EFFECTS OF A 10-WEEK PROGRESSIVE GOAL ATTAINMENT PROGRAMME ON SELECTED OUTCOMES IN PATIENTS RECEIVING CONVENTIONAL TREATMENT PROCEDURES FOR MECHANICAL LOW BACK PAIN

BY

OGUNLANA, MICHAEL OPEOLUWA

B.Sc. Physiotherapy (Ibadan), M.Sc. Epidemiology (Ibadan)

A Thesis in the Department of Physiotherapy,
Submitted to the Faculty of Clinical Sciences
In partial fulfillment of the requirements for the Degree of

DOCTOR OF PHILOSOPHY (Orthopaedic, Sports and Recreational Physiotherapy)

Of the

UNIVERSITY OF IBADAN

SEPTEMBER 2015

CERTIFICATION

We certify that this work was carried out by Mr M.O Ogunlana in the Department of Physiotherapy, University of Ibadan

……..…..

SUPERVISOR

Dr. Adesola C Odole
Senior Lecturer/
Department of Physiotherapy
College of Medicine,
University of Ibadan, Nigeria;
Consultant Physiotherapist,
University College Hospital
Ibadan, Nigeria.

……

CO-SUPERVISOR

Dr. Adebayo Adejumo
Senior Lecturer
Department of Psychology
University of Ibadan, Nigeria

DEDICATION

This work is dedicated to the memory of my late parents

Mr. and Mrs. S.A Ogunlana

Who worked hard to lay a solid foundation for all my life pursuit.

ABSTRACT

Mechanical Low Back Pain (MLBP) is a major cause of disability and may be influenced by psychosocial variables such as fear-avoidance beliefs, catastrophic thinking, reduced self-efficacy and perceived disability. Progressive Goal Attainment Programme (PGAP) is an activity-based cognitive behavioural therapy that is often administered by physiotherapists as an adjunct therapy to improve treatment outcomes for individuals with MLBP. Prospective studies on the effects of PGAP on treatment outcomes in individuals with MLBP are not readily available globally while information on its therapeutic effects on psychosocial variables in patients with MLBP in Nigeria is scarce. The effects of a 10-week PGAP adjunct therapy on selected pain and psychosocial characteristics in patients with MLBP were investigated in this study.

This quasi-experimental study involved seventy (42 females; 28 males) consecutively selected individuals with newly diagnosed MLBP at the physiotherapy clinic, Federal Medical Centre Abeokuta. Participants were screened for Pain Catastrophising (PC) and kinesiophobia using Pain Catastrophising Scale and Tampa Scale for Kinesiophobia. Participants were alternately assigned into experimental group (EG) and control group (CG). The EG received PGAP and conventional treatment for MLBP while the CG received only conventional treatment. The PGAP included a walking program, activity scheduling and monitoring. Conventional treatment entailed routine medical treatment (Paracetamol and Ibruprofen) and physiotherapy care (soft tissues mobilisation and Mckenzie exercise). Both groups received treatment thrice weekly for 10 weeks. Participants' pain intensity (PI), PC, Kinesiophobia, Perceived Disability (PD) and Self-Efficacy were assessed at baseline, end of 5^{th} and 10^{th} week of intervention using Visual Analogue Scale, Pain Catastrophising Scale, Tampa Scale for Kinesiophobia, Revised Oswestry Disability Questionnaire and Self-Efficacy in Rehabilitation Scale, respectively. Participants were re-assessed 12 weeks after intervention. Data were analyzed using t-test, Mann-Whitney U and Freidman's ANOVA at p=0.05.

The ages of EG (44.97±8.29years; n=35) and CG (47.43±7.54years; n=35) were comparable. At baseline, scores for PI (9.4±0.9; 9.1±0.9); PC (33.6±9.9; 33.0±5.3), kinesiophobia (41.4±7.7; 41.5±3.0); PD (59.1±12.8; 55.5±12.3); self-efficacy (81.4±9.5; 81.2±12.0) for EG and CG were not significantly different. Between-group comparison at the end of 5^{th} week revealed that scores for PC (22.2±11.2; 27.9±8.8),

kinesiophobia (37.3±7.5; 42.2±3.2), self-efficacy (94.4±14.5; 80.0±20.1) for EG and CG respectively were significantly different but PI (4.9±1.9; 5.0±2.8) was not significantly different. Between-group comparison at the end of 10th week revealed that the scores for PI (3.6±1.6; 3.1±1.8), PC (23.0±9.42; 23.0±8.4); kinesiophobia (34.4±6.8; 36.9±3.7), self-efficacy (94.4±11.5; 94.1±9.4) for EG and CG were not significantly different. At the end of 5th and 10th weeks, scores for perceived disability for EG (42.6±11.1; 41.1±8.5) were significantly lower than CG (57.8±8.9; 45.3±7.3) respectively. At 12 weeks follow-up, EG had lower scores for PI (3.8±1.6; 5.0±1.6); PC (21.7±9.5; 27.5±5.8), kinesiophobia (29.1±6.3; 35.8±6.6), PD (33.0±7.0; 43.4±7.6) and significantly higher score for self-efficacy (101.2±11.5; 92.3±9.3) than CG.

Addition of Progressive Goal Attainment Programme to conventional treatment is effective in achieving sustained reduction in perceived disability among patients with mechanical low back pain. This study serves as evidence for incorporating Progressive Goal Attainment Programme into treatment for patients with mechanical low back pain having psychosocial overlay.

Keywords: Mechanical Low Back Pain, Psychosocial factors, Progressive Goal Attainment Programme, Perceived disability.

WORD COUNT: 499

ACKNOWLEDGEMENT

I want to appreciate my Head of Department Professor T.K Hamzat and am grateful to all my lecturers (Professor Arinola O. Sanya, Dr. Aderonke Akinpelu, Dr. B.O.A Adegoke, Dr. Ayanniyi, Dr. A.F Adeniyi, Dr. Omoyemi O. Ogwumike, Dr. A.A Fabunmi, Revd. A.O Jaiyesimi, Mrs. Nse Odunaiya, Dr. Olubukola A. Olaleye, Dr. A. Akinremi) for their contributions to this endeavor.

My profound gratitude goes to my Teachers, Mentors and Supervisors Dr. Adesola Christiana Odole and Dr. Adebayo Adejumo for their relentless encouragement and support. Words are not sufficient to express my gratitude.

Sincere thanks to my nuclear and extended family, I owe you my sincere appreciation for your understanding and help.

I say a big thank you to friends and colleagues whose contributions are worthy of note.

To all the individuals who participated in this study, I say thank you.

Above all, I give thanks to God almighty who gave me the grace and strength to complete this work.

TABLE OF CONTENTS

Title Page		i
Certification		ii
Dedication		iii
Abstract		iv
Acknowledgement		vi
Table of Contents		vii
List of Tables		xi
List of Figures		xii
CHAPTER ONE:	INTRODUCTION	1
1.1	Introduction	1
1.2	Statement of Problem	4
1.3	Aims of Study	5
1.4	Hypotheses	5
1.4.1	Major Hypotheses	5
1.4.2	Sub-hypotheses	6
1.5	Delimitation of study	7
1.6	Limitation of study	8
1.7	Significance of study	8
1.8	Definition of Terms	8
CHAPTER TWO:	REVIEW OF LITERATURE	10
2.1	Definition of low back pain	10
2.1.1	Epidemiology of low back pain	10
2.1.2	Predisposing factors for low back pain	12
2.1.3	Other conditions that make people susceptible to low back pain	13

2.1.4 Risk factors for low back pain 17

2.2 Treatment of low back pain 17

2.2.1 Prevention of low back pain 19

2.2.2 Models for clinical classification of low back pain 20

2.3 The nature of pain 23

2.3.1 From the Biomedical model to the Biopsychosocial model 23

2.3.2 The Relationship between pain and disability 26

2.4 Psychosocial influences of pain on disability 26

2.4.1 Pain Catastrophising 27

2.4.2 Fear as a predictor of pain and disability 29

2.4.3 Self Efficacy and disability 30

2.5 Cognitive Behavior therapy 31

2.5.1 Psychologically informed practice in Physiotherapy 33

2.6 Progressive Goal Attainment Programme (PGAP) 34

2.7 Review of Previous Studies on Psychological interventions in patients with low back pain 38

CHAPTER THREE: MATERIALS AND METHODS 54

3.1 Participants 54

3.2 Instruments 54

3.3 Setting for the Study 57

3.4 Methods 57

3.4.1 Research design 57

3.4.2 Sampling Technique 57

3.4.3 Sample size determination 57

3.5 Procedure for Data collection 58

3.5.1 Ethical Approval 58

3.5.2 Recruitment procedure 58

3.5.3 Screening procedure 58

3.5.4 Translation of Instruments 59

3.5.5 Pilot testing of instruments 59

3.5.6 Data collection 60

3.5.7 Intervention 60

3.6 Data analysis 64

CHAPTER FOUR: RESULTS AND DISCUSSION 67

4.1 Results 67

4.1.1 Demographic and clinical characteristics of participants 67

4.1.2 Comparison of selected pain-related and psychosocial variables of participants in the experimental and control groups at baseline. 71

4.1.3 Changes in selected pain related and psychosocial variables of participants in the experimental group … 73

4.1.4 Changes in selected pain related and psychosocial variables of participants in the control group … 75

4.1.5 Between-group comparison of selected pain related and psychosocial variables of participants in the experimental and control groups at the end of 5th week of the study. 77

4.1.6 Between-group comparison of selected pain related and psychosocial variables of participants in the experimental and control groups at the end of 10th week of the study. 79

4.1.7 Between-group comparison of selected pain related and psychosocial variables of participants in the experimental and control groups at the end of 10th week of the study. 81

4.2 Hypotheses Testing 88

4.3 Discussion 96

4.3.1 Comparison of demographic and selected clinical variables of Participants in Control and Experimental Groups. 96

4.3.2 Comparison of selected pain related and psychosocial variables of participants in the experimental and control groups 96
4.3.3 Changes in selected pain related and psychosocial variables of participants. 97
4.3.4 Accounting for research bias 99
CHAPTER FIVE: SUMMARY, CONCLUSION AND RECOMMENDATION
5.1 Summary 101
5.2 Conclusion 102
5.3 Recommendations 103
References 104
Appendices 128

LIST OF TABLES

TABLE		PAGE
2.1	Review of previous studies	41
4.1	Comparison of demographic variables	69
4.2	Comparison of demographic and selected clinical variables of experimental and control groups at baseline	70
4.3	Between group comparison of selected pain related and psychosocial variables of experimental and control groups at baseline	71
4.4	Changes in selected pain related and psychosocial variables of participants in the experimental group	74
4.5	Comparison of changes in selected pain related and psychosocial variables of participants in the control group	76
4.6	Between-group comparison of selected pain related and psychosocial variables of experimental and control groups at the end of 5th week	78
4.7	Between-group comparison of selected pain related and psychosocial variables of experimental and control groups at the end of 10th week	80
4.8	Between-group comparison of selected pain related and psychosocial variables of experimental and control groups at three months follow-up	82

LIST OF FIGURES

FIGURE		PAGE
2.1	The biopsychosocial model by Engell	22
2:2	A conceptual model of the biopsychosocial interactive process	36
2.3	Conceptual model for the study	37
3.1	Flowchart of study	66
4.1	Trend of Pain Intensity scores in the EG and CG	83
4.2	Trend in Pain Catastrophising in the EG and CG	84
4.3	Trend in Kinesiophobia in the EG and CG	85
4.4	Trend in pain related disability in the EG and CG	86
4.5	Trend in Self Efficacy in the EG and CG	87

CHAPTER ONE
INTRODUCTION

1.1 Introduction

Low back pain (LBP) affects 85% of the world population at some time (Deyo et al, 2006) and is one of the most frequent reasons both for consulting a primary care physician and for taking time off work (Deyo et al, 2006). Patients with LBP not only suffer from physical discomfort, but also functional limitation that might cause disability and interfere with their quality of life (Horng et al, 2005, Ogunlana et al, 2012a). Chou and Hoffman (2007) recommended that patients with LBP can be classified into one of three broad categories: nonspecific low back pain also called Mechanical Low Back Pain (MLBP), back pain potentially associated with radiculopathy or spinal stenosis and back pain potentially associated with another specific spinal cause. Mechanical Back Pain refers to any type of back pain caused by any abnormal stress and strain on muscles and ligaments of the vertebral column (back region) (Akinbo, 2014). Typically, mechanical back pain results from poor posture, poorly-designed seats, incorrect bending and lifting motions (Akinbo, 2014).

Low Back Pain can interfere with activities that range from basic activities of daily living such as walking and dressing to many work-related functions. It appears that pain determines disability in patients with LBP but studies (Pincus et al, 2002; Nachemson,1992) have shown that the intensity of pain and the degree of disability do not correlate well and are associated with different risk factors (Kovacs et al, 2005). Different therapeutic interventions are available for the treatment of MLBP and these often include psychosocial interventions. The usage of psychosocial interventions is premised on the fact that pain and its resulting disability are not only influenced by somatic pathology if diagnosed but by psychological and social factors (Ostelo et al. 2008). Mechanical low back pain is a physical problem that may be influenced by psychosocial variables like fear-avoidance beliefs, catastrophic thinking and perceived disability (Brunner et al, 2012). These psychosocial variables are also termed “yellow flags” and their definition is now confined to psychological risk factors that may be considered essentially ‘normal’ but unhelpful psychological

reactions to musculoskeletal symptoms (Nicholas et al., 2011). Pain catastrophising and kinesiophobia have been reported to be major predictors of persistence of pain and disability in patients with pain problems (Picavet et al. 2002). Painful conditions eventually results in reduction in self efficacy and performance of physical activities (Adegoke & Ezeukwu, 2010). Pain-catastrophising is a significant cognitive component of the pain experience involving 'an exaggerated negative orientation to aversive stimuli' (Sullivan et al., 1995). It consist of three elements that include ruminating about pain, appraising pain in a manner that magnifies its threat value, and devaluing resources available to cope with it (Sullivan et al., 2001). Kinesiophobia describes fear of movement and fear of re-injury (Vlaeyen et al, 1995). It is "an irrational and debilitating fear of physical movement and activity resulting from a feeling of vulnerability to painful injury or (re)injury" (Kori et al., 1990). Self-efficacy roughly corresponds to a person's belief in their own competence. It is described as the belief that one is capable of performing in a certain manner to attain a certain set of goals (Bandura, 1997).

Psychosocial treatment for pain-related conditions has typically taken the form of Cognitive–Behavioural pain-management Therapies (CBT) (Morley et al, 1999; Lefort et al, 1998). The term ''cognitive–behavioural'' refers not to a specific intervention but to a class of intervention strategies that may include self-instruction (e.g., motivational self-talk), relaxation or biofeedback, exposure, developing coping strategies (e.g., distraction, imagery), increasing assertiveness, minimizing negative or self-defeating thoughts, changing maladaptive beliefs about pain, and setting goals (Linton, 2002). Three behavioural treatment approaches can be distinguished vis-a-vis: operant, cognitive and respondent (Vlaeyen, 1995). Each of these focus on the modification of one of the three response systems that characterise emotional experiences which are: behaviour, cognition and physiological reactivity (Ostelo et al. 2008). As a function of the profile of presenting problems, a client participating in a cognitive behavioural intervention may be exposed to varying selections or combinations of these strategies. Traditionally, cognitive behavioural pain management programmes have been delivered by psychologists or other rehabilitation professionals with a background in mental health (Linton, 2002). Given the strategic position of the physiotherapist as a first-line health care professional for problems associated with musculoskeletal injury, it has been suggested that physiotherapists

might be ideally suited to intervene on psychosocial barriers to rehabilitation progress (Linton, 2002). This strategy may reduce the stigma associated with visiting a psychologist or psychiatrist for therapy.

Progressive Goal Attainment Programme (PGAP) designed by Sullivan et. al, (2006) is an activity-based CBT intervention that consists of goal-attainment techniques, activity and mobilization strategies designed to target psychosocial risk factors of pain and disability. It is based on the biopsychomotor model of pain (Sullivan, 2008) that incorporates a central role for behaviour. The gate control theory and the neuromatrix model of pain were silent on the role of behaviour in the pain system. The bio-psychomotor model considers two main intra-individual behavioural systems (the communicative and protective behaviour system) in addition to the sensory component of the pain system (Sullivan, 2008).

The PGAP consists of a maximum of 10 weekly contacts between a trained PGAP provider and a patient with pain. Progressive goal attainment programme incorporates a variety of techniques that have been shown to either reduce catastrophising or reduce the negative impacts of catastrophising. Disclosure techniques are used to reduce pain severity and emotional distress that might be contributing to high levels of catastrophising (Sullivan et. al 2006). Activity and mobilization techniques are used to create a more enriched environment that will reduce the frequency and impact of catastrophic thoughts. Fear reduction techniques and belief change techniques are incorporated to indirectly target catastrophic thinking and kinesiophobia (Sullivan et. al 2006).

In a study on patients with chronic cervical pain, individuals who were participating in a functional restoration physical therapy program were compared with a sample of individuals who received PGAP in addition to the same physical therapy intervention (Sullivan et. al 2006). The results showed that, at treatment termination, there were no significant differences in pain severity or pain-related fear. However, the individuals who received PGAP showed greater reductions in catastrophizing and were more likely to return to work. More recently, Sullivan and Adams, (2010) examined the added value of including PGAP in the rehabilitation of individuals with recent onset (<12weeks) musculoskeletal pain conditions. At 1 year follow-up, individuals who

received PGAP, compared with physiotherapy alone, required fewer additional treatment sessions, required less pain medication and were more likely to return to work. However, the two groups did not differ significantly on their self-reported pain severity. These results suggest that programmes like PGAP might not reduce pain significantly, but might prevent disability associated with chronic pain and improve patients' self-efficacy.

This study was designed to investigate the effect of PGAP alongside conventional treatment on pain intensity, pain catastrophising, kinesiophobia, disability and self-efficacy among patients presenting with mechanical low back pain.

1.2 Statement of the problem.

Management of mechanical low back pain (MLBP) is a challenge for healthcare professionals as well as the healthcare system because of its high incidence and prevalence (Ostelo et al. 2008). It is a major cause of medical expenses, absenteeism and disablement (Van Tudder, 1995, Odole et al, 2012, Ogunlana et al, 2012b). A large variety of therapeutic interventions are available for treatment of MLBP and psychosocial interventions are commonly used. The usage of psychosocial interventions is premised on the fact that pain and its resulting disability is not only influenced by somatic pathology if diagnosed, but by psychological and social factors (Ostelo et al. 2008). MLBP is a physical problem that may be influenced by psychosocial variables like fear-avoidance beliefs, catastrophic thinking and perceived disability (Brunner et al, 2012).

Pain catastrophising (PC) and kinesiophobia have been shown to correlate positively with many aspects of pain experience, including pain intensity, emotional distress, pain-related disability, health services use, pain behaviour, and reliance on medication (Linton et al., 1998; Sullivan et al., 1998; Sullivan and Neish, 1999; Sullivan et al., 2001; Goubert et al., 2002; Picavet et al. 2002; Goubert et al., 2004). Studies from developed countries (Rosenstiel and Keefe, 1983; Sullivan et al., 1998; Sullivan et al., 2001) have shown that measures of catastrophizing are significantly correlated with measures of disability. The therapeutic effects of PGAP on PC, kinesiophobia and patients' self-efficacy have not been documented in patients with mechanical LBP in

Nigeria. It appears that the only study (Sulivan and Adams, 2010) that has documented the efficacy of PGAP in patients with mechanical LBP was retrospective in design. Prospective studies on the effects of PGAP on treatment outcomes in individuals with MLBP are not readily available globally while information on its therapeutic effects on psychosocial variables in patients with MLBP in Nigeria is scarce. Cultural differences could modify the effect of PGAP on LBP patients' pain experience as studies have established culture as a main determinant of pain behaviour (Baker and Green, 2005; Lebovits, 2005). This study therefore answered the following questions:

1. What were the effects of PGAP on pain intensity, pain related disability, PC, Kinesiophobia and self-efficacy in patients with MLBP?
2. What would be the carry over effects of PGAP on pain intensity, pain related disability, PC, Kinesiophobia and self-efficacy in patients with MLBP at three-month post intervention?

1.3 Aims of study.

i. To determine the effects of PGAP on pain intensity, pain related disability, PC, kinesiophobia and self-efficacy in patients with MLBP.
ii. To determine the carry over effects of PGAP on pain intensity, pain related disability, PC, kinesiophobia and self-efficacy in patients with MLBP at three-month post intervention.

1.4 Hypotheses

1.4.1 Major Hypotheses

1. A 10-week PGAP would have no significant effect on pain intensity, PC, kinesiophobia, self-efficacy and pain related disability in individuals with MLBP.
2. A 10-week PGAP would have no significant effect on pain intensity, PC, kinesiophobia, self-efficacy and pain related disability in individuals with MLBP at three-month follow up.

1.4.2 Sub-hypotheses

1. There would be no significant difference in pain intensity scores of individuals in the experimental group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
2. There would be no significant difference in the PC scores of individuals in the experimental group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
3. There would be no significant difference in the kinesiophobia scores of individuals in the experimental group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
4. There would be no significant difference in the disability scores of individuals in the experimental group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
5. There would be no significant difference in the self-efficacy scores of individuals in the experimental group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
6. There would be no significant difference in the pain intensity scores of individuals in the control group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
7. There would be no significant difference in the PC scores of individuals in the control group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
8. There would be no significant difference in the kinesiophobia scores of individuals in the control group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
9. There would be no significant difference in the disability scores of individuals in the control group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
10. There would be no significant difference in the self-efficacy scores of individuals in the control group with MLBP across baseline, end of fifth week, end of tenth week and at three-month follow up.
11. There would be no significant difference between the pain intensity scores of individuals in the experimental and control groups with MLBP at baseline, end of fifth week, end of tenth week and at three-month follow up.

12. There would be no significant difference between the PC scores of individuals in the experimental and control groups with MLBP at baseline, end of fifth week, end of tenth week and at three-month follow up.
13. There would be no significant difference between the kinesiophobia scores of individuals in the experimental and control groups with MLBP at baseline, end of fifth week, end of tenth week and at three-month follow up.
14. There would be no significant difference between the disability scores of individuals in the experimental and control groups with MLBP at baseline, end of fifth week, end of tenth week and at three-month follow up.
15. There would be no significant difference between the self-efficacy scores of individuals in the experimental and control groups with MLBP at baseline, end of fifth week, end of tenth week and at three-month follow up.

1.5 Delimitation of study

This study was delimited to the following:

(i) All consenting individuals diagnosed with MLBP

(ii) The use of the Visual Analogue Scale (VAS), Pain Catastrophizing Scale (PCS), Tampa Scale for Kinesiophobia (TSK), Revised Oswestry Disability Questionnaire (RODQ) and Self-Efficacy in rehabilitation Scale (SES).

(iii) Selected pain characteristics of pain intensity, pain related disability and duration of pain onset.

(iv) Selected psychosocial risk factors of kinesiophobia, pain catastrophising and self-efficacy.

1.6 Limitation of study

It was difficult to get all patients to attend the follow up assessment after three months especially for the participants that had complete recovery from the MLBP episode, though the researcher tried locating some of the participants through their mobile phones and home addresses. This reduced the number of participants that were assessed at three months follow-up.

Also it was difficult to monitor the oral analgesic intake of the participants even though paracetamol and or Ibruprofen were the standard prescription by the referring medical practitioner.

1.7 Significance of study

The outcome of this study revealed that addition of Progressive Goal Attainment Programme to conventional medical and physiotherapy treatment is effective in achieving sustained reduction in perceived disability among patients with mechanical low back pain. It should be incorporated into treatment for patients with mechanical low back pain and psychosocial overlay.

This outcome also supports the initiation of psychologically-informed physiotherapy practice in clinical practice.

This study provides evidence for the usage of Progressive Goal Attainment Programme as an adjunct treatment programme in the reduction of perceived disability among patients with MLBP.

1.8 Definition of terms

Mechanical LBP: Pain between the costal margins and the inferior gluteal folds, usually accompanied by painful limitation of movement, often influenced by physical activities and posture, and which may be associated with referred pain in the leg. This pain is not related to conditions such as fractures, spondylitis, direct trauma, or neoplastic, infectious, vascular, metabolic, or endocrine-related processes (Deyo, 2001; Chou et al, 2007).

Perceived disability: This refers to a person's appraisal or belief about his or her level of activity limitation (Sullivan, 2010).

Disability: For the purpose of this study is defined from the pragmatic perspective as behaviour of reduced participation in activities of daily living (Sullivan, 2010).

Psychosocial: The term psychosocial refers to the interaction between the person and his social environment, and its influences on his behaviour (Kendall et. al., 1997).

Selected Treatment Outcomes: For the purpose of this research, selected treatment outcomes refer to pain intensity, extent of pain catastrophizing, kinesiophobia, disability and self-efficacy.

CHAPTER TWO
LITERATURE REVIEW

2.1 Introduction

Low Back pain is pain, muscle tension or stiffness, localized in the back below the costal margin and above the gluteal folds with or without leg pain (Smiths et al, 2001). LBP can be classified by diagnosis as specific or non-specific and by symptom duration as acute, sub-acute or chronic (Koes et al, 2006; Vas et al, 2006). Specific low back pain is attributed to a structural problem, such as a herniated nucleus pulposus, fracture, arthritis, tumor, or infection. Patients with complaints of specific LBP typically present with signs suggestive of the underlying structural problem. These signs might include axial pain, radiculopathy, or an abnormal neurological examination (Abenheim, 2000; Smith et al, 2001). Non-specific LBP is associated with vague and diffuse complaints of pain, and neurologic examinations are generally normal. Acute LBP is LBP that has been present for less than four weeks; sub-acute LBP is present for four to less than twelve weeks, while chronic LBP is LBP persisting for at least twelve weeks (Abenheim, 2000; Smith et al, 2001).

2.1.1 Epidemiology of Low Back Pain

Review of literature describing LBP point prevalence in the developed world have reported varying estimates of prevalence rates (Leboeuf-Yde & Lauritsen, 1997; Loney & Stratford , 1995). In the studies considered by Looney and Stratford to be methodologically sound, the LBP point prevalence was estimated to be 6.8% in North America, 12% in Sweden, 13.7% in Denmark, 14% in the United Kingdom, 28.4% in Canada, and 33% in Belgium (Loney & Stratford , 1995). The size of the difference between the LBP point prevalence in North America estimated by Deyo and Tsui-Wu at 6.8% (Deyo & Tsui-Wu, 1987) and that of Canada at 28.4% illustrates the variability attributable, in unknown proportion, to sample and sampling differences.

In a review of world prevalence data, Volinn (1997) suggested that there were lower rates of prevalence in developing countries than in developed countries, but did not determine whether differences reflect demographic, cultural or research method factors. Walker (2000) conducted a systematic review of the Australian LBP prevalence literature 1966–1998, and also concluded that the true prevalence of LBP in Australia remained confounded by methodological flaws in previous studies. Walker et al. (2004), subsequently surveyed 3000 Australian adults using contemporary epidemiological methods, and estimated the point prevalence of LBP at 25.5%, six-month period prevalence at 64.6% and lifetime prevalence at 79.2%. The retrospective one-year first incidence of LBP in the sample was 8.0%. These data suggest that LBP is common in the Australian population, with four out of five adults experiencing LBP in their life and approximately one in 12 experiencing a new episode of LBP over a 12-month period. A large difference between the point prevalence and the six-month prevalence of LBP in Walker's finding is also seen in other epidemiological studies (Louw et al, 2007) and probably reflects the fluctuating, episodic nature of most LBP.

This review did not reveal evidence of gender differences in LBP prevalence in adults sampled from the USA (Deyo & Tsui-Wu, 1987) Canada, Nordic countries and Australia (Walker et al. 2004), nor in a Finish sample of children and adolescents (Taimela et al.1997). The prevalence of LBP in children is low (1%-6%) but increases rapidly (18%–50%) in the adolescent population (Taimela et al, 1997). The prevalence of LBP peaks around the end of the sixth decade of life. For example, in a prospective 12-month study of 4501 adults in the South Manchester region of the United Kingdom, the age distribution of LBP was unimodal, with the peak prevalence occurring in those aged 45 to 59 years old. This is similar to USA epidemiological data describing the peak point prevalence, period prevalence and lifetime prevalence all within ages 55 to 64 years (Deyo & Tsui-Wu, 1987). Though some age-specific back pain cost data had a bimodal distribution with a peak for women over 75 years of age, it is likely that this did not represent an increase in the prevalence of non-specific back pain but the prevalence of serious pathology (including compression fracture).

Louw et al, (2007) reviewed the prevalence of low back pain in Africa using 27 eligible epidemiological studies. The majority of the studies (63%) were conducted in

South Africa (37%) and Nigeria (26%). The most common population group from the review involved workers (48%) and scholars comprised 15% of the population. 67% of the studies were found to be methodologically sound. The mean LBP point prevalence among the adolescents was 12% and among adults was 32%. The average one year prevalence of LBP among adolescents was 33% and among adults was 50%. The average lifetime prevalence of LBP among the adolescents was 36% and among adults was 62%. The study concluded that the global burden of disease of LBP is increasing even in Africa and that further research is needed to identify the most effective strategies to prevent and manage LBP in Africa.

