a number of failed previous treatments, their beliefs about the pain and hopes for its management are more likely to be negative e.g. 'I have no control over my pain', and it may therefore be more difficult to engage the patient in self-management.

Barnes, Smith, Gatchel & Mayer (1989) looked at psychosocioeconomic predictors of treatment outcome in 150 chronic low back pain patients following a 3-week functional restoration rehabilitation programme. They aimed to identify possible factors associated with treatment success or failure, at 1- and 2-year follow-up. The Millon Behavioural

Health Inventory (MBHI; Millon, Green & Meagher, 1982), the BDI, self-report indices of pain and disability, and socioeconomic and demographic information were utilised in the study. Also, variables such as the number of prior spinal surgeries were examined as it was hypothesised that these would influence participation in an active rehabilitation setting and therefore may influence treatment response. Patients were divided into three outcome groups (success, failure, and dropout) based on outcome criteria, and then analyses aimed to show whether certain variables could predict outcome group. They revealed that the number of previous spine surgeries a person had was predictive of worse outcome. Barnes et al. (1989) suggest that this may be because during treatment, surgery may remain a viable treatment option in the patients' minds, and so as the activity demands of treatment increase they may be more tempted by a surgical cure.

Summary

In summary, many personal and medical variables have been examined with the aim of identifying possible predictors of treatment outcome. It can be seen that some variables have been investigated as possible predictors of outcome by many studies, while other variables have been left poorly researched or not researched at all.

The review has shown that the influence of the variables of age, marital status, and minority group status on treatment outcome has been under researched. For that reason, no firm conclusions can be drawn from the literature regarding the role of these demographic variables in predicting treatment outcome.

For variables where more research was available for review, in general, findings across different studies are shown to be inconsistent, and the possibility of any particular variable being a predictor of treatment outcome therefore appears to remain unclear. For example, the proposed association between gender and treatment outcome has not been shown to be theory driven, and results across studies are not in agreement. However, gender may be a variable that influences other predictors of outcome, i.e. it moderates the relationship between other variables and treatment outcome, as indicated by MacFarlane, Thomas, Croft, Papageorgiou, Jayson & Silman (1999). The role of gender as a moderating variable for treatment outcome therefore warrants further investigation.

However, this review showed that the variables of SES, social support, and applying for compensation or disability pension constantly predicted outcome in the reviewed studies. These findings contrast with those of Hamilton and Dobson (2002). Their review of predictors of response to cognitive therapy (CT) for depression found that from all demographic variables only marital status was found to be a significant predictor of response to treatment.

Specific Variables

The roles of a large number of psychological constructs (e.g. level of depression, treatment expectancy beliefs, and catastrophising) on chronic pain treatment outcome have been investigated in recent years, the most researched of which will be presented here. A review of the literature revealed that researchers tend to have defined psychological constructs in different ways (e.g. negative affect vs. depression), and

showed that many constructs overlap. This made it difficult to summarise the findings

in this area into distinct categories and so overlaps between the categories may exist.

Depression

Depression is one of the most frequently hypothesised and tested influences on

persistent pain outcome (e.g. Burns, Johnson, Mahoney, Devine & Pawl, 1998; Malone

& Strube, 1988). However, for clarity just two of the most cited studies are reviewed in

more detail, as they illustrate the inconsistency amongst findings. These studies aimed

to test the hypothesis that symptoms of depression mediate treatment response for

persistent pain patients, as individuals with high levels of depression have maladaptive

appraisals and are believed to be less likely to engage in and use the cognitive and

behavioural strategies introduced to them over the course of therapy.

Dworkin, Richlin, Handlin & Brand (1986) investigated this hypothesis in an early study

when they aimed to test whether short-term and long-term treatment responses in

depressed and non-depressed chronic pain patients differed. A sample of 454 chronic

pain patients was included in the study looking at short-term improvement. A sub-

sample of 82 patients were contacted by telephone an average of 18-months after the

beginning of treatment to gather long-term follow-up data on treatment response.

Treatment response for each patient was rated on a five point scale, from none to

complete improvement. These ratings were made based on the patient's pain complaint,

activity level, and medication use. The study revealed that short-term and long-term

treatment responses for patients with persistent pain, who also experienced depression

(diagnosed using DSM-II criteria), did not differ significantly from persistent pain patients not experiencing depression. However, it was shown that short-term treatment response in these two groups of pain patients was predicted by different patterns of variables. Predictors of good treatment outcome for non-depressed patients were found to be increased treatment visits, fewer previous treatments for their persistent pain, pain being located in the lower back area rather than other locations, and patients not receiving worker's compensation benefits. In contrast, outcome for persistent pain patients suffering with depression appeared to be influenced by employment status at the beginning of treatment (better to be employed) and the duration of persistent pain (the shorter the better).

Lofland, Burns, Tsoutsouris, Laird, Blonsky & Hejna (1997) later conducted a study looking at predictors of outcome following multidisciplinary treatment of persistent pain, looking specifically at the effects of changes in perceived disability and depression on treatment outcome. One hundred and twenty eight persistent pain sufferers admitted to a multidisciplinary pain management programme were originally asked to participate in the study, but the final sample included was 82 patients, due to non-attendance and missing measures. In contrast to the findings of Dworkin et al. (1986), their study showed that decreased depression and decreased perceptions of disability each made significant contributions to improvement in pain severity. Decreased depression also significantly predicted improvement in daily activity.

Pain Beliefs

The cognitive-behavioural approach to pain management assumes that it is cognitive change which underlies the other changes that occur following treatment (Williams et al., 1993). It is hypothesised according to the CBT approach that the effectiveness of pain management programmes is attributable to their influence on pain-related beliefs. The umbrella term of 'pain beliefs' is used here to incorporate the following variables: self-efficacy of pain; fear of pain or re/injury; pain tolerance; pain helplessness; pain catastrophising; and perceived disability. Each of these will be discussed in turn.

Richardson and Richardson (1999) examined whether cognitive change mediated successful treatment outcome following cognitive-behavioural pain management in a sample of 833 severely impaired persistent pain patients. They used the Pain Cognitions Questionnaire (PCQ: Boston et al., 1990), a self-rated measure of pain-related cognitions, to examine if change in the PCQ pre- to post-treatment was predictive of levels of pain, depression, medication use and general functioning at one-month follow-up. They revealed that cognitive change accounted for up to 15% of the variance in outcome and concluded that the study provided supportive evidence for the influence of cognitive change on outcome from cognitive-behavioural pain management.

A study by Burns, Glenn, Bruehl, Harden & Lofland (2003) used a cross-lagged panel design to investigate whether early treatment cognitive change predicted late treatment index change, but not vice versa. Based on other psychological findings, they aimed to determine whether the process of cognitive change is an active therapeutic ingredient of treatment programmes, while controlling for depression change. Their study was conducted using 90 persistent pain patients from a 4-week intensive multidisciplinary

programme. Measures of catastrophising, pain helplessness, depression, pain,

interference, and activity level were taken pre-treatment (1 week before the programme

began), mid-treatment (5th day of second week) and post-treatment (5th day of forth

week). Findings suggested that early treatment catastrophising (as measured by the

Catastrophising Subscale (CS) of the Coping Strategies Questionnaire; Rosentiel &

Keefe, 1983) and pain helplessness changes (as measured using an adapted version of

The Arthritis Helplessness Index; Nicassio, Wallston, Callahan, Herbert, & Pincus,

1985, in which the authors substituted the word arthritis with the word pain) predicted

late-treatment outcome (as measured by the Multidimensional Pain Inventory; Kerns,

Turk, & Rudy, 1985), but not vice versa, even with changes in depression controlled for.

This indicates that cognitive changes during treatment have a significant effect on

treatment outcome, regardless of changes in level of depression. They showed that pain

helplessness reduction predicted less than 10% of the variance in outcome gains.

Burns, Kubilus, Bruehl, Harden & Lofland (2003) later replicated and extended their

investigation of the role of cognitive factors contribution to treatment outcome in

persistent pain patients. 65 patients who entered a 4-week multidisciplinary programme

for the treatment of their persistent pain participated in the study. Burns et al. (2003)

revealed that early-treatment reductions in catastrophising and pain-related anxiety

predicted late-treatment improvements in pain severity, but not vice versa. Further

support that cognitive change may influence treatment changes in outcome has come

from the study of randomised control trials.

A study by Smeets, Vlaeyen, Kester & Knottnerus (2006) offers further support for this hypothesis. 211 patients with non-specific chronic low back pain took part in their randomised control design study. Using four treatment groups: active physical treatment; CBT; combined active physical treatment and CBT; and waiting list control, they investigated whether treatments based on different theories changed pain catastrophising and internal control of pain. They also examined whether changes in these factors mediated treatment outcome. Participants in all three active treatment groups were found to decrease on the measure of pain catastrophising and were shown to improve on the measures of pain experience. It was further implied that change in pain catastrophising mediated the reduction of disability, pain complaints, and pain intensity. In the active physical treatment group only, pain catastrophising was also found to mediate the reduction in depression. Smeets et al. (2006) concluded that pain catastrophising can be reduced even when it is not a direct target of therapy and that changes in pain catastrophising mediate treatment outcome.

Spinhoven, Kuile, Kole-Snijders, Mansfeld, Ouden & Vlaeyen (2004) conducted a study to examine whether changes in pain coping and cognition during multidisciplinary treatment for chronic low back pain mediated treatment outcome. They tried to improve on methodology from previous studies such as Jensen et al. (2001) by including a no-treatment control group and other forms of active treatment. It was hypothesised that changes in pain and adjustment to pain would be mediated by an enhanced perceived internal control of pain and a reduction in catastrophising of pain. Their RCT, involving 148 patients, revealed that the operant-behavioural treatment plus cognitive coping skills training group resulted in a short-term and long-term decrease in catastrophising and an

enhancement of internal pain control. They further identified that changes in catastrophising and internal pain control mediated the reduction in level of depression and pain behaviour following treatment. This study corroborates that having a focus on decreasing catastrophising thoughts and promoting internal expectations of pain control during treatment is important to bring about change.

Malone and Strube (1988) earlier hypothesised that reducing patients' fear of pain, rather than reducing the pain itself, should be the key aim of psychological treatments. McCracken and Gross (1998) later studied the role of pain-related anxiety reduction in the outcome of a 3-week multidisciplinary treatment programme for 79 chronic low back pain patients. They found that decreased pain-related anxiety significantly predicted improvement on a range of outcomes, including measures of pain severity, disability level, general affective distress and level of daily activity. Change in pain-related anxiety was identified to explain the variance in outcome even when controlling for change in depression. They concluded that resolution of fear is the key to success and reductions in pain-related fear predict improved treatment outcomes.

McCracken & Turk (2002) went on to conduct a follow-up study and showed that the role of reduced pain-related anxiety in treatment outcome was independent of change in physical capacity. They found that changes in pain-related anxiety during treatment accounted for more variance in the prediction of outcome than change in physical capacity. This casts doubt on the hypothesis that patients should adhere to doing exercises following treatment, as if a client becomes more active on a daily basis another model is that they would no longer need to do the prescribed exercises.

Lofland et al. (1997) looked at whether changes in perceived disability accounted for the variance in pre- to post-treatment improvements in outcome variables in 82 patients receiving multidisciplinary treatment for persistent pain. Regression analyses revealed that change scores on the measure of perceived disability (the Pain Disability Index) predicted unique variance in pain severity (as measured by the Pain Severity Subscale of the Multidimensional Pain Inventory) and functional level changes (patients' abilities to lift, carry, push, and pull weights as evaluated on a scale of 1 to 8). This result stood when controlling for changes in depression. The authors concluded that improvements in outcome variables may be mediated by decreases in perceived disability.

Hildebrandt et al.'s (1997) study also looked at the role of perceived disability on treatment outcome on chronically disabled low back pain patients who completed an 8-week functional restoration and behaviour support programme. They again identified that successful treatment outcome was predicted most clearly by a reduction in patients' subjective feelings of disability.

Finally, Burns et al. (1998) investigated whether cognitive and physical capacity changes that occur through multidisciplinary pain treatment contribute uniquely to long-term treatment outcome. 94 persistent pain patients participated in the study and measures of pain helplessness, lifting capacity, walking endurance, depression, pain severity, and activity levels were collected at three time points: pre-treatment; post-treatment; and 3- to 6-months follow-up. Evidence for the contribution of cognitive changes to long-term outcome was indicated as decreases in pain helplessness (as

measured by an adapted version of the Arthritis Helplessness Index) were found to be linked to reductions in pain severity (as measured by the Pain Severity subscale of the Multidimensional Pain Inventory). These results were shown even after controlling for the effects of depression decreases, indicating that decreases in pain helplessness makes a unique contribution to long-term outcome.

Acceptance of Pain

Few studies have addressed acceptance of pain as a variable influencing treatment outcome. However, a number of cognitive and behavioural therapies, such as mindfulness-based stress reduction (MBSR), have begun to focus on acceptance of pain as a factor that influences outcome. 'Acceptance' is defined as "experiencing events fully and without defence, as they are" (p.30, Hayes, Jacobsen, Follette, & Dougher, 1994), and 'acceptance of pain' is defined as acknowledging that you have pain, giving up unproductive efforts to control it, and acting as if pain does not imply disability (Hayes, Jacobson, Follette & Dougher 1994). Baer's (2003) review of empirical research on the utility of mindfulness based interventions identified four studies that had examined the effects of MBSR on patients with persistent pain. In general, findings revealed significant improvements and maintenance of improvements at follow-up, indicating that acceptance of pain may be an important variable in the prediction of treatment outcome.

A study by McCracken (1998) investigated the role of acceptance of pain on outcome using 160 chronic pain patients attending a university pain management centre. Self-

report measures including the Chronic Pain Acceptance Questionnaire, the BDI, the Pain

Anxiety Symptom Scale, and the Sickness Impact Profile were used as measures of pain

acceptance, level of depression, level of pain-related anxiety, and level of disability

respectively. The study showed that greater acceptance of pain was found to be related

to lower reports of pain, less pain-related anxiety and avoidance, less depression and

disability, and better work status. More recently, acceptance of persistent pain has also

been associated with fewer healthcare visits for pain, and fewer cases of prescribed

analgesic medications (McCracken et al., 2004).

Treatment Expectancy

Patients' pre-treatment beliefs in the success of treatment have been shown to be one of

the strongest predictors of final treatment outcome in psychotherapy research (Kirsch,

1999), but their role have only recently been attended to in the area of persistent pain.

Treatment expectancy has been referred to as the strength of expectation that

improvements will be achieved during treatment (Goosens, Vlaeyen, Hidding, Kole-

Snijders & Evers, 2005). The hypothesis that treatment expectations have an impact on

treatment outcome states that the patients' beliefs about what they think they can do are

powerful predictors of actual functioning (Lackner, Carosella & Feuerstein, 1996).

Kole-Snijders, Vlaeyen, Goossens, Rutten-van Molken, Heuts, van Breukelen & van

Eek (1999) conducted an RCT investigating the role of treatment expectancy on

outcome in patients with chronic low back pain following CBT. 148 patients

participated in the study and were assigned to an operant behavioural treatment and

cognitive coping skills training group, an operant behavioural treatment and discussion

group, or a waiting list control group. Self-report and observational measures of pain

behaviour, pain cognitions, activity tolerance, pain intensity, and negative affect were

taken pre-treatment, post-treatment, at 6-month follow-up, and at 12-month follow-up.

In addition, treatment credibility was measured at the start and end of treatment through

patients rating their personal belief that the programme would help them cope better

with pain on a visual analogue scale ranging from 'not at all' to 'very much'. Patients'

certainty score in the belief was then rated on a 5-point likert scale. Total credibility

score was calculated as a product of their belief and certainty scores. Results showed

that treatment credibility ratings were important predictors of successful outcome, as

measured by self-reported pain coping and control, and observed activity tolerance and

pain-behaviour at follow-up. Patients who believed in the credibility of treatment at the

beginning of treatment were shown to improve more on motoric behaviour and coping

control immediately after treatment.

Similarly, Williams et al. (2002) examined the influence of pre-treatment variables on

treatment outcome and found that patients' expectations of treatment benefit contributed

to outcome prediction. Also, Morley, Hussain & Williams (2005) used a sample of 1013

persistent pain patients to investigate the influence of generic pre-treatment expectation

of change on CBT treatment outcome measures measured at three time points: pre-

treatment; 1-month post treatment; and at 6, 9 or 12 months follow-up. Using multiple

regression methods they revealed that pre-treatment expectations had a significant

impact (although it explained less than 5% of the variance) on levels of pain

interference, levels of depression and physical capacity.

Furthermore, a study by Goossens et al. (2005) corroborated these findings. They aimed to examine whether patients' initial beliefs about the effectiveness of a given pain treatment influenced the final treatment outcome in patients with chronic low back pain or fibromyalgia. The extent to which treatment expectancy (as measured by a short self-report questionnaire) predicts the short-term and long-term outcome of cognitive-behavioural treatment for persistent pain was investigated employing the data of two pooled randomised control trials. Data from a total of 171 patients were available for the analysis. The analyses showed that patients with higher treatment expectancies received significantly less disability compensation and were less fearful. In addition, pre-treatment expectancy was shown to predict a small amount of variance in the four outcome measures (pain coping and control, motor behaviour, negative affect and quality of life) immediately post-treatment. The significant explanatory contribution of the pre-treatment expectancy score varied between 1% and 8%. Pre-treatment expectancy scores were also shown to predict motor behaviour, negative effect, and quality of life at 12-month follow-up. They concluded that assessing patients' expectations for CBT for their pain is necessary prior to entering treatment.

Summary

In summary, the review indicates that many studies have investigated the role of specific factors on treatment outcome and findings across studies have largely been shown to be compatible.

Changes in catastrophising, self-efficacy, pain-related fear, pain tolerance, pain helplessness and perceived disability have each been shown to consistently predict outcome. In addition, acceptance of pain, treatment expectancy, and levels of depression have each been indicated as important influences on treatment outcome. The findings show the importance of psychological variables on treatment outcome, and indicate that they should be a target on treatment.

Non-specific Variables

As the name suggests, CBT aims to bring about change in a person's thoughts and behaviours. The assumption that it is important to bring about behavioural change, either directly or indirectly via cognitive change, has been justified in other fields such as in the treatment of depression. In the field of persistent pain, behavioural change is considered as an outcome or target of treatment (e.g. increases in levels of activity, and improved physical and social functioning), and as a process variable influencing outcome (e.g. adherence to treatment recommendations). As yet it is unclear how much emphasis should be targeted towards behavioural change as a process variable in the management of persistent pain.

Adherence during treatment

Adherence during treatment refers to the undertaking of recommended behaviours (e.g. pacing, and relaxation) during the treatment period. Jensen, Turner & Romano (1994) investigated correlates of improvement in multicomponent treatment of persistent pain

using 94 persistent pain patients. They were interested in part in testing the relationships between treatment outcome and use of behavioural coping strategies. Behavioural coping strategies were measured using a questionnaire on which patients had to indicate the number of days in the preceding seven in which they had used strategies of exercise, pain-contingent rest, keeping busy, opioid medication use, and relaxation. Outcome measures of psychological functioning (measured using the BDI), physical functioning (measured using the Physical Dysfunction scale of the Sickness Impact Profile), and use of medical services (measured as number of self-reported pain-related healthcare contacts) were taken pre-treatment and 3-6 months post-treatment. They showed that increased use of behavioural coping strategies developed during the course of treatment did not explain significant amounts of improvement on outcome. Instead their study's findings implied that only changes in maladaptive and negative beliefs (measured using the Survey of Pain Attitudes) were critical in improving treatment outcome.

More recently, Heapy, Otis, Marcus, Frantsve, Janke, Shulman, Bellmore & Kerns (2005) investigated the hypothesis that adherence to treatment between sessions mediated the relationship between readiness to change and goal accomplishment in persistent pain patients. A measure of readiness to adopt a self-management approach to persistent pain, aggregated intersession adherence ratings, and mean post-treatment goal accomplishment ratings were gathered for 78 pain patients. Results indicated that pre-treatment readiness to change significantly predicted intersession adherence and goal accomplishment. Furthermore, the relationship between readiness to change and goal accomplishment was significantly enhanced when adherence between sessions was taken into account, indicating that adherence to therapist recommendations for coping

skill practice mediates readiness to change and self-reported goal attainments. However, it is worth noting that as the goal attainments measure is self-reported, it may simply be tapping the same construct again.

Adherence to Behaviour Changes after Treatment

Adherence with therapeutic recommendations subsequent to treatment termination may be an important predictor of long-term treatment gains. As Turk, Rudy & Sorkin (1993) said, 'For treatment to be effective, the patient must comply with the treatment recommendations'. Furthermore, it has been suggested that for chronic difficulties, the long-term outcome following brief treatments may be poor (even if their initial response is good) unless treatment is maintained over prolonged periods through self-management, albeit at low intensity (Roth & Fonagy, 1996). That said, limited attention has been paid to this issue in the pain literature, the evidence supporting such statements is sparse, and any attempts to examine the relationship between adherence and maintenance of treatment gains have so far used very simple models.

Turk and Rudy (1991) conducted a review of the literature in which they raised the question of the role of continued practice of pain management methods in the maintenance of treatment gains. They argue that failure to practice or employ coping strategies, exercises, relaxation techniques, and other methods taught during treatment in the follow-up period has been shown to contribute to relapse. Their review of studies on arthritis and heterogeneous pain clinic patients suggest that adherence and relapse are

related; however, this association is less well established for other problems such as headaches.

Taimela, Diederich, Hubsch & Heinricy (2000) recognised that the role of exercises on the long-term success and maintenance of outcome after the guided treatment period has ended had not been extensively investigated in chronic low back pain patients. They sought to examine to role of adherence to physical exercise on pain recurrence and absenteeism from work using 125 patients who had completed a 12-week active low back rehabilitation programme. Outcome measures of levels of subjective pain and disability were completed 14 months after treatment ended. Results showed that recurrences of persistent pain and frequencies of work absenteeism during the follow-up period were significantly fewer among those who had maintained regular exercise habits since treatment when compared to those who had been physically inactive. They concluded that the maintenance of treatment gains following active treatment is predicted by adherence to exercises after guided treatment has ended.

Summary

In summary, this area of interest appears under-researched. Few studies were found that had investigated the role of adherence during or after treatment on treatment outcome. It is important to consider also that finding associations between adherence behaviour and behavioural outcomes is to be expected if the measures of each are tapping the same thing, for example frequent exercise. The focus thus far appears to be on the impact of

adherence to behaviours rather than on the influence of adherence to cognitive strategies. This would be an exciting area for future research.

Discussion

Summary of Findings

Although CBT is effective in treating a variety of difficulties, not all persistent pain patients benefit from this form of treatment. Studies have examined a diverse range of variables that may influence treatment outcome. The review has identified that studies in the field of persistent pain have investigated a wide range of personal and medical, specific and non-specific factors that are believed to have an impact on treatment outcome following CBT for persistent pain.

Patient demographic factors identified as potential predictors of treatment outcome were SES, social support, and whether a person is applying for compensation or disability pension. Other demographic variables were not shown to consistently predict outcome. The lack of consistency among research findings means that the current results are not sufficient to guide treatment assignment and patient demographic variables therefore appear to contribute little.

Findings from studies investigating the role of non-specific variables on outcome were also found to be inconsistent across studies. Rather than concluding that these variables therefore have little or no impact on treatment outcome, the results must be interpreted

in light of considerations about the individual studies. Inconsistencies between studies

may be due in part to disparate patient samples, inconsistent research methodologies,

variability in treatments offered, different treatment settings, and divergent treatment

outcome criteria and measurement techniques between studies. For example, the

participants in the Jensen et al. (1994) study had completed an inpatient

multidisciplinary pain programme, while the participants in the Heapy et al. (2005)

study were randomly assigned to three treatment conditions (primary care CBT, CBT,

and standard care), each of which was provided in an outpatient setting.

In contrast to the findings regarding the influence of demographic and non-specific

variables on treatment outcome, the findings from research investigating the role of

psychological factors appear to be more consistent, and specific variables appear to be

significant predictors of treatment outcome. Specific predictors of treatment outcome

for persistent pain appear to include self-efficacy, catastrophising, treatment expectancy

and amount of perceived pain. It also seems that factors such as low level of depression

and high acceptance of pain are associated with positive treatment outcomes. Patients'

own beliefs and expectations about their pain and how it should be treated therefore

appear to have a considerable influence on treatment outcome.

Limitations

The review has been valuable in summarising the current research findings in this area.

However, some early studies (e.g. Dworkin et al. 1986) included in this review are out

dated and are known to be methodologically weak, and yet have been included as they

are well cited and offer much in an area of little research. Methodological weaknesses

47

of individual studies therefore need to be taken into account, and differences in methodologies between studies need to be acknowledged. Methodological weaknesses and inconsistencies between studies have an impact on the confidence that can be placed on the conclusions of individual studies and they make it difficult to make comparisons across studies. Limitations in the reviewed studies include that many of the studies used unacceptably small sample sizes, used unreliable outcome measures, and used correlation designs.

The sample sizes used in the reviewed studies varied enormously. Of particular concern is the small sample of 26 individuals used in the study by Hankin and Killian (2004). Such a small sample size obviously has implications for the power of the study, and thus on the reliability of conclusions drawn from such a study. Another concern is the self-reported nature of many of the outcome measures used by studies, which inevitably lead to bias in the results. For example, Heapy et al. (2005) used self-reported measures of intersession adherence and goal accomplishment, which simply could have tapped the same construct. Finally, the correlational nature of many studies data means that no conclusions about causal relationships between hypothesised predictors and outcome variables can me made. In other words, the fact that variables have been identified to account for some of the variance in changes in outcome measures, may simply be the result of other causative change processes. Alternative explanations for observed findings cannot be ruled out.