2.1.2 Predisposing Factors for Low Back Pain

According to Mckenzie (2010), there are basically three predisposing factors in the aetiology of NSLBP. These include: the sitting posture, loss of extension range of the lumbar spine and the frequency of flexion of the lumbar spine**.**

(i) Sitting posture:

A good sitting posture maintains the spinal curves normally present in the erect standing position. Postures which reduce or accentuate the normal spinal curves enough to place the ligamentous structures under full stretch will produce pain. Such postures are referred to as poor sitting posture (Mckenzie, 2010). A poor sitting posture will frequently enhance and always perpetuate the spine problems of patients suffering from low back pain. In relaxed sitting, the lumbar is in a fully stretched position. This will become painful if maintained for a prolonged period.

Environmental factors contribute to aetiology of low back pain due to sitting, since working platforms which are not adjusted to individual requirements; poorly designed seating for domestic, commercial and transportation purposes will promote poor sitting postures. To facilitate an efficient working position in sitting, a redesign of furniture may be necessary (Chou and Huffman, 2007). Postural factors such as certain sleeping positions as well as work-related postures may be potentially damaging and will under certain circumstances perpetuate low back pain.

(ii) Loss of Extension Range.

Mckenzie (2010) reported that a loss of lumbar extension range predisposes to low back pain production. A reduced range of extension influences the posture in sitting, standing and walking. Habitual poor posture in these positions causes the lumbar

spine to undergo adaptive changes such that the lumbar range of extension is reduced and the ability to sit with a lordosis is impaired or lost. As the loss of extension increases, the patient will be forced to walk slightly stooped. The maintenance of the slightly flexed posture creates a constant stress on the nucleus and posterior annular wall. Moving into extension normally relieves this stress, however as extension is no longer possible, lasting relief cannot be obtained. This results in adaptive changes, which extends to all periarticular structures including the apophyseal joints (Chou and Huffman, 2007).

(iii) Frequency of Flexion

According to Mckenzie (2010), frequency of flexion is a third predisposing factor in NSLBP. They examined the lifestyle of western cultures in the twentieth century concerning frequency of flexion of the lumbar spine as related to many activities of daily living. Such activities include stooping over a wash hand basin, sitting to have breakfast, sitting in travelling by bus, train or car, bending over in working either in sitting or standing. Gracovetsky, (1981) indicated that when one bends over at the waist and reaches full forward bending of flexion, the back muscles cease working because in this fully flexed position the ligaments get involved. The greatest stress is however on the superficial ligaments (the supra and interspinous ligaments) because the stress works from superficial to deeper layers of ligament (Apts, 1992). The theory therefore is that when one bends at the waist often enough, and twists frequently, the ligament are subject to too much stress. If the tensile force is high, the ligaments will start to break down and disc prolapsed may occur (Apts, 1992). In the face of this, it was recommended that patients with low back pain should extend the lumbar spine from time to time; this will theoretically reduce the stress on the posterior annular wall and simultaneously cause fluid nucleus to move anteriorly, that is away from the site of most protrusions and extrusions. Moreover, patients should sit with the lumbar spine supported in some extension as in this position, the intradiscal pressure is reduced (Delitto et al, 2012).

2.1.3 Other Conditions That Make People Susceptible To Low Back Pain.

In 85% of back pain cases, the causes are unknown. However, in most known cases of low back pain, pain begins with an injury, after lifting heavy object, or after making

an abrupt movement. A number of conditions make people more or less susceptible to low back pain from such events (Delitto, et al, 2012) and these include:

(i) Aging process

Intervetebral discs begin deteriorating and growing thinner by age 30 (Chou and Huffman, 2007). As people continue to age and the discs lose moisture and shrink, the risk for spinal stenosis increases. In women, the incidence of low back pain and sciatica increases at the time of menopause as they lose bone density. In the older adults, osteoporosis and osteoarthritis are also common. However the risk for low back pain does not mount steadily with ever-increasing age, which suggests that at a certain point, the condition causing low back pain plateaus (Delitto et al, 2012).

(ii) Genetic Factors

Many people have a genetic susceptibility to low back pain usually from inheriting spinal structural abnormalities. Marini, (2001) found that specific mutation of the COL9A gene may play a role in about 10% of cases of sciatica. This gene is normally involved in producing collagen, the protein building block in all structural tissues of the body. When defective, it may cause the disc to be less able to resist compressive forces. Marini, (2001) found that the defective gene was present in twice as many patients with disk problems as in patients without back pain.

(iii) Central Nervous System Abnormalities

After episodes of back pain, some people may experience changes in brain function that led them to chronic back pain. Such changes include an exaggerated response in nerve cells or other factors that cause a persistent perception of pain even without an actual physical injury (Foster, 2001).

(iv) Psychological and Social Factors

Psychological factors are known to play a strong influential role in the three phases of low back pain namely: onset of pain, perception of pain and chronic pain (Delitto et al, 2012). It has now been indicated that in many people, pre-existing depression and inability to cope may be more likely to predict the onset of pain than physical abnormalities (Williams and Myers, 1998). The perception of pain is affected by social and psychological factors in that people who are depressed are more likely to have vague physical symptoms, including low back pain. For example, in one study,

pilots (who generally reported "loving" their jobs) reported fewer back problems than their flight crews. Another study reported that low rank, low social support and high stress in soldiers were associated with a higher risk for disabling back pain (Reese and Mittag, 2007).

Furthermore, the way a patient perceives and copes with pain at the beginning of an acute attack may actually condition the patient to either recover or develop a chronic condition (Deyo and Weistein, 2001). Those who over-respond to pain tend to feel out of control and become discouraged thereby increasing their risk for long – term problem. In fact, some studies reported that in patients with existing back problems, the fear of pain was actually more disabling than the pain itself (Feurstein and Beattie, 1995; Williams and Myers, 1998).

(v) Pregnancy

Pregnant women are prone to back pain due to a shifting of abdominal organs, the forward redistribution of body weight and the loosening of ligaments in the pelvic area as the body prepares for delivery. Tall pregnant people are at high risk than short people (Colliton, 1996). Back pain in pregnancy may be classified into lumbar pain, sacroiliac pain and nocturnal pain. Lumbar pain can occur with or without radiation to the legs. It can stem from multiple sites but most commonly from the facet joints, paraspinal muscles, supporting ligaments or discogenic sources. In the lumbar spine due to the hormone relaxin in pregnancy, joint laxity is most notable in the anterior and posterior longitudinal ligaments both of which are pain sensitive structures. As these static supports in the lumbar spine become more lax, they cannot effectively withstand shear forces and discogenic symptoms and or pain from the facet joints may increase (Colliton, 1996).

Sacroiliac joint pain may be due to possible vertical displacement of the pubis and rotatory stress on the sacroiliac joint. In the non- pregnant state, the sacroiliac joints are extremely stable with tight anterior and posterior ligaments support and a sigmoid articular surface that limit movement. However, during pregnancy movement in the sacroiliac joints can increase dramatically hence causing discomfort when the pain sensitive ligamentous structures are stretched (Petersen et al, 1995). Nocturnal pain has been said to be probably due to circulatory changes during pregnancy. The enlarging foetus compresses the inferior vena cava when the woman is supine. This may divert

blood flow to the ascending lumbar veins, the vertebral venous plexus, the paraspinal veins and the azygous system (McCarthy et al, 1985). The intravascular volumes increases when the pregnant woman is supine and this may contribute to engorgement of the collateral neurovascular structures producing back pain at night (Fast et al, 1989).

(vi) Osteoporosis

May be a cause of low back pain when the calcium present in bones slowly decreases to the point where the bones became fragile and prone to fracture. Usually, no pain occurs about the time of menopause in women and very tiny fractures in the vertebrae caused by osteoporosis may be an undetected cause of back pain in many elderly women (Delitto et al, 2012).

(vii) Infection

Infections are a common cause of back pain. Osteomyelitis of the spine is however, a rare cause of back pain. Other infections that cause back pain include Lyme disease, septic arthritis, bacterial endocarditis, Potts disease, Reiter's syndrome, myobacterial and fungal arthritis. Chronic uterine or infections can cause low back pain in women (Nachemson, 1992).

(viii) Atherosclerosis

This is commonly called hardening of arteries and reduces blood supply in the arteries. Although mainly known as a cause of heart diseases, artherosclerosis can also reduce supply of blood to the back and cause chronic low back pain (Chou and Huffman, 2007).

(ix) Ankylosing Spondylitis

This disease, which has predilection for young men, is characterized by chronic inflammation of the spine that may gradually result in a fusion of the spine (Nwuga and Egwu, 1999). Symptoms include a slow development of back discomfort, with pain lasting for more than three months. The back is usually stiff in the morning while pain improves with exercise. In severe cases, the patients must continually stoop over. However, it can be mild and it rarely affects a person's ability to work. The disease is more common in men but about 30% of the cases are in women. Also about 20% of people with inflammatory bowel disease and about 20% of people with psoriasis develop a form of ankylosing spondylitis (Nwuga and Egwu, 1999).

(x) Other Medical Conditions

Back pain sometimes is also caused by other problems in other organs usually near the spine, which is then called referred pain. These conditions can include ulcers, kidney disease (including kidney stones), ovarian cysts and pancreatitis. Inflammatory bowel disease and rheumatoid arthritis can produce inflammation in the spine (sacroiliitis). Back pain can also be due to abscesses, blood clots and cancer. In older people, low back pain may be a sign of Paget's disease or Parkinson's (Delitto et al, 2012).

2.1.4 Risk Factors for Low Back Pain

Physical and psychosocial risk factors are known to predict occurrence of acute and chronic low back pain. Frymoyer, (1992) identified risk factors for low back pain as age, hard physical activity, prolonged driving or sitting, abnormalities of spinal canal anatomy and psychological factors. Poor lifting habits, habitual slouched posture, past injury to the spine and poor sitting posture have also been found to be linked to the low back pain syndrome (Cicinelli, 1996). Omokhodion (2002), and Omokhodion and Sanya (2003) observed that occupation and male sex were risk factors for low back pain. Psychosocial risk factors are also known to predict the occurrence of low back pain. MacGregor and Manek (2005) classified risk factors that influence low back pain. They include:

i. Individual risk factors such as age, sex, smoking, general health, birth weight, obesity and educational level.
ii. Psychosocial factors such as stress, pain behaviour, psychological distress, fear avoidance behaviour, depressive mood and somatization.
iii. Occupational factors such as manual labour, job satisfaction, monotonous tasks, control at work, social support, bending and twisting and whole body vibration.
iv. Biomechanical factors such as facet joint arthritis, annular disruption, radiographic disc space narrowing of lumbar vertebra, radiographic spondylosis and spinal instability.

2.2 Treatment of Low Back Pain

The goal of any treatment program particularly that of low back pain must be to produce remission of symptoms to the point that patients may return to the previous

level of function. Basically, the management of low back pain can be divided into the conservative and surgical treatment:

(i) Conservative management: This treatment is given without exploration of the inner structures and is divided into the pharmacological and non-pharmacological treatment.

(a) Pharmacological: This involves the use of drugs for the management of acute and chronic low back pain. Effective pain relief may involve a combination of prescription drugs and over-the-counter remedies. Patients should always check with a doctor before taking drugs for pain relief. Certain medicines, even those sold over the counter are unsafe during pregnancy, may conflict with other medications, may cause side effects including drowsiness, or may lead to liver damage (Swezey and Petrocelli, 1992). The drugs act to relieve the pain and they include analgesics, muscle relaxants and non-steroidal anti-inflammatory drugs such as aspirin, Ibuprofen, Diclofenac and Ketoprofen (Mayer, 1989).

(b) Non-Pharmacological treatment: This is the use of physical modalities in physiotherapy, chiropractic, osteopathic and psychosocial treatment protocols.

Physical modalities include Electrotherapy, Ultrasound, Cryotherapy, Therapeutic exercises and massage. Physical modalities include relaxation, biofeedback, behavioural modification et cetera. Physiotherapy is highly effective in the treatment of low back pain, and ideally all new patients diagnosed with low back pain should be seen by a physiotherapist (Dillingham, 1995). The mainstay of physiotherapy in the management of low back pain is therapeutic exercise, spinal manipulation coupled with electrotherapeutic modalities (Mckenzie, 2010). Despite this, indications, contraindications, dosage and precaution are as important in physiotherapy as in other management. Therapeutic exercises of various types and duration are prescribed for patients with low back pain. The exercises are given generally to improve blood flow, posture and mobility, decrease pain in the low back, stabilize the hypermobile vertebral segments, and to improve the fitness level of the patient. The exercises given can be classified on the basis of the movement of the spine into flexion and extension exercises. Mckenzie (2010) defined manipulation as all procedures where the hands are used to mobilize, adjust, stimulate or influence the spinal and paraspinal tissues

with the aim of relieving pain. The techniques include the vertical oscillatory pressure, transverse oscillatory pressure and lumbar rotation. The use of heat and cold therapy, ultrasound therapy, transcutaneous electrical nerve stimulation (TENS) and interferential therapy are some of the electrotherapeutic approach to the management of low back pain (Low and Reed, 1994). Other treatment approaches include, rest, lumbar traction, back supports, weight control and back schools (Chou and Huffman, 2007).

(ii) Surgical management: This is not usually carried out unless all conservative methods have failed. Although surgical treatment is performed on only two to three percents of patients with spinal disorders (Nelson, 1992), surgery has a role to play in the management of mechanical low back pain disorder (Nelson, 1992).

Conditions that require surgical interventions include:

1) Spinal stenosis: treated by decompressive laminectomy (Moore and Dalley, 1999).
2) Reccurent sciatica: which require limited laminectomy.
3) Lumbar instability: managed by spinal fusion (Nelson, 1992).
4) Herniated nucleus pulposus: that necessitates standard laminectomy or discectomy (Moore and Dalley, 1999). However, there exists indication in each condition that would make surgery the option. For example in herniated nucleus pulposus, indications for surgery include; significant straight-leg raising reduction, and failure of conservative treatments (Moore and Dalley, 1999).

2.2.1 Prevention of Low Back Pain

It has been established that the commonest cause of low back pain during activities of daily living (ADL) are attributed to poor working ergonomics and poor working posture (Delitto et al, 2012). As part of the non surgical management of patients with low back pain, back schools have been developed to educate patients to better able to manage their own back problems (Boreinstein, 1989). Preventing mechanical low back pain can therefore be achieved by taking appropriate measures to address these factors:

(a) Correct furniture design

(b) Correct working posture

Mckenzie (2010) reported that education of patients on how to avoid back problems like modification of maneuvers that are hazardous to the back will help in prevention of further occurrence of low back pain. Some of the common tips to be emphasized are good posture when standing, walking, sitting, driving, lifting and sleeping.

2.2.2. Models for Clinical Classification of Low Back Pain

The classification of low back pain into subgroups based on movement impairments has been advocated by Sahrmann (2002), and O'Sullivan (2005). Classification enables more appropriate, specific and effective interventions.

O'Sullivan (2005) suggests a classification system based on the specific mechanism underlying and driving the pain disorder. An overview of the classification model is summarized below.

Patho-anatomical model: The traditional medical approach where abnormal structural findings such as the 'disc prolapse' are assumed to be the cause of pain and treatment interventions provided on the basis of this assumption. In this model the fact that 'function affects structure' is rarely considered.

Peripheral pain generator model: Identification of pain structure based upon history, clinical examination and diagnostic blocks. Treatment such as blocks and denervation procedures address the pain symptoms without consideration for the underlying mechanism.

Neurophysiological model: Central sensitization of pain secondary to sustained peripheral nociceptotive interventions inhibit both central and peripheral processing of pain.

Psychosocial model: The impact of psychological and social factors upon the modulation of pain and in particular their capacity to increase the CNS mediated drive of pain. Poor coping strategies, anxiety, catastrophizing, hyper-vigilance tend to increase pain levels, disability and muscle guarding. Cognitive behavioural interventions can be effective. There is only a small subgroup where these factors are primary. The danger, however, is that due to lack of an alternate diagnosis, physiotherapists are tending to classify most patients with LBP as primary psychosocial driven.

Mechanical loading model: Both high and low levels of physical activity are reported risk factors for LBP; sustained end range loading: sudden and repeated loading, and related mechanical exposures are also influenced by ergonomic and environmental

factors and have the potential for ongoing peripheral nociception and need to be addressed as part of management.

Signs and symptoms model: Impairments in spinal movements and function, changes in segmental mobility, pain provocation tests; the effect of repeated movement on pain. The approaches of Maitland (1986) and Mckenzie (1981) fall into this model which is based upon biomechanical and patho-anatomical models and have led to the treatment of signs and symptoms associated with LBP. Limited evidence of efficacy may reflect research designs and neglect the biopsychosocial dimensions.

Motor control model: This model includes the approaches of Richardson and Jull (1995), Sahrmann and O'Sullivan (2000). Movement and control impairments are highly variable and their presence does not establish cause and effect. Altered motor behaviour is either protective or maladaptive which results in ongoing abnormal tissue loading and mechanically provoked pain. This group are amenable to tailored physiotherapy intervention directed at their specific physical and cognitive impairments with demonstrated positive outcomes.

Biopsychosocial model: The multidimensional approach to dealing with LBP (Engel 1977). The relative contributions of the different dimensions and their dominance will differ for each patient. Clinical reasoning allows determination as to which factors are dominant. Consideration of all factors allows for a diagnosis and mechanism based classification guiding management.

Functional movement model: This was proposed by Key (2010). It encompasses the biopsychosocial paradigm with the major focus upon improving the understanding and skill of the physical therapist in better dealing with the problem of movement dysfunction in spinal pain disorders. It sees that altered function in the posturomovement system is the primary largely responsible for the development and perpetuation of most pain syndromes. A simple clinical classification system based upon altered posturomovement function guides assessment functional diagnosis and management. Specific, appropriate treatment interventions directed to both the 'peripheral pain generator' and the altered posturomovement function improves pain and ability and helps counter the development of secondary psychosocial problems. Restoring neuromyoarticular functions helps restore the person.

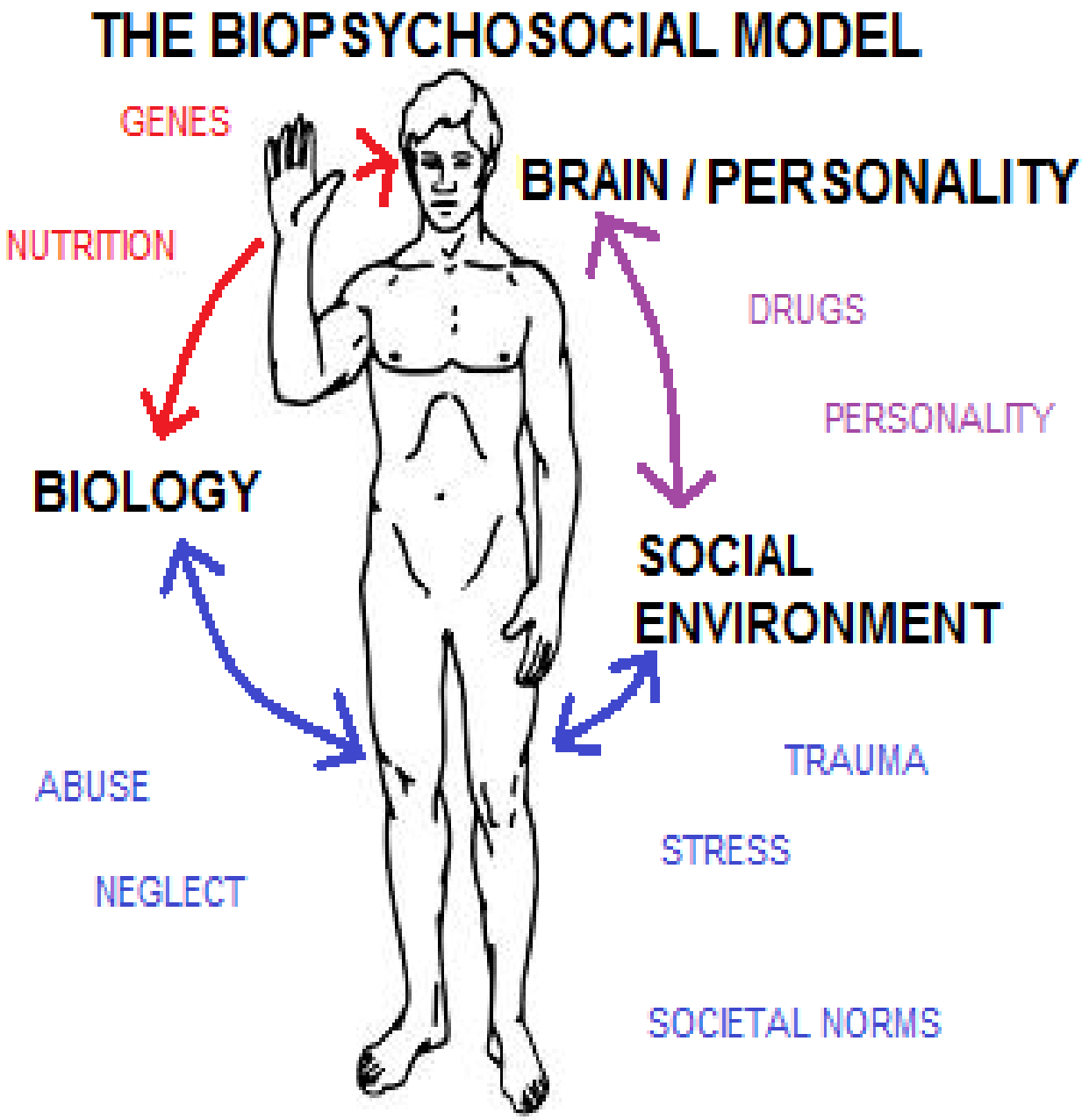


Figure 2.1 Biopsychosocial Model by Engell, 1977

2.3 The Nature of Pain

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage (Merskey and Bogduk, 1994). Pain is a ubiquitous part of life. Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery. Pain symptoms might be transient or they might persist over time. From an evolutionary perspective, pain signals have been discussed as an internal alarm mechanism that increases the probability of survival (Wall, 1999). Pain experience alerts the individual to the possibility that the integrity of the body has been compromised. Pain increases attention to the pain site and plays a role in the mobilization of behavior designed to act on the source of pain.

In 1965, Ronald Melzack and Patrick Wall proposed the Gate Control Theory (GCT) of pain. They argued that pain experience was jointly determined by physiological, motivational, cognitive and emotional factors (Melzack, 1999). The GCT helped explained clinical pain phenomena such as injuries without pain, pain that existed in the absence of discernible lesion, and psychological influences on pain (Feuerstein et al, 2006). The GCT also considered a place for behavior, but the 'action system' of the GCT operated at the spinal level; the role of behavior was relegated to the domain of reflexes. Research prompted by the GCT addressing the 'action system' of pain focused primarily on animals, and the actions studied have been reflexive withdrawal or escape responses (Wall, 1999). Melzack (1999) later proposed a 'neuromatrix' model of pain, which greatly expanded the dynamic role of networks within the brain to explain the experience of pain. In this model the brain has a neural network that integrates information from multiple sources and levels to produce the sensation of pain. This model was silent on the role of behavior in the pain system. A model of pain that does not incorporate a central role for behavior is necessarily incomplete hence the development of the biopsychomotor model (Sullivan, 2008) which considers two main intra-individual behavioral systems (the communicative and protective behavior system) in addition to the sensory component of the pain system.

2.3.1 From the Biomedical model of pain to the Biopsychosocial model of pain

The Bio-Psycho-Social Model (BPSM) of pain provides a conceptual rationale for including cognitive interventions in pain management strategies. The BPSM was first

proposed by Engel (1977) and it acknowledges biological processes, but also highlights the importance of experiential factors. Prior to the currently accepted BPSM, a biomedical model dominated all illness conceptualization for almost 300 years and still dominates in the popular imagination. The biomedical approach to pain sees a simple causal link between the amount of damage to the body and the amount of pain hence the more damage, the more pain but the experience of pain does not always correspond with amount of tissue damage. In a study exploring the important predictors of disability in workers with low back injuries, researchers found that actual physical pathology accounted for only 10% of the disability one year after the evaluation. However, 59% of the disability was explained by psychosocial variables (Burton et al, 1995). Unfortunately, despite evidence to the contrary, in many ways medicine still operates as if the physical source of the pain is the most important predictor of the experience of pain.

Another common misconception is that acute injury always produces pain. If you break your leg, everyone expects you to be in pain. The fracture can be seen on the X-ray; it is quantifiable; it is therefore considered "real", and pain is seen as justified. Nevertheless, the relationship between acute injury and the experience of pain is not as automatic as one might think. For example, during World War II, many U.S. soldiers as well as citizens were severely injured in a battle in Anzio, Italy. Frank Beecher, who was one of the medics there and later went on to become a pain researcher, observed that the meaning of the pain had a great deal to do with a person's experience of pain. Injury to the soldiers meant that they were going home, and many, even those with traumatic amputation of a limb, did not need pain medication. The citizens, on the other hand with similar injuries experienced fierce pain and required a great deal of analgesic (Beecher, 1959).

The biopsychosocial model focuses on both disease and illness, with illness being viewed as the complex interaction of biological, psychological, and social factors (Gatchel, 2005). As succinctly summarized by several authors (e.g., Gatchel, 2004a, 2004b; Turk & Monarch, 2002), disease is defined as an objective biological event involving the disruption of specific body structures or organ systems caused by one of anatomical, pathological, or physiological changes. In contrast, illness refers to a subjective experience or self-attribution that a disease is present. Thus, illness refers

to how a sick person and members of his or her family live with, and respond to, symptoms of disability. The distinction between disease and illness is analogous to the distinction that can be made between *nociception* and *pain.* Nociception involves the stimulation of nerves that convey information about potential tissue damage to the brain. In contrast, pain is the subjective perception that results from the transduction, transmission, and modulation of sensory information. This input may be filtered through an individual's genetic composition, prior learning history, current psychological status, and sociocultural influences. Loeser (1982) originally formulated a general model that delineated four dimensions associated with the concept of pain: the dimensions of nociception and pain reviewed above, *suffering* (the emotional responses that are triggered by nociception or some other aversive event associated with it, such as fear or depression), and *pain behaviour* (those things that people say or do when they are suffering or in pain, such as avoiding activities or exercise for fear of reinjury). Pain behaviours are overt communications of pain, distress, and suffering.

Waddell (1987) has emphasized that pain cannot be comprehensively evaluated without an understanding of the individual who is exposed to the nociception. Waddell also made a comparison between Loeser's (1982) model of pain and the biopsychosocial model put forth by Engel (1977). In particular, Engel proposed the important dimensions of the physical problem, distress, illness behavior, and the sick role, which corresponded to Loeser's dimensions of nociception, pain, suffering, and pain behaviour, respectively. In order to fully understand a person's perception and response to pain and illness, the interrelationships among biological changes, psychological status, and the sociocultural context all need to be considered. Any model that focuses on only one of these dimensions will be incomplete and inadequate. The BPSM encouraged broader thinking within medicine and it is now well accepted that chronic musculoskeletal pain is a multifaceted problem. It appreciates the functional interrelationships between the psyche and the soma and the consequent potential social effects that can occur in chronic pain states. The key clinical elements of this model are physical dysfunction which leads to pain. How the patient reacts to the pain will affect and be affected by the other elements like beliefs and coping, distress, illness behaviour and social interactions (Key, 2010). The adoption of the BPSM model over the biomedical disease model has given credence

to the increasing usage of cognitive behavioural approach as part of therapeutic management. Figure 2.2 shows the conceptual framework of the biopsychosocial perspective.

2.3.2 The Relationship between Pain and Disability

There is intuitive appeal to the notion that pain is the underlying cause of disability. The continued emphasis on the use of pain medication in the treatment of musculoskeletal problems reflects the commonly held belief that pain symptoms are the primary causes of disability. Pain does play a significant role in presenting symptoms of disability following soft tissue injuries to the back or neck (Cote et al, 2001). However, as the time following injury becomes prolonged, pain symptoms are no longer the most important determinants of disability. As the duration of time post-injury becomes extended, environmental, social and psychological factors become the primary determinants of disability (Waddell and Waddell, 2000). Several investigations have shown that in patients with persistent pain conditions exceeding 3 months duration, pain intensity rarely accounts for more than 10% of the variance in pain related disability (Waddell et al, 2003; Sullivan et al, 1998). Clinicians sometimes reflect that they have difficulty believing that pain accounts for only 10% of the variance in pain related disability. For every patient who indicates he or she has reduction in function due to pain, another patient with similar pain level is able to function. In other words, pain severity may not be the primary cause of disability and pain reduction may not be a solution to disability. Pain related disability has been seen as a form of behavior, and hence cannot be totally explained by pain severity (Sullivan et al, 2002). It appears that in spite of evidence indicating that symptom-focused interventions are not effective means of improving function, we continue to spend 80% of treatment–related resources on management of pain symptoms (Main et al, 2007).