Turning now to focus on understanding why few consistent findings were revealed across studies, the review highlighted that many demographic and methodological

differences existed between studies. These may in part explain the lack of consistency in findings. For example, differences between studies included that dissimilar treatments were offered, different outcome measures were used, the persistent pain populations varied, the length of follow-ups differed, and the design of studies were different.

Very different results were revealed from studies depending on whether treatment was CBT inpatient (e.g. Williams et al., 2002) or a functional restoration and behaviour support programme (e.g. Hildebrandt et al., 1997). Also, findings differed depending on whether outcome was defined as level of pain, emotional distress or return to work status. For example, Jensen et al. (2001) and Taimela et al. (2000) used early retirement and absenteeism from work respectively as their outcome measure, while Williams et al. (2002) used measures of depression and catastrophising to indicate outcome. It is therefore unsurprising that different results were obtained depending on what measure of outcome was utilised.

The type of persistent pain population recruited for studies also differed considerably. For example, Radojevic et al. (1992) focused on arthritis sufferers, while the study by Gatchel et al. (2005) used patients with spinal disorder in their research. The length of follow-up also varied amongst these, with Radojevic et al. (1992) taking measures of outcome at two-month follow-up, while Gatchel et al. (2005) took it at one-year follow-up. Again, it is not surprising that such differences in methodologies would impact on the current findings.

Arguably many of the current findings in this area lack replication and demonstration of generalisability due to the above methodological weaknesses. Finally, the review has highlighted the lack of theory-based hypothesis testing in the research. Instead, many post hoc explanations have been offered as a result of data mining for results and reporting what seem to be interesting findings. The difficulty with such methodology is that findings are unlikely to be replicated by other studies, which was shown to be the case with the demographic variables.

Ideas for Future Research

Most studies investigating the relationship between variables and treatment outcome have been shown to rely upon relatively simple conceptual models and univariate statistical approaches. Investigating the processes of change in outcome needs to continue, with the aim of specifying causal relationships.

Better models are needed of what changes and when, to move this area forward. For example, researchers need to ask questions such as what can undermine treatment efficacy, what might facilitate it, and similar questions of maintaining gains. Researchers need to develop more precise models of change, aiming to link specific outcomes with changes in particular process variables (Morley, 2004). Focused research on particular relationships will be vital in the development and specification of statistical models and to identify mediators and moderators of change following CBT for persistent pain. Future research on the process of treatment improvements in persistent pain will therefore need to use more sophisticated designs and analyses, such as crossed-lagged

panel designs as utilised by Burns et al (2003), to provide information on whether changes on process variables precede changes in outcome.

Further research will also need to address and quantify the effects of critical process variables, such as patient adherence to treatment, which as yet have been inadequately measured and researched. This will have important theoretical and clinical implications. For example, if adherence to treatment is found to predict long-term treatment gains then it suggests that appropriate maintenance of change components should be included in CBT treatments for persistent pain (Eccleston, 2001). This will be an important and valuable advance in the field as it will promote long-term behaviour change for adults with persistent pain to ensure that gains are maintained and extended after regular treatment ends. Until this issue is resolved, clinicians are unable to provide clear evidence-based recommendations to patients at the end of treatment.

The review has highlighted the lack of theory-driven hypotheses in this area, therefore theory driven research is required in the future to improve our understanding of treatment mechanisms. Also, in the current review it has been difficult to compare across studies as treatment protocols were different. Standardising treatment protocols would therefore facilitate future research. Finally, studies are needed that examine the interrelationships among identified predictor variables and outcome of CBT for persistent pain in order to better understand how these variables influence outcome. Studies need to control for other variables in their analyses of predictors of outcome, as otherwise interpretation of results is difficult.

Conclusion

It is clear that many variables appear to influence response to CBT treatment for persistent pain, although the challenge of understanding exactly why patients vary in their response to treatment remains. Great steps forward have been taken in recent years, having drawn on research findings from mental health fields. That said, improving the effectiveness of psychological treatments for persistent pain, and identifying procedures essential for therapeutic change, are challenging and yet necessary tasks, and further research is needed in this area. Developing a greater understanding of the impact and nature of these sources of variability in outcome will be an important step forward in the field of persistent pain. This would lead to advances in treatment methods, which in turn will lead to cost savings for the NHS and improvement in treatment outcome and patient satisfaction.

Williams and McCracken (In: Asmundson, Vlaeyen & Crombez, 2004) concluded that findings about predictors of treatment outcome are not yet able or ready to offer firm guidance on treatment-related decisions, and the findings of this current review confirm this status. Further research is necessary as results will improve our understanding of treatment mechanisms, and will in turn help match patients to effective and appropriate treatment, which will help to control treatment costs.

References

Baer, R. A. (2003). Mindfullness training as a clinical intervention: A conceptual and empirical review. *Clinical Psychology: Science and Practice*, 10 (2), 125-143.

Barnes, D., Smith, D., Gatchel, R. J. & Mayer, T. G. (1989). Psychosocioeconomic predictors of treatment success/failure in chronic low back pain patients. *Spine*, 14, 427-430.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J. & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561-571.

Becker, N., Hojsted, J., Sjogren, P. & Eriksen, J. (1998). Sociodemographic predictors of treatment outcome in chronic non-malignant pain patients. Do patients receiving or applying for disability pension benefit from multidisciplinary pain treatment? *Pain*, 77 (3), 279-287.

Burns, J. W., Johnson, B. J., Mahoney, N., Devine, J. & Pawl, R. (1998). Cognitive and physical capacity process variables predict long-term outcome after treatment of persistent pain. *Journal of Consulting and Clinical Psychology*, 66, 434-439.

Burns, J. W., Glenn, B., Bruehl, S., Harden, R. N. & Lofland, K. (2003). Cognitive factors influence outcome following multidisciplinary persistent pain treatment: a replication and extension of a cross-lagged panel analysis. *Behaviour Research and Therapy*, 41 (10), 1163-1182.

Burns, J. W., Kubilus, A., Bruehl, S., Harden, R. N. & Lofland, K. (2003). Do changes in cognitive factors influence outcome following multidisciplinary treatment for persistent pain? A cross-lagged panel analysis. *Journal of Consulting and Clinical Psychology*, 71, 81-91.

Dalton, J. A. & Coyne, P. (2003). CBT: tailored to the individual. *Nursing Clinics of North America*, 38 (3), 465-476.

Dworkin, R. H., Richlin, D. M., Handlin, D. S. & Brand, L. (1986). Predicting treatment response in depressed and non-depressed persistent pain patients. *Pain*, 24, 343-353.

Eccleston, C. (2001). Role of psychology in pain management. *British Journal of Anaesthesia*, 87 (1), 144-152.

Elliott A. M., Smith, B. H., Penny, K. J., Smith, W. C. & Chambers, W. A. (1999). The epidemiology of chronic pain in the community. *Lancet*, 354, 1248–52.

Elliot, A. M., Smith, B. H., Hannaford, P. C., Smith, W. C. & Chambers, W. A. (2002). The course of persistent pain in the community: results of a 4-year follow-up study. *Pain*, 99 (1-2), 299-307.

Elliott, A. M., Smith, B. H., Penny, K. I., Smith, W. C. & Chambers, W. A. (2000). Chronic Pain. *The Lancet*, 355, 234.

Evers, A. W. M., Kraaimaat, F. W., Geenen, R., Jacobs, J. W. G. & Johannes, W. J. (2003). Pain coping and social support as predictors of long-term functional disability and pain in early rheumatoid arthritis. *Behaviour Research and Therapy*, 41 (11), 1295-1310.

Gatchel, R. J., Mayer, T. G., Kidner, C. L. & McGeary, D. D. (2005). Are gender, marital status or parenthood risk factors for outcome of treatment for chronic disabling spinal disorders? *Journal of Occupational Rehabilitation*, 15 (2), 191-201.

Goossens, M. E. J. B., Vlaeyen, J. W. S., Hidding, A., Kole-Snijders, A. & Evers, S. M. A. A. (2005). Treatment expectancy affects the outcome of cognitive-behavioral interventions in persistent pain. *Clinical Journal of Pain*, 1, 18-26.

Grazzi. L., Usai. S., & Bussone. G. (2006). Chronic headaches: pharmacological and non-pharmacological treatment. *Neurological Science*, 27 (2), 174-8.

Hamilton, K. E., & Dobson, K. S. (2002). Cognitive therapy of depression: Pretreatment patient predictors of outcome. *Clinical Psychology Review*, 22, 875-893.

Hankin, H. A. & Killian, C. B. (2004). Prediction of functional outcomes in patients with persistent pain. *Work*, 22 (2), 125-130.

Hayes, S. C., Jacobson, N. S., Follette, V. M., & Dougher, M. J. (Eds.). (1994). Acceptance and change: Content and context in psychotherapy. Reno, NV: Context Press.

Heapy, A., Otis, J., Marcus, K. S., Frantsve, L. M., Janke, E. A., Shulman, M., Bellmore, W. & Kerns, R. D. (2005). Intersession coping skill practice mediates the relationship between readiness for self-management treatment and goal accomplishment. *Pain*, 118, 360-368.

Hildebrandt, J., Pfingsten, M., Saur, P. & Jansen, J. (1997). Prediction of success from a multidisciplinary treatment program for chronic low back pain. *Spine*, 22 (9), 990-1001.

International Association for the Study of Pain. (1979) - www.iasp-pain.org

Jensen, M. P., Turner, J. A., & Romano, J. M. (1994). Correlates of improvement in multidisciplinary treatment of persistent pain. *Journal of Consulting and Clinical Psychology*, Vol 62 (1), 172-179.

Jensen, I. B., Bergstrom, G., Ljungquist, T., Bodin, L. & Nygren, A. L. (2001). A randomized controlled component analysis of a behavioural medicine rehabilitation program for chronic spinal pain: are the effects dependent on gender? *Pain*, 91, 65-78.

Jensen, M. P., Nielson, W. R. & Kerns, R. D. (2003). Toward the development of a motivational model of pain self-management. *Journal of Pain*, 4, 477-492.

Keefe, F. J., Rumble, M. E., Scipio, C. D., Giordano, L. A. & Perri, L. M. (2004). Psychological aspects of persistent pain: Current state of the science. *The Journal of Pain*, 5(4), 195-211.

Keogh, E., McCracken, L. M. & Eccleston, C. (2005). Do men and women differ in their response to interdisciplinary persistent pain management? *Pain*, 114 (1-2), 37-46.

Kerns, R. D., Turk, D. C., & Rudy, T. E. (1985). The West Haven-Yale Multidimensional Pain Inventory (WHYMPI). *Pain*, 23, 345-356.

Kirsch, I. (1999). How expectancies shape experience. Washington: American Psychological Association.

Koes, B. W., van Tulder, M. W., & Thomas, S. (2006). Diagnosis and treatment of low back pain. *British Medical Journal*, 332, 1430-1434.

Kole-Snijders, A. M., Vlaeyen, J. W., Goossens, M. E., Rutten-van Molken, M. P., Heuts, P. H., van Breukelen, G. & van Eek, H. (1999). Chronic low-back pain: what does cognitive coping skills training add to operant behavioural treatment? Results of a randomised clinical trial. *Journal of Consulting and Clinical Psychology*, 67, 931-944.

Lackner, J. M., Carosella, A. M., & Feuerstein, M. (1996). Pain expectancies, pain, and functional self-efficacy expectancies as determinants of disability in patients with chronic low back disorders. *Journal of Consulting and Clinical Psychology*, 64, 212-220.

Lofland, K. R., Burns, J. W., Tsoutsouris, J., Laird, M. M., Blonsky, E. R. & Hejna, W. F. (1997). Predictors of outcome following multidisciplinary treatment of persistent pain: Effects of changes in perceived disability and depression. *International Journal of Rehabilitation and Health*, 3, 221-232.

MacFarlane, G. L., Thomas, E., Croft, P. R., Papageorgiou, A. C., Jayson, M. I. & Silman, A. J. (1999). Predictors of early improvement in low back pain amongst consulters to general practice: The influence of premorbid and episode-related factors. *Pain*, 80, 113-119.

Maixner, W., & Humphrey, C. (1993). Gender differences in pain and cardiovascular responses to forearm ischemia. *Clinical Journal of Pain*, 8, 16-25.

Malone, M. D. & Strube, M. J. (1988). Meta-analysis of non-medical treatments for persistent pain. *Pain*, 34, 231-244.

McCracken, L. M. (1998). Learning to live with the pain: acceptance of pain predicts adjustment in persons with persistent pain. *Pain*, 74, 21-27.

McCracken, L. M., Carson, J. W., Eccleston, C. & Keefe, F. J. (2004). Acceptance and change in the context of persistent pain. *Pain*, 109, 4-7.

McCracken, L. M. & Gross, R. T. (1998). The role of pain-related anxiety reduction in the outcome of multidisciplinary treatment for chronic low back pain: Preliminary results. *Journal of Occupational Rehabilitation*, 8, 179-189.

McCracken, L. M. & Turk, D. C. (2002). Behavioural and cognitive-behavioural treatment for persistent pain: Outcome, predictors of outcome, and treatment process. *Spine*, 27(22), 2564-2573.

McCracken, L. M., Vowles, K. E. & Eccleston, C. (2004). Acceptance of persistent pain: component analysis and a revised assessment method. *Pain*, 107, 159-166.

Mendelson, G. (1992). Compensation and chronic pain. *Pain*, 48: 121-123.

Millon, T., Green, C. J. & Meagher, R. B. (1982). A new psychodiagnostic tool for clients in rehabilitation settings: The Millon Behavioral Health Inventory (MBHI). *Rehabilitation Psychology, 27*(1), 23-35.

Morley, S. (2004). Editorial: Process and change in cognitive behaviour therapy for persistent pain. *Pain*, 109, 205-206.

Morley, S. & Williams, A. (2002). Conducting and evaluating treatment outcome studies. Chapter 3, In Turk and Gatchel: Psychological Approaches to Pain Management (2nd Ed). Guildford: New York.

Morley, S., Eccleston, C., & Williams, A. (1999). Systematic review and meta-analysis of randomized controlled trials of CBT and behaviour therapy for persistent pain in adults, excluding headache. *Pain*, 80, 1-13.

Morley, S., Hussain, S., & Williams, A. (2005). Testing the cognitive mediation model of change in a CBT based pain management program. Using a 3-wave panel multiple regression model to test for general and specific cognitive predictors. (In preparation)

Nicassio, P. M., Wallston, K. A., Callahan, L. F., Herbert, M., & Pincus, T. (1985). The measurement of helplessness in rheumatoid arthritis: The development of the Arthritis Helplessness Index. *Journal of Rheumatology*, 12, 462-467.

Nicholas, M. K. (1989). Self-efficacy and chronic pain. Paper presented at the annual conference of the British Psychological Society. St. Andrews.

Osborne, T. L., Raichle, K. A. & Jensen, M. P. (2006). Psychological interventions for persistent pain. *Physical Medicine and Rehabilitation Clinics of North America*, 17 (2), 415-433.

Pawlick, K. & Middaugh, S. J. (2002). Persistent pain in the older adult. Chapter 28, In Turk and Gatchel: Psychological approaches to pain management (2nd Ed). Guildford: New York.

Radojevik, V., Nicassio, P. M. & Weisman, M. H. (1992). Behavioural intervention with and without family support for rheumatoid arthritis. *Behavior Therapy*, 23, 13-30.

Rainville, J., Sobel, J. B., Hartigan, C. & Wright, A. (1997). The effect of compensation involvement on the reporting of pain and disability by patients referred for rehabilitation of chronic low back pain. *Spine*, 22, 2016-2024.

Richardson, I. H. & Richardson, P. H. (1999). Does cognitive change predict the outcome of cognitive-behavioural pain management? *Psychology, Health and Medicine*, 4 (1), 27-44.

Rosentiel, A. K., & Keefe, F. J. (1983). The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. *Pain*, 17, 33-44.

Roth, T. & Fonagy, P. (2005). What Works for Whom? (2nd Ed). Guildford Press: New York.

Smeets, R. J. E. M., Vlaeyen, J. W. S., Kester, A. D. M. & Knottnerus, J. A. (2006). Reduction of pain catastrophizing mediates the outcome of both physical and cognitive-behavioral treatment in chronic low back pain. *The Journal of Pain*, 7 (4), 261-271.

Spinhoven, P., Kuile, M., Kole-Snijders, A. M. J., Mansfeld, M. H., Ouden, D. J. & Vlaeyen, J. W. S. (2004). Catastrophizing and internal pain control as mediators of outcome in the multidisciplinary treatment of chronic low back pain. *European Journal of Pain*, 8 (3), 211-219.

Taimela, S., Diederich, C., Hubsch, M. & Heinricy, M. (2000). The role of physical exercise and inactivity in pain recurrence and absenteeism from work after active outpatient rehabilitation for recurrent or chronic low back pain: a follow-up study. *Spine*, 25 (14), 1809-1816.

Tait, R.C., Pollard, C.A., Margolis, R.B., Duckro, P.N. & Krause, S.J. (1987). The Pain Disability Index: psychometric and validity data. *Archive of Physical and Medical Rehabilitation*, 68(7):438-41.

Tollison, C.D. (1993). The comprehensive diagnosis of spinal pain: A new psychodiagnostic instrument. *Orthopedic Review*, 22, 335–340.

Tota-Faucette, M. E., Gil, K. M., Williams, D. A., Keefe, F. J. & Goli, V. (1993). Predictors of response to pain management treatment: The role of family environment and changes in cognitive processes. *Clinical Journal of Pain*, 9 (2), 115-123.

Turk, D. C. & Rudy, T. E. (1991). Neglected topics in the treatment of persistent pain patients – relapse, non-compliance, and adherence enhancement. *Pain*, 44, 5-28.

Turk, D. C., Rudy, T. E., & Sorkin, B. A. (1993). Neglected topics in chronic pain treatment outcome studies: Determination of success. *Pain*, 53, 3-16.

Vaccaro, A. R., Ring, D., Scuderi, G., Cohen, D. S. & Garfin, S. R. (1997). Predictors of outcome in patients with chronic back pain in low-grade spondylolisthesis. *Spine*, 17, 2030-2035.

Von Korff, M., Ormel, J., Keefe, F. J., & Dworkin, S. F. (1992). Grading the severity of chronic pain. *Pain*, 50, 133-149.

Wall, P. D. (1994). Introduction. (P. 1-7). In: Wall, P. D. and Melzack, R. (Eds), Textbook of pain (Third Edition). Edinburgh: Churchill Livingstone.

Ware, J. E., & Sherbourne, C. D. (1992). The MOS 36-Item Short-Form Health Survey (SF-36®): I. conceptual framework and item selection. *Medical Care*, 30(6):473-83.

Williams, A. C. DE C. (1993). Inpatient pain management of persistent pain. In: M. Hodes and S Moorey (Eds), Psychological treatment in human disease and illness. London: Gaskell Press.

Williams, A. C. DE C., Nicholas, M. K., Richardson, P. H., Pither, C. E., Justins, D. M., Chamberlain, J. H., Harding, V. R., Ralphs, J. A., Jones, S. C., Dieudonne, I., Featherstone, J. D., Hodgson, D. R., Ridout, K. L. & Shannon, E. M. (1993). Evaluation of a cognitive behavioural programme for rehabilitating patients with persistent pain. *British Journal of General Practice*, 43, 513-518.

Williams, A.C.deC., Morley, S., & Black, S. (2002). Grant report: Empirical evaluation of best practice in chronic pain management: influence of patient characteristics and treatment type on outcome. www.info.doh.gov.uk/doh/refr_web.nsf

Williams, A. & McCracken, L. M. Chapter 13 In: Asmundson, G. J. G., Vlaeyen, J. W. S., and Crombez, G. (2004). Understanding and treating pain. Oxford University Press. New York: Oxford University Press (pp. 293-312).

Winterowd, C., Beck, A. T., & Gruener, D. (2003). Cognitive therapy with chronic pain patients. Springer Publishing Company

European Guidelines on the management of chronic non-specific lower back pain (2004). www.backpaineurope.org

Part 2: Empirical paper

Cognitive-Behavioural Therapy for persistent pain: does adherence

affect outcome at one-month follow-up?

Abstract

Patients' extent of adherence to the use of cognitive techniques and behavioural changes

learnt during the course of intensive cognitive-behavioural treatment for persistent pain

have long been assumed to influence maintenance of treatment gains. However, no

research has investigated the causal influence of adherence on short-term outcome. The

aims of this paper are to assess determinants of adherence to treatment recommendations

and to examine the extent to which cognitive and behavioural adherence predicts better

outcome of cognitive–behavioural treatment for persistent pain. Longitudinal data from

a sample of 2345 persistent pain patients who attended a multicomponent treatment

programme were subjected to structural equation modeling in the form of path analyses.

Adherence emerged as a mediating factor linking post-treatment and follow-up

treatment outcome, but contributed only 3% of the variance in follow-up outcomes.

Combined end-of-treatment outcomes and adherence factors accounted for 72% of the

variance in outcome at one-month follow-up. Assuming that the measures used

adequately quantify adherence, these findings question the emphasis normally given to

adherence in the maintenance of behavioural and cognitive change, and their clinical

implications are discussed.

1. Introduction

Chronic or persistent pain is defined as continuous, long-term pain that has continued beyond three months, or beyond the expected time for healing (Elliott, Smith, Penny, Smith & Chambers, 2000). It has widespread destructive consequences often resulting in a range of emotional problems, such as depression and pain-related fear (Eccleston, 2001), and its treatment costs in the NHS are enormous (Maniadakis & Gray, 2000). Treatment options for the management of persistent pain range from invasive surgical procedures to use of medications such as opioids and other analgesic agents, alternative therapies such as needle acupuncture, and psychological therapies.

Many treatment options (e.g. surgical treatment or use of medications) aim to reduce patients' levels of pain. In contrast, when patients are referred to psychological treatment it is often for the purpose of learning behavioural and cognitive methods for reducing the experience and impact of their pain (McCracken, Carson, Eccleston & Keefe, 2004). More specifically, cognitive-behavioural therapy (CBT) aims to enhance quality of life and pain-related coping, and to reduce disability and pain-related difficulties with functioning. It is therefore a rehabilitative approach and the emphasis is placed on improvement in function despite residual pain.

Much evidence, from Cochrane and other systematic reviews (e.g. Morley, Eccleston & Williams, 1999), reveals that CBT delivered within a multicomponent treatment programme is beneficial and recommended for patients with persistent pain (Koes, van

Tulder, & Thomas, 2006). However, it has been argued that the success of therapy

should be measured by its ability both to improve patient functioning at the end of

treatment and to maintain that improvement after therapy ends (Roth & Fonagy, 2005).

An important question for all therapists is why some patients who show initial treatment

success are able to maintain their treatment gains, while other patients are not. In recent

years researchers have striven to develop their understanding of the long-term effects of

treatments and to identify both general and specific factors that may influence and

contribute to the variance in maintenance of treatment gains.

On the basis of learning theory (e.g. Bandura, 1989), one variable that has been

examined as having an influence on long-term treatment outcomes is a patient's use of

the strategies and techniques taught during the treatment programme (adherence), after

the treatment programme has ended. Unfortunately there is no consensus either on the

definition or measurement of adherence.

The World Health Organisation (2003) defined treatment adherence as the

correspondence between a regimen offered by the healthcare provider (e.g. medication

and changes in lifestyle) and the patient's behaviour to implement such a plan. In other

words, it is the degree to which patient behaviours coincide with healthcare providers'

recommendations. Self-report is widely used in the study of adherence, not least

because of its convenience, and the principal techniques used for measuring adherence

are patient self-report and the judgement of professionals (Turk & Rudy, 1991; Vitolins,

Rand, Rapp, Ribisl & Sevick, 2000).

Adherence research is vital within the field of persistent pain since rehabilitative programmes for persistent pain aim to help patients adopt methods for both physical and psychological self-management of their condition, that is, they aim to get patients to make long-term lifestyle changes. Clinicians help to plan, execute, and guide the practice of specific changes in behaviours and recommend that patients will continue to engage in those behaviours. Clinicians therefore need to know whether adhering to such recommendations has an influence on clinical outcome. If treatment adherence does, as expected, impact on clinical outcome, it suggests that further research is required to identify factors that influence treatment adherence. This question is imperative in the field of persistent pain.

<u>Does adherence to treatment recommendations influence clinical outcome?</u>

Much adherence research has focused on adherence to regimens aimed at weight loss, smoking cessation, and continued use of long-term medication (e.g. Osterberg & Blaschke, 2005; Vermeire, 2001). As expected, findings from these studies show that sustained behaviour change is required in order to maintain improvements made during treatment. Similar findings have been demonstrated in the general mental health field.

In the field of general mental health, several studies (see Antonuccio, Danton, & DeNelsky, 1995) indicate that compared to medications, CBT is associated with fewer relapses following treatment and better maintenance of treatment gains. They suggest that skill acquisition during treatment and the continued use of these skills in the follow-up period may be the cause of this difference. This finding implies that for general

mental health difficulties, behavioural and cognitive changes learnt during the course of treatment need to be sustained in order for treatment gains to be maintained and extended (Roth & Fonagy, 2005). Unfortunately, the picture is not as clear in the field of persistent pain.

Despite therapists who work with patients with persistent pain recommending that patients should continue to use techniques taught during treatment, there have been relatively few empirical studies of the therapeutic effects of adherence on follow-up outcome following treatment for persistent pain. A review article by Turk and Rudy (1991) represents the most comprehensive examination of the role of adherence on maintenance of treatment gains in the field.

Turk and Rudy (1991) conducted a review of the available literature on poor adherence and relapse in persistent pain, to examine the question of the role of continued practice of pain management methods in the maintenance of treatment gains. Their review of studies on arthritis (e.g. Basler & Rehfisch, 1991) and on heterogeneous pain clinic patients (e.g. Lutz, Silbret & Olshan, 1983) suggested that failure of adherence and relapse were related; however, this association was less well established for other disorders. They therefore argued that failure to practice or employ coping strategies, exercises, relaxation techniques, and other methods taught during treatment in the follow-up period contributed to relapse.

A further early study by Nicholas, Wilson & Goyen (1991) also showed that continued rates of practice of pain management strategies, even at modest rates, meant that post-

treatment improvements were maintained at follow-up. They agreed that maintenance of

treatment gains requires continued use of the skills and behaviours learned in treatment.

The evidence in this area is therefore sparse on the role of adherence in the maintenance

of treatment gains. The available evidence implies that adherence to treatment

recommendations may influence follow-up outcomes; although it remains unclear

whether adherence to treatment regimens is necessary to maintain treatment gains.

Factors influencing treatment adherence

In recent years a number of factors, both psychological and non-psychological, have

been hypothesised to influence extent of adherence to treatment recommendations.

However, only a handful of studies were found to have investigated predictors of

adherence in the field of cognitive and behavioural change in persistent pain.

One factor that has been investigated as having an influence on amount of adherence is

level of depression. For example, DiMatteo, Lepper & Crogham (2000) found that

patients with depressive symptoms were at greatest risk of poor adherence to medical

treatment. More specifically in the persistent pain field, Jensen, Turner and Romano

(1994) showed that reduced depression scores were associated with the continued use of

muscle strengthening exercises and cognitive techniques to challenge beliefs about pain.

Similarly, Fielding and Duff (1999) showed that high levels of psychological distress

were associated with adherence difficulties. These studies therefore suggest that

negative emotional factors, such as depressive symptoms, are associated with poor adherence to treatment.

Other psychological characteristics, such as level of self-efficacy, have also been examined for their impact upon treatment adherence. For example, Fraser, Hadjimichael & Vollmer (2003) conducted a study investigating the influence that levels of self-efficacy had on adherence in multiple sclerosis patients. They found that baseline levels of self-efficacy predicted adherence to daily self-administered injections. They concluded that low levels of self-efficacy negatively influence treatment adherence.

Other factors that have been shown to negatively influence levels of treatment adherence after treatment has ended include low expectations for treatment and poor outcomes by the end of treatment. Erlen and Caruthers (2007) suggest that for patients with physical disabilities low expectations for treatment success may lead to poor adherence to treatment recommendations, as the patient may see little point in adhering. Furthermore, Taimela, Diederich, Hubsch & Heinricy (2000) indicate that patients with less favourable outcome during treatment were less likely to participate in physical exercises post-treatment. They therefore conclude that those with good outcome in pain reduction during the treatment course were more likely to participate in physical exercise after the treatment phase had ended. Erlen and Caruthers (2007) go on to suggest that for persistent pain patients, adherence to components of their therapy such as exercise will only be sustained given adequate adherence to pain therapy in the first instance. Therefore they suggest that adherence predicts adherence.

A final factor that has been suggested to influence treatment adherence is the complexity of the treatment regimen. Erlen and Caruthers (2007) state that only 50% of patients with physical disabilities adhere to uncomplicated treatment regimens, such as taking one medication, while they believe that this figure is likely to be even less for more complex regimens. Berg, Evangelista & Dunbar-Jacob (2002) investigate this hypothesis and conclude that treatment adherence is likely to decline when the treatment regimen becomes more complex, for example if the regimen is time-consuming or involves multiple elements. This suggests that patients are more likely to adhere to a single simple recommended regimen, than to a single more complex regimen or multiple tasks.

Summary

Attempts to examine the question of how adherence to programme recommendations and practice of skills may be related to maintenance of treatment gains have so far used very simple models. It seems from the little evidence available that adherence to treatment regimens is associated with better clinical outcomes, and that many factors may influence rates of adherence. However, it has been shown that there is presently no evidence, either in the general mental health or the persistent pain fields, that any particular rate of continued practice of strategies is necessary to maintain treatment gains. Also, various methodological problems have plagued the measurement of patient adherence. Measures of adherence have focused on the self-reported frequency of adherence only, and studies have not allowed for adequate consideration of measurement error. As a consequence limited results have been produced.

No attempt has been made to examine or to clarify the nature of the link between adherence and the maintenance of treatment gains in the persistent pain field. Therefore the causal structure that accounts for any association is not known and the contribution that continued practice of pain management strategies post-treatment plays on follow-up outcome remains unclear.

The Present Study

The present study attempts to model (using substantive sample sizes and multiple measures of adherence) how adherence to treatment methods may predict follow-up treatment outcome for persistent pain sufferers.

Research in this area has important theoretical, treatment, prevention, and public policy implications. For example, if adherence to treatment is found to predict follow-up treatment maintenance then it suggests that appropriate maintenance of change components should be included in CBT treatments for persistent pain (Eccleston, 2001). Until this issue is resolved, clinicians are unable to provide clear evidence-based recommendations to patients at the end of treatment. Acquiring knowledge of the impact and of the mechanism through which adherence impacts follow-up outcome will be a valuable advance in the field as it will promote long-term behaviour change for patients with persistent pain to ensure that gains are maintained and extended after regular treatment ends.

Aims

The primary purpose of this exploratory study was to explore the relationships between post-treatment outcomes, adherence to behavioural and cognitive coping strategies, and follow-up outcomes, in patients who had completed a multicomponent pain programme. Two hypothetical causal models were proposed to explain the relationships between post-treatment outcome, adherence, and follow-up outcome. A large data-set, multiple outcome measures and multiple measures of adherence were used to generate multi-indicator latent variables using exploratory factor analysis techniques. The fit of the causal models was examined to assess to what extent, if any, adherence contributes to the prediction of outcomes at one-month follow-up.

Therefore, the present exploratory study has the following aims:

a.) To investigate the multidimensionality of adherence as a theoretical construct using exploratory factor analysis, to identify latent factors of adherence from multiple indicators;

b.) To compare several possible causal models which relate adherence to outcome at follow-up using Structural Equation Modeling (SEM).

Hypothesised causal models

There are two hypothesised causal models: adherence as a mediator of outcomes at follow-up; and adherence as an independent predictor of outcomes.

1.) Adherence as a mediator

A 'mediator' is defined as 'the generative mechanism through which the focal independent variable is able to influence the dependent variable of interest' (Baron & Kenny, 1986). In outcome research, mediators of change are those characteristics of the individual that are changed by the treatment and that, in turn, produce change in the outcome of interest (Whisman, 1993). Therefore, the logic involved in hypothesising mediating relationships is that "the independent variable influences the mediator, which in turn influences the outcome" (Holmbeck, 1997, p. 600).

It may be that adherence to treatment mediates or partially mediates the relationship between psychological state (contentment) post-treatment and psychological state at follow-up. This hypothesis is based on the premise that level of functioning and contentment at the end of treatment (post-treatment) may influence the extent to which a programme graduate adheres to treatment recommendations, which in turn will influence psychological state at follow-up. This hypothesis would suggest that patients who are severely depressed or who have low self-efficacy at the end of treatment may, through low motivation, adhere poorly to the recommended techniques, and will therefore show poorer maintenance of treatment gains at follow-up.

This model would predict the following:

a.)   that psychological state post-treatment predicts levels of adherence, with higher levels of contentment being associated with higher levels of adherence;

b.)   that adherence to treatment recommendations predicts maintenance of gains

in psychological state at follow-up, with higher levels of adherence

predicting higher levels of wellbeing at follow-up;

c.)   that the strength of the relationship between post-treatment psychological

state and psychological state at follow-up will be greater when adherence is

greater.

2.) Adherence as an independent predictor

This model suggests that level of adherence influences follow-up outcomes; but that

adherence level is not predicted by a patient's level of functioning at the end of

treatment (i.e. adherence does not mediate the change). In this model, adherence may be

predicted by some other variables, for example by psychological variables such as

motivation or social support, or by personal/medical variables such as age, gender, or

site of pain.

2   *Methods*

Sample/data

Data were extracted from a database on 3498 persistent pain patients entering a

multicomponent pain management programme between 1989 and 2004. Some patients

in the present study have been included in several prior reports, but the focus and

methods of these were different (e.g. Morley, Hussein & Williams, in preparation;

Williams, Morley & Black, 2002). Personal and medical, clinical and psychological

data were routinely collected from patients using standardised assessments.

Patients had been referred from all over the UK and attended a two or four week (9-day

or 16-day) inpatient pain management programme. The treatment aimed to restore

optimal physical function, range and level of activity, improve mood, and to reduce the

use of drugs and other health care. The programme also provided participants with a

more helpful understanding of and beliefs about pain and its implications. It aimed to

teach people habit change for life. To meet these aims, the treatment programme

included exercise and stretching routines, goal setting, pacing of activities, education

sessions, cognitive and behavioural sessions, drug reduction, relaxation, sleep

management, relapse prevention and family involvement (where appropriate).

As adherence was the variable of interest in the present study, it was decided that all

cases without adherence data at one-month follow-up would be deleted, to reduce

missing data. Although such a strategy meant that it was likely that many cases with

low adherence and lower outcome scores at the end of treatment were omitted, a

criterion for selection was that they had attended their one-month follow-up appointment

and that reports of their levels of adherence at that time were available. A sample of

2435 remained and was used in the analysis.

Randomly splitting the above sample in two (using the SPSS select random cases

function) produced two sample groups, which were treated as the exploratory group (n =

1204) and the validation group (n = 1231). The personal and medical characteristics of

the groups are listed in Table 1. No significant differences were found between the two

samples on any of these variables, except gender: the exploratory group had significantly

more females in its sample than the validation group (χ^2 (1) =8.86, p=0.003).

Table 1: Participant Demographics

Variable	Participants Exploratory Group (n=1204)	Validation Group (n=1231)	Test of mean differences χ^2/t-value (df)	p
Demographics				
Gender (% female)	59	65	8.86(1)	0.003
Mean age (years)	45	45	0.24(1112)	0.807
Ethnicity-white (%)	88	89	0.84(3)	0.839
Further education (>16years) (%)	48	48	0.24(2)	0.885
Mean months since work	62	60	1.47(901)	0.142
Mean months of pain	103	109	-1.46(1088)	0.144
Main Site of Pain				
Low back (%)	49	49	9.75(9)	0.371
Shoulder/arm/hand (%)	11	11	-	-
Hip/leg/feet (%)	10	9	-	-
Multiple sites (%)	16	15	-	-
Other (%)				
(e.g. head/neck/chest/abdomen/pelvis)	14	16	-	-
Employment Status				
Unemployed due to pain (%)	28	27	3.87(7)	0.795
Employed (%)	20	21	-	-
Permanent sick/disabled (%)	34	36	-	-
Retired (%)	11	10	-	-
Other (%)				
(student/homemaker/unemployed for other reasons)	7	6	-	-
Living Situation				
Living alone (%)	17	20	4.95(5)	0.422
Living with partner, or partner and children (%)	66	65	-	-
Other (%)				
(living with children only/friends/other relatives)	17	15	-	-

Key: n = number of participants; χ^2/t-value (df) = Chi-square/t-value and degrees of freedom; p = probability value.

Ethics

Ethical approval was granted by St Thomas' Hospital Research Ethics Committee (See

Appendix 1) and research and development approval was obtained from The Guy's and

St Thomas' NHS Foundation Trust Research and Development Department. These were granted as an amendment and an extension on approval previously obtained for research using the data sample. To ensure confidentiality, data were supplied anonymised.

Measures

The following measures were selected from a wider range of measures used routinely as part of the assessment of patients undergoing treatment at the pain unit. The selected measures were chosen for their ability to represent the constructs of interest in this study. Measures were administered at pre-treatment assessment (up to two weeks before starting treatment) (Time 1), post-treatment at discharge on the last day of treatment (Time 2), and at one-month follow-up (Time 3), unless otherwise specified. Some measures were also collected at 6-month, 9-month, and 12-month follow-up, although due to the extent of missing data these were not utilised in the current study.

Outcome Measures

Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961) was used as an index of depressive thinking and affect. The BDI consists of 21 groups of items, each focusing on a specific manifestation of depression (sadness, dissatisfaction, suicidal ideation etc). Item scores range from 0 to 3, giving a total score from 0 to 63 with higher scores indicating higher depression. The validity of the BDI for the measurement of depressive symptoms in persistent pain has however been questioned by Morley, Williams & Black (2002). They held that the BDI used with

chronic pain patients can lead to inflated depression scores, because it includes somatic

items that have a different meaning for chronic pain patients compared to depressed

patients. Factor analysis (exploratory and confirmatory) identified a factor of 'self

denigration' which they suggested could better indicate depressive thinking in persistent

pain patients. This factor consisted of items C (sense of failure), E (guilt), F

(punishment), G (self-dislike/ disappointment), H (self-blame), and N (body image

change/ unattractiveness). In this study the BDI 'self denigration' factor scores (BDI-f)

were included in the analyses as the measure of depression/mood. Scores ranged from 0

(indicating no symptoms/ positive view of self) to 18 (indicating a very negative view of

self): however, for the purposes of this study these scores were reversed so that 0 = very

negative view of self and 18 = positive view of self. This was done so that

improvements on this measure were indicated on the scale in the same direction as

improvements on other measures.

Pain Self-Efficacy Questionnaire (PSEQ; Nicholas, 2007). This self-report

questionnaire was used to assess the degree of confidence that patients have in achieving

activities in ten different areas despite their pain. Each statement (e.g. I can enjoy

things, despite the pain) is followed by a seven-point scale ranging from 0 (not at all

confident) to 6 (completely confident). Total scores range from 0 (no confidence at all)

to 60 (completely confident for all activities). This measure has been shown to have

good test-retest reliability and internal consistency (e.g. Cronbach's α was calculated at

0.92) (Asghari and Nicholas, 2001).

Pain Catastrophising Scale (PCS; Sullivan, Bishop & Pivik, 1995). This is a brief self-completion measure of catastrophising. It samples the tendency to attend to pain stimuli, to overestimate their threat value and to underestimate the ability to handle that threat. 13 statements are rated on a five-point scale ranging from 0 (not at all) to 4 (all the time), and total scores range from 0 to 52. It is a robust measure for the assessment of the cognitive construct of catastrophising and no other measure has proved as heuristic or explanatory. Internal consistency is satisfactory for the PCS total (Cronbach's $\alpha = 0.91$) and the test-retest reliability is satisfactory for the whole scale (ICC = 0.82) (Chatzidimitriou, Georgoudis, Manousou, Argira, Vadalouka, Anastasopoulou, Pavlopoulos & Siafaka, 2006). It was developed from the catastrophising subscale of the CSQ (see below: Sullivan et al., 1995). This measure was introduced as the measure of catastrophising within the pain management unit in 2000, and was taken only at Time 3 due to questionnaire burden on patients.

Coping Strategies Questionnaire (CSQ; Rosenstiel & Keefe, 1983). The catastrophising subscale of the CSQ was originally used within the service as a measure of level of catastrophising. The sub-scale includes six self-referent items such as 'I feel I can't stand it anymore'. Patients indicate on a scale of 0 (never) to 6 (always) the frequency of occurrence of making self-statements resembling each item. The total scores for the catastrophising subscale range from 0 to 36 and it has been shown to be a robust factor. This measure was again only taken at Time 3, and was replaced by the PCS as the measure of catastrophising in 2000.

As a consequence of the change in the measure of catastrophising participants in the sample were either administered the CSQ or the PCS. During data manipulation, scores on the catastrophising subscale of the CSQ were re-coded to a scale of 0-52 by multiplying scores by 52/36. A single scale of catastrophising was therefore available for all participants during data analyses. In the original scale 0 = no catastrophising and 52 = high levels of catastrophising, but this scale was reversed for the purposes of the study so that 0 = high levels of catastrophising and 52 = no catastrophising.

The combining of scores from the PCS and CSQ was justified on the basis that the PCS was developed with the CSQ authors' involvement, and because the PCS included all six items from the CSQ. In addition, research has demonstrated a high correlation between PCS and CSQ scores ($r = 0.89$) (Chatzidimitriou et al. 2006).

Adherence Measures

All measures of adherence were completed at Time 3 only.

Exercise Frequency – patients reported how often they currently carried out their circuit-exercises. These were general fitness and strength exercises such as sit-ups, which aimed to build strength and flexibility. Each patient reported adherence using one of six categories, where 1 indicated they have stopped doing the exercises completely and 6 indicated they do them at least once a day.

Stretch Frequency - patients reported how often they currently carried out their stretching (general gentle stretches to extend tendons and muscles) by ticking one of six categories, where 1 indicated they have stopped completely and 6 indicated they do them at least once a day.

Pacing Frequency - patients reported how often they were using activity pacing (breaking activities down into small manageable steps and building up activity slowly) by ticking one of six categories, where 1 indicates they had stopped completely and 6 indicates they were doing it at least once a day.

Pacing Occasion - patients reported the occasions on which they used the pacing by ticking one or more of six categories, where 1=not at all, 2=when I remember, 3=when the pain is bad, 4=for some activities, 5=indoors only and 6=as a daily approach. These categories were derived from patient reports during the programme and at follow-up. These were re-coded as follows: 1=not at all, 2=when the pain is bad, 3= when I remember, when indoors, or for some activities and 4=as a daily approach. This represents a hierarchy, derived from consultation with two experts in the field of persistent pain, such that 1 indicates the poorest level of adherence and 4 indicates the best level.

Cognitive Techniques Frequency - patients reported how often they were using the methods taught for challenging unhelpful thoughts (e.g. identifying thought biases and looking for evidence) by ticking one of six categories, where 1 indicates that they had stopped completely and 6 indicates they were doing them at least once a day.

Cognitive Techniques Occasion - patients reported the occasions when they were using

the cognitive techniques, where 1=not at all, 2=when I remember, 3=when the pain is

bad, 4=when I'm anxious, 5=when I'm depressed, 6=when someone upsets me and 7=as

a daily approach. Again following discussion with two experts in the field, items on this

scale were recoded to produce a hierarchy, where 1 indicated the poorest level of

adherence and 4 indicated the best level of adherence. The re-coded scale was as

follows: 1=not at all, 2=when the pain is bad or when I remember, 3= when I'm

depressed, when I'm anxious, or if someone upsets me, and 4= as a daily approach.

Data Analysis

SPSS (Windows version 11.0) and AMOS (Version 5.0; Arbuckle & Wothke, 1999)

software packages were used during data analysis.

Missing Data Techniques

Table 2 illustrates the percentages of missing data for each measure within each data

sample, and more specifically, Table 3 shows the number of responses within each

category for each adherence variable.

Large amounts of missing data are problematic within research studies as it makes the

sample used unrepresentative, and, if the data are not missing through random processes

but through some systematic effect, it also threatens internal validity. Table 2 reveals

that the amounts of missing data range from 0% to 4.1%, with the highest percentage

being for Mood at Time 3 for the exploratory sample. This can be considered to be well

within acceptable limits (Field, 2005). Reasons for missing patient data include (a)

measure was not administered to the patient; (b) data entry clerk did not input the data;

and (c) patient did not attend the appointment. Sorting of the data by treatment group

(patients attended their inpatient treatment in small groups, which were numbered)

revealed that data were mainly missing for whole groups of patients at each time point,

indicating that the main reason for missing data was due to reason (b), that is, due to

difficulties with staff-related data collection and entry rather than due to patient attrition.

This conclusion was confirmed through consultation with a member of staff at the pain

management unit, who revealed that pockets of data that were gathered from patients

were not entered onto the database by the data entry clerk. Therefore it appears that data

were missing at random.

Table 2: Missing Data Percentages for each Variable

Variable	Exploratory Sample (n=1204) (% data missing)	Validation Sample (n=1231) (% data missing)
Measures (time 1)		
Mood	0.0%	0.0%
Self efficacy	2.4%	2.8%
Measures (time 2)		
Mood	0.0%	0.0%
Self efficacy	2.3%	2.9%
Measures (time 3)		
Mood	4.1%	3.5%
Self efficacy	1.5%	2.1%
Non-catastrophic thinking	0.0%	0.0%
Occ. of pacing	0.0%	0.0%
Occ. of challenging thoughts	0.2%	0.2%
Frq. of stretching	0.1%	0.1%
Frq. of exercise	0.3%	0.5%
Frq. of pacing	0.7%	1.6%
Frq. of challenging thoughts	1.3%	1.0%

Key: Frq. =frequency; Occ. =occasion; n = number of participants

During analyses performed using SPSS, listwise deletion was the approach used to handle missing data. In this method, cases are deleted from the sample if they have missing data on any of the variables in the analysis to be conducted, producing a working sample with no missing data but a reduced sample size.

In contrast, AMOS uses a full information maximum likelihood (FIML) method (Arbuckle, 1999), otherwise known as direct maximum likelihood, for dealing with missing data. This means that the software recognises missing data and uses all observed data values to estimate models. This method assumes that the data have multivariate normal distribution and that the missing data mechanism can be disregarded, i.e., that the data are missing at random (Allison, 2003). FIML is the recommended estimation method of choice when the data are missing at random (Arbuckle & Wothk, 1999).

Table 3 illustrates that for all measures of adherence, across both the exploratory and validation sample groups, the majority of participants reported currently using the techniques on a daily basis. Very few participants reported having stopped using the techniques completely. This implies that most participants were adhering to treatment recommendations to a greater or lesser degree at one-month follow-up.

Table 3

Frequency and percentage of responses in each category for adherence variables

Variable Categories	Exploratory Group (n=1204)		Validation Group (n=1231)	
	N	%	N	%
Occasion of pacing				
Not at all	62	5.1	54	4.4
When the pain is bad	65	5.4	64	5.2
When I remember, when indoors, or for some activities	456	37.9	436	35.4
As a daily approach	621	51.6	677	55
Missing data	0	0	0	0
Occasion of challenging thoughts				
Not at all	137	11.4	145	11.8
When the pain is bad or when I remember	249	20.7	244	19.8
When I'm depressed, anxious, or if someone upsets me	369	30.6	421	34.2
As a daily approach	447	37.1	419	34
Missing data	2	0.2	2	0.2
Frequency of stretching				
Stopped	47	3.9	34	2.8
< once a week	34	2.8	32	2.6
1-2 per week	82	6.8	76	6.2
3-4 per week	155	12.9	162	13.2
5-6 per week	191	15.9	196	15.9
At least once a day	694	57.6	730	59.3
Missing data	1	0.1	1	0.1
Frequency of exercise				
Stopped	78	6.5	59	4.8
< once a week	51	4.2	47	3.8
1-2 per week	116	9.6	118	9.6
3-4 per week	232	19.3	264	21.4
5-6 per week	210	17.4	219	17.8
At least once a day	513	42.6	518	42.1
Missing data	4	0.3	6	0.5
Frequency of pacing				
Stopped	65	5.4	61	5
< once a week	42	3.5	34	2.8
1-2 per week	109	9.1	117	9.5
3-4 per week	187	15.5	166	13.5
5-6 per week	237	19.7	249	20.2
At least once a day	555	46.1	584	47.4
Missing data	9	0.7	20	1.6
Frequency of challenging thoughts				
Stopped	113	9.4	129	10.5
< once a week	102	8.5	97	7.9
1-2 per week	223	18.5	236	19.2
3-4 per week	188	15.6	191	15.5
5-6 per week	219	18.2	243	19.7
At least once a day	343	28.5	323	26.2
Missing data	16	1.3	12	1

<u>Data Analysis Stages</u>

The sequence of procedures carried out to arrive at my final model is as follows:

Data Cleaning

Data were coded, univariate outliers were removed and data checked so that variables likely to load together on a factor were scored in the same direction. Data were also checked for univariate normality and were not found to be problematic. Eyeballing of histograms with normal curves for each variable indicated no difficulties with normality. Also, skewness and kurtosis values indicate that data did not depart from normality; univariate skewness values range from -0.72 to 1.23, with a mean univariate skewness of -0.37, and univariate kurtosis values range from -1.25 to 0.59, with a mean value of -0.20. Tests for normality, such as Kolmogorov-Smirnov, were not used since they are overpowered for large sample sizes.

Exploration

The exploratory sample (n = 1204) was used initially to explore which variables loaded on which factors and to investigate the pathways from post-treatment outcome to outcome at one-month follow-up.

An exploratory factor analysis (EFA) was conducted in order to determine the factor structure of the construct of adherence. The aim was to account for as much variance as possible, while keeping the number of factors extracted as small as possible.

After relevant measured variables had been entered, the following procedure was used to conduct and interpret each EFA, as suggested by Dancy and Reidy (2004). Initially a matrix of correlation coefficients was produced and a set of factors was extracted. Matrix algebra was used to reveal correlations of variables with factors (the unrotated matrix). This matrix was then rotated (Varimax rotation) to produce a matrix that contained the factor loadings (correlations of variables with factors). In addition, eigenvalues representing the proportion of variance accounted for by each factor and a scree plot (a graph indicating the number of factors plotted against variance accounted for) were produced. These were used in the interpretation of the output to decide how many factors to keep.