2.4 Psychosocial Influences of Pain on Disability

Considerable research has addressed the role of psychosocial variables as risk factors for prolonged or pronounced disability (Leeuw et al, 2007; Pincus et al, 2002; Sullivan, 2003). Although the bulk of research in this area has been conducted on samples of individuals with pain related disability, research is beginning to accumulate suggesting that the same psychosocial factors might contribute to disability, regardless of the nature of the debilitating health condition (Tinetti et al,

1990; Tomassen et al, 2000). Although research points to a number of psychosocial variables that contribute to disability, three specific variables (also called yellow flags) have emerged as consistent and robust predictors of disability across a wide range of debilitating health and mental health conditions. These include catastrophic thinking, fear and perceived disability. Theoretical models of the psychology of disability suggest that individuals who engage in catastrophic or alarmist thinking about their health symptoms, who are fearful of engaging in activity that might exacerbate their symptoms and who believe themselves to be severely disabled are individuals at high risk for prolonged and pronounced disability (Sullivan et al, 2001; Turk, 2002, Vlaeyen and Linton, 2000). Research is also beginning to accumulate suggesting that the most effective rehabilitation programs will be those that effectively target these psychosocial risk factors (Spinhoven et al, 2004; Sullivan, 2006)

2.4.1 Pain Catastrophising

Catastrophising has been described as a significant cognitive component of the pain experience involving 'an exaggerated negative orientation to aversive stimuli' (Sullivan et al., 1995). Catastrophising is comprised of three elements that include ruminating about pain, appraising pain in a manner that magnifies its threat value, and devaluing resources available to cope with it (Chaves and Browne, 1987; Rosenstiel and Keefe, 1983; Sullivan et al., 1995, 2001). The term catastrophising was used by Albert Ellis, the founder of rational–emotional therapy, almost four decades ago. Ellis gave the following example of catastrophizing: "How terrible the situation is; I positively cannot stand it!" (Ellis, 1962). Beck et al.(1985) discussed catastrophising in terms of dwelling on the worst possible outcome of any situation in which there is a possibility for an unpleasant outcome. Examples of catastrophizing given by Beck et al. include the following: (1) during an airplane flight, a woman dwells on the possibility of the plane's crashing and her being killed; and (2) a college student taking an examination is preoccupied with the possibility of failing and consequently flunking out of college. Such thoughts are tied to the perception of oneself as vulnerable and as being subject to danger over which one has insufficient control.

An example of catastrophic pain thinking is seen in the writings of the novelist 'Maupassant' who described migraine as an atrocious torment, one of the worst in the world, weakening the nerves, driving one mad, scattering one's thoughts to the winds

and impairing the memory. So terrible are these headaches that I can do nothing but lie on the couch and try to dull the pain by sniffing ether." (Maupassant as quoted by Sullivan et al. 2001). Maupassant's words describe the torment of his pain, his emotional distress, and the disability that pain brings to his life. He feels overwhelmed by his pain, and he is helpless to deal with it. He surrenders to the pain and seeks chemical means of dulling it. Maupassant's words emphasize the psychological components of pain perception; the sensory, cognitive, affective, and behavioral dimensions of his experience. Specialists of the psychology of pain would argue that Maupassant's "catastrophic" orientation to his pain likely played a role in heightening the intensity of the pain he experienced (Beck et al, 1985). Catastrophic thinking has been shown to correlate positively with many aspects of the pain experience, including pain intensity, emotional distress, pain-related disability, health services use, pain behavior, and reliance on medication (Linton et al., 1998; Goubert et al., 2002, 2004; Sullivan and Neish, 1999; Sullivan et al., 1998, 2001).

Research on the nature of catastrophising (Chaves and Browne, 1987; Rosenstiel and Keefe, 1983) have shown consensus in construing catastrophising in terms of negative pain-related cognitions, they differ in their emphasis on the content of these cognitions. To address this issue, Sullivan et al.1995 developed the Pain Catastrophising Scale (PCS) using examples of catastrophic thinking drawn from each of these earlier studies (Chaves and Browne, 1987; Rosenstiel and Keefe, 1983). Factor analysis yielded a correlated three-factor solution, suggesting that catastrophizing could be viewed as a unitary construct comprising three different dimensions (i.e., magnification, rumination helplessness). The PCS is composed of three scales: Rumination (four items; e.g. 'When I am in pain, I keep thinking about how badly I want the pain to stop'), Magnification (three items; e.g. 'When I am in pain, I become afraid that the pain will get worse'), and Helplessness (six items; e.g. 'When I am in pain, I feel I can't go on').

In recent years, increasing attention has been drawn to examining the contributions of 'catastrophising' to the prediction of pain and disability in individuals suffering from chronic pain. A number of studies (Linton et al., 1998; Goubert et al., 2002, 2004; Sullivan and Neish, 1999; Sullivan et al., 1998, 2001). have also shown that measures of catastrophising are significantly correlated with objective and subjective measures

of disability. Rosenstiel and Keefe (1983) reported that the Coping Strategies Questionnaire (CSQ); which includes a catastrophising subscale accounted for 37% of the variance in patients' pain ratings, and 19% of the variance on a measure of functional capacity. Similarly, Turner and Clancy (1986) reported that the CSQ accounted for 27% of the variance in disability and psychosocial impairment, and 16% of the variance in downtime. In patients with rheumatoid arthritis and fibromyalgia, it has been shown that factor scores of the CSQ (which included the catastrophizing scale) were also predictive of functional impairment classification and pain behaviours (Keefe et al., 1987; Parker et al., 1989; Beckman et al., 1991; Nicassio et al., 1995). The available literature, therefore, points to the important role of catastrophising as a predictor of pain and disability in chronic pain patients.

2.4.2 Fear as a Predictor of Pain and Disability

Fear is an integral component of pain. Fear is the driving force of escape and avoidance: two response systems that are critical to survival when the body has been injured. In 1965, Melzack and wall first addressed the multidimensional nature of pain (Melzack, 1999). They stated that in addition to a sensory dimension, the pain system also comprised affective (emotional) and motivational dimensions. If pain signals are to serve a survival function, alerting the individual to the possibility that the integrity of the body has been compromised, the pain system must also include mechanisms by which the individual can act to escape or avoid further injury. Human behaviour is frequently driven by some form of emotion. Emotion provides the drive or motivation for action. In the case of escape or avoidance, fear is likely to be the source of the drive (Sullivan, 2010). Fear of movement or fear of re-injury is significant determinant of prolonged work disability (Vlaeyen et al, 1995). Two frequently used scales developed to assess pain related fears include the Fear-Avoidance Beliefs Questionnaire (Waddell et al, 1993) and the Tampa Scale for Kinesiophobia (Kori, 1990). A number of studies revealed that high scores on these measures were associated with longer periods of work disability. What was striking was that fear was often a better predictor of prolonged disability than pain itself (Crombez et al, 1999, Waddell et al, 2003).

Vlaeyen et al, (1995) proposed a cognitive-behavioural Fear-Avoidance Model to account for the processes by which psychological factors might adversely impact on pain and disability. This model states that individuals will differ in the degree to

which they interpret their pain symptoms in a 'catastrophic' or 'alarmist' manner. The model predicts that catastrophic thinking following the onset of pain will contribute to heightened fears of movement and increased hypervigilance to pain symptoms. In turn, fear is expected to lead to avoidance to escape of activity that might be associated with pain (Vlaeyen and Linton, 2000). Prolonged inactivity is expected to contribute to depression and disability (Sullivan et al, 2006). Hypervigilance is expected to contribute to further increases in pain severity. The model is recursive such that increased pain symptoms, distress and disability become the input for further catastrophic or alarmist thinking. If fear is a significant determinant of disability, it follows that interventions that have proven effective in the reduction of fear might be usefully applied to disability. If the fear component of disability could be reduced, then disability might be reduced as well.

2.4.3. Self-efficacy and Disability

Self-efficacy as defined by Bandura (1997) is, "a belief in one's personal capabilities," and plays an important role in human function in four major ways. This includes (1) Cognitive functioning; a person with high efficacy will have high aspirations, set difficult challenges for themselves and be committed to meeting those challenges. (2)Motivational: a person with high self-efficacy will have stronger motivation because they will be able to attain their goals and adjust them based on setbacks they may encounter. (3) Mood or Affect: High self-efficacy will lead to people lowering stress and anxiety by deflating threatening situations they may come across, along with diverting their attention, relaxing and relying on a good social network in such situations. (4)Depression: people with low efficacy self defeat their own hopes, lowering their mood, which will further weaken their efficacy, further lowering their mood. Self-efficacy is concerned with judgements of what one can do with whatever skills one possesses (Bandura, 1986). Efficacy expectations with regards to pain control, management, coping and daily functioning may help to determine the extent of disability (Arnstein et al, 1999). Self-efficacy helps to determine how well a patient adapts to pain (Anderson et al, 1995) and may explain the variability between a patients' perceived level of activity and his actual performance (Gage and Polatajko, 1994; Strong, 1995). If pain cannot totally explain disability, self-efficacy might (Anderson et al, 1995).

2.5 Cognitive Behavioural Therapy

Psychosocial treatment for pain-related conditions has typically taken the form of cognitive–behavioural pain-management programmes (Morley et al, 1999; Lefort et al, 1998). The term ‘‘cognitive–behavioural’’ refers not to a specific intervention but to a class of intervention strategies that may include self-instruction (e.g., motivational self-talk), relaxation or biofeedback, exposure, developing coping strategies (e.g., distraction, imagery), increasing assertiveness, minimizing negative or self-defeating thoughts, changing maladaptive beliefs about pain, and setting goals (Linton, 2002; Turk et al, 1983). As a function of the profile of presenting problems, a client participating in a cognitive–behavioural intervention may be exposed to varying selections or combinations of these strategies. This concept is recognized as the most promising treatment approach for chronic LBP, particularly in terms of encouraging activity and exercise (Airaksinen et al, 2006). Cognitive Behavioural Therapy (CBT) is a psychotherapeutic treatment concept comprising elements of behavioural therapy mainly based on the principle of operant conditioning and elements from cognitive therapy. Describing or framing CBT for the treatment of LBP is challenging because it tends to be an umbrella term for a broad variety of interventions. But in general, these approaches all have a common aim which is to alter maladaptive thoughts, feelings and behaviour as well as dysfunctional sensory phenomena, and thereby the experience of pain (Henschke et al, 2010). The CBT concept for chronic LBP has been distinguished into three different treatment approaches: operant, cognitive, and respondent treatment (Vlaeyen et al, 1995).

Operant treatment is based on the operant conditioning theory described by Ferster &Skinner, 1957. This treatment approach aims to reinforce healthy behaviours and reduce pain behaviours by using an exercise quota for increasing general activity levels which are gradually built up towards a realistic predefined goal. Spouses and/or family members are integrated into the therapy whenever possible and instructed to promote well behaviours of the patient (Saunders, 2002). Cognitive treatment, based on the cognitive model from Beck et al (1979), is designed to help patients modify maladaptive conceptualizations and dysfunctional beliefs about themselves and their disability (Winterowd et al, 2003). Patients learn to identify negative emotions related to pain, stressful events and associated maladaptive thoughts (Turner & Jensen, 1993).

In addition to this, they are taught to generate adaptive thoughts in order to 'counter' automatic negative cognitions (Turner & Jensen, 1993). Cognitive therapies often integrate imagery exercises, aimed at changing the pain experience by shifting attention to something other than bodily sensations (Syrjala & Abrams, 2002). Respondent treatment attempts to modify the physiological response system to pain. The theory of this approach is based on the assumption of a pain-tension cycle, where pain is viewed as cause and result of muscular tension (Henschke et al, 2010). Electromyographic (EMG) biofeedback and relaxation techniques are used to encourage the patient to identify tension-eliciting stimuli and to differentiate between muscle tension and relaxation (Vlaeyen et al, 1995).

Traditionally, cognitive–behavioural pain-management programmes have been delivered by psychologists or other rehabilitation professionals with a background in mental health (Linton, 2002). Given the strategic position of the physiotherapist as a first-line health care professional for problems associated with musculoskeletal injury, it has been suggested that physiotherapists might be ideally suited to intervene on psychosocial barriers to rehabilitation progress (Linton, 2002). Recent study has examined the effectiveness of psychosocial interventions delivered by physiotherapists. In each of the studies described below, the effectiveness of a pain-related psychosocial intervention, administered by a physiotherapist, was compared to traditional physiotherapy. Of interest in all these studies was whether the impact of physiotherapy treatment could be increased by an intervention specifically targeting psychosocial barriers to rehabilitation progress.

The effects of a cognitive–behavioural pain-management programme delivered by physiotherapists was reported by Hay et al, (2005), Physiotherapists attended a 2-day training workshop (with follow-up supervision) to develop the skill set needed to deliver a group cognitive–behavioural pain-management programme. The effects of the pain-management programme were compared to those of physiotherapy alone. The results of the study revealed that the two treatment groups did not differ significantly at post-treatment on measures of pain severity, emotional distress, or self-reported disability; however, patients in the pain-management group showed a decrease in use of health care services compared to patients in the physiotherapy group (Hay et al, 2005).

George et al, (2008) reported the results of a study comparing treatment outcomes of patients with back pain who received physiotherapy, graded activity, or graded exposure. The graded exposure intervention was intended to target pain-related fears, while the graded activity was intended to increase involvement in activity. The study report provides no information on the duration of training for physiotherapists who provided the graded exposure or graded activity interventions. Group comparisons conducted at 6-month follow-up revealed no significant differences among groups on measures of pain intensity, physical impairment, or disability. Brunner et al, 2012 reported a systematic literature review of eight studies that employed CBT-based intervention strategies. Half of the studies suffered from high risk of bias, and study characteristics varied in all domains of methodology, particularly in terms of treatment design and outcome measures. Graded activity, an operant approach based on principles of operant conditioning was identified as a CBT-based strategy with traceable theoretical justification that can be applied by physiotherapists. The systematic review concluded that operant conditioning can be integrated in ambulant physiotherapy practice and is a promising CBT-based strategy for the prevention and management of chronic LBP.

2.5.1 Psychologically-informed practice in Physiotherapy

This approach is based on the identification of normal psychological processes that affect the perception of pain and the response to it as an expected and normal part of the musculoskeletal pain experience that are potentially modifiable (Main and George, 2011). Psychologically-informed Practice in Physiotherapy (PIP) offers a "middle way" between the narrowly focused standard physiotherapy practices based on biomedical principles and the cognitive-behavioural approaches developed originally for the treatment of mental illness (Foster and Delitto, 2011). This new approach uses the "flags" framework, with psychologically informed practice requiring routine and specific consideration of "yellow flags" and "blue flags" (depending on clinical setting) for determining risk of poor outcome and identifying the potential for treatment modification but with cognizance of the overall environment or context in which the clinician must operate (Nicolas et al, 2011). This context includes professional culture, health care policy, and insurance reimbursement (potential "black flags"). The primary goal of this approach is to prevent the

development of unnecessary pain-associated activity limitations (Main and George, 2011).

2.6 Progressive Goal Attainment Programme (PGAP)

PGAP is a CBT-based intervention for anyone who is experiencing a high level of disability associated with painful health condition. By addressing psychosocial barriers to rehabilitation progress, PGAP can assist individuals in increasing their participation in life- role activities that once brought to their lives a sense of purpose and a sense of meaning. PGAP was developed in response to research showing that symptom reduction was not sufficient to achieve resumption of occupational activities. Across a variety of domains of illness and disability, research show that symptoms of different health conditions rarely account for more than 10-30% of the variance in levels of disability. It follows that symptom management approaches will be limited in their potential impact on disability (Sullivan, 2010).

PGAP proceeds from the view that 'symptoms of illnesses and 'expressions of disability' are distinct and partially independent phenomena (Sullivan, 2010). Within the conceptual framework of PGAP, 'symptoms' are relevant to what patients 'feel' while 'disability' is relevant to what patients 'do'. The primary goal of PGAP is to change what patients 'do'.

A brief overview of the structure and content of PGAP (Sullivan, 2010)

i. Education and Reassurance: The PGAP information video is used to provide the patient with education about the nature of residual symptoms associated with painful conditions.

ii. Maintaining an activity log: Since one of the goals of PGAP is to maximize activity involvement, the client is asked to complete the Activity log in the PGAP client workbook throughout the course of treatment.

iii. Activity Scheduling: Working with the PGAP provider, the client develops an activity schedule that is designed to keep him or her as active as possible. Activities may include household activities, running errands, social and recreational activities. Activities are scheduled in relation to the client's chosen participation goals and are intended to create an activity structure that will ultimately facilitate resumption of occupational activities.

iv. The walking program: A main component of PGAP is the development of a walking program. The walking program starts with one 15 minutes walk each day. As PGAP moves forward, the PGAP provider works with the client to steadily increase the distance walked each day.

v. Increasing activity involvement: Through the course of the treatment program, the PGAP provider assists the client in ways to increase activity involvement. The client is taught principles of graded activity participation to maintain momentum of recovery while minimizing the risk of symptom flare-ups. Activity planning offers opportunities for success and achievement experiences; elements that are critical for maintaining a positive and engaged orientation toward rehabilitation.

vi. Overcoming psychological obstacles to activity involvement: In the second phase of the program, the client develops skills to overcome fears of re-injury, learns to monitor and modify catastrophic thinking that may accompany distressing symptoms and leans to challenge his or her perceived limitations.

In one study of patients with chronic cervical pain, individuals participating in a functional restoration physiotherapy programme were compared to a sample of individuals who received PGAP in addition to the same physiotherapy intervention (Sullivan et al, 2006). The results showed that at treatment termination, there were no significant differences in pain severity or pain-related fear; however, the individuals who received PGAP showed greater reductions in catastrophizing and were more likely to return to work (Sullivan et al, 2006). Although research suggests that PGAP may improve rehabilitation outcomes for individuals with chronic pain, PGAP is yet to be evaluated in individuals in the sub-acute period of recovery. Data suggesting that PGAP improves clinical outcomes for individuals in the sub-acute period of recovery would point to PGAP as a potential intervention to prevent the transition to chronicity. A key question in the development of psychosocial interventions to complement physiotherapy is not only whether outcomes can be improved but, specifically, what domains of functioning are most likely to be improved with the addition of a psychosocial intervention. Sullivan and Adams (2010) used PGAP in augmenting the Physiotherapy intervention of 24 patients with disabling back pain. They concluded that PGAP intervention provided by physiotherapists can lead to meaningful reductions in psychosocial risk factors for pain and disability and may contribute to more positive rehabilitation outcomes.

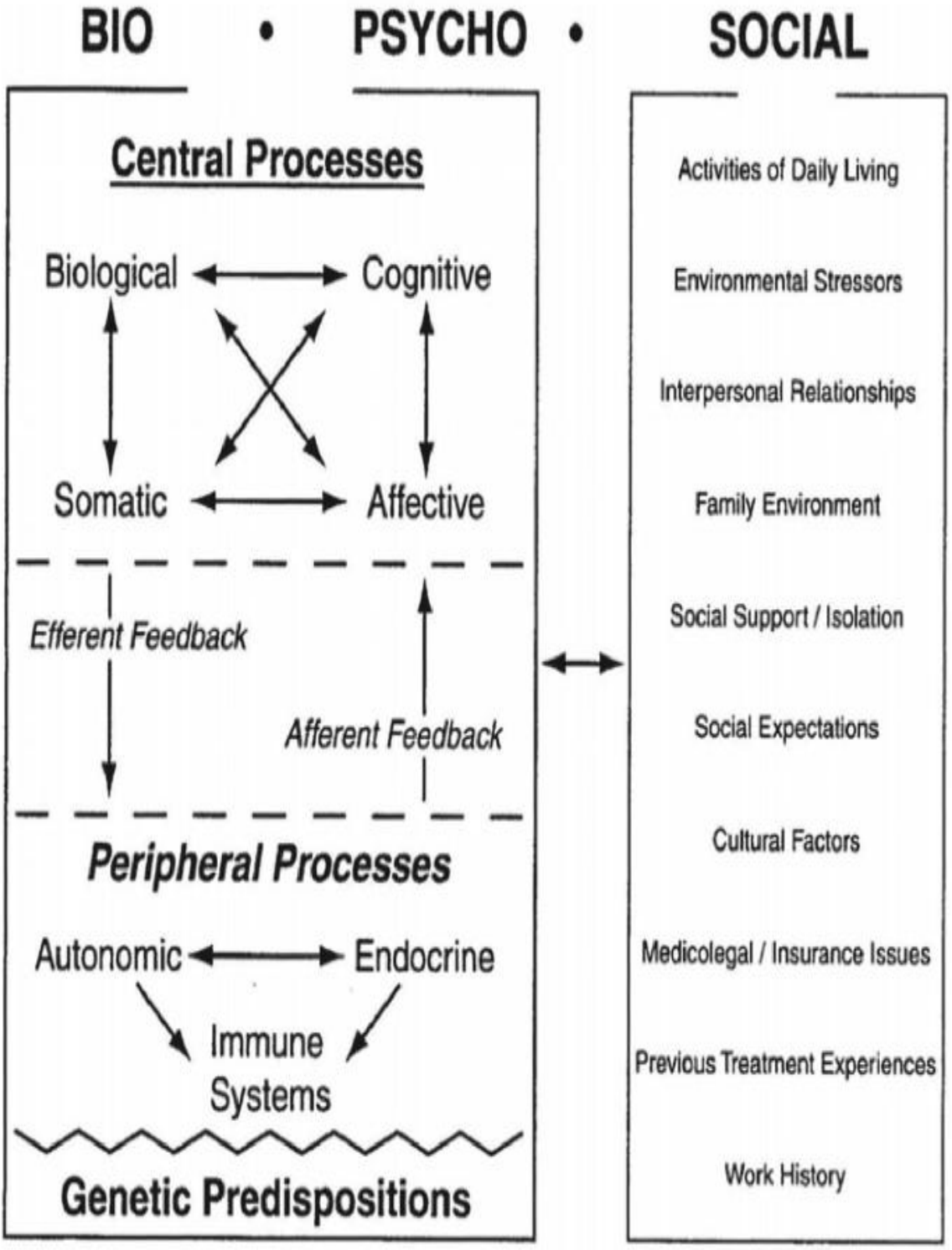


Figure 2:2 A conceptual model of the biopsychosocial interactive processes involved in health and illness. Gatchel, 2004.

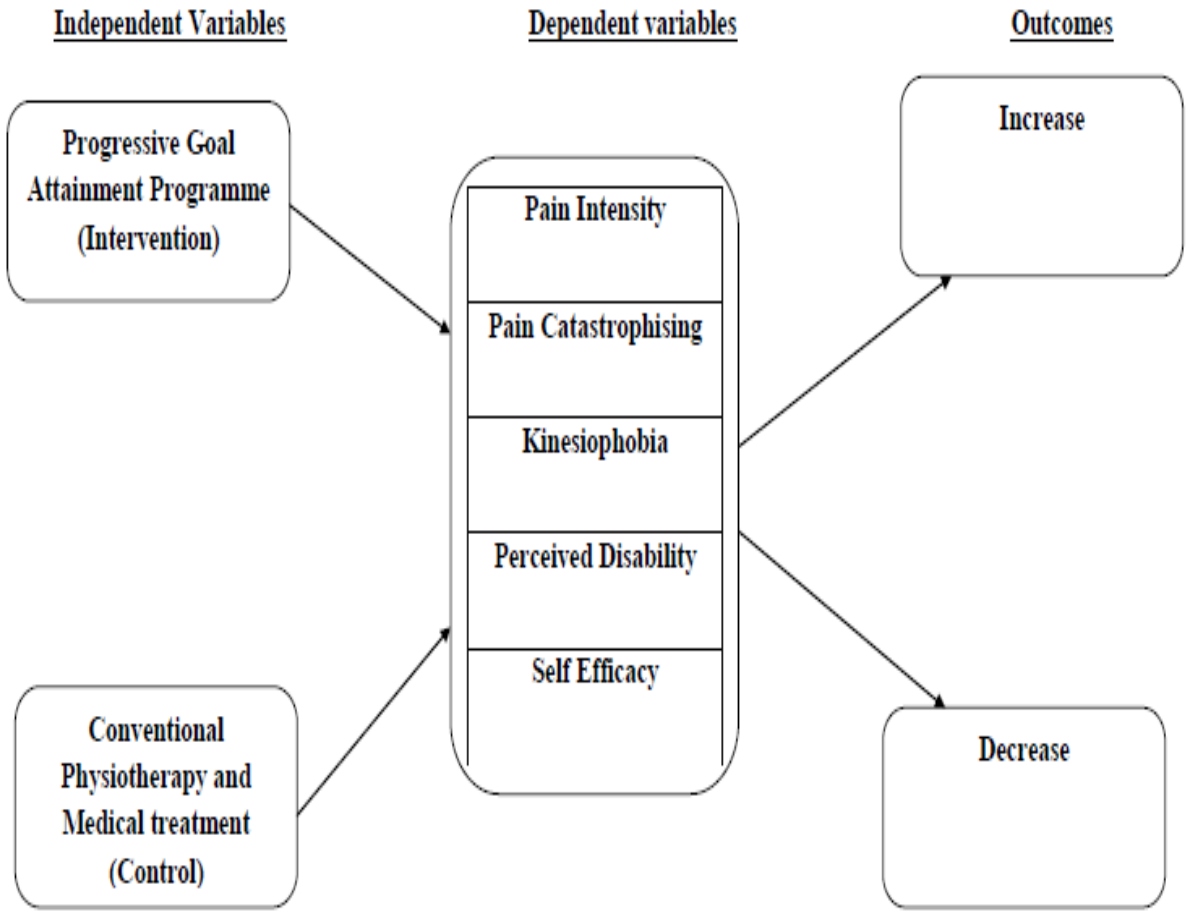


Figure 2.3 Conceptual Model for Study Titled Effects of a 10-week Progressive Goal Attainment Programme on Selected Outcomes in Patients Receiving Conventional Treatment Procedure for MLBP.

2.7 Review of Previous Studies on Psychological Interventions in Patients with Low Back Pain

Literature search of existing published studies on psychological interventions in patients with low back pain was done using five databases (Pubmed, Hinari, Medline, Google Scholar and Science Direct) and the following key words were used: psychological interventions, mechanical/non-specific low back pain and behavioural interventions. The search strings were psychological interventions RCTs and pain intensity, psychological interventions RCTs and pain related disability, psychological interventions RCTs and pain catastrophising, psychological interventions and kinesiophobia, psychosocial interventions and self-efficacy. Only studies published in English language from 1980 to 2014 were considered for inclusion in this review.

An initial search of Pubmed was undertaken using the identified search terms. This yielded 213 articles. A search of Google Scholar was done to further ensure that all relevant studies have been identified. This yielded 23,900 articles. The researcher thereafter did an analysis of the text words contained in the titles of the index terms used to describe the identified articles. A further evaluation of the abstracts or full texts of papers identified by the initial search for appropriateness to the study question and in consideration of the inclusion criteria was done. A total of 52 articles that met the inclusion criteria where identified. Data on author and year, outcome variables, types of interventions, target population, and findings were obtained. A summary of these studies are presented in Table 2.1.

All the studies were carried out in Europe, America and Asia but none in Africa. Eleven studies were carried out in the United States of America (Altmaier et al, 1992; Brox et al, 2003; Donaldson et al, 1994; Gatchel et al, 2003; Menzel and Robinson, 2006; Moore et al, 2000; Nicholas et al. 1991; Nicholas et al, 1992; Rogerson et al, 2010; Stuckey et al, 1986 and Whitfill et al. 2010). Six studies were carried out in Canada (Sullivan and Adams, 2010; Turner 1982; Turner and Clancy, 1988; Turner et al. 1990; Turner and Jensen, 1993 and Woods and Asmundson, 2008) Ten studies were conducted in the United Kingdom (Bush et al, 1985; Fairbank et al, 2005; Fersum et al, 2013; Johnson et al, 2007, McCauley et al, 1983, Poole et al, 2007; Rose et al, 1997; Hay et al, 2005; Lamb et al, 2010; Newton-John et al, 1995). Four studies

were carried out in Germany (Basler et al, 1997; Friedrich et al. 1998; Mangels et al, 2009; Schweikert et al, 2006). Two studies were carried out in Switzerland (Henchoz et al, 2010; Kool et al, 2005). Ten studies were carried out in the Netherland (Hlobil et al, 2005; Kole-snijders et al, 1996; Leeuw et al, 2008; Nouwen 1983; Smeets et al, 2006; Steenstra et al, 2006; van den Hout et al, 2003; van den Roer et al, 2008; von Korff et al, 1998; von Korff et al, 2005) Two studies were carried out in France (Jousset et al, 2004; Kaapa et al, 2006) Four studies were carried out in Sweden (Lindstrom et al, 1992; Linton et al, 1989; Linton et al, 2000; Linton et al, 2008) Two of the studies were carried out in Norway (Magnussen et al, 2005; Storheim et al, 2003). Only one of the studies was carried out in Australia (Strong 1998).

Only one study was retrospective while the remaining were randomized control trials that made use of different types of psychological intervention in addition to standard medical and physiotherapy care as interventions. The main outcomes measured in all these studies were pain intensity (measured using VAS), functional disability (measured by Oswestry Disability Index, Roland Morris Disability Questionnaire etc), time to return to work, fear avoidance beliefs, Cost effectiveness Ratio, mean number of sick leave days, quality of life and quality adjusted life years measurement and psychological indexes (catastrophising, kinesiophobia, self-efficacy, depression, anxiety). The sample size involved in all these studies ranged from 17 to 409.

The outcome of the review of these studies revealed that psychological interventions (in form of different types of Cognitive Behavioural Therapy) used alongside standard medical and physiotherapy intervention were significantly useful in reducing pain intensity and disability in patients with mechanical low back pain (Linton et al, 1989; Donaldson et al, 1994; Basler et al, 1997; Friedrich et al, 1998; Strong, 1998; Linton et al, 2000; Gatchel et al, 2003; Hay et al, 2005; Linton et al, 2008; von Korff et al, 2005; Woods and Asmundson, 2008; Henchoz et al, 2010; Fersum et al, 2013). Some authors (Altmaier et al, 1992; Brox et al 2003; Bush et al, 1985; Fairbank et al, 2005; Johnson et al, 2007; Kaapa et al, 2006; Leeuw et al, 2008; McCauley et al, 1983; Nouwen 1983; Poole et al, 2007) nevertheless concluded that psychological interventions were not significantly efficacious when used alongside standard medical and physiotherapy treatment. Operant therapy was more effective than waiting list for short-term pain relief (Kole-snijders et al, 1996; Leeuw et al. 2008). Little or no

difference exists between operant, cognitive, or combined behavioural therapy for short- to intermediate-term pain relief (Altmaier et al, 1992, Leeuw et al, 2008). Behavioural treatment was more effective than usual care for short-term pain relief but there were no differences in the intermediate- to long-term, or on functional status (Kool et al, 2005). There was little or no difference between behavioural treatment and group exercise for pain relief or depressive symptoms over the intermediate- to long-term (Lindstrom et al, 1992) and adding behavioural therapy to inpatient rehabilitation was no more effective than inpatient rehabilitation alone (Nicholas et al, 1991. Nicholas et al, 1992).

The divergent results and conclusions made from these studies could proceed from the lack of homogeneity of subjects involved as some studies involved only chronic low back pain patients while others involved all duration of low back pain patients. Some studies (Hay et al 2005; Nicholas et al, 1991. Nicholas et al, 1992) screened the subjects for the presence of yellow flags as eligibility criteria for participation. Also, the professional training of the researchers may be of great importance as more recent studies have used trained clinicians (Hay et al, 2005; Turner et al, 1990) other than clinical psychologist and psychiatrist to deliver the psychological intervention. This may influence the efficacy of the intervention among the subjects. It is important to note that none of these studies were done in Africa (Nigeria inclusive) and particularly the only study that used PGAP as a psychological intervention was carried out in Canada with a retrospective research design.

Table 2.1: Review of Previous Studies on Psychological Interventions in Patients with Mechanical Low Back Pain

S/N	Author/ Year	Design	Country	Sample Size	Variables	Interventions
1	Altmaier et al, 1992	RCT	U.S.A	47	Pain intensity and interference, return to work and disability.	Standard 3-week program with ope conditioning com Intervention grou rehabilitation prog
2	Basler et al, 1997	RCT	Germany	94	Pain intensity and control over pain in behavioural and functional domains	Cognitive behavio medical treatment group and medica medication, nerve physical therapy)
3	Brox et al, 2003	RCT	U.S.A	64	Extent of disability (ODI), Fear avoidance beliefs, Fingertip-floor distance, Lower limb pain	Cognitive interve of a lecture reinfo daily physical exe for intervention g instrumental lumb followed by post- the reference grou
4	Bush et al, 1985	RCT	U.K	72	Pain intensity, functional and psychological status.	Auditory EMG bio training in sitting intervention grou feedback of back reference group 1 control for refere
5	Donaldson et al, 1994	RCT	U.S.A	36	Scores on McGill questionnaire and pain intensity.	Progressive relaxa intervention I gro unit feedback trai intervention II gro education on ana depression and st management for treatment group
6	Fairbank et al, 2005	RCT	U.K	349	Extent of disability using Oswestry Disability Index.	Cognitive behavio identify and overc unhelpful beliefs group and spinal surgery at discreti surgeon for refere
7	Fersum et al, 2013	RCT	U.K	121	Pain intensity (NRS), Disability (ODI)	classification-base functional therapy

						group and manual exercise for the re
8	Friedrich et al, 1998	RCT	Germany	98	Pain intensity (VAS), disability scores, Modified Waddell scale for self reported compliance and motivational scales for motivation	Individual exercis and motivational intervention grou treatment was in programme.
9	Gatchel et al, 2003	RCT	U.S.A	124	Pain, disability and socioeconomic outcomes (return to work and healthcare utilization).	Intervention grou functional restora intervention, refe were ALBP patien of developing chr reference group I patients at low ris chronicity. The re did not receive ar
10	Hay et al, 2005	RCT	England	402	Change in the score on the Roland and Morris disability questionnaire at 12 months. Analysis was by intention to treat	Intervention grou management ther intervention grou therapy.
11	Henchoz et al, 2010	RCT	Switzerland	109	Functional diability (ODI), Work Status, lifting capacity, spinal ROM, trunk muscle endurance, aerobic capacity	Intervention grou multidisciplinary t Physiotherapy wh treatment group physiotherapy ou alone.
12	Hlobil et al, 2005	RCT	Netherland	134	Functional status, pain and time to return to work	Intervention group activity while refe treatment as usual
13	Johnson et al, 2007	RCT	U.K	234	Pain intensity (VAS), Functional status (Roland Morris Disability Questionnaire), Cost effectiveness Ratio	Cognitive behavio approach (eight 2- sessions over a 6- educational pack (cassette) for interv and reference trea educational pack

14	Jousset et al , 2004.	RCT	France	86	Pain intensity, quality of life, mean number of sick leave days, functional and psychological indexes.	The intervention g functional restora while the referenc active individual t
15	Kaapa et al, 2006	RCT	France	120	Back and sciatic pain intensity, disability, sick leaves, healthcare consumption, symptoms of depression, and beliefs of working ability after 2 years	The intervention g multidisciplinary r while the referen individual physiot treatment.
16	Kole-snijders et al, 1996	RCT	Netherland	148	Pain coping, control and behaviour	Operant treatmer intervention grou treatment + grou intervention grou reference treatm list control group
17	Kool et al, 2005	RCT	Switzerland	174	The number of days at work in 3 months after treatment, self-efficacy, Lifting capacity, pain intensity, mobility, strength, and global perceived effect	Intervention grou centered treatme reference treatm pain-centered ma
18	Lamb et al, 2010	RCT	England	701	Primary outcomes were the change from baseline in Roland Morris disability questionnaire and modified Von Korff scores at 12 months	Intervention grou management adv consultation in ad cognitive behavio sessions. The refe had only active m advisory consulta
19	Leeuw et al, 2008	RCT	Netherland	85	Pain intensity (MPQ), Pain catastrophising (PCS), kinesiophobia (TSK), Functional ability (QBPDS)	Exposure in vivo (therapy and educ I, Operant graded group II
20	Lindstrom et al, 1992	RCT	Sweden	103	Pain intensity, return to work and sick leaves.	Intervention grou activity and opera behavioral treatm Reference group as usual.

21	Linton et al, 1989	RCT	Sweden	66	Pain intensity (VAS), Psychological outcome measures (CSQ)	Behavioural thera in group treatmer physiotherapy int intervention grou list control with a treatment for refe
22	Linton et al, 2000	RCT	Sweden	243	Sick absenteeism, health care use, pain, function, fear-avoidance beliefs, and cognitions	Cognitive behavio treatment as usua intervention grou as usual + back pa information for th treatment group.
23	Linton et al, 2008	RCT	Sweden	46	Pain intensity (VAS), Pain related fear (TSK) and ADL assessment	Exposure in vivo f group and waiting treatment as usua group.
24	Magnussen et al, 2005	RCT	Norway	152	Pain intensity	The intervention cognitive interver (information and exercise) and trea for reference grou
25	Mangels et al, 2009.	RCT	Germany	363	Pain, disability, depression, self-efficacy, health status, life satisfaction, and coping strategies were assessed.	Intervention grou traditional orthop rehabilitation, Int II had multidisciplı (behavioral-medi rehabilitation alo Intervention grou multidisciplinary with subsequent
26	McCauley et al, 1983	RCT	U.K	17	Pain intensity (VAS) and depression	Progressive musc training and differ for intervention g hypnosis and hypı techniques for ref
27	Menzel and Robinson, 2006	RCT	U.S.A	32	Pain intensity, stress and depression scores, unscheduled work absence, disability.	Intervention grou behavioural therap as usual for refere

28	Moore et al, 2000	RCT	U.S.A	226	Back-related worry, fear-avoidance beliefs, pain ratings and interference with activities	Intervention group intervention and t usual supplement back pain care for group.
29	Newton-John et al, 1995	RCT	England	44	Pain intensity, functional status and behavioural outcomes(Coping, depression, anxiety, pain beliefs)	Intervention grou behavioural thera electromyographi and the reference waiting list contro
30	Nicholas et al, 1991	RCT	U.S.A	58	Pain intensity (6 point nominal scale), Self-rated functional status (SIP) and pain beliefs.	Intervention grou conditioning and group II had beha treatment, progr relaxation training physiotherapy,gro cognitive therapy physiotherapy, gr cognitive therapy and progressive n training. Referenc group I had physi information and h reference treatm physiotherapy tre 2hours/week for
31	Nicholas et al, 1992	RCT	U.S.A	20	Pain intensity (6 point nominal scale), Self-rated functional status (SIP) and pain beliefs.	Cognitive behavio and physiotherapy group and Physio reference treatme
32	Nouwen 1983	RCT	Netherlands	20	Pain duration and intensity	Auditory and visu biofeedback train intervention grou list control for ref treatment.
33	Poole et al, 2007	RCT	U.K	234	Pain intensity, Qol (Sf-36), functioning (ODI)	Progressive musc intervention grou reflexology for th group.

34	Rogerson et al, 2010	RCT	U.S.A	121	Pain symptoms,(MVAS), Follow up at 12 months, QALY	Cognitive behavio and Physical thera intervention grou reference group r standard care.
35	Rose et al, 1997	RCT	U.K	281	Pain intensity, functional status and psychological distress(self efficacy, depression, locus of control, somatic perception	All groups receive behavioral treatm intervention group into Part A&B. Pa interventions grou intervention group treatment and gro individual treatme intervention group that received 15-h and intervention g 30-hour program
36	Schweikert et al, 2006	RCT	Germany	409	Pain intensity, disability outcome measurs and quality adjusted life years gained.	Cognitive behavio management pro sessions + standa rehabilitation con physiotherapy for intervention grou inpatient rehabili of physiotherapy reference treatm
37	Smeets et al, 2006	RCT	Netherlands	227	Pain intensity, disability (RMDQ), QALY gained.	Cognitive behavio for intervention g combined therap physical training a behavioural thera intervention grou treatment group training and refer were waiting list c
38	Steenstra et al, 2006	RCT	Netherland	112	Number of days off work, total number of days on sick leave during follow up, functional status, and severity of pain.	Intervention grou activity while refe had treatment as
39	Storheim et al, 2003	RCT	Norway	93	Pain, disability, sick-listing and satisfaction with care, self-efficacy for pain and for function, fear-avoidance beliefs,	Intervention grou regime, intervent cognitive interver reference therapy as controls.

					emotional distress, generic health status and life satisfaction	
40	Strong 1998	RCT	Australia	30	Illness behaviour, depression and negative cognitions	Inpatient pain ma program + psycho individual treatm intervention grou pain managemen reference treatm
41	Stuckey et al, 1986	RCT	U.S.A	30	Pain intensity and ADL assessment	Relaxation trainin intervention group biofeedback traini intervention group reference treatmer EMG no feedback instructions.
42	Sullivan and Adams, 2010	Retrospectiv e	Canada	48	Severity of pain (MPQ, PRI and NRS), Physical Funtion (5 minute work and finger to floor test), Self rated disability (PDI), PCS, TSK and BDI	Intervention group Physiotherapy trea (functional restora programme) alone Intervention group Physiotherapy trea augmented with P attainment progra
43	Turner 1982	RCT	Canada	46	Pain intensity (VAS), Self rated functional impairment scale (SIP).	Progressive muscl training for interv cognitive behavio intervention group reference treatmer waiting-list contro
44	Turner & Clancy, 1988	RCT	Canada	81	McGill pain Questionnaire (MPQ), Self rated functional impairment scale (SIP).	Aerobic exercises conditioning+ par spouses for interv systematic progre relaxation and ima intervention group reference treatmer waiting-list contro
45	Turner et al, 1990	RCT	Canada	96	McGill pain Questionnaire(MPQ), Self rated functional impairment scale (SIP), Depression	Operant condition intervention grou conditioning + ae for intervention g Reference treatm aerobic exercise c reference group l list controls.

46	Turner & Jensen, 1993	RCT	Canada	102	Pain intensity (VAS), Self rated functional impairment scale (SIP)	Cognitive therapy training for interv cognitive therapy group II, progress relaxation trainin for intervention g Reference treatm list controls.
47	van den Hout et al, 2003	RCT	Netherland	115	McGill pain Questionnaire(MPQ),Rol and Morris Disability Questionnaire (RMDQ), Tampa scale for Kinesiophobia (TSK), pain catastrophising scale (PCS)	Operant therapy b cognitive problem for intervention g therapy + group e intervention group
48	van den Roer et al, 2008	RCT	Netherland	114	Functional status, pain intensity and cost effectiveness planes	Intensive group tr intervention group based treatment b physiotherapist fo treatment
49	Von korff et al, 1998	RCT	Netherland	255	Disability (RMDQ), pain intensity, Attitudes towards care and extent of worries.	Intervention group educational progra back pain self-ma reference group h usual supplement back pain care.
50	von korff et al, 2005	RCT	Netherland	240	Roland Morris Disability Questionnaire (RMDQ), pain intensity, quality of life (SF-36), Fear avoidance beliefs (FABQ)	Activating interve (addressing fears normal activities intervention group treatment was Usu prescription and n pain medications ancillary services therapy)
51	Whitfill et al, 2010	RCT	USA	105	occupational status, self-reports of pain and disability, coping ability or psychosocial functioning	Intervention group at high risk of dev receiving early int intervention group intervention with and the reference had standard care.
52	Woods and Asmundson, 2008	RCT	Canada	44	Fear of pain/movement, Fear avoidance beliefs, pain related anxiety, pain self efficacy, pain	Intervention group vivo exposure, int II had graded acti reference treatme

					catastrophising and depression.	waiting list contro

CHAPTER THREE
MATERIALS AND METHODS

3.1 Participants

The participants for this study were individuals diagnosed of mechanical LBP who gave consent to participate in the research. They were recruited from the outpatient unit of the physiotherapy department at the Federal Medical Centre Abeokuta, Ogun State.

3.1.1 Inclusion Criteria

The following categories of patients were included in this study:

i. Patients diagnosed with MLBP.
ii. Patients who had a score equal or greater than 26 on the Pain Catastrophising scale (PCS) and or equal-to or greater-than 37 on the Tampa Scale of Kinesiophobia (TSK) (Sullivan 2010).
iii. Participants who can comprehend instructions in English and or Yoruba Languages. Yoruba language is the major language of communication in South-western Nigeria where the study was carried out.

3.1.2. Exclusion Criterion

These categories of patients were excluded from the study:

i. Patients with co-morbidity that may influence overall well-being such as cancer, vertebral or spinal infections, referred pain from internal organs, psychological pathology (Appendix VIII).

3.2 Instruments

The following instruments were used for data collection during the course of carrying out this study.

i. A self-designed form: This form (consist of 11 items; Appendix I) was used in obtaining socio-demographic (gender, age, ethnic group, marital

status, religious affiliation, educational status) and clinical characteristics (onset of MLBP) of the participant.

ii. PGAP activity log: This was used to record the activity log of the participants (Appendix IX).

iii. The Visual Analog Scale (VAS): This scale (Appendix II) was used for assessing pain intensity. It is the most common scale used in pain research. It represents the intensity dimension by a 10cm pain line with two anchor points of 'no pain' and 'worst pain I ever felt.' The patient is requested to draw a line at the point that best describes his or her pain level. It is widely used in the assessment of pain in the clinical setting and has been reported to be sensitive and reliable (Odole & Akinpelu, 2009). The anchors of VAS have been translated to Yoruba language with $r=0.63$ and $p<0.05$ (Odole & Akinpelu, 2009).

iv. Pain Catastrophising Scale: The pain catastrophising scale (consist of 13 items; Appendix III) was used to measure the degree of catastrophic thoughts about pain. Sullivan et al.(1995) developed the scale with three dimensions of pain catastrophizing vis-a-vis rumination, magnification and helplessness. This 13 items 5-point Likert scale has scores ranging from 0 (not at all) to 4 (all the time), relating the items to the past painful experience. Separate sub scores for the dimensions (range, rumination 0–16; magnification 0–12; and helplessness 0–24 points) or a total score (range, 0–52 points) can be calculated for the PCS. Higher scores denote a higher degree of catastrophizing. A score of 26 differentiates between high and low scores (Sullivan et al, 1995). The PCS has been shown to have good reliability and validity in a clinical population (Van Damme et al., 2000, Crombez et al., 1998; Vlaeyen et al., 1990). There was significant positive correlation ($r=0.89$, $p=0.03$) between the Yoruba translation and the English version of the PCS.

v. Tampa Scale of Kinesiophobia (TSK): Kinesiophobia was measured using the Tampa scale of kinesiophobia (consist of 17 items; Appendix IV) which was developed to measure fear of movement in person with musculoskeletal pain. The TSK consists of 17 statements capturing the idea that pain is a signal for reinjury because of physical activity or certain movements. Respondents were asked to indicate their level of agreement

on a 4-point rating scale. A high score indicates a high level of kinesiophobia (Swinkels-meewisse et al, 2003). The TSK uses a 4-point Likert scale, with scoring options ranging from 1 = 'strongly disagree' to 4 = 'strongly agree'. A total score is calculated after inversion of the individual scores of items 4, 8, 12 and 16. The total score ranges between 17 and 68. A high value on the TSK indicates a high degree of kinesiophobia (Lundberg et al., 2004). A score of 37 differentiates between high and low scores (Vlaeyen et al., (1995). There was significant positive correlation (r=0.77, p=0.02) between the Yoruba translation and the English version of the TSK.

vi. The Revised Oswestry Disability Questionnaire (RODQ) is a LBP functional assessment tool (consist of 10 items; Appendix V). It has been shown to be a valid indicator of disability in patients with LBP. It is based on ten sections with six levels each, assessing limitations in various activities of daily living (Fairbank and Pynscent, 2000; Davidson and Keating, 2002). The range of possible values is from 0 (the best health state) to 100 (the worst health state). Scoring of this questionnaire was done by computing the disability index percent (DIP). For each section of the questionnaire total possible score is five. The first statement was scored 0 and consecutive statements were scored 1 to 5. The total score was then divided by the total possible score and expressed in percentage to produce the disability index percent. The DIP was interpreted as 0-20% – Minimal disability, 21-40% – Moderate disability, 41-60% – Severe disability, 61-80% – Crippled and 81-100% – Bed bound or exaggerated symptoms. The RODQ was administered by interview to the participant. There was significant positive correlation (r=0.86, p=0.01) between the Yoruba translation and the English version of the RODQ.

vii. Self-efficacy in Rehabilitation for LBP (SER): This comprise of 12 statements regarding one's ability to perform activities related to the treatment of the back (Appendix VI). A low score relates to low perceived self-efficacy, while a high score predicts high perceived self-efficacy. SER has excellent internal consistency (α=0.88) and good test-retest reliability (r=0.88) (Woby et al. 2007). Each item was scored on an 11-point scale ranging from 0 to 10, where zero correlates with the statement,

"I cannot do it," and 10 means, "I am certain I can do it" (Woby et al. 2007). There was significant positive correlation (r=0.76, p=0.03) between the Yoruba translation and the English version of this questionnaire.

3.3 Setting for the Study

The study was carried out in the Physiotherapy outpatient clinic, of the Federal Medical Centre Abeokuta, Ogun State.

3.4 Methods

3.4.1 Research design

This study was a quasi-experimental design with control group (CG) and experimental group (EG).

3.4.2 Sampling Technique

A consecutive sampling technique was used to invite participants for the study. The subjects were screened by a physiotherapist in order to determine whether they met the inclusion criteria for the study. They were then assigned by a research assistant to either of the two groups - PGAP and Conventional Treatment (EG) and Conventional Treatment only (CG) as they became available using the toss of a coin. The subjects were blinded to the group they were assigned to and were not allowed to choose a group. Blinding was achieved by not allowing the subjects know what tossing head or tail of the coin translated to. The first available pair of participants was assigned into either of the two groups using the toss of the coin where the person who tossed tail was assigned to the experimental group and the head to the control group. Consecutive participants were then assigned alternately to either of the groups as they became available.

3.4.3 Sample Size Determination

The following equation was used to calculate the sample size for this study:

$$N = n\left(\frac{Z_{1-\alpha/2} + Z_{1-\beta}}{ES^2}\right)^2 \quad \text{and} \quad ES = \frac{\mu_d}{\sigma_d} \qquad \text{(Sullivan, 2012)}$$

Where

N = Sample size

n (number of groups) = 2

α (Selected level of significance) = 5%

$Z_{1-\alpha/2}$ (Standard normal distribution holding 1-α/2 below it) = 1.96

1-β (Selected power) = 80%

$Z_{1-\beta}$ (Standard normal distribution holding 1-β below it) = 0.84

ES = Effect size = 0.7

μ_d (mean difference in outcome measures scores after intervention that is assumed significant) = 14 points

$\mathfrak{v}_d$ (assumed standard deviation in the difference score) = 20 units

Sample size for each group was 32 participants making a total of 64 participants for the two groups.

10% of the calculated sample was added to make-up for participants lost to follow-up giving a desired sample size of 70 participants (35 subjects per group).

3.5 Procedure for data collection

3.5.1 Ethical approval

Ethical approval for the study was sought and obtained from the Ethics Committee of the University of Ibadan and University College Hospital, Ibadan. Ethical approval was also obtained from the Health Research Ethics Committee of the Federal Medical Centre Abeokuta. Permission was obtained from the Head of Physiotherapy Department, Federal Medical Centre Abeokuta.

3.5.2 Recruitment procedure

Participants were newly diagnosed or newly referred patients with MLBP who presented at the physiotherapy clinic for treatment. Patients interested in participating after due consultations with the researcher, having found to be eligible, were guided through the informed consent process (Appendix VII). This was followed by random allocation into either the experimental or control group which was done by the research assistant and the researcher scheduled appointments for the participants when their groups had been determined (Figure 3.1).

3.5.3 Screening procedure

All consenting participants in this research were screened for red and yellow flags on their first appointment by the researcher following these steps (Sullivan, 2010):

1. Assessing the appropriateness of PGAP for the participant through a screening evaluation using the PCS and TSK.
2. Clinical assessment to ascertain the diagnosis of mechanical low back pain using screening guideline for "red flags" in LBP (Appendix VIII).

   Socio-demographic and baseline data of all consenting participants in this study was obtained after the screening.

3.5.4 Translation of instruments

All the paper and pen research instruments were translated into Yoruba language through a cross cultural adaptation and validation process (Beaton et al, 2000). The original versions of the instrument were given to two experts (one of the expert was a Yoruba lecturer and the other a public health Nurse) in the Yoruba language for forward translation of the instruments. Both experts in each language then compared their versions to identify discrepancies indicative of ambiguous wording in the original instrument. A third person who is verse in English and Yoruba language and a specialist in Orthopaedics and Sports Physiotherapist then mediated to develop a consensus version of the translated instrument. A fourth expert (another lecturer of Yoruba Language) in Yoruba language translated the new instrument back into English and compared it to the original instrument. The translated instruments were Pain Catastrophising Scale (Appendix XI), Tampa Scale of Kinesiophobia (Appendix XII), Revised Oswestry Disability Questionnaire (Appendix XII), and Self-efficacy in Rehabilitation Scale (Appendix XIV). Several discrepancies observed in the backward translation were then harmonised by a panel of experts that comprised of all the translators and the researcher.

3.5.5 Pilot testing of instruments

The translated instruments were administered to a sample of 12 bilingual (English and Yoruba languages) patients with MLBP to ascertain the comprehension of the translated instruments. Majority of the respondents at this stage revealed that they understood the items and responses in both versions of the questionnaires. The queries of two respondents on the appropriate translation of Low Back Pain was easily attended to as the expert panel fully attended to this during their meeting.

3.5.6 Data Collection:

The following data were collected and recorded at baseline, end of the 5th and 10th week of the study.

Socio-demographic and clinical characteristics of Participants: These were obtained by interview using a self-designed form (Appendix I). The socio-demographic variables that were obtained in this study were gender, age, ethnic group, marital status, religious affiliation, educational status, social class. The main clinical characteristic measured was the onset of LBP which was used in classifying the participants into acute, sub-acute and chronic LBP cases.

Symptom and Disability Profile

i. Pain intensity: The visual analog scale (Appendix II) was used to assess pain intensity.
ii. Disability: This was measured using the Revised Oswestry Disability Questionnaire (RODQ) (Appendix V). It was used as a LBP functional assessment tool.

Psychosocial Risk Factors

i. Pain Catastrophizing: The pain catastrophizing scale (Appendix III) was used to measure the degree of catastrophic thoughts about pain.
ii. Fear of Movement: Tampa scale of kinesiophobia (Appendix IV) was used to measure fear of movement in the participants (Swinkels-meewisse et al, 2003).
iii. Self-efficacy: The SER for LBP (Appendix VI) was used in assessing the self-efficacy of the participants (Woby et al. 2007).

3.5.7 Intervention

The Experimental Group: This group participated in PGAP in line with the protocols of Sullivan (2010) alongside the conventional treatment for low back pain. The Back school book by Odebiyi (2004) was given to each of the participants for education and advice on low back pain. Measurement of selected symptom/disability profile and psychosocial risk factors were taken at baseline and at the end of the 5th and 10th week.

The Control Group: This group participated in conventional treatment for LBP. The Back school book by Odebiyi (2004) was given to each of the

participants for education and advice on LBP. Measurement of selected symptom/disability profile and psychosocial risk factors were taken at baseline and at the end of the 5th and 10th week.

All participants in the experimental group of this study were treated with the Progressive Goal Attainment Programme (PGAP). The PGAP comprised of 10 sessions.

A brief overview of each session are indicated below:

Screening and engagement (Session 1):

Assessment was conducted to determine a participant's suitability for participation in PGAP.

Session 2 (Week 1):

The researcher:

i. Built therapeutic relationship.
ii. Examined life impact of participant's MLBP.
iii. Established pre-injury or pre-illness activity repertoire.
iv. Provided subject with instructions on completion of workbook.

Session 3 (Week 2):

The researcher;

i. Examined participant's work book during the 1st week.
ii. Examined life-role interference resulting from participant's MLBP.
iii. Introduced waking and walking routine.
iv. Introduced activity planning.
v. Worked on re-establishing pre-injury activity structure.

Session 4 (Week 3):

The researcher;

i. Reviewed participant's planned and completed activities during the 2nd week.
ii. Introduced importance of pre-determine duration of activity involvement for all new activities planned.
iii. Introduced goal setting.
iv. Assisted participant in translating goals into specific activities.

v. Assisted participant in incorporating goal-relevant activities in schedule of planned activities for coming week.

Session 5 (Week 4):

The researcher;

i. Reviewed participant's planned and completed activities during the 3rd week with particular emphasis on goal-relevant activities.
ii. Discussed participant's perception of important changes since the beginning of treatment.
iii. Assisted the participant in scheduling goal-relevant and role-relevant activities for the coming week.

Session 6 (Week 5):

The researcher;

i. Reviewed participant's planned and completed activities during the 4th week with particular emphasis on goal-relevant and role-relevant activities.
ii. Began establishing links to re-employment resources.
iii. Began exposure techniques to facilitate resumption of discontinued activities.
iv. Assisted the participant in planning activities for the coming week focusing on resumption of discontinued activities.

Mid-treatment assessment was completed by the research assistant.

Session 7(Week 6):

The researcher;

i. Reviewed participant's planned and completed activities during the 5th week with particular emphasis on resumption of discontinued activities.
ii. Planned focus of intervention for remaining weeks of treatment.
iii. Introduced participant to thought monitoring techniques for controlling negative or pessimistic cognitions that might be impacting negatively on rehabilitation progress.
iv. Assisted the participant in planning activities for the coming week focusing on resumption of discontinued activities.

Session 8 (Week 7):

The researcher;

i. Reviewed participant's planned and completed activities during the 6th week with particular emphasis on resumption of discontinued activities.
ii. Reviewed the participant's thought monitoring exercise.
iii. Assisted the participant in examining multiple response options to stressful situations.
iv. Discussed contact with re-employment resources.
v. Assisted the participant in planning activities for the coming week focusing on employment relevant activities.

Session 9 (Week 8):

The researcher;

i. Reviewed participant's planned and completed activities during the 7th week with particular focus on employment relevant activities.
ii. Examined feared activities associated with return to work.
iii. Prepared participant to be fully involved in establishing a modified work re-entry plan.
iv. Reviewed thought monitoring exercises.
v. Assisted the participant in planning activities for the coming week focusing on employment-relevant activities.