A number of statistical criteria were used in combination with researcher judgement to decide how many factors to retain. In keeping with convention, any factor with an eigenvalue greater than 1.00 was kept, and values just below 1.00 were inspected for possible retention. Also, the scree plots were viewed to see where the slope became a plateau, and the amount of variance accounted for by each factor was considered. The aim was to explain as much variance with the least number of factors, and to account for approximately 75% of the variance.

Finally, the factors were named using shared characteristics among the constituent variables. A minimum factor loading of approximately 0.4 to 0.5 was chosen as a criterion on which to base a decision about whether an item should be included in the naming process and interpretation of factors, although this figure is arbitrary (Dancy & Reidy, 2004). The names of the latent variables in this investigation are based on the items that operationalise them, as determined by researcher judgement.

Test of the Measurement Model

Structural Equation Modeling (SEM) is a collection of multivariate statistical techniques that allow a set of hypothesised relationships between independent variables and dependent variables to be examined. The same standard assumptions of the general linear model hold: normality, homogeneity of error variance, and independence of errors.

Using SEM, Confirmatory Factor Analysis (CFA) was initially used to develop a measurement model that demonstrated an acceptable fit to the data, to ensure that measurement of each latent variable (i.e. the unobserved factors determined from the EFA) was psychometrically sound, that is, that it tested the validity of the indicator variables (i.e. the observed/measured variables). Therefore, the measurement portion of the model reflects the degree to which the indicators represent a latent construct (i.e. factor loadings). CFA tests whether the indicator variables relate only to the latent variables they purport to represent and whether error terms only relate to each other in

the ways that have been anticipated. In the measurement model every latent variable was allowed to correlate with every other latent variable. The measurement model was identified by constraining one unstandardised factor loading for each factor and for all the error terms to 1.0. This was necessary to set the measurement scale and to ensure that the errors had the same scale of measurement as the variables. The measurement model provided the full model against which the more parsimonious structural models were later compared.

Test of the Structural Model

The measurement model was modified so that it represented the alternative theoretical models proposed (the structural models). The structural portion of the model reflects the interrelationships between constructs (e.g. parameter estimates). Linear equations are represented with arrows from the independent to the dependent variables. Path analysis was used to explore the hypothesis that adherence mediates the relationship between post-treatment psychological state and psychological state at one-month follow-up. Therefore the fit of the whole model (measurement and structural) was assessed.

Criteria for Mediation

Baron and Kenny (1986) describe a three step process for determining mediation. Firstly, the proposed mediators are regressed on the predictor. Secondly, the criterion is regressed on the predictor. Finally, the criterion is regressed on both the predictor and the mediators. These steps are illustrated using path diagrams in Figure 1. Mediation

would be implied in the present study if a.) adherence factors are found to be significantly correlated to psychological state post-treatment, b.) psychological state at one-month follow-up is significantly related to psychological state post-treatment, and c.) the relationship between psychological state post-treatment and psychological state at one-month follow-up decreases (or goes to zero) when adherence factors are entered into the equation. Similarly, Holmbeck (1997) stated that a pre-condition for examining mediated relationships is that the independent variable is significantly associated with the dependent variable prior to testing any model for mediating variables. He stated that the extent to which the introduction of the hypothesised mediating variables reduces the magnitude of any direct influence is of importance.

Estimation and Model Fit

Maximum likelihood estimation (MLE) methods were used to estimate all model parameters simultaneously; this is the most commonly used algorithm in model fitting programmes (Kline, 1998). As opposed to ordinary least squares estimation (which seeks to minimise the sum of squared distances of the data points to the regression line), MLE seeks to maximise the log likelihood (the odds) that the observed values of the dependent may be predicted from the observed values of the independents. MLE is an iterative process and is appropriate for use with a large sample size and when data are normally distributed and continuous (Hox & Bechger, 1998). Goodness-of-fit indices were chosen from the array provided by AMOS after consideration regarding critical factors such as the large sample size, the complexity of the model, and the estimation

87

procedure. The chi-square test of fit (CMIN) (χ^2), the goodness-of-fit index (GFI), the

comparative fit index (CFI; Bentler, 1990), the Tucker-Lewis index (TLI; Bentler &

Bonett, 1980), and the root-mean square error of approximation (RMSEA; Browne &

Cudeck, 1993) with a 90% confidence limit were used. Chi-square difference tests were

used to compare nested models. Critical ratios (CRs) were examined to evaluate the

significance of model parameters. The statistics used to evaluate the models are shown

in Table 4.

Figure 1 – Path diagrams illustrating Baron and Kenny's (1986) steps for determining

mediation

Step 1 -
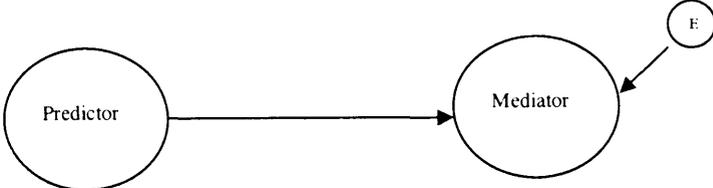
Step 2 –
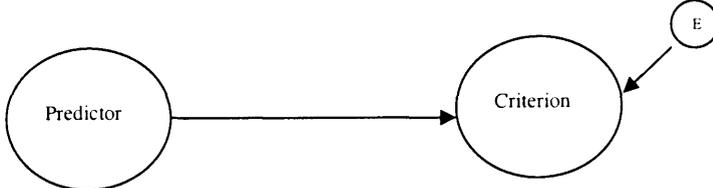
Step 3 –
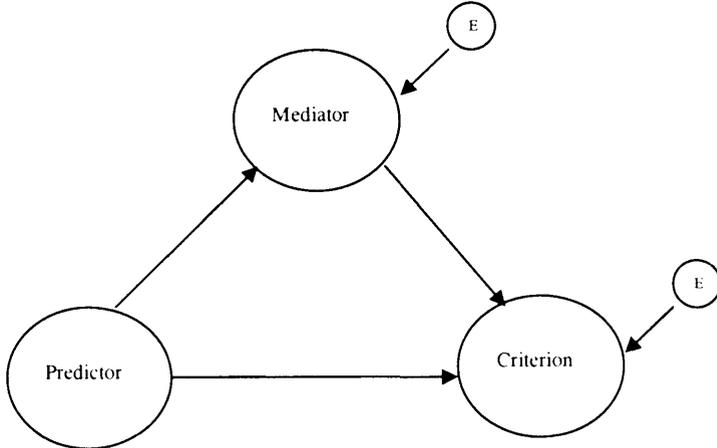
Key: Circles represent latent (unobserved) variables; E = Residual (error in the prediction of endogenous variables)

Table 4: Statistics used to evaluate model fit

| Index	Type	Description of measures of fit/ratios	Good fit/significance indicated by
χ^2 (CMIN)	Likelihood ratio test	Represents the discrepancy between the unrestricted sample covariance matrix (saturated model) and the restricted covariance matrix (hypothesised model)	Not significant value
CR	Comparative index	Critical ratio is an estimate of each path coefficient divided by its standard error and is used to evaluate the significance of model parameters	>1.96 are significant at 0.05 level
GFI	Comparative index	Goodness of fit index (Jöreskog & Sörbom, 1989) measures the relative amount of variance and covariance in the sample covariance matrix	>0.90
CFI	Comparative index	Comparative fit index (Bentler, 1990) compares the hypothesised model to the independence model, giving a measure of complete covariation in the data	>0.90 (Bentler, 1992)
TLI	Comparative index	Tucker-Lewis index (Bentler & Bonett, 1980) coefficient compares the fit of the model to the independence model	>0.95
RMSEA		Root mean square of approximation (Browne & Cudeck, 1993), measures discrepancy per degree of freedom	<0.05 (Browne & Cudeck, 1993)
Hoelter's		Hoelter's critical N is the largest sample size at which the researcher could accept the model at the .05 and .01 levels	>200

The validation sample (n = 1231) was used to cross-validate the final structural model. The sensitivity of the final model was also investigated. Sensitivity testing has been recommended as a second stage in model evaluation (Leamer, 1985), to investigate whether the inclusion or exclusion of other variables in the model may have biased parameter estimates (missing variable bias). For instance, the exclusion of important variables could have inflated the estimate of the effect of adherence on psychological state.

3   *Results*

Descriptives and Correlational Analyses

Table 5 shows the descriptive statistics for all outcome and adherence variables. Intercorrelations between these variables are shown in Table 6. 67 of 78 correlations can be seen to reach significance at p<0.05. This level of significance was chosen as it is the most common level of significance reported in research (Dancy & Reidy, 2004). It means that results are considered significant if they would occur by chance 5% of the time or less.

Table 6 shows the correlations between outcome and adherence measures. Pre-treatment outcome measures, mood at time 1 and self-efficacy at time 1, were each shown to be related to outcome measures post-treatment (at time 2) and at one-month

follow-up (at time 3). Also, each post-treatment outcome variable (at time 2) was shown to be significantly associated with outcome at one-month follow-up (at time 3). This implies that psychological state pre-treatment has an impact on psychological state post-treatment, which in turn is associated with psychological state at one-month follow-up.

The relevant correlations between outcome measures and adherence variables are also shown in Table 6. Two findings are of particular interest here: a.) that pre-treatment variables did not appear to be related to adherence; and b.) that post-treatment and one-month follow-up outcome variables each showed a strong relationship with adherence.

a.) Pre-treatment scores on outcome measures were not consistently related to measures of adherence. Mood at time 1 was not significantly associated with any of the adherence variables, except frequency of stretching (r=0.11, n=1203, p<0.01) and frequency of pacing (r=0.08, n=1195, p<0.01). Also, the only measure of adherence significantly related to self-efficacy at time 1 was frequency of stretching (r=0.09, n=1174, p<0.01). This indicated that psychological state pre-treatment shared little variance with participants' extent of adherence after treatment has ended.

b.) Mood and self-efficacy post-treatment were however significantly related to all measures of adherence, except frequency of challenging thoughts. Also, there were significant correlations between mood and self-efficacy at one-month follow-up and all adherence measures. This implies that participants are more likely to adhere if they had higher post-treatment scores, and that

they were more likely to do better at follow-up if they have adhered.

Alternatively, these results could imply that participants are more likely to

report higher levels of adherence if they were doing well at follow-up.

Finally, Table 6 shows that all adherence variables were positively correlated with one

another. This indicates that an individual who adheres to one technique is likely to

adhere to the others.

Differences in the mean scores of outcome variables over time were investigated using

repeated measures ANOVAs, to reveal whether significant improvements in outcome

were shown between pre-treatment, post-treatment, and one-month follow-up.

Therefore, analyses were performed to compare levels of mood and self-efficacy over

the three time points. Analyses revealed significant differences in levels of mood

$(F(2,1153) = 0.244$, $p<0.001)$ and levels of self-efficacy $(F(2,1134) = 0.422$, $p<0.001)$

between these time points. On average, pre-treatment levels of mood were lower

(M=13.98, SD=3.43) than at post-treatment (M=15.70, SD=2.76). Similarly, pre-

treatment mean levels of self-efficacy were lower (M=24.48, SD=12.04) than at post-

treatment (M=34.55, SD=12.97). Pre-treatment levels of mood and self-efficacy were

also shown to be lower than at one-month follow-up, M=15.50, SD=3.02, and M=33.06,

SD=13.57 respectively. These results imply that improvements in mood and self-

efficacy had taken place by the end of treatment, and that these improvements were

maintained at one-month follow-up.

Table 5: Descriptive Statistics for Outcome and Adherence Variables

Variable	Participants Exploratory Group (n=1204)			Validation Group (n=1231)		
	Mean	SD	N (% data missing)	Mean	SD	N (% data missing)
Measures (time 1)						
Mood	13.98	3.43	1204 (0)	14.14	3.44	1231 (0)
Self efficacy	24.48	12.04	1175 (2.4)	24.25	11.88	1196 (2.8)
Measures (time 2)						
Mood	15.70	2.76	1204 (0)	15.89	2.61	1231 (0)
Self efficacy	34.55	12.97	1176 (2.3)	34.38	12.94	1195 (2.9)
Measures (time 3)						
Mood	15.50	3.02	1155 (4.1)	15.59	2.86	1188 (3.5)
Self efficacy	33.06	13.57	1186 (1.5)	33.10	13.67	1205 (2.1)
Non-catastrophic thinking	35.80	12.86	1204 (0)	36.02	12.66	1231 (0)
Occasion of pacing	3.36	0.81	1204 (0)	3.41	0.78	1231 (0)
Occasion of challenging thoughts	2.94	1.02	1202 (0.2)	2.91	1.00	1229 (0.2)
Frequency of stretching	4.07	1.36	1203 (0.1)	4.15	1.28	1230 (0.1)
Frequency of exercise	3.65	1.52	1200 (0.3)	3.71	1.43	1225 (0.5)
Frequency of pacing	3.80	1.46	1195 (0.7)	3.87	1.43	1211 (1.6)
Frequency of challenging thoughts	3.12	1.65	1188 (1.3)	3.06	1.65	1219 (1.0)

Key: N/n = number of participants; SD = standard deviation

Table 6

Intercorrelations Between All Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
Outcome Variables													
1. Mood (time 1)	1												
2. Self efficacy (time 1)	.370**	1											
3. Mood (time 2)	.506**	.267**	1										
4. Self efficacy (time 2)	.232**	.534**	.390**	1									
5. Mood (time 3)	.553**	.255**	.625**	.346**	1								
6. Self efficacy (time 3)	.258**	.538**	.340**	.735**	.452**	1							
7. Non-catastrophic thinking (time 3)	.317**	.292**	.337**	.460**	.480**	.547**	1						
Adherence variables													
8. Occ. of pacing (time 3)	.055	.036	.085**	.160**	.135**	.203**	.151**	1					
9. Occ. of challenging thoughts (time 3)	.028	.036	.065*	.110**	.105**	.202**	.145**	.338**	1				
10. Frq. of stretching (time 3)	.107**	.094**	.108**	.196**	.148**	.264**	.155**	.347**	.246**	1			
11. Frq. of exercise (time 3)	.048	.039	.067*	.151**	.122**	.242**	.148**	.361**	.227**	.661**	1		
12. Frq. of pacing (time 3)	.083**	.004	.088**	.131**	.125**	.180**	.163**	.682**	.329**	.407**	.422**		
13. Frq. of challenging thoughts (time 3)	.019	.010	.010	.055	.064*	.161**	.096**	.286**	.734**	.260**	.254**	.377**	1

(*$p<.05$. **$p<.01$. All tests were two-tailed).

Key: Occ = occasion; Frq = Frequency

Exploratory Factor Analysis

Adherence scores were submitted to principal components factor analysis (Varimax rotation). Three orthogonal factors were extracted and a summary of the output is shown in Table 7. The rotated solution accounted for all 6 capacities (extraction communalities ranged from 0.83 to 0.87), explained 85% of rating variance, and yielded two factors with eigenvalues over 1.0. The first factor, termed Cognitive Adherence (eigenvalue = 2.98) accounted for 50% of the variance and included two capacities: occasion of challenging thoughts and frequency of challenging thoughts. A second factor, Physical Adherence (eigenvalue = 1.23), accounted for 21% of the variance and included two capacities: frequency of stretching and frequency of exercise. A third factor, Pacing Adherence (eigenvalue = 0.87) was also extracted. Although this factor did not yield an eigenvalue greater than 1.0, it accounted for 15% of the variance and included two capacities: occasion of pacing and frequency of pacing. The clarity of the loadings and the absence of substantial cross-loadings indicated that this solution was clean. The solution showed that the six adherence measures loaded onto three factors, and these were used throughout the remainder of the analyses as latent variables of adherence.

Post-treatment and one-month follow-up outcome variables were each combined into a single latent variable called 'psychological state', with two and three observed variables respectively. This decision was based on the premise that it made theoretical sense to combine these variables, and because the significant correlations in Table 6 corroborated this.

96

Table 7: Factor Loadings, Communalities, Eigenvalues, and Percentage of Variance for each adherence factor

Item	Factor 1: Cognitive Adherence	Factor 2: Physical Adherence	Factor 3: Pacing Adherence	Communalities
Occ. of challenging thoughts	0.91	0.10	0.18	0.87
Frq. of challenging thoughts	0.91	0.14	0.16	0.87
Frq. of stretching	0.13	0.88	0.19	0.83
Frq. of exercise	0.10	0.88	0.22	0.83
Occ. of pacing	0.15	0.17	0.90	0.86
Frq. of pacing	0.21	0.26	0.85	0.83
Eigenvalue	2.98	1.23	0.87	
Percent of Variance	49.64	20.50	14.54	**Total Variance = 84.68**

Key: Occ = Occasion; Frq = Frequency

Model Estimation

Structural equation modeling was used to investigate the relationships among psychological state post-treatment, adherence factors and psychological state at follow-up. A two-stage SEM approach was adopted in order to first test the quality of the measurement items before proceeding to the estimation of the structural model (Hulland, Chow, & Lam, 1996). Based on the findings from the EFA and correlations, latent variables were constructed, with indicators, and a Measurement Model A was specified. Table 8 shows the chi-squared values and goodness of fit indices for each model tested.

Measurement Model

The hypothesised Measurement Model A (MMA) illustrating the hypothesised links between psychological state post-treatment, psychological state at 1-month follow-up, and adherence, is presented in Figure 2. In this model, circles represent unobserved variables (factors and error terms), while observed variables (scale scores) are represented by rectangles. The two-headed arrows linking each of the latent variables represent the correlations among the factors.

The latent factor 'Psychological state Time 2' is comprised of the observed indicators Mood at time 2 (Mood2) and Self Efficacy at time 2 (Selfef2). Similarly, the latent factor 'Psychological state Time 3' is comprised of the observed indicators Mood at time 3 (Mood3) and Self Efficacy at time 3 (Selfef3). In addition, the scale of Non-catastrophic Thinking at time 3 (Uncata3) also loads on the psychological state factor at time 3. The adherence indicators of occasion of challenging thoughts

(cthocc3), frequency of challenging thoughts (cthoug3), frequency of stretching

(stretc3), frequency of exercise (exerc3), occasion of pacing (paceoc3), and

frequency of pacing (pace3) load onto the three adherence factors as described by the

exploratory factor analysis. Err1-err11 are the error terms for the measures.

Figure 2: Measurement Model A, showing standardised coefficients. *p<0.05.
**p<0.01.
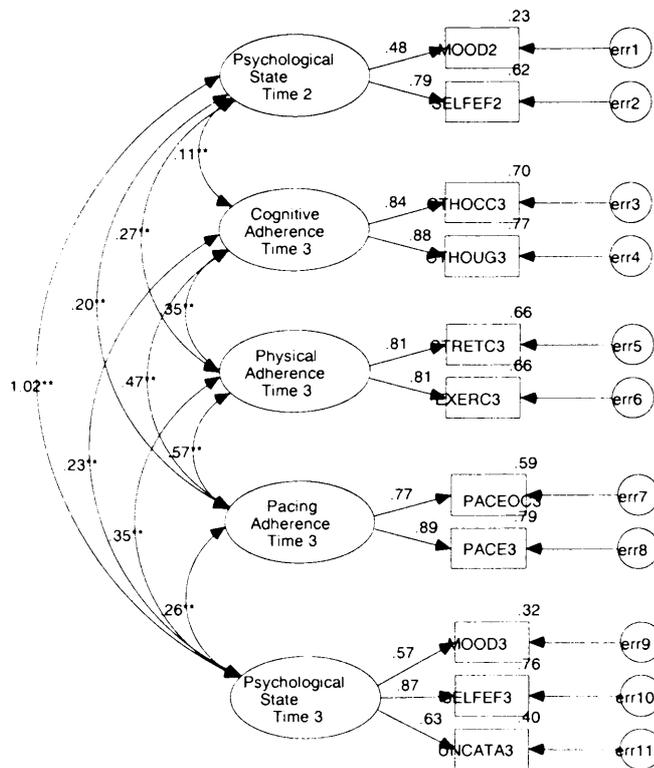
The confirmatory factor analysis resulted in a statistically significant chi-square value for this model (χ^2 (34) =573.58, p<0.001). The fit of the Measurement Model A was also shown to be 'not positive definite', indicating that it was clearly miss-specified and that the solution was inadmissible. Examination of the correlations shown in Figure 2 between the latent factors 'Psychological state Time 2' and 'Psychological state Time 3' showed the correlation to be 1.02. This correlation being greater than 1.0 indicated a problem of multicollinearity (Byrne, 2001), whereby the observed variables were so highly correlated that they seemed to represent the same underlying construct. This was not surprising given that the underlying constructs in question were the same, but measured at different time points. Therefore post hoc modifications were required to find a measurement model that adequately fitted the data.

Post Hoc Adjustments

The measurement model was re-specified as shown in Measurement Model B (MMB) in Figure 3. As the same measures were administered at different time points for the psychological state factors, two correlations among their disturbances were allowed and tested. The two-headed arrows linking err1 and err9, and err2 and err10 represent the autocorrelations over time for the error terms for the Mood-time 2 and Mood-time 3 variables, and for the Self-efficacy variables at each time point.

Figure 3: Measurement Model B, showing standardised coefficients. *p<.05. **p<0.01.
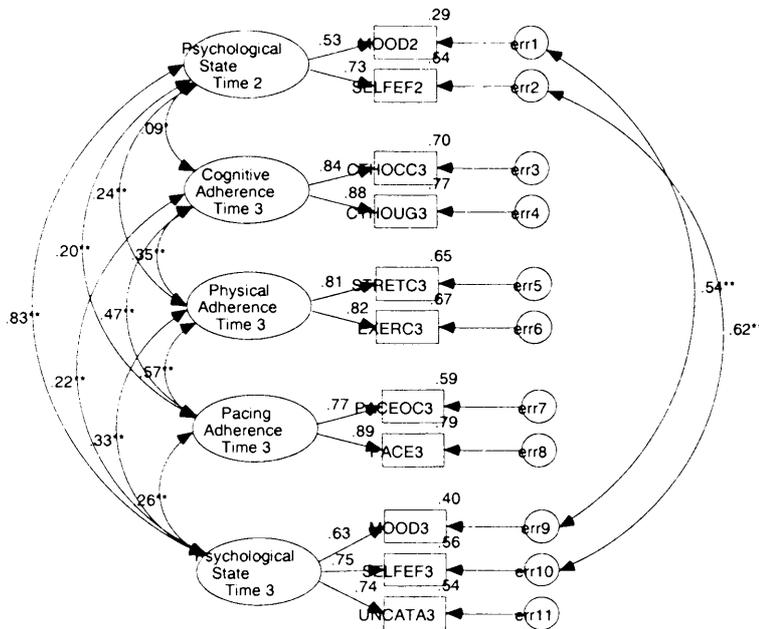

Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Cthocc3 =Occasion of challenging thoughts (one-month follow-up); Cthoug3 =Frequency of challenging thoughts (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err 1-11=Error terms

A statistically significant chi-square value was again produced for this model (χ^2 (32) = 96.64, p<0.001). However, the chi-square statistic is unreliable for such large samples; therefore the fit of the revised Measurement Model B was assessed using a range of goodness of fit indices as shown in Table 8. Each of these implied a good fit; the χ^2/df ratio was 3.02, the CFI was 0.99 and the RMSEA was 0.04. All of these values indicated that the re-specified measurement model achieved a good fit of the data.

101

The standardised estimates show that the value 0.83 is the correlation between Psychological state Time 2 and Psychological state Time 3. This indicates a tendency for patients who have higher levels of wellbeing post-treatment also to have higher levels of wellbeing at one-month follow-up. Figure 3 reveals that the psychological state post-treatment was significantly correlated with each adherence factor, indicating a tendency for patients who had higher levels of wellbeing to be more likely to adhere. It can be seen that all correlations between the latent factors in the model were highly significant, with only the correlation between psychological state post-treatment and cognitive adherence being weaker, although still significant (r=0.09, p=0.026). As expected, significant correlations were shown between the error terms of mood and self-efficacy at the two time points. This implies that the scales of mood and self-efficacy shared some variance over time that was not related to the psychological state factor. The R^2 values for the mood measure at both time points indicated that 29% and 40% of the variance in this measure was shared with the psychological state factor at time 2 and time 3 respectively. These values therefore represented the reliability estimates of the Mood 2 and Mood 3 indicator variables respectively. All other indicator variables were seen to show much greater estimates of reliability (R^2 values range from 54% to 79%), indicating that they more reliably measure the factors implied in the model. Since mood and self-efficacy scales are not designed to measure the same construct, it seems reasonable to believe that differences between them are due to more than just measurement error. Therefore error terms represent not only measurement error, but every other variable that might affect scores on them besides psychological state, the major variable that affects them both.