Session 10 (Week 9):

The researcher;

i. Reviewed participant's planned and completed activities during the 8th week with particular focus on employment relevant activities.
ii. Addressed psychosocial challenges of work resumption where necessary.
iii. Assisted participant in generating multiple response options to work re-entry stresses.

Session 11(Week 10):

The researcher;

i. Reviewed participant's planned and completed activities during the 9th week.
ii. Discussed participant's perception of important changes occurring through the course of treatment.
iii. Provided participant with assessment feedback.
iv. Discussed participant's ongoing involvement in goal setting.
v. Discussed discharge and follow up plans.

Final assessment was completed by the research assistant.

Conventional treatment for MLBP: This included drug treatment in form of analgesics (paracetamol, and Ibuprofen) and muscle relaxant (Norflex) as prescribed by the attending medical practitioner. Arrangements were made with the medical practitioner to ensure that patients used the same oral medications during the study duration. Also standard Physiotherapy care which is a combination of several interventions like soft tissue mobilization, TENS therapy, lumbar traction, isometric trunk muscle strengthening exercises, flexibility and coordination exercises and ergonomics counselling. The patients with MLBP of acute onset received Cryotherapy and TENS therapy for 15 minutes, Patients with chronic MLBP received Infra-red therapy and TENS for 15 minutes. All the patients had Soft tissue mobilisation with analgesic cream and McKenzie exercises within the allowance of their pain threshold. Patients received physiotherapy treatment thrice in a week. Lumbar traction for 30 minutes was used when there was evidence of nerve root impingement occasioned by pain radiculopathy. Home programmes were mainly a combination of McKenzie exercises and soft tissue mobilisation.

Follow up Assessment: Participants in the two groups were booked for a three month follow-up assessment during which all the initial assessment at baseline were repeated.

3.6 Data Analyses

The data collected were analyzed as follows:

i. Descriptive statistics of frequency distribution, mean, standard deviation and percentages was used in summarizing the socio-demographic and clinical characteristics of the participants.

ii. Chi-square and independent t-test were used in comparing the demographic variables.

iii. Friedman Analysis of Variance (FANOVA) was used to compare participants' pain intensity, PC score, kinesiophobia score, disability score and self-efficacy score within each group at baseline and after intervention at the end of the fifth and tenth weeks. Percentage change values on measures of pain intensity, PC, kinesiophobia, disability, and self-efficacy was assessed in order to compare the magnitude of change within each group.

iv. Man Whitney U test was used to compare the pain intensity, PC score, kinesiophobia score, disability score and self-efficacy score between the two groups at baseline, and after intervention at the end of the fifth and tenth weeks. Level of significance was set at alpha = 0.05.

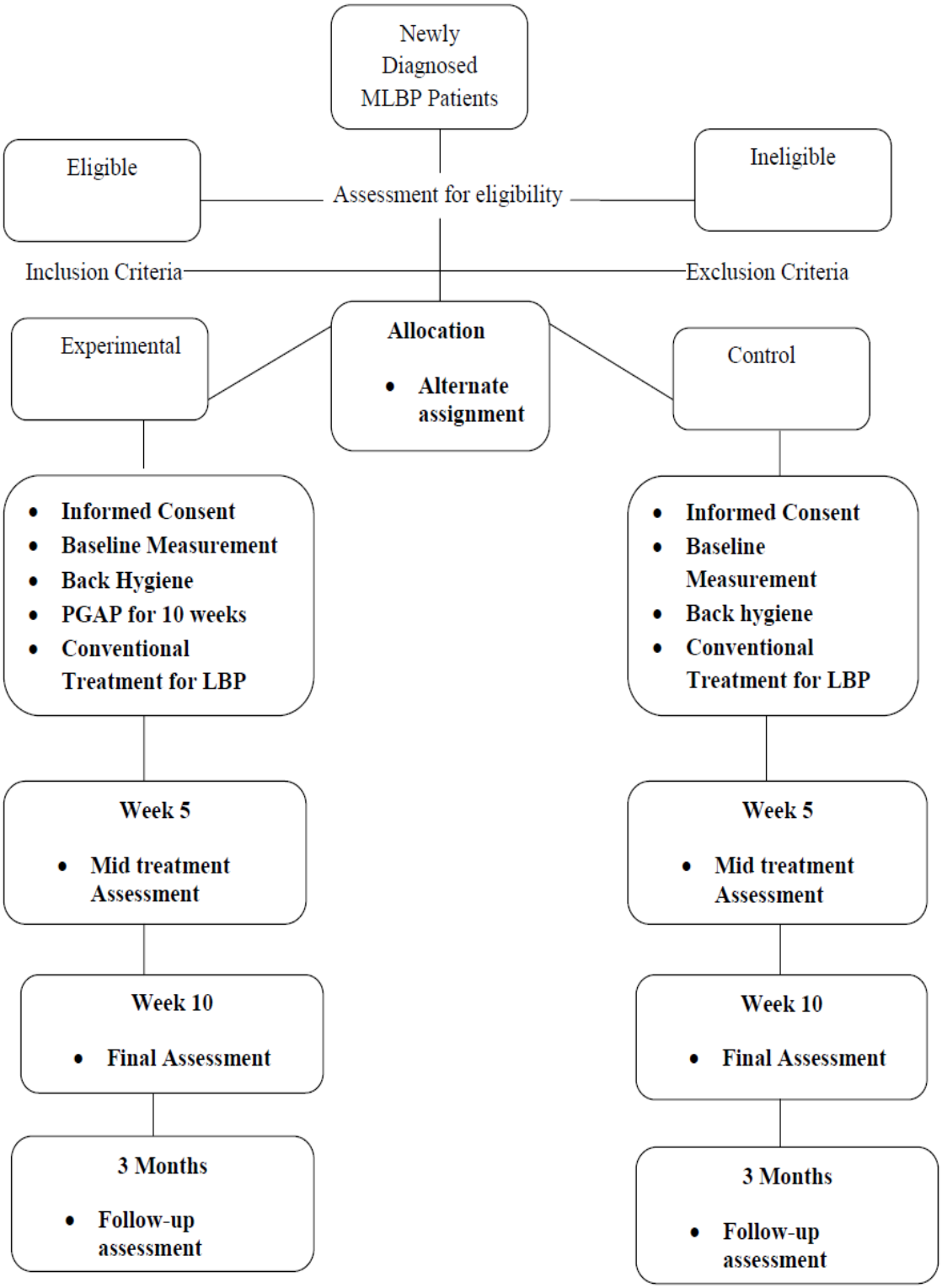


Figure 3.1 Flow chart for the study

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 RESULTS

4.1.1 Demographic and Clinical Characteristics of Participants

One hundred and eighteen patients presenting with low back pain were invited to participate in this study, only eighty seven met the inclusion criteria after screening and a total of 70 participants (35 participants in each of the experimental and control groups) took part in the study. The demographic and clinical variables of the two groups are shown in table 4.1 and 4.2. Twenty three (65.7%) participants in the experimental group were males while twelve (34.3%) were females. In the control group, five (14.3%) of the participants were males while thirty (85.7%) were females. Four (11.4%) of the participants' in the experimental group had primary education as the highest educational status attained. Six (17.1%) of the participants' in the experimental group had secondary education as the highest educational status attained. Twenty five (71.4%) of the participants' in the experimental group had tertiary education as the highest educational status attained. None of the participants' in the control group had primary education as the highest educational status attained. Four (11.4%) of the participants' in the control group had secondary education as the highest educational status attained. Thirty one (88.6%) of the participants' in the control group had tertiary education as the highest educational status attained (Table 4.2).

All the participants were married. Twenty one (60%) of the participants in the experimental group were christians while fourteen (40%) were muslims. In the control group, all the participants were christians. Thirty one (88.6%) participants in the experimental group were of the Yoruba tribe while four (11.4%) were of the Hausa/Fulani tribe. All participants in the control group were of the Yoruba tribe.

Fifty six (80%) of all the participants were public servants with white collar occupation group

while 14(20%) participants were traders who were self-employed (Table 4.2). The mean age, height and weight of the participants were 44.9±8.3years; 1.7±0.1m; 74.9±11.2kg for the experimental group and 47.4±7.5years; 1.6±0.1m; 81.1±9.5kg for the control group respectively. Four (11.4%) of the participants' in the experimental group's duration of pain was less than 6 weeks. Eleven (31.4 %) of the participants' in the experimental group had pain duration between 6 and 12 weeks and twenty (57.1%) of the participants in the experimental group had pain duration of more than 12 weeks. Four (11.4%) of the participants' in the control group's duration of pain was less than 6 weeks. Thirteen (37.1 %) of the participants' in the control group had pain duration between 6 and 12 weeks and eighteen (51.4%) of the participants in the control group had pain duration of more than 12 weeks (Table 4.1).

Twenty two of the participants (63%) in the experimental group and 25 (71%) in the control group were available for the three-month follow up assessment. Twenty three (33%) participants were not available for the follow-up assessment. Fifteen of these participants reported that they had pain relief and had returned to their locations, while eight of these participants could not be traced for follow-up at the period of this report.

Table 4.1 Comparison of Demographic Variables of Experimental and Control Groups using Independent t-test statistics

Variables	EG n=35 $\bar{x}$±S.D	CG n=35 $\dot{x}$±S.D	t-value	p-value
Age (yrs)	44.97± 8.29	47.43±7.54	1.29	0.19
Height (m)	1.66±0.92	1.61±0.82	2.40	0.02*
Weight (kg)	74.89±11.23	81.14±9.51	2.52	0.01*
BMI (Kg/m^2)	27.24±3.26	31.57±4.26	4.78	<0.01*

Key: * = Significant at $p<0.05$

Table 4.2 Comparison of Demographic and selected Clinical Variables of Experimental and Control groups at Baseline using Chi-square statistical test

Variables	Characteristics	Freq.(%)	Freq.(%)	test-stat.	p-value
	—	EG n=35	CG n=35	χ^2	
Sex	Male	23(65.7)	5(14.3)	19.29	<0.01*
	Female	12(34.3)	30(85.7)		
Educational Status	Primary	4(11.4)	0(0)	-	-
	Secondary	6(17.1)	4(11.4)		
	Tertiary	25(71.4)	31(88.6)		
Religion	Christianity	21(60)	35(100)	-	-
	Islam	14(40)	0(0)		
Tribe	Yoruba	31(88.6)	35(100)	-	-
	Hausa	4(11.4)	0(0)		
Duration LBP	Acute	4(11.4)	4(11.4)	0.27	0.87
	Sub-acute	11(31.4)	13(37.3)		
	Chronic	20(57.1)	18(51.4)		

KEY: * = Significant at $p < 0.05$

\- = invalid chi-square

4.1.2 Comparison of Selected Pain related and Psychosocial Variables of Participants in the Experimental and Control Groups at Baseline of Study

The mean values of the selected pain-related and psychosocial variables for both groups are shown in table 4.3. The mean pain intensity score for the experimental group was 9.4±0.9 while that for the control group was 9.1±0.9. No significant difference was observed in the pain intensity score of both the experimental and the control groups at baseline (U = 473, p= 0.07). The mean score on the pain catastrophizing scale for the experimental group was 33.6±9.9 and 33.0±5.3 for the control group. No significant difference was observed in mean score on the pain catastrophizing scale of both the experimental and the control groups at baseline (U =529.5, P=0.33). The mean score on the TSK scale for the experimental group was 41.4±7.7 and 41.5±2.9 for the control group. No significant difference was observed in mean score on the TSK scale of both the experimental and the control groups at baseline (U = 527.5, P= 0.31). The mean disability index score of the experimental (59.1±12.8) and control groups (55.5±12.3) were not significantly different at baseline (U = 527.5, p= 0.32). The mean score on the self-efficacy in rehabilitation scale of the experimental (81.4±9.5) and control groups (81.2±12.0) were not significantly different at baseline (U = 594, p= 0.83) (Table 4.3).

Table 4.3 Between-Group Comparison of selected Pain related and Psychosocial Variables of Experimental and Control Groups at Baseline using Man-Whitney U statistical test

Variables	EXPERIMENTAL n=35 x±S.D	CONTROL n=35 x̄±S.D	U-value	p-value
PIS	9.4±0.9	9.1±0.9	473.0	0.07
PCS	33.6±9.9	33.0±5.3	529.5	0.33
TSKS	41.4±7.7	41.5±2.9	527.0	0.31
DIS	59.1±12.8	55.5±12.3	527.5	0.32
SES	81.4±9.5	81.2±12.0	594.0	0.83

KEY:

PIS: Pain Intensity Score

PCS: Pain Catastrophising Score

TSKS: Tampa Scale for kinesiophobia Score

DIS: Disability Index Score

SES: Self-efficacy in rehabilitation Score

U-value: Man-Whitney U

4.1.3 Changes in Selected Pain related and Psychosocial Variables of Participants in the Experimental group across baseline, 5th week, 10th weeks and 3 months follow-up

Comparison of changes in Selected Pain related and psychosocial variables of participants in the experimental group across baseline, end of 5th and 10 week are presented on table 4.4. There was a significant difference in the pain intensity score of the experimental group across baseline, end of 5th week, 10th week and 3 months follow-up with Fr=61.16, and $p < 0.01$. There was a significant difference (Fr = 43.40, $p < 0.01$) in the PC score of the experimental group across baseline, end of 5th week, 10th week and 3 months follow-up (see Table 4.4). There was a significant difference (Fr = 26.31, $p < 0.01$) in TSK score of the experimental group across baseline, end of 5th week, 10th week and 3 months follow-up. The extent of pain related disability (Mean DIS) in the experimental group was significantly different (Fr = 50.28, p=0.25) across baseline, end of 5th week, 10th week and 3 months follow-up. There was a significant difference (Fr = 32.19, p=0.19) in the self-efficacy score of the experimental group at baseline, end of 5th week, 10th week and 3 months follow-up (See Figure 4.1 to 4.5).

Table 4.4 Changes in selected Pain related and Psychosocial Variables of Participants in the Experimental Group across Baseline, 5th week, 10th week and 3 months Follow-up using Friedmann ANOVA statistical test

Variables	Baseline n= 35 x±S.D	5th Week n=35 x±S.D	10th Week n=35 x±S.D	3months n=22 x±S.D	Fr	p-value
PIS	9.4±0.9	4.9±1.9	3.6±1.6	3.8 ±1.6	61.16	<0.01*
PCS	33.6±9.9	22.2±11.2	23.0±9.4	21.7±9.5	43.40	<0.01*
TSKS	41.4±7.7	37.3±7.5	34.4±6.7	29.1±6.3	26.31	<0.01*
DIS	59.1±12.8	42.6±11.1	41.1±8.5	33.0±6.9	50.28	<0.01*
SES	81.4±9.5	94.4±14.5	94.4±11.5	101.2±11.5	32.19	<0.01*

KEY:

* = Significant at $p < 0.05$

PIS: Pain intensity score

PCS: Pain catastrophising score

TSKS: Tampa scale for kinesiophobia Score

DIS: Disability Index Score

SES: Self-efficacy in rehabilitation Score

Fr: Friedmann ANOVA

4.1.4 Changes in Selected Pain related and psychosocial variables of Participants in the Control group across baseline, 5th week, 10th weeks and 3 months follow-up

Comparison of changes in selected pain related and psychosocial variables of participants in the control group across baseline, end of 5th, 10th week and 3 months follow-up are shown in table 4.5. There was significant difference (Fr = 52.41, p = 0.01) in pain intensity scores of the control group across baseline, end of 5th week, 10th week and 3 months follow-up. There was significant difference (Fr = 42.61, p<0.01) in the PC scores of the control group across baseline, end of 5th week, 10th week and 3 months follow-up. There were significant differences (Fr = 31.66, p = 0.37) in TSK scores of the control group across baseline, end of 5th week, 10th week and at 3 months follow-up. The extent of pain related disability (Mean DIS) in the control group was significantly different (Fr = 30.09, p<0.01) across the baseline, end of 5th week, 10th week and 3 months follow-up. There was significant difference (Fr = 32.53, p<0.01) in the self-efficacy score of the control group across baseline, end of 5th week, 10th week and 3 months follow-up (see Table 4.5 and Figure 4.1 to 4.5).

Table 4.5 Comparison of changes in selected Pain related and Psychosocial Variables of Participants in the Control Group across baseline, 5th week, 10th weeks and 3 months follow-up using Friedmann ANOVA statistical test

Variables	Baseline n= 35 x ± S.D	5th Week n= 35 x ± S.D	10th Week n =35 x ± S.D	3months n=25 x ± S.D	Fr	p-value
PIS	9.1± 0.9	5.0±2.8	3.1±1.8	4.9±1.6	52.41	<0.01*
PCS	33.0±5.3	27.9±8.8	23.0±8.4	27.5±5.8	42.61	<0.01*
TSKS	41.5±2.9	42.2±3.2	36.9±3.7	35.8±6.6	31.66	<0.01*
DIS	55.5±12.3	57.8±8.9	45.3±7.3	43.4±7.6	30.09	<0.01*
SES	81.2±12.0	80.0±20.1	94.1±9.4	92.3±9.3	32.53	<0.01*

KEY:

* = Significant at $p < 0.05$

PIS: Pain intensity score

PCS: Pain catastrophising score

TSKS: Tampa scale for kinesiophobia Score

DIS: Disability Index Score

SES: Self-efficacy in rehabilitation Score

Fr: Friedmann ANOVA

4.1.5 Between-group Comparison of Selected Pain related and Psychosocial Variables of Participants in the Experimental and Control Groups at the end of 5th week of the Study

At the end of 5th week of the study, the mean values of the selected pain related and psychosocial variables for both groups are shown in table 4.6. The mean pain intensity score for the experimental and control groups did not differ significantly at the end of 5th week (U = 593.5, p= 0.82). The mean score on the pain catastrophizing scale for the experimental and control groups was significantly different at the end of 5th week (U = 434.5, p= 0.04). The mean score on the TSK scale for the experimental and control groups was significantly different at the end of 5th week (U = 357.0, p= 0.03). The mean disability index score for the experimental and control groups was significantly different at the end of 5th week (U = 141.5, p= 0.01). The mean score on the self-efficacy in rehabilitation scale for the experimental and control groups was significantly different at the end of 5th week (U = 377.0, p=0.01).

Table 4.6 Between-Group Comparison of Selected Pain related and Psychosocial Variables at the end of 5th Week using Man-Whitney U statistical test

Variables	EXPERIMENTAL n=35 x±S.D	CONTROL n=35 x±S.D	U-value	p-value
PIS	4.9±1.9	5.0±2.8	593.5	0.82
PCS	22.2±11.2	27.9±8.8	434.5	0.04*
TSKS	37.3±7.5	42.2±3.2	357.0	0.03*
DIP	42.6±11.0	57.8±8.8	141.5	0.01*
SES	94.4±14.5	80.0±20.1	377.0	0.01*

KEY:

* = Significant at $p < 0.05$

PIS: Pain intensity score

PCS: Pain catastrophising score

TSKS: Tampa scale for kinesiophobia Score

DIS: Disability Index Score

SES: Self-efficacy in rehabilitation Score

U-value: Man-Whitney U

4.1.6 Between-group Comparison of Selected Pain related and Psychosocial variables of Participants in the Experimental and Control Groups at the end of 10th week of the Study

The mean values of the selected pain related and psychosocial variables for both groups at the end of 10th week are presented in table 4.7. There was no significant difference in the pain intensity score of both groups at the end of 10th week (U=477.5, p=0.10). The mean score on the pain catastrophizing scale for the experimental group was not significantly different from that of the control groups at the end of 10th week (U = 554.0, p= 0.49). The mean score on the TSK scale was not significantly different for both the experimental and the control groups at the end of 10th week (U=498.0, p=0.18). The mean disability index score on for the experimental group was 41.1±8.5 and 45.3±7.3 for the control group. There was a significant difference observed in the mean disability index score of both the experimental and the control groups at the end of 10th week (U = 428.5, P= 0.03) with the experimental group having significantly lower scores on disability. The mean score on the self-efficacy in rehabilitation scale for the experimental group was 94.4±11.5 and 94.1±9.4 for the control group. There was no significant difference between the two groups at the end of 10th week (U = 604.5, p=0.93).

Table 4.7 Between-group Comparison of Selected pain related and Psychosocial Variables at the end of 10th week using the Man-Whitney U statistical test

Variables	EXPERIMENTAL n=35 x±S.D	CONTROL n=35 x±S.D	U-value	p-value
PIS	3.6±1.6	3.1±1.7	477.5	0.10
PCS	23.0±9.4	23.0±8.4	554.0	0.49
TSKS	34.4±6.8	36.9±3.7	498.0	0.17
DIS	41.1±8.5	45.3±7.3	428.5	0.03*
SES	94.4±11.5	94.1±9.4	604.5	0.93

KEY:

* = Significant at $p < 0.05$

PIS: Pain intensity score

PCS: Pain catastrophising score

TSKS: Tampa scale for kinesiophobia Score

DIS: Disability Index Score

SES: Self-efficacy in rehabilitation Score

U-value: Man-Whitney U

4.1.7 Between-group Comparison of Selected Pain related and Psychosocial Variables of Participants in the Experimental and Control Groups at three months follow-up.

The mean values of the selected pain related and psychosocial variables for both groups at 3 months follow-up are presented in table 4.8. There was a significant difference in the pain intensity score of the experimental and the control groups at three months follow-up (U =175.0, p=0.03) with the experimental group having significantly lower pain intensity score. There was a significant difference in the mean score on the pain catastrophizing scale of the experimental and the control groups at three months follow-up (U = 176.5, p= 0.04) with the experimental group having significantly lower PC score. The mean score on the TSK scale for the experimental group was 29.1±6.3 and 35.8±6.6 for the control group. There was a significant difference in the mean score on the TSK of the experimental and the control groups at three months follow-up (U = 116.5, p<0.01) with the experimental group having significantly lower score on the TSK. The mean disability index score on the experimental group was 33.0±6.9 and 43.4±7.6 for the control group. There was a significant difference in the mean disability index score of the experimental and the control groups at three months follow-up (U = 89.0, p<0.01) with the experimental group having significantly lower disability index score. The mean score on the self-efficacy in rehabilitation scale for the experimental group was 101.2±11.5 and 92.3±9.3 for the control group. There was a significant difference between the two groups at three months follow–up (U = 141.5, p<0.01) with the experimental group having higher score on the self-efficacy in rehabilitation scale.

Table 4.8 **Between-group Comparison of Selected pain related and Psychosocial Variables at three months follow-up using Man-Whitney U statistical test**

Variables	EXPERIMENTAL n=22 $\bar{x} \pm$ S.D	CONTROL n=25 $\bar{x} \pm$ S.D	U-value	p-value
PIS	3.8 ±1.6	4.9±1.6	175.0	0.03*
PCS	21.7±9.5	27.5±5.8	176.5	0.04*
TSKS	29.1±6.3	35.8±6.6	116.5	<0.01*
DIS	33.0±6.9	43.4±7.6	89.0	<0.01*
SES	101.2±11.5	92.3±9.3	141.5	<0.01*

KEY:

* = Significant at $p < 0.05$

PIS: pain intensity score

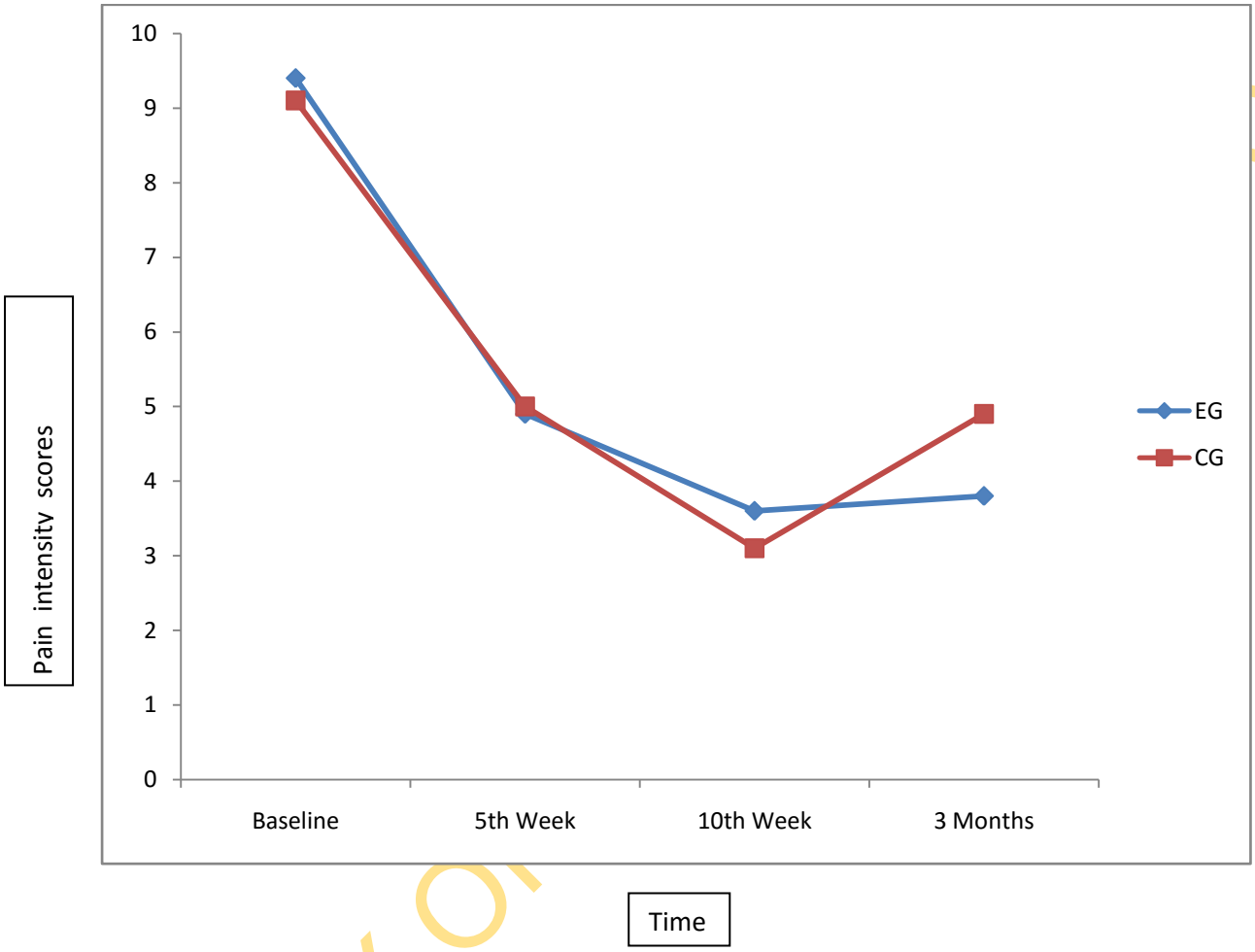
PCS: Pain catastrophising score

TSKS: Tampa scale for kinesiophobia Score

DIS: Disability Index Score

SES: Self-efficacy in rehabilitation Score

U-value: Man-Whitney U

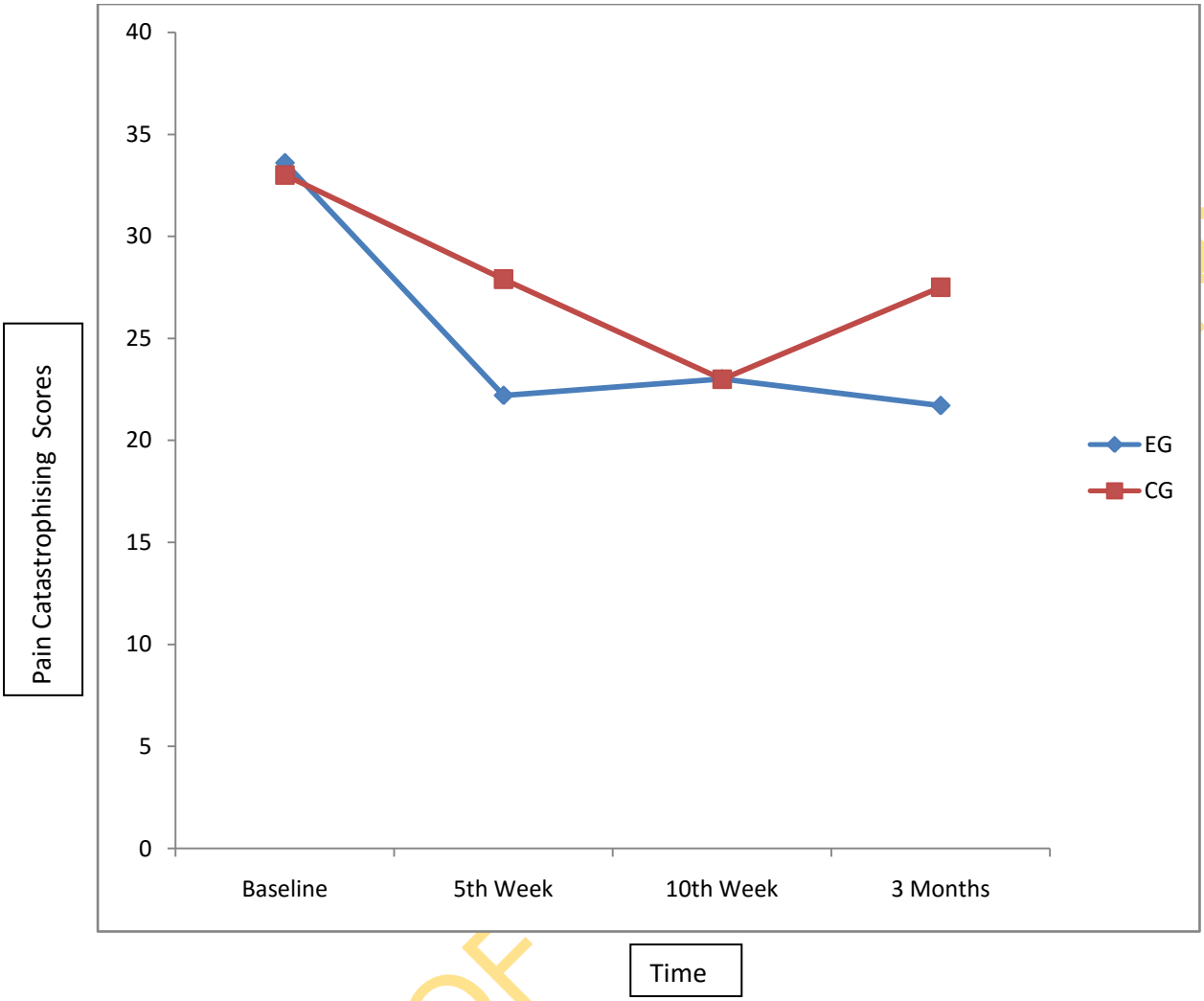


Key:

EG: Experimental group

CG: Control group

Figure 4.1: Trend of Pain Intensity scores of EG and CG

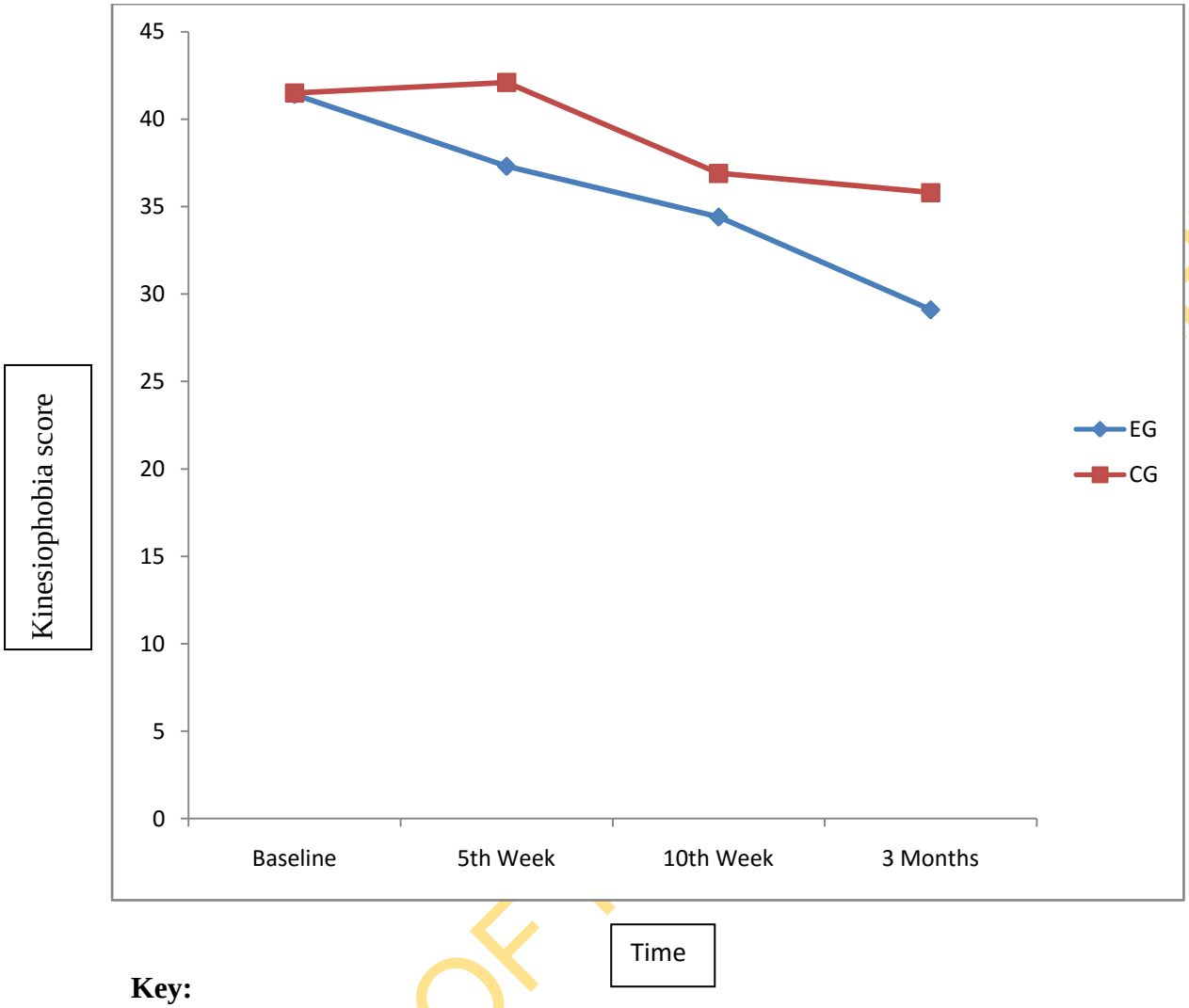


Key:

EG: Experimental group

CG: Control group

Figure 4.2: Trend in Pain Catastrophising of EG and CG

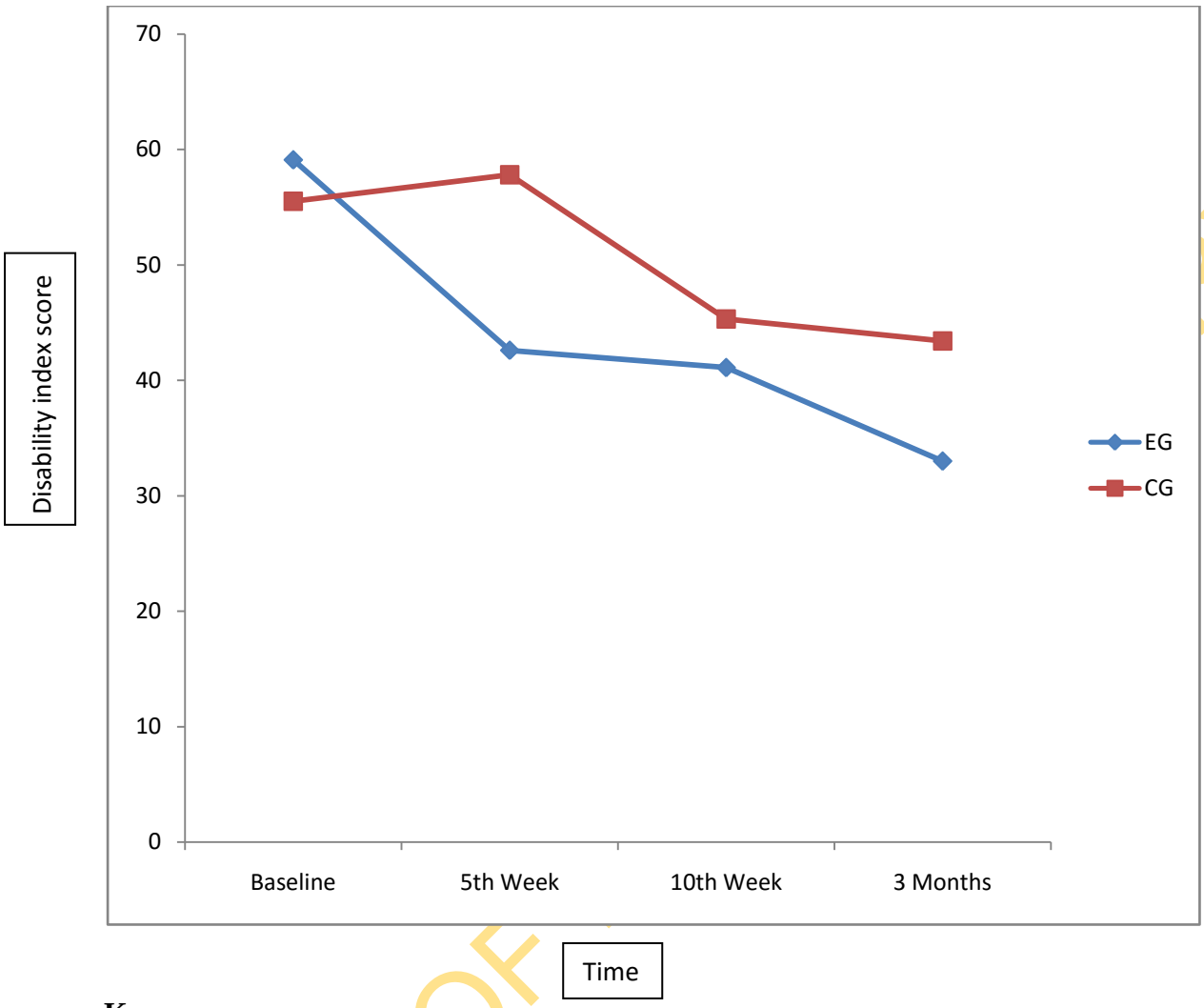


Key:

EG: Experimental group

CG: Control group

Figure 4.3: Trend in Kinesiophobia of EG and CG

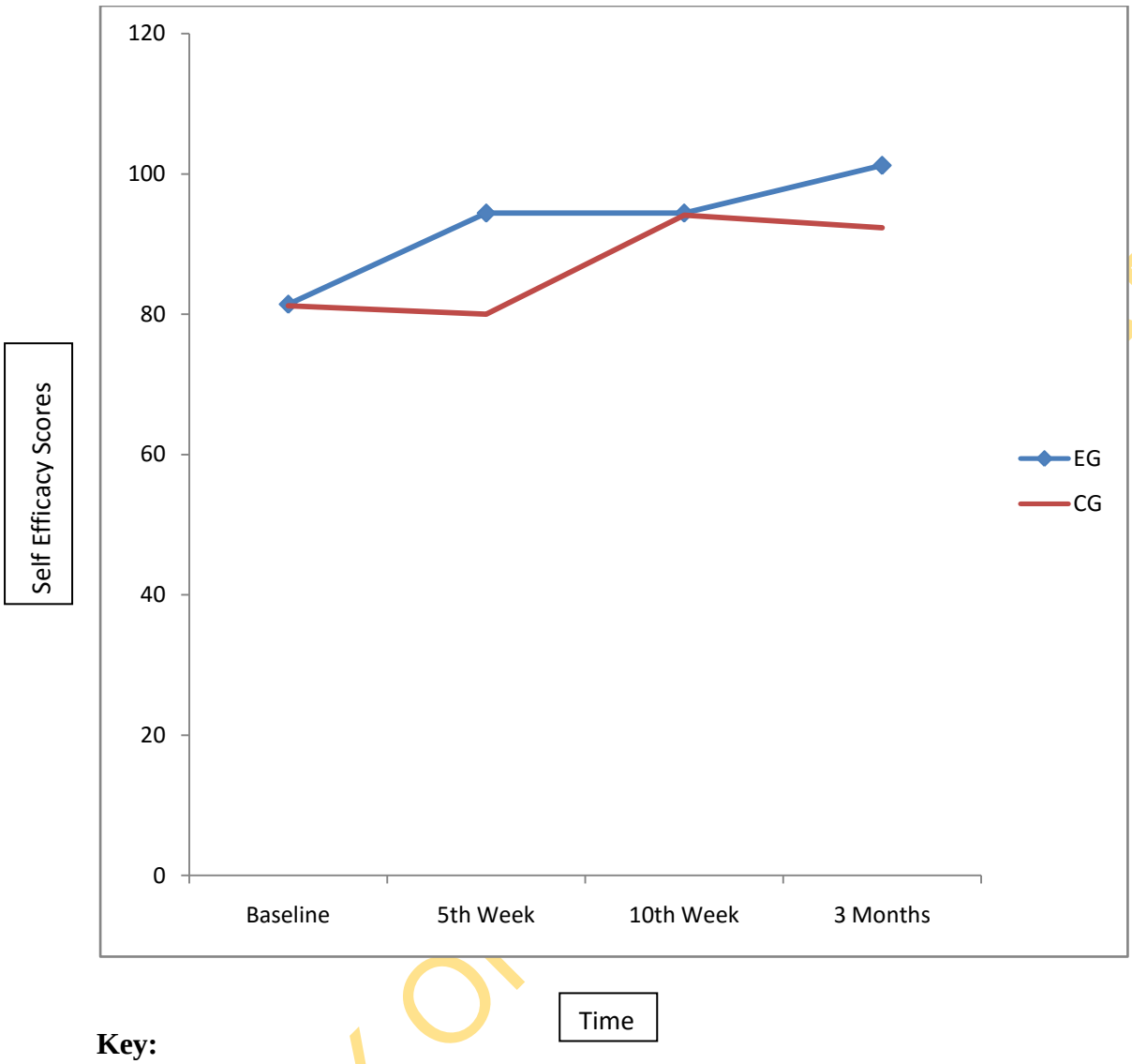


Key:

EG: Experimental group

CG: Control group

Figure 4.4: Trend in pain related disability of EG and CG



Key:

EG: Experimental group

CG: Control group

Figure 4.5: Trend in Self-efficacy of EG and CG

4.2. Hypotheses Testing

Sub hypothesis 1

Statement: There would be no significant difference in the pain intensity scores of individuals in the experimental group with MLBP across baseline, fifth week, tenth week and at three-month follow up.

Alpha level = 0.05

Test Statistics: Friedman ANOVA

Conclusion: Pain intensity score of Experimental group: $p < 0.01$

Sub hypothesis 1 was REJECTED

Sub hypothesis 2

Statement: There would be no significant difference in the PC scores of individuals in the experimental group with MLBP across baseline, fifth week, tenth week and at three-month follow up.

Alpha level = 0.05

Test statistics: Friedman ANOVA

Conclusion: PC score of Experimental group: $p < 0.01$

Sub hypothesis 2 was REJECTED

Sub hypothesis 3

Statement: There would be no significant difference in the TSK scores of individuals in the experimental group with MLBP across baseline, fifth week, tenth week and at three-month follow up.

Alpha level = 0.05

Test statistics: Friedman ANOVA

Conclusion: TSK score of Experimental group: $p < 0.01$

Sub hypothesis 3 was REJECTED

Sub hypothesis 4

Statement: There would be no significant difference in the mean disability index percent of individuals in the experimental group with MLBP across baseline, fifth week, tenth week and at three-month follow up.

Alpha level = 0.05

Test statistics: Friedman ANOVA

Conclusion: Mean disability index score of Experimental group: $p < 0.01$
Sub hypothesis 4 was REJECTED

Sub hypothesis 5

Statement: There would be no significant difference in the mean score in the self-efficacy in rehabilitation (SER) scale of individuals in the experimental group with MLBP across baseline, fifth week, tenth week and at three-month follow up.
Alpha level = 0.05
Test statistics: Friedman ANOVA

Conclusion: Mean score on SER scale of Experimental group: $p < 0.01$
Sub hypothesis 5 was REJECTED

Sub hypothesis 6

Statement: There would be no significant difference in the pain intensity scores of individuals in the control group with MLBP across baseline, fifth week, tenth week and at three-month follow up.
Alpha level = 0.05
Test statistic: Freidman ANOVA

Conclusion: Pain intensity score of control group: $p < 0.01$
Sub hypothesis 6 was REJECTED

Sub hypothesis 7

Statement: There would be no significant difference in the PC scores of individuals in the control group with MLBP across baseline, fifth week, tenth week and at three-month follow up.
Alpha level = 0.05
Test statistic: Freidman ANOVA

Conclusion: PC score of control group: $p < 0.01$
Hypothesis 7 was REJECTED

Sub hypothesis 8

Statement: There would be no significant difference in the TSK scores of individuals in the control group with MLBP across baseline, fifth week, tenth week and at three-month follow up.
Alpha level = 0.05
Test statistic: Freidman ANOVA

Conclusion: TSK score of control group: $p < 0.01$
Sub hypothesis 8 was REJECTED

Sub hypothesis 9

Statement: There would be no significant difference in the mean disability index percent of individuals in the control group with MLBP across baseline, fifth week, tenth week and at three-month follow up.
Alpha level = 0.05
Test statistic: Freidman ANOVA

Conclusion: Mean disability index percent of control group: $p < 0.01$
Sub hypothesis 9 was REJECTED

Sub hypothesis 10

Statement: There would be no significant difference in the mean score in the self-efficacy in rehabilitation (SER) scale of individuals in the control group with MLBP across baseline, fifth week, tenth week and at three-month follow up.
Alpha level = 0.05
Test statistic: Freidman ANOVA

Conclusion: Mean score in SER scale of control group: $p < 0.01$
Sub hypothesis 10 was REJECTED

Sub hypothesis 11

a. Statement: There would be no significant difference between the pain intensity scores of individuals in the experimental and control groups with MLBP at baseline.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: Pain intensity score of experimental and control groups: $p= 0.07$

Sub hypothesis 11a was NOT REJECTED

b. Statement: There would be no significant difference between the pain intensity scores of individuals in the experimental and control groups with MLBP at the end of fifth week.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: Pain intensity score of experimental and control groups: p= 0.82

Sub hypothesis 11b was NOT REJECTED

c. Statement: There would be no significant difference between the pain intensity scores of individuals in the experimental and control groups with MLBP at the end of tenth-week.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: Pain intensity score of experimental and control groups: p=0.10

Hypothesis 11c was NOT REJECTED

d. Statement: There would be no significant difference between the pain intensity scores of individuals in the experimental and control groups with MLBP at 3 months follow-up.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: Pain intensity score of experimental and control groups: p=0.03

Hypothesis 11d was REJECTED

Sub hypothesis 12

a. Statement: There would be no significant difference between the PC scores of individuals in the experimental and control groups with MLBP at baseline.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: PC score of experimental and control groups: p = 0.33

Hypothesis 12a was NOT REJECTED

b. Statement: There would be no significant difference between the PC scores of individuals in the experimental and control groups with MLBP at the end of fifth week.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: PC score of experimental and control groups: p = 0.04

Sub hypothesis 12b was REJECTED

c. Statement: There would be no significant difference between the PC scores of individuals in the experimental and control groups with MLBP at the end of tenth week.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: PC score of experimental and control groups: p = 0.49

Sub hypothesis 12c was NOT REJECTED

d. Statement: There would be no significant difference between the PC scores of individuals in the experimental and control groups with MLBP at 3 months follow-up.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: PC score of experimental and control groups: p = 0.04

Sub hypothesis 12d was REJECTED

Sub hypothesis 13

a. Statement: There would be no significant difference between the TSK scores of individuals in the experimental and control groups with MLBP at baseline.
   Alpha level = 0.05
   Test statistic: Man Whitney U

Conclusion: TSK score of experimental and control groups: p = 0.31

Sub hypothesis 13a was NOT REJECTED

b. Statement: There would be no significant difference between the TSK scores of individuals in the experimental and control groups with MLBP at the end of fifth week.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: TSK score of experimental and control groups: $p < 0.01$

Hypothesis 13b was REJECTED

c. Statement: There would be no significant difference between the TSK scores of individuals in the experimental and control groups with MLBP at the end of tenth week.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: TSK score of experimental and control groups: $p = 0.18$

Hypothesis 13c was NOT REJECTED

d. Statement: There would be no significant difference between the TSK scores of individuals in the experimental and control groups with MLBP at 3 months follow-up.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: TSK score of experimental and control groups: $p < 0.01$

Hypothesis 13d was REJECTED

Sub hypothesis 14

a. Statement: There would be no significant difference between the mean disability index percent of individuals in the experimental and control groups with MLBP at baseline.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: Mean disability index score of experimental and control group: $p = 0.32$

Sub hypothesis 14a was NOT REJECTED

b. Statement: There would be no significant difference between the mean disability index percent of individuals in the experimental and control groups with MLBP at the end of fifth week.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: Mean disability index score of experimental and control group: $p < 0.01$

Sub hypothesis 14b was REJECTED

c. Statement: There would be no significant difference between the mean disability index percent of individuals in the experimental and control groups with MLBP at the end of tenth week.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: Mean disability index score of experimental and control group: $p = 0.03$

Sub hypothesis 14c was REJECTED

d. Statement: There would be no significant difference between the mean disability index percent of individuals in the experimental and control groups with MLBP at 3 months follow-up.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: Mean disability index score of experimental and control group: $p < 0.01$

Sub hypothesis 14d was REJECTED

Sub hypothesis 15

a. Statement: There would be no significant difference between the mean score in the self-efficacy in rehabilitation (SER) scale of individuals in the experimental and control group with MLBP at baseline.

Alpha level = 0.05

Test statistic: Man Whitney U

Conclusion: Mean score in SER scale of experimental and control groups: $p = 0.83$

Sub hypothesis 15a was NOT REJECTED

b. Statement: There would be no significant difference between the mean score in the self-efficacy in rehabilitation (SER) scale of individuals in the experimental and control group with MLBP at the end of fifth week.
Alpha level = 0.05
Test statistic: Man Whitney U

Conclusion: Mean score in SER scale of experimental and control groups: $p < 0.01$

Sub hypothesis 15b was REJECTED

c. Statement: There would be no significant difference between the mean score in the self-efficacy in rehabilitation (SER) scale of individuals in the experimental and control group with MLBP at the end of tenth week.
Alpha level = 0.05
Test statistic: Man Whitney U

Conclusion: Mean score in SER scale of experimental and control groups: $p = 0.93$

Sub hypothesis 15c was NOT REJECTED

d. Statement: There would be no significant difference between the mean score in the self-efficacy in rehabilitation (SER) scale of individuals in the experimental and control group with MLBP at the 3 months follow-up.
Alpha level = 0.05
Test statistic: Man Whitney U

Conclusion: Mean score in SER scale of experimental and control groups: $p < 0.01$

Sub hypothesis 15d was REJECTED

4.3 Discussion

4.3.1 Comparison of demographic and selected clinical variables of Participants in Control and Experimental groups at Baseline of Study

There was no significant difference between the mean ages and onset of MLBP of participants in both groups. There was significant difference in the mean height,

weight, and BMI and sex distribution of participants in the two groups. This shows that the two groups were comparable in age distribution and onset of MLBP but not comparable in anthropometric parameters like height and weight and sex distribution. Differences in sex (Keefe et al, 2000) and age group (Ogunlana et al, 2012b) have been documented as a major determinant of patients' response in certain painful scenarios. Hence the lopsidedness in sex distribution might be a source of misclassification bias for this study. The evenness in age distribution ensured that the EG and CG are comparable. No study known to the researcher has documented the influences of anthropometric parameters like height, weight and BMI on psychosocial variables of patients with MLBP.

4.3.2 Comparison of Selected Pain related and psychosocial variables of Participants in the Experimental and Control Groups at Baseline of Study

There was no significant difference between the mean scores of pain intensity, PC score, TSK score, DIS and SES of participants in the experimental and control groups of this study. This suggests that the two groups are comparable in the above selected pain related and psychosocial variables; and any change observed in these variables at the end of the assessment period could not be due to differences in these variables between the two groups at baseline. The consecutive assignment of participants to the EG and CG ensured even distribution of psychosocial and pain related variables in the research groups. Consecutive assignment of research subjects ensures even numbers of participants in study groups but not necessarily even distribution of extraneous variables (Schulz & Grimes, 2002) hence it is not used in randomized controlled trials.

4.3.3. Changes in Selected Pain related and psychosocial variables of Participants in both groups at baseline, end of 5th week, 10th week and 3 months follow-up.

There were significant decreases in the within-group pain intensity for the participants in the experimental and control groups. This supports the fact that both intervention categories are effective in reducing pain intensity in MLBP. It could also support the popular belief that patients with MLBP will achieve pain relief irrespective of the treatment intervention (Waterschoot et al., 2014, Ehrlich, 2003). Comparing the two groups did not produce significant differences in pain intensity at the end of the study (10th week) but at 3 months follow-up the experimental group had significantly lower pain intensity compared to the control group. This may suggest that the EG were able to cope with pain and its reoccurrence than the control group. Psychological

interventions like PGAP have been said to show short and long term effects on pain intensity (Sturgeon 2014). In this study the lack of significant difference at the end of the intervention may stem from the fact that conventional physiotherapy treatment as practiced in this study employed some unstandardized psychological methods like relaxation techniques, patient education and counselling which is described as the respondent type of cognitive behavioural therapy (Brunner, 2012). Standardized psychological intervention like PGAP have been shown to enhance pain reduction and patient's coping strategy (Sullivan and Adams, 2010), this may explain the significantly low pain intensity score of the ËG at 3 months follow-up as the participants in this reported lesser pain intensity because of increased pain coping ability.

On the effect of PGAP on fear of movement and re-injury (kinesiophobia); there was a significant decrease in the within-group kinesiophobia score for the experimental and control groups and there was significant difference in the between-group kinesiophobia score at the end of the fifth week of treatment but not at the end of the tenth week of treatment. At the end of fifth week of treatment the experimental group had significant reduction in fear of reinjury and movement. This difference was not sustained at the end of the tenth treatment session but was again apparent at 3 months follow-up. Hence findings of the present study suggest that kinesiophobia might resolve earlier when treatment is augmented with PGAP as observed in the experimental group after the fifth week of intervention. This result corresponds with the work of Sullivan and Adams (2010) but the lack of sustained reduction in kinesiophobia by the EG is contrary to the work of Sullivan and Adam (2010). Also the lack of significant difference between the EG and CG on kinesiophobia at 10^{th} week may be explained by the usage of unstandardized psychological techniques (patient counselling) in conventional physiotherapy treatment which the two groups of participants were exposed to. The 10^{th} week of treatment may correspond to the time when patients with MLBP will have pain relieve irrespective of type of intervention (Ehrlich, 2003), hence when there is pain relieve this may translate to reduction in kinesiophobia.

There was significant difference in the within-group and between-group scores of pain catastrophizing. This suggest that conventional treatment may ensure reduction

in pain catastrophizing but addition of PGAP enhanced earlier reduction in pain catastrophizing as the experimental group had significant reduction in pain catastrophizing at the end of the fifth week. Brunner et al. (2012) and Ostelo et al. (2008) in their systematic reviews revealed that using techniques that target psychosocial risk factors like PGAP enhanced reduction in PC thereby reducing pain related disability. Pain catastrophising was not significantly reduced at 10th week of this study in the EG compared to the CG. At 3 months follow-up the participants in the EG had significantly lower extend of catastrophic thinking than the CG. This suggests that their ability to cope with pain may have improved when compared with the CG. Reese & Mittag, (2013) in a systmatic review of psychological interventions like PGAP confirmed the effectivesness of these interventions in improving the coping strategies of patients with LBP.

There was significant difference in the within-group and between-group scores in the disability index score. Sullivan and Adams (2010) had significant reduction in pain related disability in a sample of non-specific LBP patients who received standard treatment augmented with PGAP compared with a sample of non-specific LBP patients who received symptom focused interventions. Brunner et al. 2012 and Ostelo et al. 2008 in their systematic reviews also emphasized that using techniques that target psychosocial risk factors like PGAP enhanced reduction in pain related disability. Results from this present study corroborate the assertions by Sullivan and Adams (2006), Brunner et al. (2012) and Ostelo et al. (2008). There was a decrease in the mean DIS score of both groups; but the experimental group had significantly lower levels of perceived disability throughout the study. Progressive goal attainment programme is an intervention targeting activity limitation and the outcome of this study may be useful as a proof of effectiveness of PGAP.

Painful conditions are associated with reduction in self-efficacy and performance of physical activities (Adegoke & Ezeukwu, 2010; Arnstein, 2000). The results of this study reveal that self-efficacy as measured by the SER questionnaire was significantly different within-group and between-group. Conventional treatment for MLBP increased the functional self-efficacy of the participants significantly as seen in the within-group analysis but the addition of PGAP significantly improved the functional self-efficacy of the participants at the end of the fifth week of intervention. Evidence

has shown that painful conditions reduce self-efficacy, self acceptance and results to activity limitation (Sturgeon, 2014). Participant in the experimental group had significantly better self-efficacy at the fifth session of PGAP intervention and at 3 months follow-up.