Table 8
Chi-Squared Values and Fit Indices for All Models

|	χ^2	df	cmin/df	CFI	TLI	RMSEA	Hoelter's
Measurement Models							
MMA	575.58	34	16.93	0.90	0.81	0.12	102
MMB	96.64	32	3.02	0.99	0.98	0.04	576
MMC	34.28	6	5.71	0.99	0.97	0.06	442
MMD	4.18	5	0.84	1.00	1.00	0.00	3185
Structural Models							
SM1	4.56	2	2.28	1.00	0.99	0.03	1581
SM2a	4.95	2	2.48	1.00	0.99	0.04	1456
SM2b	9.62	5	1.92	1.00	0.99	0.03	1385
SM2c	25.97	10	2.60	1.00	0.99	0.04	849
SM2d	49.41	11	4.49	0.99	0.97	0.05	480
SM2e	37.40	11	3.40	0.99	0.98	0.05	633
SM3a	0.90	1	0.90	1.00	1.00	0.00	5120
SM3b	6.40	4	1.60	1.00	0.99	0.02	1785
SM3c	13.06	9	1.45	1.00	0.10	0.02	1559
SM3d	25.15	10	2.51	1.00	0.99	0.04	876
SM3e	40.98	10	4.10	0.99	0.97	0.05	538
SM4a	0.82	1	0.82	1.00	1.00	0.00	5674
SM4b	23.21	4	5.80	0.99	0.95	0.06	492
SM4c	31.73	9	3.53	0.99	0.98	0.05	642
SM4d	53.11	10	5.31	0.99	0.96	0.06	415
SM4e	65.88	10	6.59	0.98	0.95	0.07	335
SM5a	40.00	15	2.67	0.99	0.98	0.04	752
SM5b	65.38	22	2.97	0.99	0.98	0.04	625
SM5c	91.10	35	2.60	0.99	0.98	0.04	658
SM5d	119.77	36	3.33	0.98	0.97	0.04	513
SM5e	123.87	36	3.44	0.98	0.97	0.05	496
Validation	87.44	35	2.50	0.99	0.98	0.04	701
Sensitivity	207.08	71	2.92	0.98	0.96	0.04	533

Key: χ^2 = Chi square value; df = degrees of freedom; cmin/df = χ^2/degrees of freedom ratio; CFI = Comparative fit index; TLI = Tucker-Lewis index; RMSEA = Root mean square error of approximation; Hoelter's = Hoelter's critical sample size

The Structural Models

Direct Pathway

In accordance with Holmbeck (1997), the first step in developing models which test

mediation was to show that there was a direct causal effect between post-treatment

outcome and one-month follow-up outcome to be mediated. Figure 4 shows

Structural Model 1 (SM1) illustrating the hypothesised effect of post-treatment

outcome on follow-up outcome. The output for this model is also illustrated in

Figure 4. In SM1, the correlation among the factors has been replaced by a causal

path (indicated by a one-headed arrow): the causal effect of psychological state post-

treatment on psychological state at one-month follow-up. For identification, an error

term was added to the dependent variable, while other procedures for identification

remained the same as for the measurement model.

Figure 4: Structural Model 1, showing standardised coefficients. *p<.05. **p<0.01.
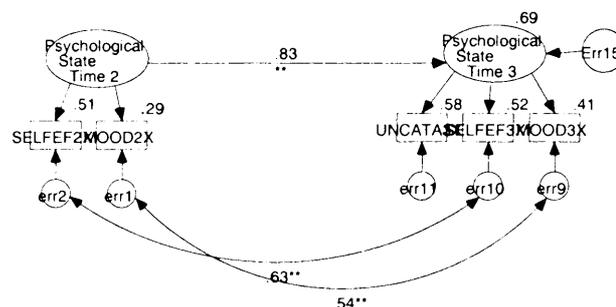
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Err =Error terms

Fit indices (see Table 8) for Model SM1 indicated an excellent fit. The causal effects

of psychological state post-treatment on psychological state at one-month follow-up

were significant. Psychological state at the end of treatment was a statistically significant predictor of psychological state at one-month follow-up (β = 0.83, SE =0.07, p<0.001). Therefore, a significant direct pathway from post-treatment outcome to one-month follow-up outcome was shown to exist. The regression analyses revealed that psychological state at the end of treatment explained 69% of the variance (R^2=0.69) in psychological state scores at one-month follow-up.

Mediating Models

A series of models explored the mediating influences of cognitive adherence, physical adherence, and pacing adherence between post-treatment outcome and follow-up outcome individually, to initially investigate whether any of the types of adherence had a mediating impact on the relationship between psychological state at the end of treatment and at follow-up.

1.)   Does Cognitive Adherence have a Mediating Role?

In line with Baron and Kenny's (1986) steps to test for mediation, three separate causal models (path analyses) were run: see Models SM2a, SM2b and SM2c in Figures 5, 6 and 7 respectively. Model SM2a tests for an association between psychological state post-treatment and cognitive adherence, Model SM2b tests for the influence of cognitive adherence on psychological state at one-month follow-up, and Model SM2c tests the mediating pathway. Model SM2c is therefore based on the assumption that both psychological state post-treatment and cognitive adherence make independent contributions to psychological state at follow-up, and that cognitive adherence is predicted by psychological state post-treatment.

*Figure 5*: Model SM2a, showing standardised coefficients. *p<.05. **p<0.01.
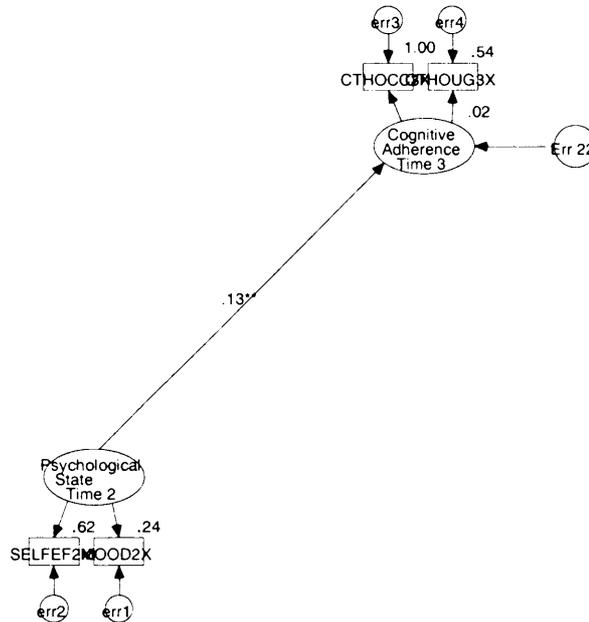
Model SM2a shows a direct path from psychological state post-treatment to

cognitive adherence. The total variance in cognitive adherence accounted for by this

model was only 2% (R^2=0.02), although fit indices (see Table 8) suggested a good

fit. The model showed that psychological state post-treatment had a significant

direct path to cognitive adherence (ß=0.13, p<0.001).

In model SM2b cognitive adherence is assumed to predict psychological state at

follow-up. The causal effect of cognitive adherence on psychological state at follow-

up was 0.22 (p<0.01). The total variance in follow-up outcome accounted for by this

model was 5% (R^2=0.05), and again fit indices showed good fit. The model

illustrated that cognitive adherence had a significant direct path to psychological

state at follow-up.

Figure 6: Model SM2b, showing standardised coefficients. *p<.05. **p<0.01.
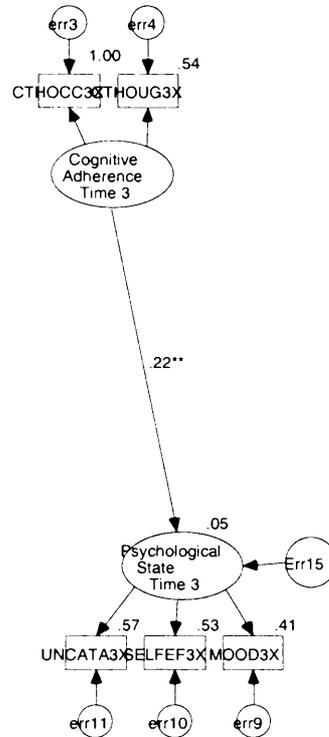
Fit indices for Model SM2c show that this model also has good fit. Model SM2c output indicates that when the cognitive adherence factor was entered, psychological state post treatment was still a statistically significant predictor of psychological state at one-month follow-up ($\text{ß}=0.81$, $p<0.01$) at one-month follow-up, although the strength of the relationship has decreased. From Figure 7 it is apparent that the introduction of the mediating variable is accompanied by a drop in the values of the regression coefficient ($\text{ß}=0.81$, while in Model SM1 $\text{ß}=0.83$) representing the direct effect of psychological state post treatment on psychological state at one-month follow-up. This suggests that cognitive adherence partially mediates the relationship between these two variables. The total variance in psychological state at follow-up

accounted for by this model was 70% (R²=0.70). Also, Figure 7 shows that the

addition of cognitive adherence factor to the model only contributes an additional 1%

of the explained variance in psychological state at one-month follow-up.

Figure 7: Model SM2c, showing standardised coefficients. *p<.05. **p<0.01.
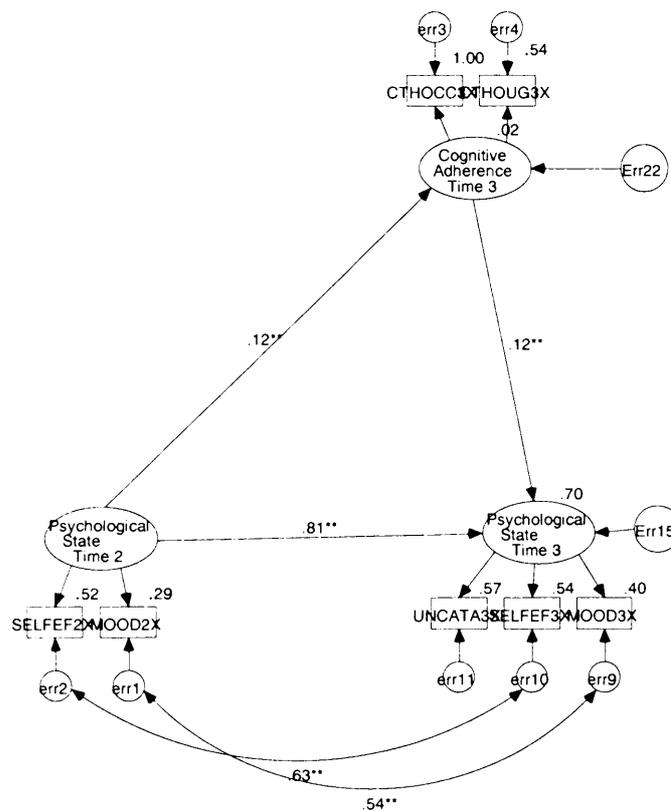
*Key:*   Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment);
Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Cthocc3 =Occasion of challenging thoughts (one-month follow-up); Cthoug3 =Frequency of challenging thoughts (one-month follow-up); Err =Error terms

Comparison with nested models

Because the mediating role of cognitive adherence was not the only plausible model,

model SM2c was compared with two nested models representing alternative

hypotheses. Model SM2d (shown in Figure 8) assumes that psychological state time

2 predicts cognitive adherence and psychological state time 3, but that no path

between cognitive adherence and psychological state time 3 exists. By comparison,

in Model SM2e (shown in Figure 9) psychological state time 2 and cognitive

adherence are assumed to be related to psychological state time 3, but there is no link

between psychological state time 2 and cognitive adherence. Fit indices reveal that

both Model SM2d and SM2e fit the data adequately.

Figure 8: Model SM2d, showing standardised coefficients. *p<.05. **p<0.01.
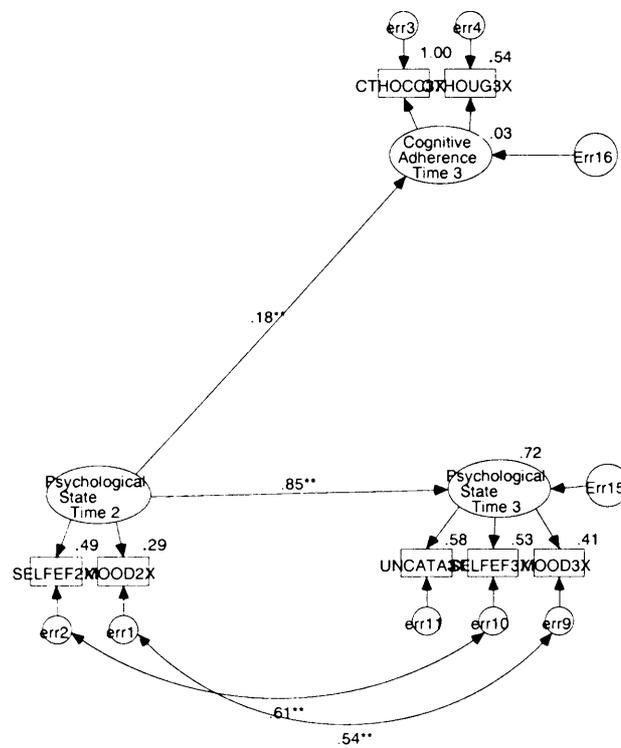
Figure 9: Model SM2e, showing standardised coefficients. *p<.05. **p<0.01.
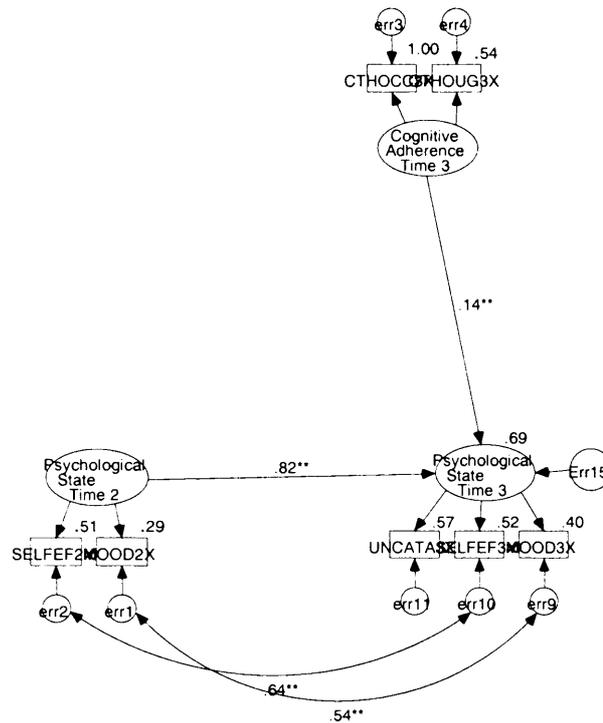
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Cthocc3 =Occasion of challenging thoughts (one-month follow-up); Cthoug3 =Frequency of challenging thoughts (one-month follow-up); Err =Error terms

Because of differences in degrees of freedom between models SM2c and SM2d and between SM2c and SM2e, it was possible to compare these two pairs on the basis on change in χ^2. Model SM2c was a significantly better fit over Model SM2d, χ^2diff (1) = 23.44, p<.001, and over Model SM2e, χ^2diff (1) = 11.44, p<.001. Comparing fit indices also revealed that Model SM2c had a better fit. This again suggests that cognitive adherence has a mediating role in the relationship between psychological state post-treatment and psychological state at one-month follow-up.

Does Pacing Adherence have a Mediating Role?

The path analyses reported above were re-run using the pacing adherence factor, to investigate whether it too had a mediating effect. Models SM3a, SM3b and SM3c are shown in Figures 10, 11 and 12 respectively. Each model was found to provide an excellent fit with the data, as shown from the fit indices detailed in Table 8.

Model SM3a in Figure 10 predicts that psychological state at time 2 effects level of pacing adherence. The total variance accounted for by this model was 4% (R^2=0.04) and fit indices suggested an excellent fit. This model therefore implies that psychological state at time 2 has a significant direst path (β=0.21) to pacing adherence.

Figure 10: SM3a, showing standardised coefficients. *p<.05. **p<0.01.
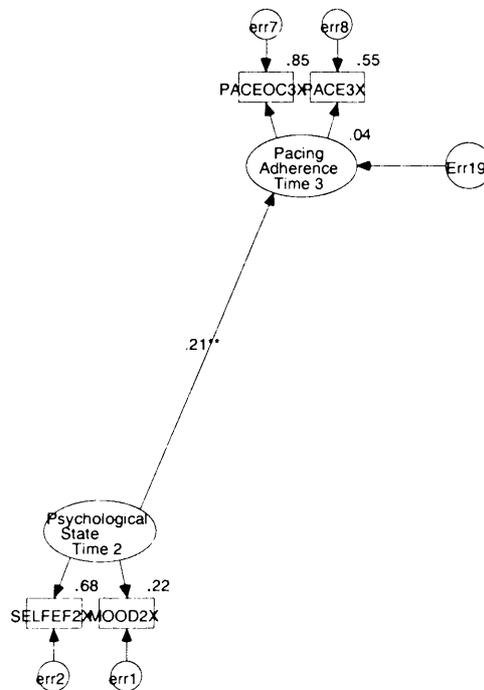
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err =Error terms

Figure 11: SM3b, showing standardised coefficients. *p<.05. **p<0.01.
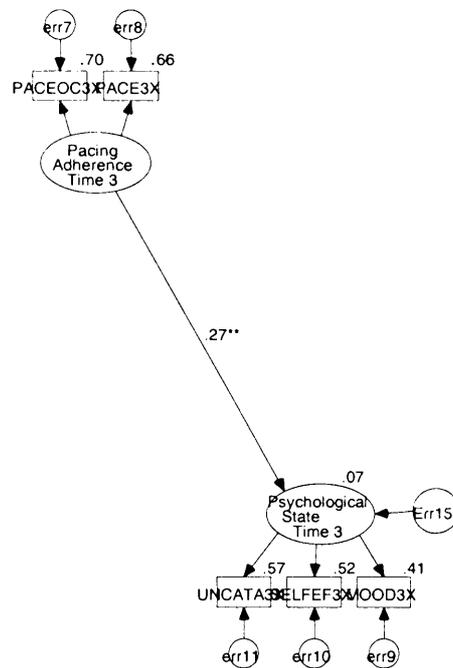
Key: Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err =Error terms

Model SM3b in Figure 11 implies that pacing adherence has a direct association with psychological state at follow-up. The analyses reveals that pacing adherence is significantly related to psychological state at follow-up (ß=0.27), and that pacing adherence accounts for 7% of its variance (R²=0.07). This implies that pacing adherence influences follow-up psychological state.

Finally, Model SM3c in Figure 12 illustrates the hypothesised mediating model; that psychological state at the end of treatment (post-treatment) directly effects psychological state at follow-up, but also that psychological state at the end of treatment effects psychological state at follow-up indirectly via pacing adherence. This model again showed good fit with the data. Psychological state post-treatment

is seen to be a statistically significant predictor of psychological state at follow-up

(ß=0.81), and of pacing adherence (ß=0.21). Pacing adherence was also shown to

significantly predict psychological state at follow-up (ß=0.10). Psychological state

post-treatment and pacing adherence together account for 70% of the variance in

psychological state at follow-up (R^2=0.70).

Figure 12: SM3c, showing standardised coefficients. *p<.05. **p<0.01.
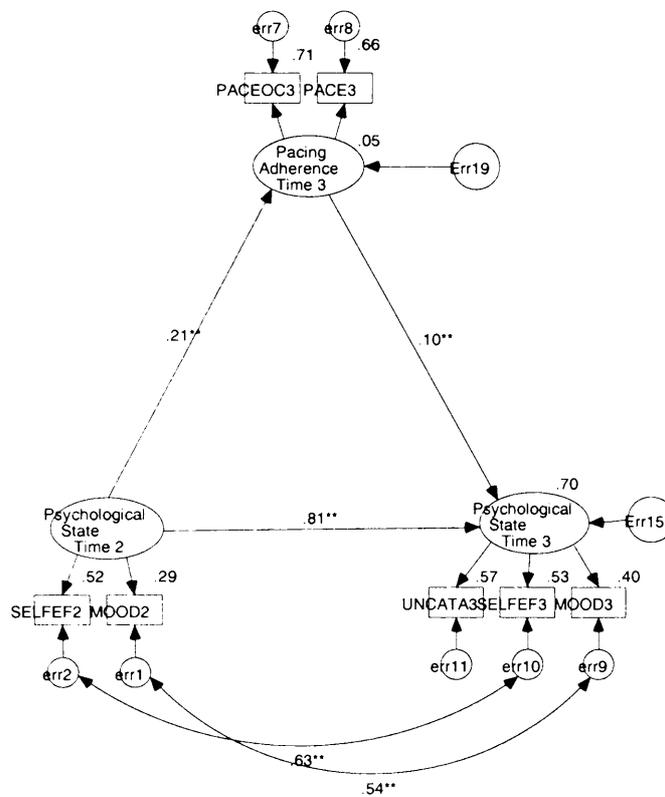
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err =Error terms

Comparison with nested models

Models SM3d and SM3e illustrated in Figures 13 and 14 again illustrate nested

models representing competing causal hypotheses. Each model showed good fit as

indicated by the fit indices in Table 8. However, examination of the χ^2 differences

113

between each of these models and SM3c revealed that model SM3c was a significant improvement on each of them (Comparing SM3c and SM3d, χ^2diff (1) = 12.08, p<.001, and comparing SM3c and SM3e, χ^2diff (1) = 27.92, p<.001). This confirms, as expected, that model SM3c, which assumes that pacing adherence has a mediating role between psychological state post-treatment and psychological state at follow-up, represents the best fitting model.

Figure 13: SM3d, showing standardised coefficients. *p<.05. **p<0.01.
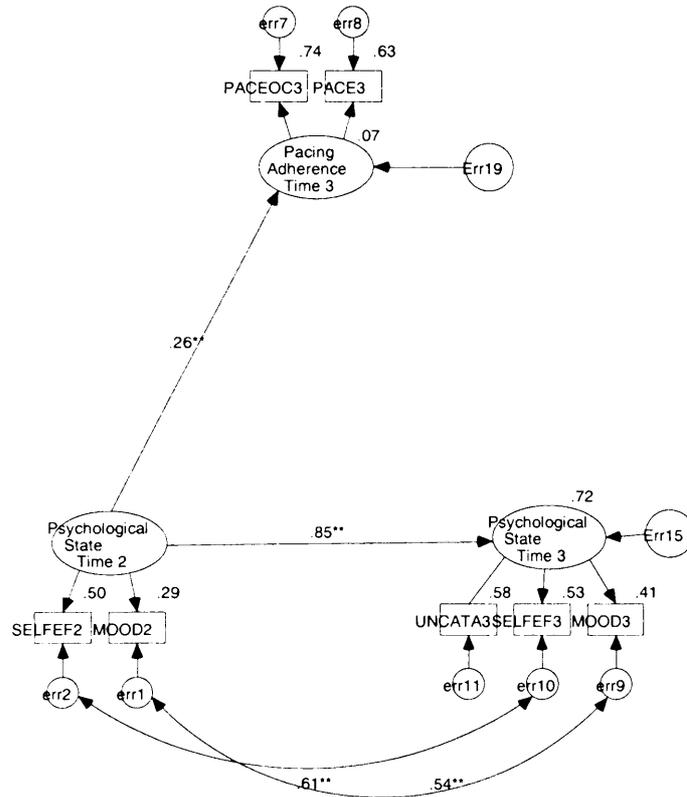
Figure 14: SM3e, showing standardised coefficients. *p<.05. **p<0.01.
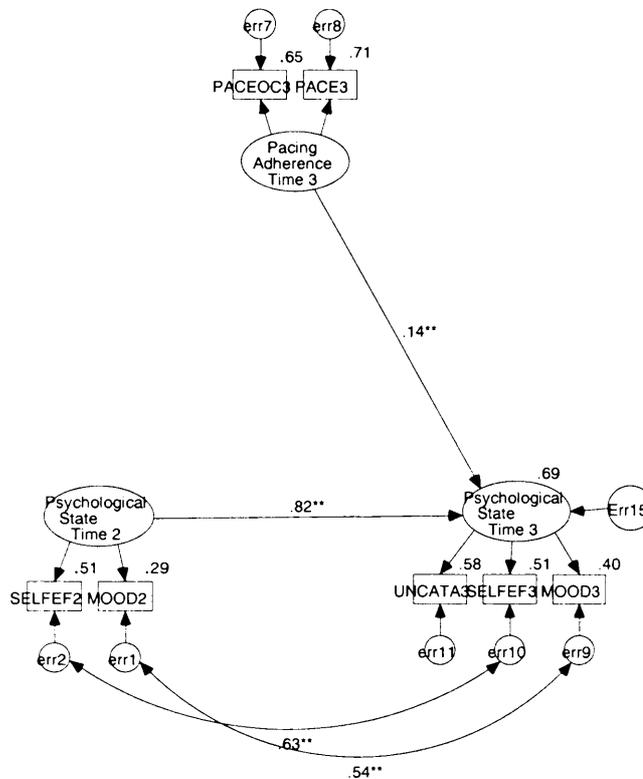
*Key:*   Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err 1-11=Error terms

Does Physical Adherence have a Mediating Role?

As an initial step to explore the hypothesised mediating role of physical adherence on the relationship between psychological state post-treatment and psychological state at follow-up, physical adherence was regressed on psychological state post-treatment as shown in Model SM4a (Figure 15). Fit indices for this model indicated good fit with the data, and a significant direct path from psychological state post-treatment to physical adherence was implied (ß=0.25). In addition, 6% of the variance in physical adherence was accounted for by this model (R^2=0.06).

Furthermore, Model SM4b in Figure 16 examines whether physical adherence predicts psychological state at one-month follow-up. It can be seen from this model that a significant direct relationship between these factors is implied (ß=0.32), and that 10% of the variance in follow-up psychological state is accounted for.

Finally, Model SM4c (Figure 17) investigates the existence of a mediating effect of physical adherence on the relationship between psychological state post-treatment and psychological state at follow-up. The analyses revealed each direct path to be significant. Most importantly, the direct effect of psychological state post-treatment to psychological state at follow-up was shown to be reduced (ß=0.80) in this model when compared to model SM1, in which no mediating variable is included. This implies that physical adherence partially mediates the relationship between these two variables. Also, the addition of physical adherence into the model results in 2% increase in the percentage of explained variance in psychological state at follow-up, with this model explaining 71% of its variance (R^2=0.71).