4.3.4 Accounting for Research Bias

The outcome of this study may be subject to a number of biases. Firstly, the lack of randomization reduces the external validity of the results. This is evident in the misclassification bias occasioned by the lopsidedness in demographic distribution of the experimental and control groups. It is common to have heterogeneous groups in quasi-experimental design which makes the true experimental design of superior evidence level. Secondly, the researcher was not blinded to the two groups as he was involved in the treatment of the two groups thereby subjecting the outcome of this study to the possibility of a researcher's bias. This may affect the internal validity of the study. The effect of researcher's bias was minimized by ensuring that the participants were blinded to the groups and a trained research assistant took the measurements throughout the study. Thirdly, attrition bias was evident at the three months follow-up with attrition rate of 33%: more than pre-estimated 10% attrition rate. Attrition was almost evenly distributed in the experimental and control groups hence may not have significantly affected the outcome of this study. In spite of these biases, the research concludes that combining standard treatment protocol for MLBP with intervention strategies designed to target psychosocial risk factors for pain and disability may represent one of the most effective approaches in the management of these patients. Routine evaluation of psychosocial risk factors can facilitate identification of patients who are at risk of chronicity, and providing at risk patients with interventions that specifically target these risk factors may prevent the development of chronicity (Sullivan and Adams, 2010). The outcomes of this study revealed that MLBP patients with psychosocial risk factors benefited from the PGAP particularly for the reduction in pain related disability, fear of movement/reinjury, pain catastrophising and improvement of functional self-efficacy.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATIONS

5.1 SUMMARY

The purpose of this study was to investigate the effect of Progressive Goal Attainment Programme (PGAP) combined with conventional treatment on pain intensity, pain catastrophizing, kinesiophobia, disability and self-efficacy in patients presenting with mechanical low back pain (MLBP). The presence of psychosocial risk factors (yellow flags) like heightened level of kinesiophobia, catastrophising, low self-efficacy and increased levels of functional disability alongside pain in MLBP has been shown to explain the progression of acute pain to chronic pain. The use of the biomedical treatment approach alone may not aid the prevention of chronicity of MLBP. The biopsychosocial treatment approach is widely accepted not only because of its sound theoretical and conceptual framework but because of its effectiveness in secondary prevention when used in the management of painful syndromes. The implementation of the biopsychosocial treatment approach has been deterred by the lack of multidisciplinary care, high cost of funding of holistic care and stigma when accessing psychosocial treatment in cultures settings like Nigeria. The introduction of the PGAP as an adjunct that can be administered by any trained rehabilitation clinician may aid the implementation of the biopsychosocial treatment approach in management of MLBP thereby reducing activity limitation and chronicity. Progressive Goal Attainment Programme being an activity based psychosocial intervention can be administered by a trained clinician (this time a Physiotherapist) for patients with painful syndromes presenting with yellow flags. The aim of this present study was to investigate the effectiveness of PGAP as an adjuvant in the management of patients with MLBP who have heightened levels of yellow flags.

A thorough review of related literature was attempted to cover low back pain, its incidence and prevalence, pathophysology, psychoscocial risk factors like

catastrophising, kinesiophobia, self-efficacy, perceived functional disability, and cognitive behavioural interventions. Empirical findings were critically reviewed along major variables of interest in the study. The research design for this study was quasi-experimental. Ethical approval was sought and obtained from UI/UCH Ethics Committee and the Federal Medical Centre Abeokuta Health Research Ethics Committee. Signed informed consents forms of all the participants who were referred for physiotherapy or presented on first contact at the Federal Medical Centre Abeokuta, Ogun State were obtained.

A total number of 70 MLBP patients after screening for eligibility were consecutively assigned to the EG and CG of the study and their baseline data were measured. The control group were given the conventional treatment while the experimental group were given the same conventional treatment alongside PGAP. The age, gender, weight, height, educational status, duration of pain were measured at baseline. Pain intensity score, pain catastrophising score, kinesiophobia score, disability index score and self-efficacy score were measured at baseline, 5^{th} week, 10^{th} week and 3 months follow-up. Data was summarized using descriptive statistics of mean, standard deviation and percentages. Inferential Statistics of Mann-Whitney U and Friedmann ANOVA were used to analyse data. Level of significance was set at p=0.05.

At baseline the mean scores of pain intensity (9.4±0.9; 9.1±0.9); PCS (33.6±9.9; 33.0±5.3), TSK (41.4±7.7; 41.5±2.9); DIS (59.1±12.8; 55.5±12.3); SES (81.4±9.5; 81.2±12.0) for EG and CG respectively were not significantly different. Between group comparison at the end of the 10^{th} week revealed that the mean scores of pain intensity (3.6±1.6; 3.1±1.8), PCS (23.0±9.4; 23.0±8.4); TSK (34.4±6.8; 36.9±3.7), SES (94.4±11.5; 94.1±9.4) for EG and CG respectively were not significantly different but the mean DIS for EG (41.1±8.5) was significantly lower than CG (45.3±7.3). At three-month follow-up EG had significant reduction in mean scores for pain intensity (3.8±1.6; 4.9±1.6); PCS (21.7±9.5; 27.5±5.8), TSK (29.1±6.3; 35.8±6.6); DIS (33.0±6.9; 43.4±7.6); than the CG. Also the EG had significant increase in SES (101.2±11.5; 92.3±9.3) than the CG at three months follow-up.

The outcome of this study revealed that Progressive Goal Attainment Programme is efficacious in achieving sustained reduction in extent of disability when used to augment conventional treatment in patients with Mechanical Low Back Pain.

5.2 CONCLUSIONS

The following specific conclusions were drawn from the findings of the study:

1. Pain intensity, Pain catastrophising, kinesiophobia, self-efficacy and pain-related disability improved in the EG than the CG at 3 months follow-up.
2. Addition of Progressive Goal Attainment Programme to conventional medical and physiotherapy treatment is effective in achieving sustained reduction in perceived disability among patients with mechanical low back pain.

5.3 RECOMMENDATIONS

Based on the findings of this study, the following recommendations are proposed:

1. Progressive goal attainment programme should be incorporated into treatment for patients with mechanical low back pain with psychosocial overlay.
2. Further studies may be necessary to adapt the PGAP intervention so that it will be easy to administer in the clinic.

REFERENCES

Abenheim, L. Rossignol, M. Valat, J.P. Nordin, M. Avovac Blotman, F. Charlot, J. Dreiser R.L. Legrand, E. Rozenberg, S. and Vautravers, A. 2000. The role of activity in the therapeutic management of back pain. Report of the international Paris Task force on back pain *Spine* 25: 15-335.

Adegoke, B.O.A and Ezeukwu, A.O. 2010. Pain intensity, self-efficacy and physical performance in patient with chronic low back pain. *International Journal of Therapy and Rehabilitation* , Vol.17 No.10.

Airaksinen O, Brox JI, Cedraschi C, Hildebrandt J, Klaber-Moffett J, Kovacs F, Mannion AF, Reis S, Staal JB, Ursin H and Zanoli G, 2006. COST B13 Working Group on Guidelines for Chronic Low Back Pain. Chapter 4. European guidelines for the management of chronic nonspecific low back pain. *European Spine Journal* ;15 Suppl 2:S192–S300.

Akinbo S.R.A, 2014. Unseen but felt: The torment of a painful back. University of Lagos Inaugural Lecture series. *Unilag Press and Bookshop Ltd*.

Allan, D.B and Waddell, G.1989. An historical perspective on low back pain disability. *Acta Orthopaedic Scandinavian :* Suppl. 234: 1-23.

Altmaier, E.M, Lehmann, T.R, Russell, D.W, Weinstein, J.N and Feng Kao C. 1992. The effectiveness of psychological interventions for the rehabilitation of low back pain: a randomized controlled trial evaluation. *Pain*.**49**:329–35.

Anderson, K.O. Dowds, B.N. Robyn, P.E. Edwards, W.T and Peeters-Asdoyuriam, C. 1995. Development and initial validation of a scale to measure self-efficacy beliefs in patients with chronic pain. *Pain* 63(1): 77-84.

Andersson, G.B.1999. Epidemiologic features of chronic low back pain. *Lancet* Aug 14;354 (9178): 581-5.

Apts, D.W.1992. Back Injury and Prevention Handbook. *Lewis Publishers*. Chelsea, 50-55.

Arnstein, P. 2000. The mediation of disability by self-efficacy in different samples of chronic pain patients. *Disability and Rehabilitation*. 22(17): 794-801.

Azize, K., Sultan, K., Aysel, A and Nevin, D. 2009. Low Back Pain Prevalence and Associated Risks Factors among Hospital Staffs. *Journal of Advanced Nursing*. 65(3), 516-524.

Baker, T.A and Green C.R. 2005. Intra-race Differences Among Black and White Americans Presenting for Chronic Pain Management: The Influence of Age, Physical Health, and Psychosocial Factors. *Pain Medicine*: Vol.6 (1).

Bandura, A. 1986. *Social foundations of thought and action: A social cognitive theory*. Prentice hall, Englewood cliffs: 19-26.

Bandura, A. 1997. Insights. Self-efficacy. *Havard mental health letter*, 13(9), 4-6. Retrieved from CINAHL.

Basler, H.D, Jäkle, C and Kröner-Herwig B. 1997. Incorporation of cognitive-behavioural treatment into the medical care of chronic low back patients: a controlled randomized study *Patient Education & Counseling* 1997;**31**:113–24.

Beaton, D.E. Bombardier, C. Guilleminm F and Ferraz, M.B. 2000. Guidelines for the Process of cross-cultural Adaptation of self-report measures. *Spine* Vol.25:24, 3186-3191.

Beck, A, Rush, J, Shaw, B and Emery G.1979. *Cognitive therapy of depression*. New York: The Guilford Press

Beck, A.T. Emery, G and Greenberg, R.L. 1985. *Anxiety disorders and phobias*. New York: Basic Books.

Beckman, J.C. Keefe, F.J. Caldwell, D.S and Roodman, A.A. 1991. Pain coping strategies in rheumatoid arthritis: Relationships to pain, disability, depression and daily hassles. *Behavioural Therapy* :22 (113-124).

Beecher, H.K. 1959. *Measurement of subjective responses*. New York: Oxford University Press.

Borenstein, D.G and Wiessel, S.W. 1989. *Medical Diagnosis and Comprehensive Management of Low Back Pain*. Philadelphia, W.B. Saunders 34, 46-58, 551-570.

Brox, J.I, Sørensen, R, Friis A, Nygaard, Ø, Indahl, A, Keller, A, Riise, R and Reikerås, O 2003. Randomized clinical trial of lumbar instrumented fusion and cognitive intervention and exercises in patients with chronic low back pain and disc degeneration. *Spine* **28**(17):1913–21.

Brunner, E, De Herdt, A, Minguet P, Baldew, S and Probst M. 2012. Can cognitive behavioural therapy based strategies be integrated into physiotherapy for the prevention of chronic low back pain? A systematic review. *Disability & Rehabilitation*, DOI: 10.3109/09638288.

Burton, A.K., Tillotson, K.M., Main, C.J and Hollis, S. 1995. Psychosocial predictors of outcome in acute and sub acute low back trouble. *Spine*, 20, 722-728.

Bush, C, Ditto, B and Feuerstein, M. A 1985. Controlled evaluation of paraspinal EMG biofeedback in the treatment of chronic low back pain. Health Psychology **4**:307–21.

Chaves, T.S and Brown, J.M. 1987. Spontaneous cognitive strategies for the control of clinical pain and stress. *Journal of Behavioural Medicine* ;10:263-76.

Chou, R and Huffman, L.H. 2007. Guideline for the evaluation and management of low back pain: Evidence Review. American Pain Society Glenview. chronic

low back pain: a randomised controlled trial. *European Spine Journal*. **17**:1193–200.

Cicinelli, L. 1996. Back pain, In tuner, A; Foster, M. and Johnson, S.E 1996. 4th Ed., *Occupational Therapy and Physical Dysfunction*. Edinburg. Churchill Livingstone, pg 67-69.

Colliton, J. 1996. Back Pain and Pregnancy: Active management strategies. *The Physician and Sports medicine*. (24), 7, 1-7.

Cote, P. Hogg-Johnson, S. Cassidy, J.D. Carrol, L and Frank, J.W. 2001. The association between neck pain intensity, physical functioning, depressive symtomatology and time-to-claim-closure after whiplash. *Journal of Clinical Epidemiology*. 54(3):275-286.

Crombez, G. Vlaeyen, J.W. Heuts, P.H and Lysens, R. 1999. Pain related fear is more disabling than pain itself: evidence on the role of pain-related fear in chronic back pain disability. *Pain;* 80:329-39.doi:10.1016/s0304-3959 (98)00229-2.

Davidson, M and Keating, J.A. 2002. Comparison of five low back disability questionnaires reliability and responsiveness. *Physical therapy* 82:8-24.

Delitto A, George S.Z,Van Dillen L, Whitman J.M, Sowa G, Shekelle P, Denninger T.R and Godges J.G. 2012. Low Back Pain:Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopaedic Section of the American Physical Therapy Association. *Journal of Orthopaedics and Sports Physical Therapy*: 42(4):A1-A57. doi:10.2519/jospt.0301

Deyo, R.A and Bass, J.E. 1989. Lifestyle and low back pain: the influence of smoking and obesity. *Spine*. 14: 501-506.

Deyo, R.A. Mirza, S. and Martin, B. 2006. Back pain prevalence and visit rates: estimate from US National Surveys, 2002. *Spine* 31: 2724-27.

Deyo, R.A and Weistein, J.N. 2001. Low back pain. *New England Journal of Medicine*. 344 (5), 363-370.

Deyo, R.A and Tsui-Wu, Y. 1987. Descriptive epidemiology of low back pain and its related medical care in the United States. *Spine*.12:264-268.

Dillingham T.1995. Evaluation and management of low backpain: A overview. *State of the art reviews* 9(3):559–74.

Donaldson, S, Romney, D, Donaldson, M and Skubick D. 1994. Randomized study of the application of single motor unit biofeedback training to chronic low back pain. *Journal of Occupational Rehabilitation*. **4**:23–37.

Ehrlich, G. E. 2003. Low back pain, *Bulletin of WHO*:81(03), 671–676.

Ellis, A. 1962. *Reason and emotion in psychotherapy*. New York: Lyle Stuart.

Engel GF, 1977. The need for a new medical model: a challenge for biomedicine. *Science*. ;196:129-36.

Fairbank J, FrostH, Wilson-MacDonald J, Yu LM, Barker K and Collins R, 2005. Spine Stabilisation Trial Group. Randomised controlled trial to compare surgical stabilisation of the lumbar spine with an intensive rehabilitation programme for patients with chronic low back pain: the MRC spine stabilisation trial. *British Medical Journal*. **330**(7505):1233–9.

Fairbank, J.C.T and Pynsent, P.B. 2000. The Oswestry Disability Index. *Spine*. 25(22):2940-2953.

Fast, A. Weiss, L and Parikh, S. 1989. Night backache in pregnancy: hypothetical pathophysiological mechanisms. *American Journal of Physical Medicine and Rehabilitation*. 68 (5), 227-229.

Ferster CB and Skinner BF, 1957. *Schedules of reinforcement*. New York: Appleton-Century-Crofts, Inc.

Feurstein, M and Beattie, P. 1995. Biobehavioral factors affecting pain and disability in low back pain: mechanisms and assessments. *Physical Therapy*. 75, 267-280.

Foster NE and Delitto A. 2011. Embedding psychosocial perspectives within clinical management of low back pain: integration of psychosocially informed management principles into physical therapist practice–challenges and opportunities. *Physical Therapy* ;91:790–803.

Foster, L. 2001. Botulinium toxin and chronic low back pain: A randomized double blind study. *Neurology*. 56, 1290-1293.

Friedrich M, Gittler G, Halberstadt Y, Cermak T and Heiller I. 1998. Combined exercise and motivation program: effect on the compliance and level of disability of patients with chronic low back pain: a randomized controlled trial. *Archives of Physical Medicine and Rehabilitation* :**79**:475–87.

Frymoyer, J.W. 1992. Can low back disability be prevented? In : Nordin, M., Vischer, T.L. (eds) *Common Low back Pain: Prevention of Chronicity*. London, Bailere Tindall.38-46.

Frymoyer, J.W. 1998. Back pain and sciatic. *New England Journal of Medicine*. 318:291-300.

Gage, M. and Polatojko, H.J. 1994. Enhancing occupational performance through an understanding of perceived self-efficacy *American Journal of Occupational Therapy*: 48 (5): 452-61.

Gatchel RJ, Polatin PB, Noe C, Gardea M, Pulliam C and Thompson J, 2003. Treatment- and cost-effectiveness of early intervention for acute lowback pain patients: a one-year prospective study. *Journal of Occupational Rehabilitation* 2003; 13:1–9.

Gatchel, R. J. 2004a. Award for Distinguished Professional Contributions to Applied Research. *American Psychologist*, 59, 794–805.

Gatchel, R. J. 2004b. Comorbidity of chronic pain and mental health: The biopsychosocial perspective. *American Psychologist*: 59, 792–794.

Gatchel, R. J. 2005. *Clinical essentials of pain management*. Washington,DC: American Psychological Association.

George, S.Z. Zeppieri, G. J. Cere, A.L. Cere, M.R. Borut, M.S. and Hodges, M.J. 2008. A randomized trial of behavioural physical therapy interventions for acute and subacute low back pain. (NC00373867). *Pain* 140:145-67.

Goubert, L. Crombez, G. and Van Damme, S. 2004. The role of neuroticism, pain catastrophizing and pain-related fear in vigilance to pain: a structural equations approach. *Pain*; 107:234-41.

Goubert, L. Francken, G. Crombez, G. Vansteenwegen, D. and Lysens, R. 2002. Exposure to physical movement in chronic back pain patients: no evidence for generalization across different movements. *Behavioural Research Therapy*; 40:415-29.

Gracovetsky, S. 1981. The mechanism of the lumbar spine. *Spine* 3, (6): 249-262.

Hay, E.M. Mullis. R. Lewis, M. Volora, K. Main, C. Watson, P.J. Dziedzic KS, Sim J, Minns Lowe C and Croft PR. 2005. Comparison of physical treatments versus a brief pain management programme for back pain in primary care: a randomised clinical trial in physiotherapy practice. *Lancet* 365:2024-30.

Henschke N, Ostelo RW, van Tulder MW, Vlaeyen JW, Morley S, Assendelft WJ and Main CJ. 2010. Behavioural treatment for chronic low-back pain. *Cochrane Database* Syst Rev;7

Hlobil H, Staal JB, Twisk J, Köke A, Ariëns G, Smid T and van Mechelen W. 2005. The effects of a graded activity intervention for low back pain in occupational health on sick leave, functional status and pain: 12-month results of a randomized controlled trial. *Journal of Occupational Rehabilitation* **15**:569–80.

Hoogendorn, W.E., Van Poppel, M.N., Bongers, P.M., Koes, B.W. and Bouter L.M. 1999. Physical load during work and leisure time asrisk factors for back pain. *Scandinavian Journal of work environment and health* 25(5), 387-403.

Horng, Y.S. Hwang, Y.H. Wu, H.C. Liang, H.W. Jang, Y. Twu, F.C and Wang J.D. 2005. Predicting health related quality of life in patients with low back pain. Spine,30:551r.-5.
in German pain treatment centers. *Patient Education & Counseling:* **31**:113–24.

Johnson, R.E, Jones, G.T, Wiles, N.J, Chaddock, C, Potter, R.G, Roberts, C, Symmons, D.P, Watson, P.J, Torgerson, D.J and Macfarlane, G.J. 2007. Active exercise, education, and cognitive behavioral therapy for persistent disabling low back pain: a randomized controlled trial. *Spine.* **32** (15):1578–85.

Johnstone R, Donaghy M and Martin D, 2004. A pilot study of a cognitive–behavioural therapy approach to physiotherapy, for acute low back pain patients, who show signs of developing chronic pain. *Advanced Physiotherapy* :2:182–188.

Jousset N, Fanello S, Bontoux L, Dubus V, Billabert C, Vielle B, Roquelaure Y, Penneau-Fontbonne D and Richard I 2004. Effects of functional restoration versus 3 hours per week physical therapy: a randomized controlled study. *Spine* **29**(5):487–93.

Kääpä EH, Frantsi K, Sarna S and Malmivaara A. 2006. Multidisciplinary group rehabilitation versus individual physiotherapy for chronic nonspecific low back pain: a randomized trial. *Spine* **31**:371–6.

Keefe, F. J., Lefebvre, J. C., Egert, J. R., Af, G., Sullivan, M. J and Caldwell, D. S. 2000. The relationship of gender to pain , pain behavior , and disability in osteoarthritis patients : the role of catastrophizing. *Pain*: 87, 325–334.

Keefe, F.J. Caldwell, D.S. Queen, K.T. Gil, K.M. Martinez S. Crisson, J.E. Ogden, W. and Nunley, J.1987. Pain coping strategies in osteoarthritis patients. *Journal of Consulting and Clinical Psychology* 55:208-212.

Kendall, N A S, Linton, S J and Main, C J 1997. *Guide to Assessing Psycho-social Yellow Flags in Acute Low Back Pain: Risk Factors for Long-Term Disability and Work Loss.* Accident Compensation Corporation and the New Zealand Guidelines Group, Wellington, New Zealand. (Oct, 2004 Edition).

Key, J. 2010. The problem of back pain. *Back pain: A movement problem.* A clinical approach incorporating relevant research and practice. 1st ed. Elsevier UK Chapter 2 pg. 4-15.

Koes, B.W., Van Tulder, M.W and Thomas, S. 2006. Diagnosis and treatment of low back pain. *British Medical Journal*. 332 (1555): 1430-4.

Kole-Snijders, A.M.J., Vlaeyen, J.W.S., and Goossens, M.E.J.B.1999.Chronic low back pain: what does cognitive coping skills training add to operant behavioral treatment? Results of a randomized clinical trial. *Journal of Consulting and Clinical Psychology*: **67**: 931–44.

Kool, J.P, Oesch, P.R, Bachmann, S, Knuesel, O, Dierkes, J.G, Russo, M, de Bie, RA and van den Brandt PA 2005.Increasing days at work using function centered rehabilitation in non-acute nonspecific low back pain: a randomized controlled trial. *Archives of Physical Medicine & Rehabilitation*. **86**:857–64.

Kori, S. Miller, R and Todd, D. 1990. Kinesiophobia: A new view of chronic pain behaviour. *Pain management*. (Jan):35-43.

Kovacs, F.M., Abiara, V., Zamora, J. and Fernandez, C. 2005. The Spanish Back Pain Research Network. The transition from acute to sub-acute chronic low back pain. A study based on determinants of quality of life and prediction of chronic disability. *Spine:* 30: 1786-1792.

Lamb SE, Hansen Z, Lall R, Castelnuovo E, Withers EJ, Nichols V, Potter R and Underwood MR. 2010. Group cognitive behavioural treatment for low-back pain in primary care: a randomised controlled trial and cost-effectiveness analysis. *Lancet* **375**:916–23.

Lebourt-Yde, C., Lawrisen, M.J. and Lauristen, T. 1997. Why has the search for causes of low back pain being largely inconclusive. *Spine*. 22(8): 881-7.

Lebovits A. 2005. The Ethical Implications of Racial Disparities in Pain: Are Some of Us More Equal? *Pain Medicine* Vol. 6(1).

Leeuw M, Goossens MEJB, van Breukelen GJP, de Jong JR, Heuts PHTG, Smeets RJEM, Köke AJ and Vlaeyen JW. 2008. Exposure in vivo versus operant graded activity in chronic low back pain patients: results of a randomized controlled trial. *Pain*. **138**:192–207.

Leeuw, M., Goossens, M.E., Linton, S.J., Crombez, G., Boeersma, K. and Vlaeyen, J.W. 2007. The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *Journal of Behavioural Medicine:* 30(1):77-94.

Lefort, S.M., Gray-Donald, K., Rowat, K.M and Jeans, M.E. 1998. Randomized controlled trial of a community based psychoeducation program for the self management of chronic pain. *Pain:* 74:297-306. doi:10.1016/S0304-3959(97)00190-5.

Lindström I, Öhlund C, Eek C, Wallin L, Peterson LE, Fordyce WE and Nachemson AL. 1992. The effect of graded activity on patients with subacute low back pain: a randomized prospective clinical study with an operant-conditioning behavioural approach. *Physical Therapy*: **72**(4):279–90.

Linton SJ and Andersson T. 2000. Can chronic disability be prevented? A randomized trial of a cognitive-behavioral intervention and two forms of information for patients with spinal pain *Spine*; **25**(21):2825–31.

Linton SJ, Boersma K, JanssonM, Overmeer T, Lindblom K and Vlaeyen JWS. 2008. A randomized controlled trial of exposure in vivo for patients with spinal pain reporting fear of work related activities. *European Journal of Pain*. **12**:722–30

Linton SJ, Bradley LA, Jensen I, Spangfort E and Sundell L.1989. The secondary prevention of low back pain: a controlled study with follow-up. *Pain*. **36**:197–207.

Linton, S.J. 2002. New avenues for the prevention of chronic musculoskeletal pain and disability. *Journal of Amsterdam*: Elservier. 3467(07)60096-2, doi:10.1016/S0899.

Linton, S.J., Buer, N., Vlaeyen, J. and Hellsing A. 1998. Are fear-avoidance beliefs related to the inception of an episode of back pain? A prospective study. *Psychology and Health*; 14:1051-9.

Loeser, J. D. 1982. Concepts of pain. In J. Stanton-Hicks & R. Boazb(Eds.), *Chronic low back pain* (pp. 109–142). New York: Raven Press.

Louw, Q.A., Morris, L.D. and Grimmer-Somers K. 2007. The prevalence of low back pain in Africa: a systemic review. *BMC Muscukoskeletal Disorders* 8:105.

Low, J. and Reed, A. (1999). *Electrotherapy Explained*. 4th edition, Oxford, Butterworth Heinemann. 179-219.

Lundberg, M.K.E., Styf, J. and Carlsson, S.G. 2004. A psychometric evaluation of the Tampa Scale for Kinesiophobia- from a physiotherapeutic perspective. *Physiotherapy Theory and Practice*. 20:121-133.

MacCarthy, S.M., Stark, D.D. and Filly, R.A. 1985. Obstetrical magnetic resonance imaging: maternal anatomy. *Radiology*. 154, (2), 421-425.

Macgregor, A. and Manek, C. 2005. Structural, Psychological and Risk factors of low back pain. *Spine*: 59(2), 160-167.

Magnussen L, Rognsvåg T, Tveito TH and Eriksen HR. 2005. Effect of a brief cognitive training programme in patients with long-lasting back pain evaluated as unfit for surgery. *Journal of Health Psychology*. **10**:233–43.

Main C.J and George S.Z. 2011. Psychologically Informed Practice for Management of Low Back Pain: Future Directions in Practice and Research. *Physical Therapy*; 91:820-824.

Main, C., Sullivan, M.J.L. and Watson P.J. 2007. *Pain Management: practical applications of the biopsychosocial perspective in clinical and occupational settings*. Second ed. edinburg, Scotland.:Churchill Livingstone.

Maitland J. 1986. *Vertebral Manipulation*. London: Butterworths.

Mangels, M, Schwarz, S, Worringen, U, Holme, M and Rief, W. 2009.Evaluation of a behavioral-medical inpatient rehabilitation treatment including booster sessions: A randomized controlled study. *Clinical Journal of Pain*. **25**:356–64.

Marini, J.C. 2001. Genetic risk factors for lumbar disc disease. *Journal of American Medical Association*. 285, 1886-1888.

Marras, W.S., Lavender, S.A. and Leurgans, S.E. 1993. The role of dynamic three-dimensional trunk motion in occupational related back disorders. *Spine* 18:617-628.

Mayer, T. J. 1989. Function restoration for patients with chronic low back pain: in Weistein J.D and Wiesel S.W (1990): *The Lumbar Spine*. Philadelphia, WB Saunders Company. 336-340.

McCauley JD, Thelen MH, Frank RG,Willard RR and Callen E. 1983.Hypnosis compared to relaxation in the outpatient management of chronic low back pain. *Archives of Physical Medicine and Rehabilitation*. **64**:548–52.

Mckenzie R. 1981. *The lumbar spine: Mechanical diagnosis and treatment*. Waikanae New Zealand: Spinal Publications.

Mckenzie R, 2010 Treat your own back New Waikanae New Zealand: Spinal Publications.

Melzack R. 1999. From the gate to the neuromatrix. *Pain*. 6:S121-S126.

Melzack, R. and Wall P. 1965. Pain mechanisms: a new theory. *Science*; 150:971-979.

Menzel NN and Robinson ME. 2006. Back pain in direct patient care providers: early intervention with cognitive behavioural therapy. *Pain Management Nursing*. **7**:53–63.

Merskey, H. and Bogduck, N. 1994. *Classification of Chronic Pain, Descriptions of Chronic Pain Syndromes and Definitions of Pain Terminology.* 2nd Ed, Seattle. IASP Press, 11-12.

Moore JE, Von Korff M, Cherkin D, Saunders K and Lorig K. 2000. A randomized trial of a cognitive-behavioral program for enhancing back pain self care in a primary care setting. *Pain*. **88**:145–53.

Moore, L.K and Dalley, F.A, 1999. *Clinically Oriented Anatomy* 4th Edition. Lippincott Williams and Wilkins 432.

Morley, S., Eccleston, C. and Williams, A. 1999. A systemic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and

behaviour therapy for chronic pain in adults, excluding headache. *Pain* 80: 1-13, doi: 10.1016/S0304-3959(98)00255-3.

Nachemson, A.L. 1992. Newest knowledge of low back pain: A critical look. Clinical Orthopeadics. 279: 8-20.

Nelson, M.A. 1992. Surgery, an overview. In Jayson M.I 1992: *The Lumbar Spine and Back Pain*. 4th Ed, Churchill Livingstone. 387-405.