Figure 15: SM4a, showing standardised coefficients. *p<.05. **p<0.01.
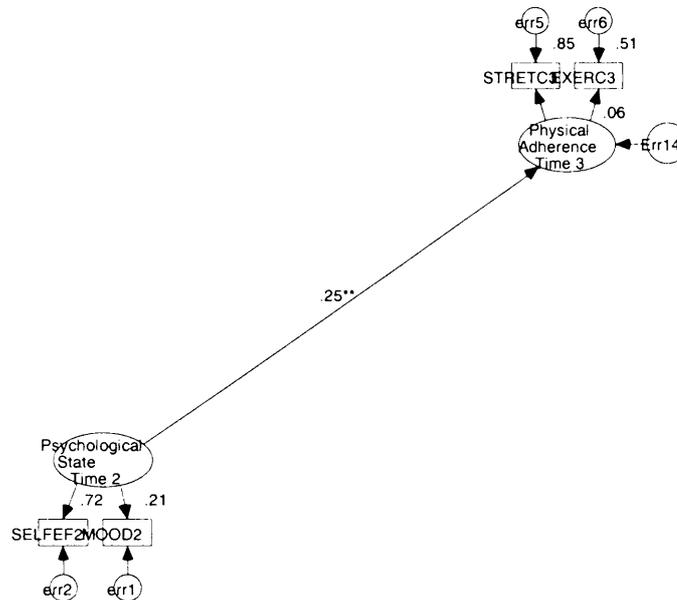
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Err =Error terms

Figure 16: SM4b, showing standardised coefficients. *p<.05. **p<0.01.
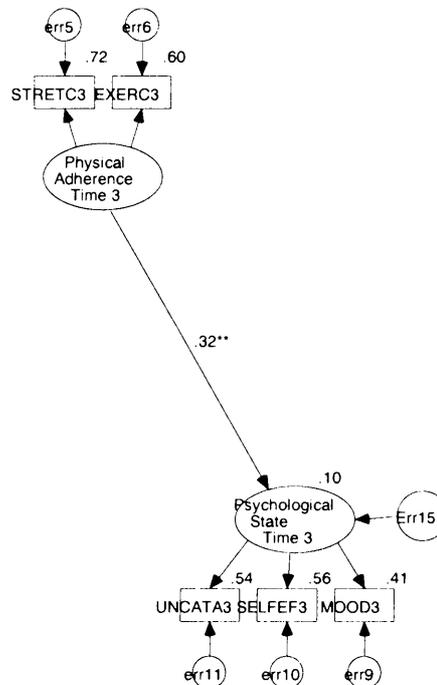
Key: Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Err =Error terms

117

Figure 17: SM4c, showing standardised coefficients. *p<.05. **p<0.01.
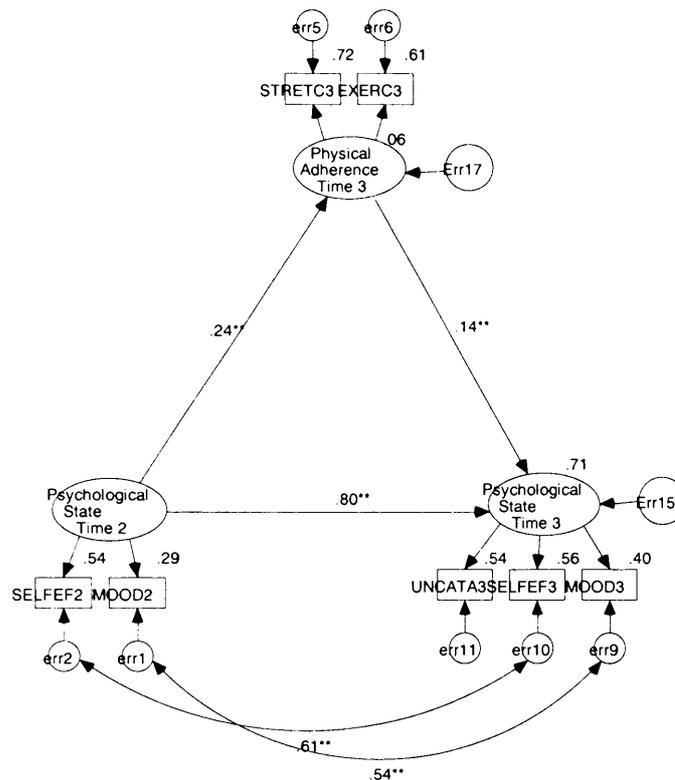
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Err =Error terms

Comparison with nested models

Models SM4d and SM4e illustrated in Figures 18 and 19 again illustrate nested

models representing the competing causal hypotheses. Examination of the χ^2

differences between each of these models and SM4c (the mediating model) revealed

that model SM4c was a significant improvement on each of them (Comparing SM4c

and SM4d, χ^2diff (1) = 21.34, p<.001, and comparing SM4c and SM4e, χ^2diff (1) =

34.15, p<.001). Fit indices also point to model SM4c being the best fit with the data.

This implies that physical adherence has a mediating role between psychological

state post-treatment and psychological state at follow-up, and that Model SM4c

represents the best fitting model.

Figure 18: SM4d, showing standardised coefficients. *p<.05. **p<0.01.
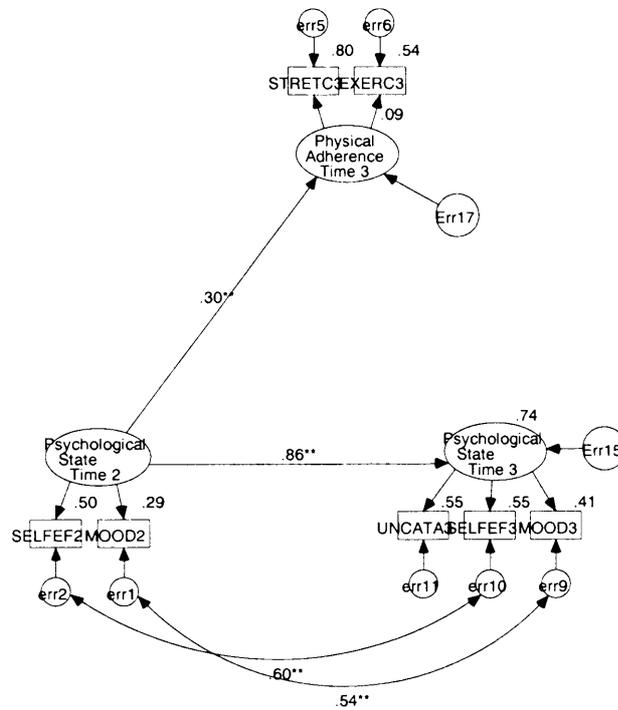
Figure 19: SM4e, showing standardised coefficients. *p<.05. **p<0.01.
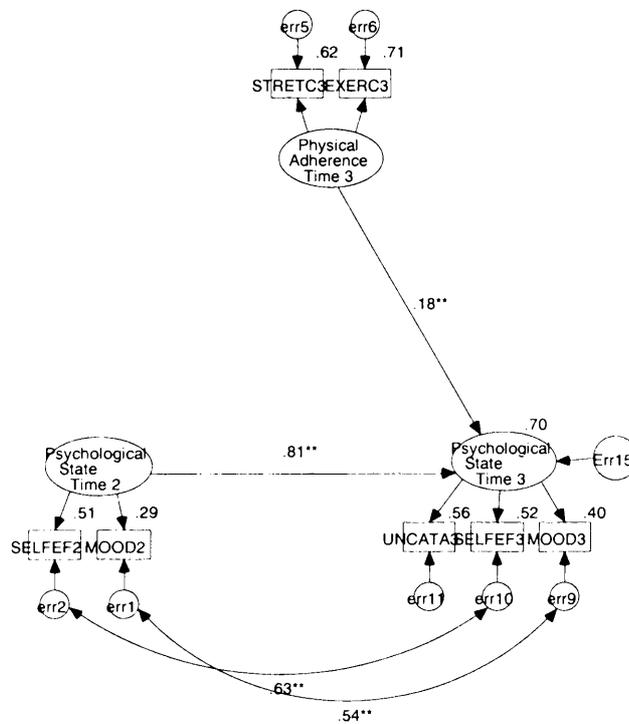
*Key:* Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Err =Error terms

Does Overall Adherence have a Mediating Role?

The results of the previous models suggest that each adherence factor (cognitive adherence, pacing adherence, and physical adherence) independently have a partial mediating effect on the relationship between post-treatment and follow-up outcomes. These path analyses therefore formed the basis for a final set of analyses to model the potential pathway from psychological state time 2 to psychological state time 3, with 'overall adherence' mediating. A second order factor, 'Overall Adherence', was therefore developed prior to being used in this final set of analyses.

Second-Order Confirmatory Factor Analysis (CFA) Model

Model MMC, shown in Figure 20, hypothesised that responses to individual adherence measures could be explained by three first-order factors (cognitive adherence, pacing adherence, and physical adherence), and by one second-order factor (overall adherence). Error terms associated with each item were hypothesised to be uncorrelated and covariation among the three first-order factors would be explained fully by their regression on the second-order factor.

Model MMC showed reasonable fit with the data as indicated by fit indices in Table 8. However, given that the items 'occasion of pacing' and 'occasion of challenging thoughts' had both been rescaled within the study, the specification of an error correlation between them was substantiated. A correlation between err3 and err7 was therefore allowed and the model was re-estimated in an effort to improve its fit, as illustrated in Model MMD (Figure 21).

Figure 20: MMC, showing standardised coefficients. *p<.05. **p<0.01.
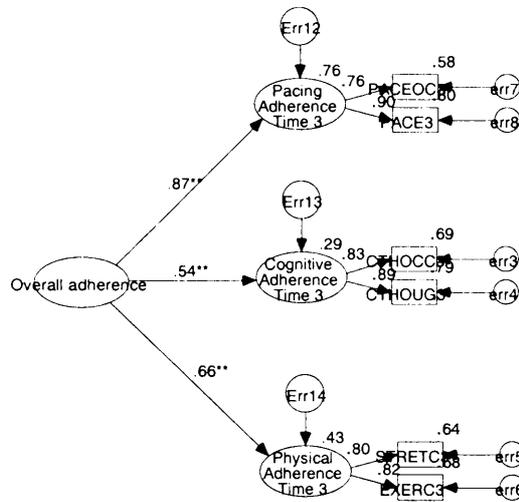
Figure 21: MMD, showing standardised coefficients. *p<.05. **p<0.01.
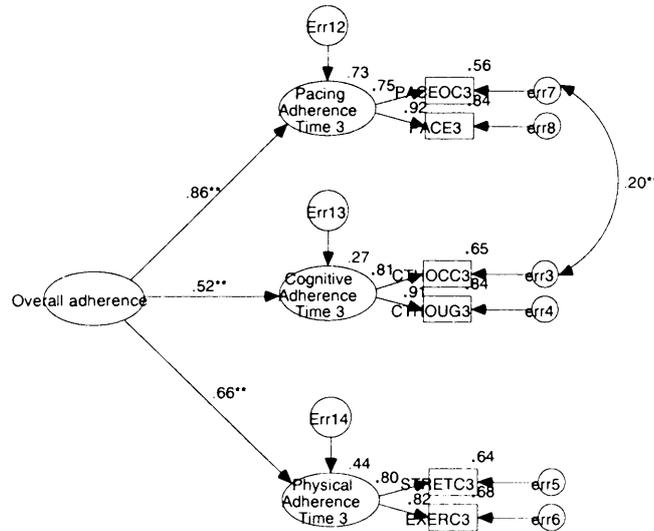
Key: Cthocc3 =Occasion of challenging thoughts (one-month follow-up); Cthoug3 =Frequency of challenging thoughts (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err =Error terms

Model MMD showed an excellent fit, as shown by the χ^2-value and fit indices for this model in Table 8. The standardised estimates show that a significant correlation exists between the error terms err3 and err7. All factor loadings are also statistically

significant. Therefore, on the basis of the adequacy of the fit statistics and standardised solutions, and because there is no justification for making further modifications, this model is considered to best represent the second-order structure of adherence items. It was therefore used as the basis of all subsequent structural models.

The first structural model to be tested that included the second-order factor 'overall adherence' is shown in Figure 22. Model SM5a examines the hypothesis that overall adherence is predicted by psychological state post-treatment. This model shows good fit with the data as indicated by fit indices in Table 8. 6% of the variance in overall adherence is accounted for by psychological state post-treatment (R^2=0.06), and a significant regression coefficient (β=0.25) is shown.

*Figure 22*: SM5a, showing standardised coefficients. *p<.05. **p<0.01.
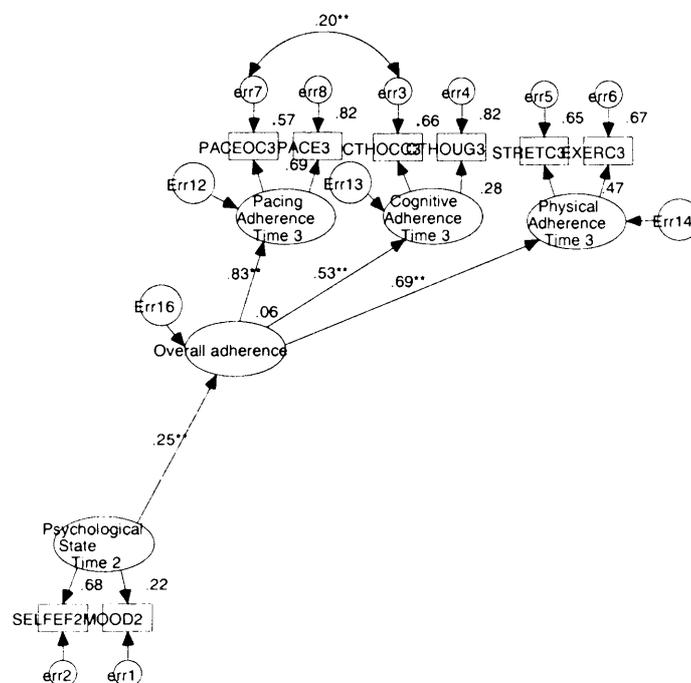
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Cthocc3 =Occasion of challenging thoughts (one-month follow-up); Cthoug3 =Frequency of challenging thoughts (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err =Error terms

Model SM5b (Figure 23) went on to test for an association between overall

adherence and psychological state at follow-up. The total variance accounted for by

this model was 13% (R^2=0.13), and a significant direct path from overall adherence

to psychological state at follow-up was found to exist.

Figure 23: SM5b, showing standardised coefficients. *p<.05. **p<0.01.
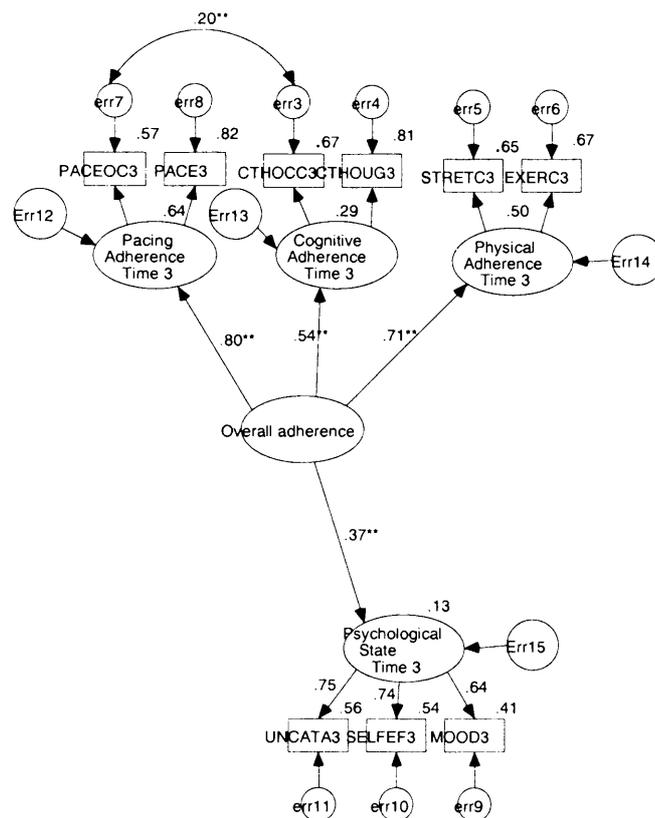
Figure 24 shows Model SM5c, which tests whether direct and indirect pathways exist

between levels of psychological state at the two time points. This model represents

the mediating model, i.e. that overall adherence has a mediating role in the

relationship. Fit indices for Model SM5c show that this model has good fit. All

standardised regression coefficients were shown to be significant indicating the

following effects:

- that psychological state post-treatment significantly predicts psychological state at follow-up;

- that psychological state post-treatment significantly predicts overall adherence;

- that overall adherence significantly predicts psychological state at follow-up.

The decrease in the strength of the relationship between psychological state post-treatment and psychological state at follow-up, indicated by the drop in the regression co-efficient from 0.83 in SM1 to 0.79 in SM5c, highlights the partially mediating role that overall adherence plays. Psychological state post-treatment and overall adherence is seen to account for 72% ($R^2 = 0.72$) of the variance in follow-up wellbeing. This indicates that overall adherence explains an additional 3% of the variance in psychological state at follow-up that is not accounted for by previous psychological state.

Figure 24: SM5c, showing standardised coefficients. *p<.05. **p<0.01.

Comparison with nested models

Because the mediating role of overall adherence was not the only plausible model, model SM5c was compared with two nested models representing alternative hypotheses. Model SM5d (shown in Figure 25) assumes that psychological state time 2 predicts overall adherence and psychological state time 3, but that no path between overall adherence and psychological state time 3 exists. By comparison, in Model SM5e (shown in Figure 26) psychological state time 2 and cognitive adherence are assumed to be related to psychological state time 3, but there is no link between psychological state time 2 and cognitive adherence. Fit indices reveal that both Model SM5d and SM5e fit the data adequately.

That said, change in χ^2 between each of these models and SM5c showed that each was significantly worse when compared to SM5c. Model SM5c was a significant improvement over SM5d, χ^2diff (1) = 28.67, p<.001. Similarly, Model SM5c was a significant improvement over SM5e, χ^2diff (1) = 32.77, p<.001. Comparison of various fit indices also reveals that Model SM5c is a better fit. These results were consistent with the hypothesis that adherence is influenced by psychological state, and that adherence has a causal effect on later changes in psychological state.

These results suggest Model SM5c should be considered to be the final overall causal model in the study. This model implies that psychological state at the end of

treatment is directly related to psychological state at one-month follow-up, and that

this relation also functions via overall adherence. All causal paths in this model were

shown to be significant, suggesting that there is a strong relationship between

psychological state at the two time points and overall adherence. This model

therefore suggests that patients who adhere to treatment recommendations are likely

to maintain their treatment gains while patients who do not as less likely to maintain

their gains. Of course, the effect of adherence does not disqualify the effect of

psychological state post-treatment having a strong direct causal effect on

psychological state at follow-up.

*Figure 25*: SM5d, showing standardised coefficients. *p<.05. **p<0.01.

Figure 26: SM5e, showing standardised coefficients. *p<.05. **p<0.01.

Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment);
Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Cthocc3 =Occasion of challenging thoughts (one-month follow-up); Cthoug3 =Frequency of challenging thoughts (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err =Error terms

Model Validation

Cross-validation of the final model (SM5c) was conducted using the split sample

(N=1231) as post-hoc modifications had been made to the model. Figure 27

illustrates the final model, tested on the validation sample. Fit indices revealed of

this model again showed good fit. All pathways remained significant in this model.

The similarity between the outputs from this final model between the exploratory and

validation groups indicates that this model is valid beyond the sample it was

developed on.

127

Figure 27: Validation Model, showing standardised coefficients. *p<.05. **p<0.01.
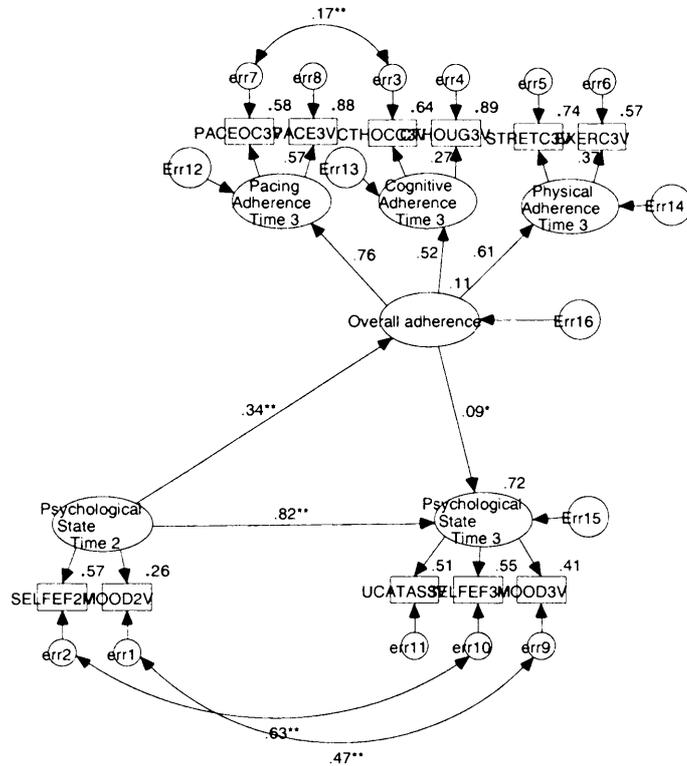
Sensitivity Testing

As a test of sensitivity, the following demographic variables were included in the

model: age; gender; education level; and site of pain. These variables were

correlated with psychological state post-treatment and had causal effects on

adherence and on psychological state at one-month follow-up, as shown in Figure 28.

As no differences were detected between the outcomes of the final model when

tested on the two distinct samples, only the exploratory sample was used during the

sensitivity testing stage.

The fit of this model was good. This sensitivity test indicated that the effect of psychological state post-treatment and overall adherence on psychological state at follow-up was independent of the effects of the demographic variables. This can be concluded as parameter estimates do not appear to have been influenced by the inclusion of the other variables. The causal effect of overall adherence on psychological state time 3 remained significant (CR=5.33, p<.0001); and the parameter value of 0.17 (SE =0.08) was also comparable. Neither age nor sex were shown to be significant predictors of psychological state at follow-up, and neither site of pain nor age left school were significantly related to overall adherence. This implies that psychological state at follow-up and overall adherence are not influenced by these demographic variables.

*Figure 28:* Final Model with Sensitivity Testing, showing standardised coefficients. *p<.05. **p<0.01.
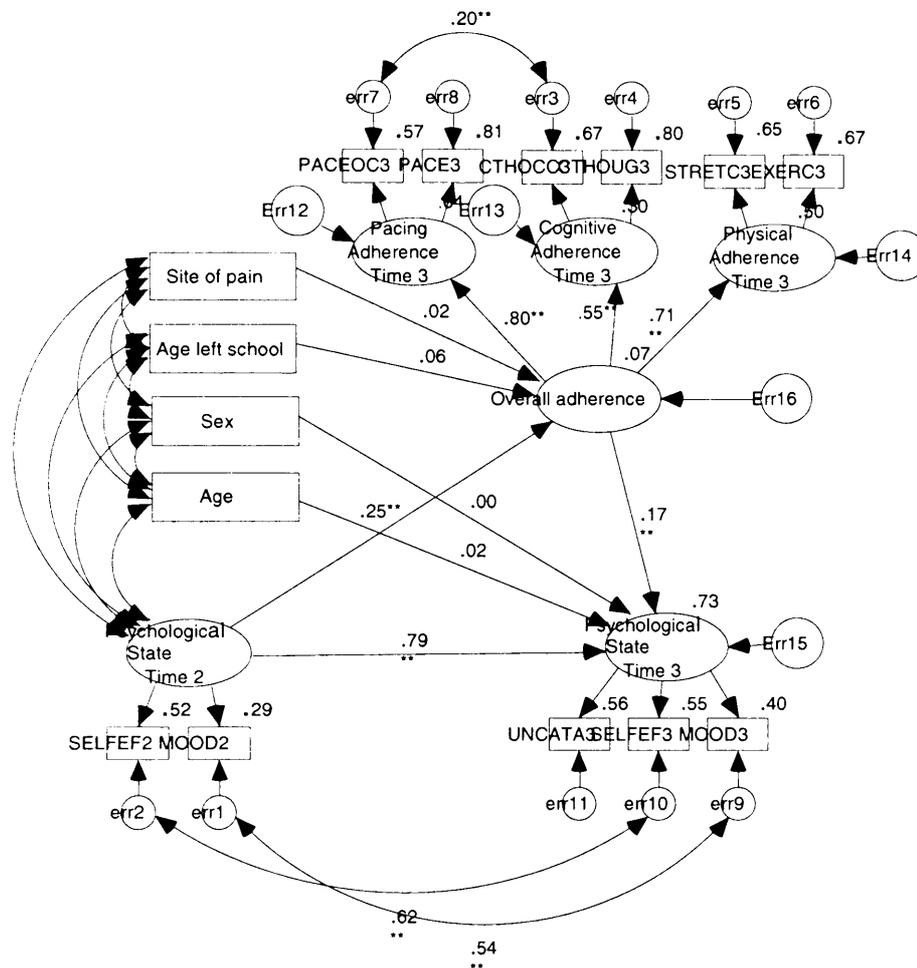
Key: Mood2 =Mood at time 2 (post-treatment); Selfef2 =Self efficacy at time 2 (post-treatment); Mood3 =Mood at time 3 (one-month follow-up); Selfef3 =Self efficacy at time 3 (one-month follow-up); Uncata3 =Non-catastrophic thinking at time 3 (one-month follow-up); Cthocc3 =Occasion of challenging thoughts (one-month follow-up); Cthoug3 =Frequency of challenging thoughts (one-month follow-up); Stretc3 =Frequency of stretching (one-month follow-up); Exerc3 =Frequency of exercise (one-month follow-up); Paceoc3 =Occasion of pacing (one-month follow-up); Pace3 =Frequency of pacing (one-month follow-up); Err =Error terms

4   Discussion

The purposes of this study were to investigate the multidimensionality of adherence as a theoretical construct using exploratory factor analysis, and to compare several causal models which relate adherence to outcome. The study aimed to examine the

extent to which cognitive adherence (continued use of thought challenging techniques) and behavioural adherence (continued use of stretch, exercise, and activity pacing) after discharge from cognitive-behavioural rehabilitative group treatment for persistent pain, predicts outcome at one-month follow-up. Structural equation modeling was used to assess to what extent, if any, adherence contributes to the prediction of outcomes at one-month follow-up. The research highlights the successful application of complex modeling techniques to meet these aims.

The first finding of this study concerned the multidimensionality of adherence as a construct. The exploratory factor analysis provided a clean solution indicating that the adherence measures loaded on to three adherence factors: physical adherence; pacing adherence; and cognitive adherence. The clarity of loadings and the absence of substantial cross-loadings identified this solution to be excellent, and so it was used confidently throughout the subsequent analyses.

The primary finding of the present study was that each factor of adherence was independently shown to partially mediate the relationship between psychological state post-treatment and psychological state at one-month follow-up.
In support of the findings of Fielding and Duff (1999), and Fraser et al. (2003), levels of mood and self-efficacy were shown to contribute to the prediction of adherence. Similarly, in line with the findings of Nicholas et al. (1991), the results obtained are supportive of the hypotheses that cognitive adherence, pacing adherence and physical adherence each contribute to the prediction of one-month follow-up outcomes. That said, the percentages of variance in one-month follow-up outcome accounted for by the adherence factors were shown to be small, with each type of adherence only

accounting for an additional 1-2% of the explained variance in psychological state at follow-up.

Experts in the field of persistent pain have reported that from clinical experience patients often identify activity pacing as the strategy which most helps them post-treatment. Various reasons can be provided for this, including that pacing gives patients a scheme to work to which may reduce worrying and rumination. It can also enable patients to plan better and to do more than they feel capable of judging by pain alone. This in turn helps patients with persistent pain to avoid vigilance to and monitoring of pain, which may contribute to catastrophising. Finally, pacing provides patients with permission to stop before completing tasks without accusing themselves of failure. It is interesting therefore that pacing adherence did not account for a more substantial percentage of variance in follow-up outcome than revealed by this study. It seems that despite patients placing more emphasis on pacing as the method that helps, each type of adherence (physical, cognitive, and pacing) has an equal impact on influencing maintenance of treatment gains. However, some patients may be using activity pacing counterproductively to keep their activity levels low rather than to build up, and some have few activities that they enjoy. These issues must be taken into account when interpreting the findings.

A third finding was that overall adherence was shown to partially mediate the relationship between psychological state post-treatment and at one-month follow-up. When psychological state at follow-up was regressed on psychological state post-treatment, psychological state post-treatment was revealed to account for 69% of the variance in psychological state at follow-up. However, when overall adherence was

added into the model, 72% of the variance in psychological state at follow-up was accounted for, indicating that overall adherence accounted for an additional 3% of the variance. This finding highlights the relatively small yet significant impact that adherence has on follow-up outcome.

In hypothesising about why the amount of variance accounted for by adherence was shown to be small, one argument stands out. It may be that improvement after the end of treatment happens over a longer time span, and that more variance in follow-up outcome may be attributable to adherence over 3 months, for example. The present study was unable to examine this hypothesis because of the extent of missing data at longer-term follow-up.

The results of this study suggest that a patient's psychological state post-treatment has more influence on follow-up psychological state than the extent to which s/he adheres to treatment recommendations. This suggests that it may be that getting patients to as high a level as possible at discharge is more productive than in allocating more time and resources to encouraging patients to adhere to treatment methods.

Validation of the model was important as empirically based re-specifications to the model could have led to capitalization on chance and over-fitting of the model. The cross-validation strategy used in this study meant that the model was developed with a split-half of the sample, and it was validated using the remainder of the sample. Testing of the final model on a split half of the sample revealed that the validity of the model was good. However, model fit was cross-validated using a nested sample,

which has implications for the generalisability of findings. For example, the results of this study can only be used to draw conclusions about the impact of this particular treatment programme on adherence and outcomes. The validity of the model can only be demonstrated by the weight and convergence of a future body of research, and cross-validation with an independent sample is required to test whether the findings of this research are truly generalisable to other populations.

Finally, the sensitivity of the model was shown to be good, as the addition of these other variables in the model did not reveal any missing variable bias. This implies that these were not important missing variables from the model which might have biased results. The sensitivity testing was also important to indicate that psychological state at follow-up is not dependent on age and sex. This has clinical implications, as it suggests that services should not exclude patients from treatment on the basis of age or sex.

SEM was the method chosen for testing for the mediation role of adherence on follow-up outcome. It combines multiple regression and factor analysis procedures, it simultaneously estimates multiple regression equations, and it avoids problems of over- and under-estimating effects by controlling for measurement error (Baron & Kenny, 1986). SEM techniques were used as opposed to regression methods as they provide a less biased assessment of mediator effects, and because it is the preferred approach when multiple indicators are available for each latent variable (Tabachnick & Fidell, 2001). It also allowed for the estimation of a model that modelled for multicollinearity. Alternative statistical procedures such a multiple regressions would have required separate tests of model components conducted on an equation-

by-equation basis, rather than of the model as a whole. This would have had implications for type one error.

Limitations

The results of the study should be interpreted in view of the following limitations.

Research design and sample limitations

The ability to infer causality is tied most fundamentally to research design, not statistical analyses. One concern is that measures of one-month outcomes and adherence were obtained concurrently. Participants were therefore asked to recall their current levels of adherence at the same point at which follow-up outcome data were collected. This raises two important considerations. Firstly, as noted by Cole and Maxwell (2003), a retrospective measure of adherence may well be biased by the outcome that it claims to predict. Secondly, it was not possible to conclude whether adherence actually caused psychological state at follow-up or vice versa. Therefore, the direction of causality cannot be determined definitely. Ideally, adherence should have been measured at a time before the outcome variables, so that data would have been collected in a fashion that allowed time to elapse between the theoretical cause (adherence) and its effect (long-term outcome). To infer causality, it must be demonstrated that the cause precedes the effect. Further analyses would be needed to substantiate that adherence caused follow-up outcome. For example, use of a cross-lagged panel design could have been used to infer causal paths, to show that adherence caused outcome and not vice-versa.

Although utilising a large data sample during the analyses brought with it many advantages, it is important to also note the problems that may have arisen from using such a large sample. Firstly, the correlation analyses revealed statistically significant correlations between variables, but substantive significance should not be assumed merely because statistical significance was demonstrated. For large samples, even very weak relationships may be shown to be statistically significant. Secondly, the fit of the models was assessed using the likelihood ratio chi-square statistic, and other indices of fit. The problem of the chi-square fit index is that the larger the sample, the more likely the rejection of the model and the more likely the Type II error. This index is also very sensitive to violations of the assumption of multivariate normality, and it is affected by the size of the correlations in a model (Tabachnick & Fidell, 2001). In this study, the large sample size and the strong correlations between latent variables may have contributed to the large, significant chi-squared values. The fit indices utilised to indicate model fit should also not be regarded as perfect measures of model suitability. These indices were simply a tool to enable the conclusion that a well-fitting model is one plausible representation of the underlying structure.

The current study only utilised follow-up data collected one-month after treatment had ended. Although some long-term follow-up outcome data were available, it was decided that it was not appropriate to use it in the current study due to large percentages of missing data and inconsistencies in the data collection. The present study was therefore unable to examine the role that adherence may play in longer-term outcome. The analysis of long-term follow-up data (e.g. collected one year after treatment) would have enabled the final model to be tested to examine whether adherence to treatment recommendations continues to have an influence on

maintaining treatment gains in the long-term. This of course would have been of benefit in the current study. Although extended follow-up periods are suggested to research the long-term outcome of treatments, the longer the follow-up period, the harder it is to determine whether outcome is due to the given factor under investigation. Therefore, results from very long-term follow-up studies may be difficult to interpret and caution would have been necessary when interpreting findings on the influence of adherence on longer-term outcome.

The present study examined the role that adherence to treatment methods had in relation to psychological state only. It was useful to illustrate that psychological state does not only arise from using cognitive methods, but from an interaction of various behavioural and cognitive strategies. However, what is missing from the current research findings is information concerning the relationship of adherence to other important outcomes such as physical wellbeing and experience of pain.

The study was not able to test the hypothesis of Erlen and Caruthers (2007), that adherence predicts adherence. Further research is necessary to focus on and examine the influence that adherence has on other outcome domains, and to examine whether adherence at one point is associated with levels of adherence at a later point.

Despite the very good fit of the models examined in this study and the use of sensitivity testing, it is possible that important constructs (e.g. motivation level or amount of social support) may have been omitted from the model that in reality are implicated in the causal structure. Therefore the final model from this study is unlikely to provide a complete account of the causes of one-month follow-up

outcome. Although residual variance and covariance terms allowed the modeling of the effects of omitted variables, if important variables were omitted, inaccuracies on estimates, standard errors and inferences about causal structure are likely. Sensitivity analyses were used to assess the possible biases induced by omitting variables on regression coefficients, although only a handful of possible omitted variables were tested.

Measurement Limitations

Measurement methods must be valid, reliable and sensitive to change if they are to be employed with confidence (Tabachnick & Fidell, 2001). A number of measurement limitations may have contributed to bias in the current research findings, or may have restricted the conclusions that are able to be drawn from this study. Two of these limitations will be discussed in detail below.

The first measurement limitation concerns the choice of measures available for the study. As this was a study using existing data there was a limited choice of process and outcome measures available. Models therefore had to be developed around what measures were available, rather than the hypothesised model dictating which measures should be used. This meant that the development of latent constructs and models was restricted. For example, the latent construct of psychological state was very broad as only one measure of each more distinct latent construct (e.g. depression) was taken. Normally latent variables are developed by having multiple measures of the same distinct construct. For example, Hoyle and Gregory (1994) proposed that each latent variable should be uniquely represented by three or more

indicators. As these were not available the reliability of the indicators, particularly for the psychological state constructs, could be called into question. Being constrained by available measures also meant that other potentially theoretically interesting process factors, such as perceptions of helplessness, could not be included in models. Even where crucial measures were available for use, they were not without their limitations.

Secondly, it is crucial to note that self-report measures of adherence were used in the study. Although self-report measures have the advantage of ease of administration and coding, the use of a self-report measure of adherence assumes that a participant truthfully reports his/her adherence. The disadvantages of self-report data include the response biases of social desirability and demand characteristics. For example, using a self-report method it has been shown that patients under-report non-adherence, especially if there is a therapist expectation of high adherence. Self-report also requires accurate recall by participants. In this study specifically, patient reporting of adherence may have been influenced by their psychological state at follow-up, as they were measured concurrently. For example, a participant feeling in a particularly good mood may be more likely to report good adherence. It has been argued that self-reporting therefore overestimates adherence (Kenna, Labbe, Barrett, & Pfister, 2005), and the reliability and validity of the measures of adherence could be questioned, because of response biases such as social desirability, recency effects, and acquiescence. To increase the reliability of adherence measures, if running this study again, additional adherence ratings from a partner or friend of the participant would be beneficial. Alternatively, overt behaviours may have been assessed more objectively by an independent check or use of electronic readers such as pedometers.

Finally, thought diaries and physical exercise records could also have been utilised in the study to provide more detailed records of adherence over a given period. Such methods would be preferable as they do not depend on recalling levels of adherence retrospectively. Although time consuming and costly, such modifications may have resulted in more reliable measures of adherence. However, these methods also put considerable burden on the participant, which may limit their use to highly motivated, literate participants who are doing well after treatment. Consequently, they may not represent the full sample of treated patients. The best strategy for assessing adherence may have involved a combination of both self-report and objective measures.

Clinical Implications

In spite of the above limitations, this study made unique contributions: It was the first study to demonstrate using structural equation modeling the possible linkages among adherence and psychological state at different time points. The current study indicated that psychological state at the end of treatment was associated with adherence and that adherence was related to follow-up levels of wellbeing. It also demonstrated the influence that psychological state post-treatment has directly on psychological state at one-month follow-up. The use of a large clinical dataset ensured that the results had maximum external validity and clinical relevance.

These findings contribute to a theoretical framework which has implications for treatment content and for pain management practice and education. For example, to improve patient adherence staff might increase the emphasis they place on encouraging and educating patients on the importance of adhering to treatment

recommendations. The findings can therefore inform efforts to enhance patient adherence.

The results of this investigation also indicate that it is important for patients to reach as high a psychological state by the end of treatment as possible. This information could be used to educate and motivate patients during the treatment phase, and it also has implications for informing treatment delivery. For example, intensive treatment programmes would be suggested as they have been shown to result in increased gains by the end of treatment, when compared with less intensive, outpatient programmes (e.g. Williams, Richardson, Nicholas, Pither, Harding, Ridout, Ralphs, Richardson, Justins & Chamberlain, 1996). The argument for an intensive treatment approach would endorse that the better the state of a patient by the end of treatment, the more likely it may be that everyday life presents reinforcements for managing pain successfully and for returning to a more normal lifestyle. For example, lifestyle changes could render exercises unnecessary in terms of formal practice as taught on the programme, because the patient is more active on a day-to-day basis. In this way, the gains of treatment may be sustained, not by the specific means taught on the programme, but by everyday exercise such as walking the dog or climbing stairs. However, such a proposal contradicts the recent government initiative to priorities Expert Patient Programmes, in the form of self-management groups, as the answer to all chronic physical problems (Department of Health, 2001).

Future Research

The links between adherence and psychological state at follow-up definitely warrant further exploration and several recommendations can be made to direct future

investigations in this area. Firstly, there is clearly a need for validated measures of adherence to be developed as adherence is a multidimensional construct that may apply somewhat differently across various components of treatment. If better methods of measurement were available, it may be possible to examine whether there is a minimum level of adherence which is necessary for maintenance, and a maximum beyond which no further gains are made. There is also the possibility of an interaction effect, where those doing well at the end of treatment might need to adhere less than those who are doing less well, as everyday life may involve activities that promote maintenance for the former. The examination of hypothesised non-linear relations using SEM could be the focus of further investigations (Bauer, 2005).

Secondly, the examination of the final model's applicability in other samples would be useful. It is recommended that the model be tested using different independent data sets to examine the validity and reliability of its inferences further. Replication will be important to validate and support the findings of this study.

Once the findings of this study have been replicated with an independent sample, future research could focus on further developing the structural model to include other important variables that were omitted in the current study. For example, more focus could be put on identifying predictors of adherence, such as motivation levels and treatment satisfaction levels. In addition, change scores from pre- to post-treatment could be incorporated into the model to examine whether the amount of change achieved during treatment influences adherence. The model could also be examined to compare whether different lengths of treatments influence adherence.

Finally, adding pre-treatment measures into the model could help to understand more about change that happens during treatment, rather than just about change after treatment. Another method that could be used to shed light on the trajectory of change during treatment is the use of case tracking methods. For example, by taking session by session counts of exercise.

Future studies could examine the influence of adherence on longer-term follow-up outcome. Predicting long-term psychological state would require that a range of individual, familial, social, community and cultural factors be taken into account in the model. Future research should investigate the influence of adherence overtime more robustly.

Finally, SEM is unable to confirm definitely that a given model is a correct representation of the measurement or causal structure in the population. Equivalent models should be considered. It is suggested that future studies should be conducted, explicitly designed to discriminate plausible alternatives to the target model.

Conclusions

In summary, the present study is the first to investigate the predictive role of adherence on follow-up outcomes, using complex statistical procedures. Patients with persistent pain are a diverse group, so large sample sizes were necessary. The current data set was helpful in addressing the research questions and was valuable in providing evidence in support of the hypothesis that adherence is influenced by psychological state at the end of treatment, and it in turn influences follow-up

outcomes. Adherence was revealed to partially mediate the relationship between psychological state post-treatment and psychological state at one-month follow-up.

SEM methods represented an appropriate method to the study of mediation. To obtain more conclusive results regarding the causal associations between adherence and follow-up outcomes more highly refined studies will be needed to confirm these results in the future. Further research will also be necessary to investigate the potential role that adherence may play to longer-term follow-up outcomes.

References

Allison, P. D. (2003). Missing data techniques for structural equation modeling. *Journal of Abnormal Psychology*, 112(4), 545-557.

Antonuccio, D. O., Danton, W. G. & DeNelsky, G. Y. (1995). Psychotherapy versus medication for depression: Challenging the conventional wisdom with data. *Professional Psychology: Research and Practice, 26*, 574-585.

Arbuckle, J. (1999). *Amos 4.0 user's guide*. Chicago, Ill: Marketing Department, SPSS Inc.

Arbuckle, J. L. & Worthke, W. (1999). *Amos 4.0 User's Guide*, Small Waters Corporation, Chicago.

Asghari, A. & Nicholas, M. K. (2001). Pain self-efficacy beliefs and pain behaviour. A prospective study. *Pain,* 94(1):85-100.

Bandura A. (1989). *Social learning theory*. Englewood Cliffs, NJ: Prentice-Hall.

Baron, R. & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology*, 51(6), 1173-1182.

Basler, H. D. & Rehfisch, H. P. (1991). Cognitive-Behavioral therapy in patients with ankylosing spondylitis in a German self-help organisation. *Journal of Psychosomatic Research*, 35, 345–54.

Bauer, D. J. (2005). A semiparametric approach to modeling nonlinear relations among latent variables. *Structural Equation Modeling, 12*(4), 513–535.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4, 561-571.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*, 238-246.

Bentler, P.M. (1992). On the fit of models to covariances and methodology to the Bulletin. *Psychological Bulletin,* 112, 400–404.

Bentler, P. M. & Bonett, D. G. (1980). Significance tests and goodness-of-fit in the analysis of covariance structures. *Psychological Bulletin, 88,* 588-606.

Berg, J., Evangelista, L. S. & Dunbar-Jacob, J. (2002). Compliance. In I. M. Lukin and P. Larsen (Eds) *Chronic Illness: Impact and interventions* (5th edition, pp 203-232). Boston, MA: Jones and Bartlett Publishers.

145

Browne, M. W. & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. Bollen & S. Long (Eds.), *Testing structural equation models* (pp. 136-162). Newbury Park, NJ: Sage.

Byrne, B. M. (2001). Structural Equation Modeling with AMOS: Basic concepts, applications, and programming. Lawrence Erlbaum Associates: London.

Chatzidimitriou, A., Georgoudis, G., Manousou, A., Argira, E., Vadalouka, A., Anastasopoulou, D., Pavlopoulos F. & Siafaka, I. (2006). 918 Cognitive pain assessments in Greek patients: The case of the pain catastrophising scale. *European Journal of Pain*, 10, Supplement 1, S237-S238.

Cole, D. A. & Maxwell, S. E. (2003). Testing mediational models with longitudinal data: Questions and tips in the use of structural equation modeling. *Journal of Abnormal Psychology*, *112*, 558–577.

Concise Oxford Dictionary of Current English (8th edition). (1990). Allen, R. E. (Eds). Clarendon Press: Oxford.

Dancy, C. P. & Reidy, J. (2004). Statistics without maths for psychology, using SPSS for Windows (3rd Edition). Pearson.

Department of Health (2001). *The expert patient*: a new approach to chronic disease management for the 21st century. www.doh.gov.uk.

Dimatteo, M. R., Lepper, H. S. & Croghan, T. W. (2000). Depression is a risk factor for noncompliance with medical treatment: Meta-analysis of the effects of anxiety and depression on patient adherence. *Archives of Internal Medicine,* 160, 2101–2107.

Eccleston, C. (2001). Role of psychology in pain management. *British Journal of Anaesthesia,* 87 (1), 144-152.

Elliott, A. M., Smith, B. H., Penny, K. I., Smith, W. C. & Chambers, W. A. (2000). Chronic pain. *The Lancet*, 355, 234.

Erlen, J. A. & Caruthers, D. (2007). Adherence to medical regimens. In: Kennedy, P., Psychological management of physical disabilities: A practitioner's guide. Routlege.

Field, A. P. (2005). *Discovering statistics using SPSS (Second Edition)*. London: Sage.

Fielding, D. & Duff, A. (1999). Compliance with treatment protocols: interventions for children with chronic illness. *Archives of Disease in Childhood*, 80, 196-200.

Fraser, C., Hadjimichael, O. & Vollmer, T. (2003). Predictors of adherence to glatiramer acetate therapy in individuals with self-reported progressive forms of multiple sclerosis. Journal of Neuroscience Nursing, 35(3), 163-170.

Holmbeck, G. N. (1997). Toward terminological, conceptual, and statistical clarity in the study of mediators and moderators: Examples from child-clinical and paediatric psychology literatures. *Journal of Consulting and Clinical Psychology*, 4, 599-610.

Hox, J. J. & Bechger, T. M. (1998). An introduction to structural equation modeling. *Family Science Review*, 11, 354-373.

Hoyle, R. H. & Gregory, T. S. (1994). Formulating clinical research hypotheses as structural equation models: A conceptual overview. *Journal of Consulting and Clinical Psychology*, 62(3), 429-440.

Hulland, J., Chow, Y. H. & Lam, S. (1996). Use of causal models in marketing research: a review. *International Journal of Research in Marketing*, 13, 181-197.

Jensen, M. P., Turner, J. A. & Romano, J. M. (1994). Correlates of improvement in multidisciplinary treatment of persistent pain. *Journal of Consulting and Clinical Psychology*, 62 (1), 172-179.

Jöreskog, K. G. & Sörbom, D. (1989). *LISREL 7: A guide to the program and applications* (2nd Ed.). Chicago: SPSS Inc.

Kenna, L. A., Labbé, L., Barrett, J. S. & Pfister, M. (2005). Modeling and simulation of adherence: Approaches and applications in therapeutics. *American Association of Pharmaceutical Scientists,* 7(02), 390-407.

Kline, R. B. (1998). Principles and practice of structural equation modeling. New York: The Guildford Press.

Koes, B. W., van Tulder, M. W., & Thomas, S. (2006). Diagnosis and treatment of low back pain. *British Medical Journal*, 332, 1430-1434.

Leamer, E. E. (1985). Sensitivity analyses would help. *American Economic Review* 75, 308–13.

Lutz, R. W., Silbret, M. & Olshan, N. (1983). Treatment outcome and compliance with therapeutic regimens: long-term follow-up of a multidisciplinary pain program. *Pain*, 17, 301-308.

McCracken, L. M., Carson, J. W., Eccleston, C. & Keefe, F. J. (2004). Acceptance and change in the context of persistent pain. *Pain*, 109, 4-7.

Maniadakis, N. & Gray, A. (2000). The economic burden of pain in the UK. *Pain,* 84, 95-103.

Morley, S., Eccleston, C., & Williams, A. (1999). Systematic review and meta-analysis of randomized controlled trials of CBT and behaviour therapy for persistent pain in adults, excluding headache. *Pain*, 80, 1-13.

Morley, S., Williams, A. C. de C. & Black, S. (2002). A confirmatory factor analysis of the Beck Depression Inventory in chronic pain. *Pain*, 99(1), 289-298.

Morley, S., Hussain, S., & Williams, A. (2005) Testing the cognitive mediation model of change in a CBT based Pain Management Program. Using a 3-wave panel multiple regression model to test for general and specific cognitive predictors. (In preparation)

Nicholas, M. K. (2007). The pain self-efficacy questionnaire: Taking pain into account. *European Journal of Pain*, 11(2), 153-163.

Nicholas, M.K., Wilson, P.H. & Goyen, J. (1991). Operant-behavioural and cognitive-behavioural treatment for chronic low-back pain, *Behaviour Research Therapy*, 29, 238–255.

Osterberg, L. & Blaschke, T. (2005). Review article: adherence to medication. The *New England Journal of Medicine*, 353(5), 487-497.

Rosentiel, A. K. & Keefe, F. J. (1983). The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. *Pain*, 17, 33-44.

Roth, T. & Fonagy, P. (2005). What works for whom? (2nd Ed). Guildford Press: New York.

Sullivan, M. J. L., Bishop, S. & Pivik, J. (1995). The Pain Catastrophizing scale: development and validation. *Psychological Assessment*, 7, 524-32.

Tabachnick, B. G. & Fidell, L. S. (2001). Using multivariate statistics (4th edition). Boston: Allyn and Bacon.

Taimela, S., Diederich, C., Hubsch, M. & Heinricy, M. (2000). The role of physical exercise and inactivity in pain recurrence and absenteeism from work after active outpatient rehabilitation for recurrent or chronic low back pain: a follow-up study. *Spine*, 25 (14), 1809-1816.

Turk, D. C. & Rudy, T. E. (1991). Neglected topics in the treatment of persistent pain patients – relapse, non-compliance, and adherence enhancement. *Pain*, 44, 5-28.

Vermeire, E., Hearnshaw, H., Van Royen, P. & Denekens, J. (2001). Patient adherence to treatment: three decades of research. A comprehensive review. *Journal of Clinical Pharmacy and Therapeutics*, 26 (5), 331–342.

Vitolins, M. Z., Rand, C. S., Rapp, S. R., Ribisl, P. M. & Sevick, M. A. (2000). Measuring adherence to behavioral and medical interventions. *Control Clinical Trials*, 21(5 Suppl), 188-194.

Whisman, M. A. (1993). Mediators and moderators of change in cognitive therapy of depression. *Psychological Bulletin*, 114, 248-265.

Williams, A. C. de C., Richardson, P. H., Nicholas, M. K., Pither, C. E., Harding, V. R., Ridout, K. L., Ralphs, J. A., Richardson, I. H., Justins, D. M. & Chamberlain, J.

H. (1996). Inpatient vs. outpatient pain management: results of a randomised controlled trial. *Pain*, 66, 13-22.

Williams, A. C. de C., Morley, S. & Black, S. (2002). Grant report: Empirical evaluation of best practice in chronic pain management: influence of patient characteristics and treatment type on outcome.
www.info.doh.gov.uk/doh/refr_web.nsf

World Health Organisation (2003). *Adherence to long-term therapies*: Evidence for action. Geneva, Switzerland: World Health Organisation.

Part 3: Critical Appraisal

Introduction

My experience of conducting this piece of research can only be described as a roller coaster ride, with emotions ranging from despair, frustration, and utter confusion, to joy, satisfaction and relief – and not necessarily in that order! Completing the appraisal will give me the opportunity to discuss some general concerns about using an existing data set for the study. It will also allow me to explain why certain decisions were made during the process and to share some of the lessons I will be taking away with me from this experience. Finally, the appraisal will provide me with a chance to reflect on the process of conducting this piece of research.

General Concerns

One of the distinguishing features of this study was that it utilised an existing data set, rather than data that were collected by me, specifically for this study. Although using an existing data set brought with it many advantages, one issue that stands out for me is how difficult it was to familiarise myself with the data. Thinking back, using the existing data set would have been impossible had my supervisor not had detailed knowledge of the data, and the service from which it came. Because of her expertise with the data and of the procedures used at the pain management programme concerning patient care and data collection I was able to develop my own understanding of the data relatively quickly. However, it was vital to be able to have discussions with my supervisor regarding any issues that raised concerns as and when they arose. One such concern was about whether my research would meet the criteria for a Doctor of Clinical Psychology (D.Clin.Psy) thesis.

As a result of using an existing data set it was a requirement that I made my statistics 'complex', 'sophisticated', and impressive enough to compensate for not collecting the data myself. This raised alarm bells for me as unfortunately it was not clear what level of sophisticated statistics would be 'good enough' to meet the requirements for a D.Clin.Psy. This study was different to many D.Clin.Psy. projects as in my study the majority of time was spent planning, executing, and interpreting the statistical analyses, rather than on planning and executing data collection. However, it made sense for me to use a huge existing data set, where there was the possibility to use much neglected large sample statistical procedures. The alternative would have been to collect my own small sample, which is often the only option given the limited time-frame for completing a D.Clin.Psy. research project.

A final general concern that stands out for me is the rather shocking lack of valid and reliable measures that are found within adherence research. It seems astounding to me that despite CBT expecting lasting change in behaviour(s) and cognition, no standard ways of assessing whether those are happening seem to exist. Where measures of adherence do exist, they focus on frequency of adherence, rather than for example on the quality, effectiveness, or the appropriate use of the strategies. The development of reliable and standardised measures of adherence behaviour will be essential in the future if research findings are to be comparable across studies.

Dilemmas

Many important decisions had to be taken over the course of the research period. These decisions included ones about which statistics techniques, statistical software, and cases and measures from the archival data to use. I would like to try to explain why such decisions were made.

One of the first decisions to be made was to decide whether SEM was the most appropriate statistical approach to investigate my hypotheses with the given data. There seemed to be so much to think about. The more I read, the more I felt confused and worried that it may not be an appropriate approach to use. My decision to use SEM as opposed to multiple regression procedures was made based primarily on the fact that SEM could simultaneously estimate multiple regression equations, while also controlling for measurement error. This ensured that my findings were as unbiased as possible.

When SEM had finally been decided upon, the next decision concerned choosing which statistical software package to use to conduct the analyses. I remember feeling unqualified to be making such a decision, especially when it was important to also consider financial costs. LISREL, EQS and AMOS statistical packages were each investigated and all had their advantages and disadvantages (Tabachnick & Fidell, 2001). AMOS was the package of choice because of its graphical interface, because of its ability to handle missing data, and because it was already available for use within the University College London (UCL) clinical psychology department.

Next, the decision about which data to use had to be made. For example, data were available on patients who had taken part in a 4-week (16-day) and a 2-week (9-day) inpatient programme, and I had to decide whether to use a combined set rather than only one or other of them. The decision to include data from the 4-week inpatient treatment group and the 2-week inpatient treatment group combined was difficult to make. For maximum homogeneity of experience I understand that it would have been beneficial to include only participants from one of these groups, and to have dropped the data from the other. However, this would have meant that much valuable data on adherence would have been lost. My decision to include both of these populations as a single group was based on my understanding that both were inpatient groups, in which massed practice behavioural change was encouraged. Also, as my interest was in adherence after treatment, rather than change during it, my decision to include both populations seemed justified. Another strategy may have been to include both treatment groups but to include treatment group as a variable of interest, i.e. to compare the model between the two groups. However, as no previous research in persistent pain has explored the relationship between adherence and follow-up outcome it was important to include as much information as possible in developing an initial model. It was also thought that this would help to increase the generalisability of the research findings. Other information that informed this decision included that the 2-week inpatient group was much smaller than the 4-week sample, and that there was no clear hypothesis about what the differences would be between these two treatment groups. It was therefore decided that it would be more beneficial to look at the relationship of adherence to outcome overall in the first instance, using as large a data sample as possible. Perhaps a comparison of the role of adherence to follow-up outcome within different treatment groups could be conducted as a follow-up study.

A huge array of measures was collected routinely from patients and I deliberated about which measures it would be best for me to use. In many cases I felt that I had to make the best of measures available, but I also had to make the difficult decision about which potentially valuable and interesting measures to drop. One example of having to make the best of what I had can be seen in the decision to combine scores from the pain catastrophising scale (PCS; Sullivan et al. 1995) and the coping strategies questionnaire (CSQ; Rosenstiel & Keefe, 1983). Had scores on only the PCS or only the CSQ been used the final dataset would have contained a vast amount of missing data. This would have meant that valuable information on the influence of catastrophising would have been unnecessarily lost.

In terms of dropping variables, reported frequency of relaxation, quality of life data (as measured by the SF-36; Ware & Sherbourne, 1992), and data on physical functioning (as measured by walk distance) were each dropped from the study. Relaxation was left out as it was decided that there was no way to tell exactly how individuals were using it. The SF-36 quality of life measure was dropped as this information was not collected consistently from clients over the given time period. For example, the measure was not taken post-treatment. Also, eyeballing of the data available revealed that the data contained repeated subject numbers. This meant that the data were not considered to be reliable. Finally, walk distance was initially recorded in 10-min, and was then changed to 5-min after the first 700 patients. The reliability of these data were also called into question as eyeballing of the data revealed that many patients with chronic pain had been recorded as having walked over 600m in 5-minutes. These distances seemed overly large. As there was no way

to double check the reliability of these data it was decided that it was safer to drop this measure.

My decision to cut the sample size from its original size of 3948 participants to 2345 was based on concerns about the amount of missing data within the sample. The decision was therefore made to only keep cases for which adherence data were available. It was decided that as adherence was my variable of interest it made little sense to include cases which included data on outcome, if they then did not have adherence data. Although this reduced the sample size and meant that some potentially valuable information was lost, it was necessary to lower the percentages of missing data to acceptable levels for analysis. This still left a substantial sample size of 2435 participants, which was more than adequate to conduct my analyses.

Concerns about missing data also had a part to play in determining which data-collection time-points would be used in the study. Data were available at pre-treatment assessment (5 days before treatment), at post-treatment assessment (last day of treatment), and then they were available at one-month, six-month, nine-month and 12-month follow-up. It was decided that pre-treatment outcomes would not be modelled as my interest was in change after treatment, rather than in change during it. The decision was made to use only the measures collected at post-treatment and one-month follow-up (and to model the relationship between them), for the following reasons. Firstly, it was only data collected at post-treatment and at one-month follow-up that were collected consistently over the course of the data collection period. By contrast, data were only occasionally collected at 6-month, 9-month and 12-month follow-up and never at all three time-points, which meant that any long-term follow-

up data would have had to have been a combined form from each of these time points. For example, 6-month and 12-month follow-up data were only collected for the first 700 patients, with only 20% of these having completed the measures. 1000 participants had completed some outcome measures at 9-month follow-up, although, again the percentages of missing data for individual measures were very high (50-80% missing data). The amount of missing data at these later time-points was therefore great, and the decision to leave them out was made. The decision to drop the long-term follow-up data was also based on two considerations: that the missing data at that time-point was not missing at random; and that there may have been too little variance at this point as only those doing well may have come back. In addition, long-term follow-up data were not used as any model examining change over that time span would have needed to model the influence of many external variables which were not measured. For example, variables such as partner/family support and employment prospects may have had a significant impact on longer-term follow-up outcomes. This would have meant that the effect of adherence would have been weaker. Finally, increasing heterogeneity at that time would have meant that my final model was inadequate to test the influences of adherence on outcome at long-term follow-up. A more inclusive model of what leads to long-term behaviour change would be required, which was beyond the scope of the current study.

Another crucial decision had to be made regarding the methods utilised in determining latent variable structure. Factor analysis was chosen as the method for the adherence measures. This was appropriate as multiple measures of adherence were available and factor analysis illustrated clearly the factors that those measures loaded on to. In contrast, for the psychological state measures, only one outcome

measure for each distinct latent construct (e.g. depression) was available and too few measures, even of the broader latent variables, were taken at each time point to allow for the use of factor analysis. My decision to load mood and self-efficacy together was therefore justified on the grounds that they were identified to correlate well. In addition, it was considered acceptable as it made logical and theoretical sense to combine measures as I did. It could be argued that the construct of 'psychological state' used in my study was too broad as mood and self-efficacy measures are designed to measure different things. I realise that this situation was not ideal, but as only one indicator for each specific construct was available I had to make do.

Finally, when first thinking about potential hypothesised models to examine, very elaborate models were conjured up. I wanted to include everything in my model. I wanted to test how the latent construct of psychological state was related to physical wellbeing and experience of pain, and how each of these was associated with each type of adherence. I wanted to include how patient satisfaction and expectations for treatment influenced these associations. However, I soon realised that I had unrealistic expectations of what could be achieved. After much consideration I decided that in order for the examiners and me to understand and follow the research process I was going to have to simplify things and lower my expectations of what could be achieved within the time scale, and with my knowledge of SEM.

I decided that rather than throwing everything into my models, I would build them up slowly so that I could assess at each point where any difficulties lay. Looking back, my original strategy of throwing everything into a model would surely have left me feeling overwhelmed and unable to explain the output. My approach instead allowed

the models to be developed in discrete, logical steps, and allowed me to tell a story in the research. It also helped me to not over complicate the models, which I hope made it easier to read.

Overcoming Hurdles

Many hurdles presented themselves during the course of conducting this study. One such obstacle concerned gaining ethical clearance for the research. Previous ethical and research and development approval had been gained for previous research involving the same data set. This meant that only extensions of approval for each were required for my study, as amendments to the original study, rather than having to complete the NHS ethics form. I assumed that obtaining this amendment would not be stressful or time consuming as it had to be passed only by the amendment subcommittee. However, this process was slow and it took months to learn that ethical consent and research and development clearance had been granted. Thinking back over the research process, this meant that the momentum of the project development was interrupted, and it was very difficult to persevere with the study at that time without the certainty of having ethical approval. The reassurance and continuous support that I received from my research supervisor at that time, and throughout the research, was very helpful.

Another hurdle to overcome was how to gain my knowledge of SEM and AMOS. At the start of the project my knowledge and experience of using SEM techniques and of using AMOS was zero. I wanted to attend a training course run by AMOS to teach me the basics on top of which I would be able to build independently. I recall being

disappointed when I was informed that I would not be able to go on the AMOS

training course as it would cost too much. It felt like my research was being put in

jeopardy, simply due to financial restraints, and I felt powerless to do anything about

it. I was therefore forced to set about learning how to use AMOS by reading and by

doing examples. The AMOS user guide (Arbuckle, 1999) became my bible.

Although completely overwhelming initially, I am struck now by the user-friendliness

of AMOS. For example, AMOS gave written prompts when running analyses such as

prompting me that a model was unidentified, or that pairs of latent exogenous

variables were not correlated. This was helpful when first learning how to use the

programme. It took much time to learn to use the AMOS graphics package, but

AMOS's simple visual representations were helpful for me to get a grasp of the

difficult statistical concepts and analyses, and made it much easier to explain and

understand the models to be tested. All the constructs and their interrelationships

were clearly displayed which was really helpful to understand what was happening. I

really liked the way that the multiple regression equations could be represented by a

diagram, and it took the fear out of performing very sophisticated statistics. Drawing

figures and having the output displayed on the diagrams also made it easier to

understand the results and to be able to explain the results to others, who may not

have had as much knowledge. On reflection, having a good research design made

using the AMOS statistics package a challenging, yet achievable experience, rather

than the punishing experience it could well have been.

Retrospectively, I think it would have been difficult for me to make use of the AMOS

training course opportunities before I had got up to my elbows in analysis. Having to

learn through reading and doing examples myself meant that my understanding of my

data and what the package could offer grew quickly. In turn, I noticed that my confidence increased, and knowing that I achieved the knowledge independently has filled me with a sense of satisfaction and achievement.

Lessons Learnt

Many lessons will be taken away with me from this experience. Two of the most important of these lessons, and the ones that will be presented here, are the lesson I learnt about my own personal strengths, and a lesson regarding the constraints within which all research has to be conducted.

Firstly, on a personal note, I have recognised many of my own strengths and skills that I was unaware of prior to this experience. In particular, I have been struck by my ability to problem solve, to persevere, and to be flexible when faced with hurdles and difficult decisions. In addition, my time management and organisational skills have been vital, as it was necessary to prioritise and plan tasks carefully in order to meet my deadlines. I also think that my computer skills have flourished, as well as my ability to communicate complicated material as simply as possible. Finally, the research has shown me that not only am I capable of working independently to meet goals, but I have thrived on the satisfaction and the sense of achievement that completing such a task independently has given me. I'm sure there were times when I wanted to back out early on, and wanted to admit that I could not cope. However, looking back it seems that the enormity of my task seemed to motivate me and spurred me on to persevere and learn, rather than to throw in the towel. This taught

me a lot about my own self diligence and the drive I have to succeed, even when faced with difficulties.

Secondly, on reflection, the experience of having to learn how to do structural equation modeling independently from books, rather than from a taught course, taught me a great deal. It forced me to look for alternative strategies to gain the knowledge that I needed. In addition, it taught me the important lesson that all research within the NHS has to be conducted under constraints, whether it is time constraints and/or financial ones. I learnt how it is necessary to problem solve any hurdles that you are faced with, rather than to give in to them.

Finally, completing this research I also learnt hands-on about the sort of compromises and decisions that have to be made when using real life clinical data, with missing values and discontinuities.

Personal Reflections

Looking back, my journey began on a high. I had secured the amazing opportunity to use a large set of archival data in an area of psychology that fascinates me. The available literature and research on adherence in persistent pain was very elementary, meaning that there was huge scope for me to do some ground breaking research. It felt like the world was my oyster in terms of what I wanted to investigate, and what statistical methods I would use to do it. However, the reality of what this would actually entail soon set in. As I have said, my statistical analyses needed to be 'very sophisticated', and the logistics of getting my head around such an enormous data set

and of deciding on and learning how to use complex statistical procedures was very frightening. The words 'structural equation modelling' and 'multivariate analysis' filled me with fear, and in the beginning, reading about them only seemed to heighten this.

The period of time between my research proposal being submitted and returned was filled with anticipation. I recall the relief I felt when the proposal was accepted, while also remembering the feeling of fear that I would actually have to do what I said I could do. I was sure that my incompetence would be revealed at any point.

When I first came face to face with the data I have to admit that I felt completely overwhelmed. It seemed an impossible task to get my head around what was there, to familiarise myself with the data, to decide what I wanted to keep, and then to arrange this data into a suitable form for my analyses. This process at the time seemed slow, and never ending. Data was saved in numerous different files, so the merging of files was the first necessary but very time consuming step of turning the data into a single workable file. It was helpful to map out clearly what data was available at each time point, in order for me to fathom how to begin this immense task. Data were then meticulously checked by hand to ensure that all cases with no data were removed and to ensure that there were no repeated subject numbers. Looking back, this long process was vital to ensure that the data set was clean and as complete as possible for analysis. It was also vital for me to familiarise myself with the data set. My knowledge of the data and its meaning was strengthened, and I think that completing this process made it possible for me to go on and perform the analyses efficiently.

I recall that as I read the AMOS user guide (Arbuckle, 1999) and a book on structural equation modeling (Byrne, 2001) for the first time I waited patiently for that light bulb to come on, hoping that at each turn of the page it would suddenly all make sense to me. I highlighted everything that seemed important (the whole book was highlighted!) and I remember closing the back cover when I had finished the book and being filled with dread that the 'Geronimo' moment had not come. Looking back, there never was a distinct moment when I can say 'I got it'. Instead, the learning curve in developing my knowledge and skills in conducting my analyses was slow, steady, and continuous.

I still feel that I have much to learn about this area of statistics, although I can also now see how far I have come in terms of my knowledge and confidence. I can now quickly and confidently draw, specify and estimate structural models with ease, as well as interpret their output. This seemed like an impossible task when the project was embarked upon. The words structural equation modeling and multivariate statistics no longer fill me with fear, which illustrates for me how far I have come.

Final Comments

I am very pleased with what I have achieved. This research made good use of complex statistical methods, using a big sample and based on theory driven hypotheses. I feel that I have been able to add a great deal in an area of little research. The results have shown important effects, which have clinical implications. That said, my only lasting frustration is that I am unable to conclude that my final model is definitely right, only that it is the most well fitting one tested. My research

does not in anyway prove a causal pathway, and I am forced to concede that many equivalent models will exist. However this research has provided an adequate basic model on which to build in future research.

Using AMOS graphics has increased my interest in quantitative analysis and it opened up the possibility for me to overcome any fears I had of statistics. My experience of using AMOS and of completing this research can be summarised as hard work, but most of all satisfying and rewarding.

References

Arbuckle, J. (1999). *Amos 4.0 user's guide*. Chicago, Ill: Marketing Department, SPSS Inc.

Byrne, B. M. (2001). Structural Equation Modeling with AMOS: Basic concepts, applications, and programming. Lawrence Erlbaum Associates: London.

Rosentiel, A. K. & Keefe, F. J. (1983). The use of coping strategies in chronic low back pain patients: Relationship to patient characteristics and current adjustment. *Pain*, 17, 33-44.

Sullivan, M. J. L., Bishop, S. & Pivik, J. (1995). The Pain Catastrophizing scale: development and validation. *Psychological Assessment*, 7, 524-32.

Tabachnick, B. G. & Fidell, L. S. (2001). Using Multivariate Statistics (4th edition). Boston: Allyn and Bacon.

Ware, J. E., Sherbourne, C. D. (1992). The MOS 36-Item Short-Form Health Survey (SF-36®): I. conceptual framework and item selection. *Medical Care*, 30(6):473-83.

Appendices

Appendix 1 Ethical approval confirmation

Appendix 2 Participant questionnaires:

1.) Personal/Medical information sheet

2.) Beck Depression Inventory (BDI)

3.) Pain Self-Efficacy Questionnaire (PSEQ)

4.) Pain Catastrophising Scale (PCS)

5.) Coping Strategy Questionnaire (CSQ)

6.) Adherence Questionnaire

Appendix 1 – Ethical approval confirmation

St Thomas' Hospital Research Ethics Committee

19 September 2006

Dr Amanda C de C Williams
Reader in Clinical Health Psychology
Sub-Department of Clinical Health Psychology
University College London

Dear Dr Williams

Study title:	**Estimating the magnitude of change following, and modelling change processes in, cognitive behaviour therapy for chronic pain**
REC reference:	**04/Q0702/54**
Protocol Amendment Detail:	**Part of the data analysis to be done by Charlotte Curran, for her thesis for a doctorate of clinical psychology 'CBT for persistent pain - how well can long-term treatment gains be predicted on the basis of adherence to treatment?' under the supervision of Dr Amanda C de C Williams**

Amendment date: **05 May 2006**

Thank you for your letter of 05 May 2006, notifying the Committee of the above amendment.

The amendment has been considered by Dr AJ Williams, the Co-Chair and by   ',
Expert Member.

The Committee does not consider this to be a "substantial amendment" as defined in the Standard Operating Procedures for Research Ethics Committees. The amendment does not therefore require ethical review by the Committee and may be implemented immediately, provided that it does not affect the research governance approval for the research given by the R&D Department for the relevant NHS care organisation.

Documents received

The documents received were as follows:

Document	Version	Date
Covering email		05 May 2006

Appendix 2 – Participant questionnaires

INPUT Pain Management Programme
Patient Questionnaire

Name: _____

Address: _____

_____

_____

Postcode: _____

Telephone: _____ Date of birth:_____

In order to make a proper assessment of your pain problem we need as much information as possible about you and your pain. Some of this can be gained from the interview, but we also find it helpful for you to fill out this questionnaire before you come.

The questionnaire is intended to give us some information about your pain, such as when it started and what treatments you may have had, as well as some details about you and your background.

Your answers to all of these questions will help us to get a general picture of you and your pain problem. Please be assured that your answers are totally confidential.

If you have difficulty with any of the questions just leave them blank - we can discuss them during your assessment.

GP Name: _____

Address: _____

_____

_____

Postcode: _____

Telephone: _____

Treatment for pain problems

Please indicate if you have had any of the following treatments by circling your answer:

1. medicines (any type)...   **no**   **yes**
2. surgery..   **no**   **yes**
3. injections/nerve blocks/epidurals...............................   **no**   **yes**
4. TENS...   **no**   **yes**
5. acupuncture..   **no**   **yes**
6. bed rest (in hospital)...   **no**   **yes**
7. physiotherapy..   **no**   **yes**
8.   traction...   **no**   **yes**
9.   hydrotherapy...   **no**   **yes**
10.   Alexander technique..   **no**   **yes**
11. saw a psychiatrist..   **no**   **yes**
12. saw a psychologist...   **no**   **yes**
13.   relaxation..   **no**   **yes**
14.   biofeedback..   **no**   **yes**
15.   hypnosis..   **no**   **yes**
16. osteopathy..   **no**   **yes**
17. chiropractic...   **no**   **yes**
18. faith healing..   **no**   **yes**
19. other _____

Have you had private treatment for the pain?	**no** **yes**	
It will help us to know approximately how much it cost:	less than £500	1
	£500 to £1000	2
	£1000 to £5000	3
	more than £5000	4

Do you have private medical insurance?   **no**   **yes**

Do you take medicines for the pain?   **no**   **yes**

If so, do you take them: when needed   **no**   **yes**

                   regularly by the clock   **no**   **yes**

                   both   **no**   **yes**

Please list the medicines you currently take for the pain

Drug	Dose	Times daily

Please list any other drugs you take (not for pain)

_____ _____

_____ _____

_____ _____

_____ _____

1351098579

FOLLOW-UP REVIEW - 07

Name... Date...............................

Please fill in the applicable boxes, to let us know how, and how often, you are using what you learned on the programme.

STRETCH

How often do you do stretches?

stopped completely ☐
less than once a week ☐
1 - 2 times a week ☐
3 - 4 times a week ☐
5 - 6 times a week ☐
at least once a day ☐

EXERCISES

How often do you do exercise?

stopped completely ☐
less than once a week ☐
1 - 2 times a week ☐
3 - 4 times a week ☐
5 - 6 times a week ☐
at least once a day ☐

RELAXATION

How often do you relax?

stopped completely ☐
less than once a week ☐
1 - 2 times a week ☐
3 - 6 times a week ☐
at least once a day ☐

When do you use relaxation?
(You can fill in more than one box.)

rests and breaks ☐
increased pain ☐
when stressed ☐
during activities ☐

PACING

How often do you use pacing?

stopped completely ☐
less than once a week ☐
1 - 2 times a week ☐
3 - 6 times a week ☐
at least once a day ☐
most of the time ☐

When do you use pacing?
(You can fill in more than one box.)

not at all ☐
when I remember ☐
when the pain is bad ☐
for some activities ☐
indoors only ☐
as a daily approach ☐

CHALLENGING THOUGHTS

How often do you check your thoughts, and challenge them if necessary?

stopped completely ☐
less than once a week ☐
1 - 2 times a week ☐
3 - 6 times a week ☐
at least once a day ☐
most of the time ☐

When do you do this?
(You can fill in more than one box)

not at all ☐
when I remember ☐
when the pain is bad ☐
when I'm anxious ☐
when I'm depressed ☐
when someone upsets me ☐
as a daily approach ☐