Newcomer KL, Vickers Douglas KS, Shelerud RA, Long KH and Crawford B, 2008. Is a videotape to change beliefs and behaviors superior to a standard videotape in acute low back pain? A randomized controlled trial. *Spine Journal* ;8:940–947.

Newton-John TR, Spence SH and Schotte D. 1995. Cognitive behavioural therapy versus EMG biofeedback in the treatment of chronic low back pain. *Behavioural Research Therapy*. **33**:691–7.

Nicassio, P.M., Schoenfeld-smith, K., Radojevic, V. and Schuman C. 1995. Pain coping mechanisms in fibromyalgia: Relationship to pain and functional outcomes. *Journal of Rheumatology*. 22:1552-1558.

Nicholas M.k, Linton S.J, Watson P.J and Main C.J. The 'Decade of the Flags' working group. 2011. Early Identification and Management of Psychological Risk Factors ("Yellow Flags") in Patients With Low Back Pain: A Reappraisal. *Physical Therapy*; 91:737-753.

Nicholas MK, Wilson PH and Goyen J. 1991. Operant-behavioural and cognitive behavioural treatment for chronic low back pain. *Behavioural Research Therapy*. **29**:225–38.

Nicholas MK, Wilson PH and Goyen J. 1992. Comparison of cognitive behavioral group treatment and an alternative non-psychological treatment for chronic low back pain. *Pain*.**48**:339–47.

Nouwen A. 1983. EMG biofeedback used to reduce standing levels of paraspinal muscle tension in chronic low back pain. *Pain*.**17**:353–60.

Nwuga, V.C. and Egwu, M.O. 1999. *Back Pain: Causes, Treatment and Prevention*: clean print ventures. 1st Ed; Pg 6-12.

O'Sullivan P, 2005. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as an underlying mechanism. *Manual Therapy*. ;10:242-55.

Odebiyi, D.O. 2004. *How to avoid back pain at work and rest – A handbook of the Nigerian Back School. Lagos;* Crowntex Printing Press, pg. 1-20.

Odole, A. C., and Akinpelu, A. O. 2009. Translation and Alternate Forms Reliability of the Visual Analogue Scale in the Three Major Nigerian Languages. *The internet Journal of Allied Health Sciences and Practice* 7(3), 1–7.

Odole, A.C, Akinpelu A.A, Adekanla B. A and Obisanya O.B. 2012. Economic Burden of Low Back Pain on Patients Seen at the Outpatient Physiotherapy Clinics of Secondary and Tertiary Health Institutions in Ibadan. *Journal of the Nigeria Society of Physiotherapy:* 18 & 19 pg 44-48.

Ogunlana M.O, Odunaiya N.A, Dairo M.D and Ihekuna O 2012. Predictors of health-related quality of life in patients with non-specific low back pain. *African Journal of Physiotherapy and Rehabilitation Sciences* Vol. 4, Nos. 1 & 2, June, pp. 15 – 23.

Ogunlana, M. O., Davis, O. A., Thanni, A. I., Lateef, R. O., Solabi, A., Alimi, T. J., and Olasore, O. D. 2012b. Determinants of Low Back Pain Disability Among Hospital Workers, *Journal of the Nigeria Society of Physiotherapy:* 20, 15–20.

Omokhodion, F.O. 2002. Low back pain in a rural community in South Western Nigeria. *West African Journal of Medicine*. 21(2), 87-90.

Omokhodion, F.O. and Sanya, A.O. 2003. Risk factors for low back pain in office Workers. *Journal of Occupational Medicine* 53**:**287–289.

Ostelo, R.W.J.G. Van Tulder, M.W, Vlaeyan, J.W.S. Linton, S.J. Morley, S. and Assendelft, W.J.J. 2008. Behavioural treatment for chronic low-back pain (Review) *The Cochrane Collaboration* http://www.thecochranelibrary.com

Parker, J.C., Smarr, K.L., Buesher, K.L., Phillips, L.R., Frank, R.G., Beck, N.C., Anderson, S.K. and Walker, S.E. 1989. Pain Control and rational thinking: Implications for rheumatoid arthritis *Arthritis and Rheumatology*. 32 (984-990).

Petersen LK, Vogel I, Agger AO, Westergård J, Nils M and Uldbjerg N. 1995. Variations in serum rellaxin (nRLX-2). *Acta Obstetrics and Gynecology Scandanavian.* Apr;74(4):251-6

Picavet, H.S., Vlaeyen, J.W and Schouten, J.S. 2002. Pain catastrophizing and kinesiophobia: predictors of chronic low back pain. *American Journal of Epidemiology*. 156(11):1028-1034.

Pincus, T., Burton, A.K., Vogel, S and Field, A.P. 2002. A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine* ;27(5):E109-120.

Poole H, Glenn S and Murphy P. 2007. A randomised controlled study of reflexology for the management of chronic low back pain. *European Journal of Pain*. **11**:878–887.

Reese, C and Mittag, O. 2013. Psychological interventions in the rehabilitation of patients with chronic low back pain: evidence and recommendations from systematic reviews and guidelines. *International Journal of Rehabilitation Research*.: 36(1), 6–12. doi:10.1097/MRR.0b013e32835acfec

Richardson CA and Jull GA 1995. Muscle control – pain control. What exercises should you prescribe. *Manual Therapy* 1(1):2-10.

Rogerson MD, Gatchel RJ and Bierner SM. 2010. A cost utility analysis of interdisciplinary early intervention versus treatment as usual for high-risk acute low back pain patients. *Pain and Practice* ;10:382–395.

Rose MJ, Reilly JP, Pennie B, Bowen-Jones K, Stanley IM and Slade PD. 1997. Chronic low back pain rehabilitation programs: a study of the optimum duration of treatment and a comparison of group and individual therapy. *Spine* **22**:2246–53.

Rosenstiel, A.K. and 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.

Sahrmann SA, 2002. *Diagnosis and Treatment of Movement Impairment Syndromes.* St. Louis: Mosby.

Sanders SH. 2002. Operant conditioning with chronic pain: back to basics. In: Turk DC, Gatchel RJ. *Psychological approaches to pain management: a practitioner's handbook.* 2nd ed. New York: The Guilford Press, p128–137.

Schulz, K. F and Grimes, D. A. (2002). Epidemiology series Allocation concealment in randomised trials : defending against deciphering. *Lancet:* 359, 614–618.

Schweikert B, Jacobi E, Seitz R, Cziske R, Ehlert A, Knab J and Leidl R. 2006. Effectiveness and cost-effectiveness of adding a cognitive behavioral treatment to the rehabilitation of chronic low back pain. *Journal of Rheumatology* **33**: 2519–26.

Smeets RJEM, Vlaeyen JWS, Hidding A, Kester ADM, van der Heijden GJMG, van Geel ACM and knotterus JA. 2006. Active rehabilitation for chronic low back

pain: cognitive behavioral, physical, or both? First direct post-treatment results from a randomized controlled trial. *BMC Musculoskeletal Disorders* **7**:5.

Smith, B.H., Elliot, A.M., Chambers, W.A., Hannaford, P.C. and Penny, K. 2001. the impact of chronic pain in the community. *Family practice.* 18(3): 292-9.

Spinhoven, P., Ter Kuile, M., Kole-Snijders, A.M., Hutten Mansfield, M., Den Ouden, D.J., and Vlaeyen, J.W. 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.

Steenstra IA, Anema JR, Bongers PM, de Vet HCW, Knol DL and van Mechelen W. 2006. The effectiveness of graded activity for low back pain in occupational healthcare. *Occupational and Environmental Medicine.* **63**:718–725.

Steenstra IA, Anema JR, van Tulder MW, Bongers PM, de Vet HC and van Mechelen W. 2006. Economic evaluation of a multi-stage return to work program for workers on sick-leave due to low back pain. *Journal of Occupational Rehabilitation*; 16:557–578.

Storheim K, Brox JI, Holm I, Koller AK and Bo K. 2003. Intensive group training versus cognitive intervention in sub-acute low back pain: short-term results of a single-blind randomized controlled trial. *Journal of Rehabilitation Medicine*; 35:132–140.

Strong J. 1998. Incorporating cognitive-behavioural therapy with occupational therapy: a comparative study with patients with low back pain. *Journal of Occupational Rehabilitation:*. **8**:61–71.

Strong, J. 1995. *Self-efficacy and the patients with chronic pain* In: Schacklock M(ed). Moving in on pain. Butterworth-Heinemann, Melbourne.

Stuckey SJ, Jacobs A and Goldfarb J. 1986. EMG biofeedback training, relaxation training, and placebo for the relief of chronic back pain. *Perception and Motor Skills* **63**:1023–36.

Sturgeon, J. A. 2014. Psychological therapies for the management of chronic pain, *Psychology Research and Behavioural Management*: 7,115–124.

Sullivan, L.M. 2012. Power and sample size determination. *Essentials of biostatistics in public health*. Ontario , Jones and Bartlett Learning, pg169-188.

Sullivan, M.J.L and Neish N. 1999. The effects of disclosure on pain during dental hygiene treatment: the moderating role of catastrophizing. *Pain*: 79(2-3):155-163.

Sullivan, M.J.L. 2003. Emerging trends in the secondary prevention of pain-related disability. *Clinical Journal of pain* ;19:77-79.

Sullivan, M.J.L. 2006. Psychology of Pain: Assessment of Cognitive Variables. In R.F. Schmidt and W.D. Willis (Eds.). *The encyclopedic Reference of Pain*. New York: Springer.

Sullivan, M.J.L. 2008. Toward a biopsychomotor conceptualisation of pain. Clin J *Pain*.24:282-290.

Sullivan, M.J.L. 2010. *Progressive Goal Attainment Program*. Treatment manual 3rd edition.

Sullivan, M.J.L and Adams, H. 2010. Psychosocial Treatment Techniques to augment the impact of physiotherapy interventions for low back pain. *Physiotherapy Canada*. 62: 180-189.

Sullivan, M.J.L., Adams, H., Rhodenizer, T and Stanish, W.D. 2006. A psychosocial risk factor-targeted intervention for the prevention of chronic pain and disability following whiplash injury. *Physical Therapy:*.86:8-18.

Sullivan, M.J.L., Bishop, S and Pivik, J. 1995. The pain catastrophizing Scale: Development and validation. *Psychology Assessment*. 7:524-532.

Sullivan, M.J.L., Rodgers, W.M., Wilson, P.M., Bell, G.J., Murray, T.C and Fraser, S.N. 2002. An eperimental investigation of the relation between catastrophizing and activity intolerance. *Pain* 100(1-2):47-53.

Sullivan, M.J.L., Stanish, W.D., Waite, H., Sullivan, M and Tripp, D.A. 1998. Catastrophizing, pain and disability in patients with soft tissue injuries. *Pain* 77(3):253-260.

Sullivan, M.J.L., Thorn, B., Haythornwaite, J.A., Keefe, F., Martin, M., Bradley, L.A and Lefebvre, J.C. 2001. Theoretical perspectives on the relation between catastrophizing and pain. *Clinical Journal of Pain*. 17(1):52-64.

Swezey, R.L and Petrocelli, A.R. 1992. Conservative Treatment of Back pain. In Jayson M.I (1992) T*he lumbar spine and back pain* 4th Ed. Churchill Livingstone. 387-405.

Swinkels-Meewisse, E., Swinkels, R., Verbeek, A., Vlaeyen, J and Oostendorp, R. 2003. Psychometric properties of the Tampascale for Kinesiophobia and the fear-avoidance beliefs for questionnaire in acute low back pain. *Manual Therapy* 8(1):29-36.

Syrjala KL and Abrams JR.2002. Hypnosis and imagery in the treatment of pain. In: Turk DC, Gatchel RJ. *Psychological approaches to pain management: a practitioner's handbook.* 2nd ed. New York: The Guilford Press, p 187–209.

Taimela, S., Kujala, U.M., Salminen, J.J and Vijamen, T. 1997. Prevalence of low back pain among children and adolescents: a nationwide, cohort-based questionnaire survey in finland. *Spine*. 22(10) 1132-1136.

Tinetti, M.E., Richman, D and Powell, L. 1990. Falls efficacy as a measure of fear of falling. *Journal of Gerontology*. 45:P239-P243.

Tomassen, P.D., Post, M.W and Van Asbeck, F.W. 2000. Return to work after spinal cord injury. *Spinal Cord* 38: 51-55.

Turk, D. C and Monarch, E. S. 2002. Biopsychosocial perspective on chronic pain. In D. C. Turk & R. J. Gatchel (Ed.), *Psychological approaches to pain management: A practitioner's handbook* (2nd ed., pp. 3–30). New York: Guilford Press.

Turk, D., Meichenbaum, D and Genest, M. 1983. *Pain and behavioural Medicine: a cognitive-behavioural-perspective. NewYork*: Guilford doi:10.1080

Turk, D.C. 2002. A diathesis-stress model of chronic pain and disability. *Pain Research Management*.;7;9-19.

Turner JA, Clancy S, McQuade KJ and Cardenas DD. 1990. Effectiveness of behavioral therapy for chronic low back pain: a component analysis. *Journal of Consulting and Clinical Psychology*. **58**: 573–9.

Turner JA and Clancy S. 1988. Comparison of operant behavioural and cognitive-behavioral group treatment for chronic low back pain. *Journal of Consulting Clinical Psychology*. **56**:261–66.

Turner JA and Jensen MP. 1993. Efficacy of cognitive therapy for chronic low back pain. *Pain*. **52**:169–77.

Turner JA. 1982. Comparison of group progressive-relaxation training and cognitive-behavioral group therapy for chronic low back pain. *Journal of Consulting and Clinical Psychology* **50**:757–65.

Turner, J.A and Clancy, S. 1986. Strategies for coping with chronic low back pain : Relationship to pain and disability, *Pain* 24 (355-366).

Van Damme, S., Crombez, G. and Ecclestron, C. 2002. Disengagement from pain cues: the effects of pain catastrophizing and pain expectancy. *Pain* 100(1-2)111-118.

van den Hout JHC, Vlaeyen JWS, Heuts PH, Zijlema JH and Wijnen JA. 2003. Secondary prevention of work-related disability in nonspecific low back pain: does problem solving therapy help?. *Clinical Journal of Pain*. **19**(2):87–96.

van der Roer N, van Tulder MW, Barendse JM, Knol D, van Mechelen W and de Vet HCW 2008. Intensive group training protocol versus guideline physiotherapy for patients with chronic low back pain: a randomised controlled trial. *European Spine Journal; 17:1193*–200.

Van Tulder, M.W. Koes, B.W. and Bouter, L.M. 1995. A cost-of- illness study of back pain in the Netherlands. *Pain*: 62:233-240.

Vas, J. Perea-Milla, E. Mendez, C. Silva, L. Galante, A.H. Regules, J.M. Barquin, D.M. Aguilar, I. and Faus, V. 2006. Efficacy and safety of acupuncture for the treatment of non-specific acute low back pain: A randomized controlled multicentre trial protocol. *BMC complementary alternative medicine*. 6:14.

Vlaeyen JW, Haazen IW, Schuerman JA, Kole-Snijders AM and van Eek H.1995. Behavioural rehabilitation of chronic low back pain: comparison of an operant treatment, an operant-cognitive treatment and an operantrespondent treatment. *British Journal of Clinical Psychology*;34 (Pt 1):95–118.

Vlaeyen, J.W and Linton, S.J. 2000. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. *Pain*. 85(3):317-332.

Vlaeyen, J.W., Kole-Snijders, A.M., Boeren, R.G. and van Eek, H. 1995. Fear of movement/(re)injury in chronic low back pain and its relation to behavioural performance. *Pain*. 62(3):363-372.

Volinn, E 1997. The epidermiology of low back pain in the rest of the world. A review of surveys in low-and middle-income countries. *Spine*; 22:1747-54.

Von Korff M, Balderson BHK, Saunders K, Miglioretti DL, Lin EHB, Berry S, Moore JE and Turner JA. 2005. A trial of an activating intervention for chronic back pain in primary care and physical therapy settings. *Pain*. **113**:323–30.

Von Korff M, Moore JE, Lorig K, Cherkin DC, Saunders K, Gonzalez VM, Laurent D, Rutter C and Comite F. 1998. A randomized trial of a lay person-led self-management group intervention for back pain patients in primary care. *Spine*. **23**(23):2608–15.

Waddell, G. 1987. A new clinical method for the treatment of low back pain. *Spine*, 12, 632–644.

Waddell, G. and Waddell, H. 2000. Social influences on neck and back pain. In: Nachemson A, Jonsson E, eds. Neck and Back pain: *The scientific Evidence of Causes, Diagnosis and Treatment.* New York Lippincott Williams and Wilkins.

Waddell, G., Burton, A. and Main, C. 2003. *Screening to identify people at risk of long term incapacity for work*. London, UK: Royal Society of Medicine Press.

Waddell, G., Neuton, M., Henderson, I., Somerville, D. and Main, C.J. 1993. A fear-avoidance beliefs questionnaire (FABQ) and the role of fear avoidance beliefs in chronic low back pain and disability. *Pain* 52:157-168.

Walker, B. 2000. The prevalence of low Back pain: A systematic review of the literature from 1966 to 1998. *Journal of spinal disorders* 13(3): 205-217.

Walker, B., Muller, R. and Grant, W. 2004. Low back pain in Australian adults. Prevalence and associated disability. *Journal of manipulative and physiological Therapeutics* 27: 238-44.

Wall, P. 1999. Pain: *The science of suffering*. London.: Weidenfelfd and Nicolson.

Waterschoot, F. P. C., Dijkstra, P. U., Hollak, N., de Vries, H. J., Geertzen, J. H. B and Reneman, M. F. 2014. Dose or content? Effectiveness of pain rehabilitation programs for patients with chronic low back pain: a systematic review. *Pain*, 155(1), 179–89. doi:10.1016/j.

Whitfill T, Haggard R, Bierner SM, Pransky G, Hassett RG and Gatchel RJ. 2010. Early intervention options for acute low back pain patients: a randomized clinical trial with one-year follow-up outcomes. *Journal of Occupational Rehabilitation* ;20:256–263.

Williams, R.M. and Meyers, A.M. 1998. Functional abilities confidence scale: a clinical measure for injured workers with acute low back pain. *Physical Therapy*. 78,624-634.

Winterowd C, Beck AT and Gruener D, 2003. *Cognitive therapy with chronic pain patients*. New York: Springer Publishing Company.

Woby, S.R., Roach, N.K., Urmston, M. and Watson, P.J. 2007. The relation between cognitive factors and levels of pain and disability in chronic low back pain patients presenting for physiotherapy. *European Journal of Pain*. 11, 869-877.doi:10.1016/j.ejpain.2007.01.005

Woods MP and Asmundson GJG. 2008. Evaluating the efficacy of graded in vivo exposure for the treatment of fear in patients with chronic back pain: A randomized controlled clinical trial. *Pain*. **136**:271–80.

APPENDIX I

SOCIO-DEMOGRAPHIC CHARACTERISTICS OF PARTICIPANTS

Demographic and Clinical data. Today's date.....

Please respond to the following questions by filling in the space provided or circling the correct response. The answers you give shall be regarded as anonymous and kept in strict confidence.

What is your gender? 1. Male 2. Female

What is your date of birth[if known] ....

Day Month Year

Age ..... Weight ..... Height .....

What is the highest formal education you have received? 1. None 2. Primary 3. Secondary

4. Tertiary 5. Other

[specify]----

What is your occupation?

What is your marital status? 1. Single 2. Married 3. Divorced 4. Widowed

Which tribe are you from? 1. Yoruba 2. Hausa 3. Igbo 4. Others[specify]

What is your religion or denomination? 1. Christianity

2. Islam

3. Traditional

4. None

5. Others [please specify]

How long have you had this present episode of low back pain?

1. Less than six weeks 2. Between 6 weeks and 12 weeks

3. More than 12 weeks

APPENDIX II

VISUAL ANALOGUE SCALE

Patient Name_____

Date_____

No pain _____
worst possible pain
0 1 2 3 4 5 6 7 8 9 10

VISUAL ANALOGUE SCALE (Yoruba version)

(Validated by Odole and Akinpelu, 2009)

Orúkọ ẹni tí ó ń gba ìwòsàn:_____ Déètì òní:_____

Ko si inira _____
Irora ti
0 1 2 3 4 5 6 7 8 9 10 mo ni
to buruju

APPENDIX III

Pain Catastrophizing Scale

Sullivan MJL, **Bishop** S, **Pivik** J. (1995)

Name:	**Age:**	**Gender:**	**Date:**
----------	----------	☐Male ☐Female	----------

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

Instructions:
We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

RATING	**0**	**1**	**2**	**3**	**4**
MEANING	Not at all	To a slight degree	To a moderate degree	To a great degree	All the time

When I'm in pain …

Number	**Statement**	**Rating**
1	I worry all the time about whether the pain will end.	
2	I feel I can't go on.	
3	It's terrible and I think it's never going to get any better	
4	It's awful and I feel that it overwhelms me.	
5	I feel I can't stand it anymore	
6	I become afraid that the pain will get worse.	
7	I keep thinking of other painful events	
8	I anxiously want the pain to go away	
9	I can't seem to keep it our of my mind	
10	I keep thinking about how much it hurts.	
11	I keep thinking about how badly I want the pain to stop	
12	There's nothing I can do to reduce the intensity of the pain	
13	I wonder whether something serious may happen.	

APPENDIX IV

Tampa Scale for Kinesiophobia

(Miller , Kori and Todd 1991)

1 = strongly disagree
2 = disagree
3 = agree
4 = strongly agree

1. I'm afraid that I might injury myself if I exercise	1	2	3	4
2. If I were to try to overcome it, my pain would increase	1	2	3	4
3. My body is telling me I have something dangerously wrong	1	2	3	4
4. My pain would probably be relieved if I were to exercise	1	2	3	4
5. People aren't taking my medical condition seriously enough	1	2	3	4
6. My accident has put my body at risk for the rest of my life	1	2	3	4
7. Pain always means I have injured my body	1	2	3	4
8. Just because something aggravates my pain does not mean it is dangerous	1	2	3	4
9. I am afraid that I might injure myself accidentally	1	2	3	4
10. Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening	1	2	3	4
11. I wouldn't have this much pain if there weren't something potentially dangerous going on in my body	1	2	3	4
12. Although my condition is painful, I would be better off if I were physically active	1	2	3	4
13. Pain lets me know when to stop exercising so that I don't injure myself	1	2	3	4
14. It's really not safe for a person with a condition like mine to be physically active	1	2	3	4
15. I can't do all the things normal people do because it's too easy for me to get injured	1	2	3	4
16. Even though something is causing me a lot of pain, I don't think it's actually dangerous	1	2	3	4
17. No one should have to exercise when he/she is in pain	1	2	3	4

APPENDIX V

(Fairbank and Pynscent, 2000; Davidson and Keating, 2002)

THE REVISED OSWESTRY LOW BACK PAIN QUESTIONNAIRE

PATIENT NAME: _____ **DATE**: _____

Please read: This questionnaire is designed to enable us to understand how much your low back pain has affected your ability to manage your everyday activities. Please answer each section by circling the ONE CHOICE that most applies to you. We realize that you may feel that more than one statement may relate to you, but PLEASE, JUST CIRCLE THE ONE CHOICE WHICH MOST CLOSELY DESCRIBES YOUR PROBLEM RIGHT NOW.

SECTION 1 - Pain Intensity

A The pain comes and goes and is very mild.
B The pain is mild and does not vary much.
C The pain comes and goes and is moderate.
D The pain is moderate and does not vary much.
E The pain comes and goes and is severe.
F The pain is severe and does not vary much.

SECTION 2 - Personal Care

A I do not have to change my way of washing or dressing in order to avoid pain.
B I do not normally change my way of washing or dressing even though it causes some pain.
C Washing and dressing increases the pain but I manage not to change my way of doing it.
D Washing and dressing increases the pain and I find it necessary to change my way of doing it.
E Because of the pain I am unable to do some washing and dressing without help.
F Because of the pain I am unable to do any washing and dressing without help.

SECTION 3 - Lifting

A I can lift heavy weights without extra pain.
B I can lift heavy weights but it causes extra pain.
C Pain prevents me from lifting heavy weights off the floor.
D Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, e.g., on a table.
E Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.
F I can only lift very light weights at the most.

SECTION 4 - Walking

A I have no pain on walking.
B I have some pain on walking but it does not increase with distance.
C I cannot walk more than one mile without increasing pain.
D I cannot walk more than 1/2 mile without increasing pain.
E I cannot walk more than 1/4 mile without increasing pain.
F I cannot walk at all without increasing pain

SECTION 5 - Sitting

A I can sit in any chair as long as I like.
B I can sit only in my favorite chair as long as I like.
C Pain prevents me from sitting more than one hour.
D Pain prevents me from sitting more than 1/2 hour.
E Pain prevents me from sitting more than 10 minutes.
F I avoid sitting because it increases pain straight away.

SECTION 6 - Standing

A I can stand as long as I want without pain.
B I have some pain on standing but it does not increase with time.
C I cannot stand for longer than one hour without increasing pain.
D I cannot stand for longer than 1/2 hour without increasing pain.
E I cannot stand for longer than 10 minutes without increasing pain.
F I avoid standing because it increases the pain immediately.

SECTION 7 - Sleeping

A I get no pain in bed.
B I get pain in bed but it does not prevent me from sleeping well.
C Because of pain my normal night's sleep is reduced by less than 1/4.
D Because of pain my normal night's sleep is reduced by less than 1/2.
E Because of pain, my normal night's sleep is reduced by less than 3/4.
F Pain prevents me from sleeping at all.

SECTION 8 - Social Life

A My social life is normal and gives me no pain.
B My social life is normal but increases the degree of my pain.
C Pain has no significant effect on my social life apart from limiting my more energetic interests, e.g., dancing, etc.
D Pain has restricted my social life, and I do not go out very often.
E Pain has restricted my social life to my home.
F I have hardly any social life because of the pain.

SECTION 9 - Travel

A I get no pain while traveling.
B I get some pain while traveling, but none of my usual forms of travel make it any worse.
C I get extra pain while traveling, but it does not compel me to seek alternative forms of travel.
D I get extra pain while traveling, which compels me to seek alternative forms of travel.
E Pain restricts all forms of travel.
F Pain prevents all forms of travel except that done lying down.

SECTION 10 - Changing degree of pain

A My pain is rapidly getting better.
B My pain fluctuates but overall is definitely getting better.
C My pain seems to be getting better but improvement is slow at present.
D My pain is neither getting better nor worse.
E My pain is gradually worsening.
F My pain is rapidly worsening.

APPENDIX VI

(Swinkels-meewisse et al, 2003)

SELF-EFFICACY FOR REHABILITATION OUTCOME SCALE

INSTRUCTIONS: The Self-Efficacy for Rehabilitation Outcome Scale (SER) provides 12 statements that conclude the sentence, "During my rehabilitation, I believe I can do ..." Please complete this survey by choosing the most appropriate number for each statement. This scale is rated on an 11-point scale ranging from 0 (I cannot do it) to 10 (Certain I can do it). If you have any questions, please ask for clarifications.

0 1 2 3 4 5 6 7 8 9 10

I cannot do it Certain I can do it

During my rehabilitation, I believe I can do ...

1. Therapy that requires me to stretch my back .....
2. Therapy that requires me to lift my back .....
3. Therapy that requires me to bend my back .....
4. Therapy that requires me to stand .....
5. Therapy that requires me to walk .....
6. All of my therapy exercises during my rehabilitation .....
7. My therapy every day that it is scheduled .....
8. The exercises my therapists say I should do, even if I don't understand how it helps me .....
9. My therapy no matter how I feel emotionally .....
10. My therapy no matter how tired I may feel .....
11. My therapy even though I may already have other complicating illnesses .....
12. My therapy regardless of the amount of pain I am feeling .....

APPENDIX VII

INFORMED CONSENT FORM

IRB Research approval number_____.

This approval will elapse on : _____.

EFFECTS OF PROGRESSIVE GOAL ATTAINMENT PROGRAMME ON SELECTED PAIN CHARACTERISTICS AND PSYCHOSOCIAL FACTORS IN PATIENTS WITH MECHANICAL LOW BACK PAIN

This study is being conducted by Mr Ogunlana Michael Opeoluwa, a post graduate student of the physiotherapy department university of Ibadan. I am conducting a study to investigate the effect of progressive goal attainment program (PGAP) on selected pain characteristics and psychosocial factors in people with Mechanical low back pain. This study is being carried out in partial fulfillment of the requirement for the award of Master of Philosophy/Doctor of Philosophy (Physiotherapy) degree of the College of Medicine University of Ibadan. You may be required to participate in a treatment program that will span ten weeks (one session peer week) alongside your regular treatment for low back pain.

All the information you give will be confidential and used for the purpose of the research only. The information you and others give will help me to document the efficacy of PGAP on low back pain. Please note that participation in this study is voluntary and you are free to decline from participating. You are also free to withdraw your participation at any instance. I will be grateful if you will help by completing the questionnaire and participate in the study. Your participation in this research will not cost you anything, and any information collected during the course of this study will be treated confidentially by using code numbers, there won't be any record of your name or any form of identifier used in any publication or reports from this study. Your participation in this research is voluntary and if you choose not to participate, it will not affect your treatment in any way. You can also choose to withdraw at any time during the course of this study but the initial information that has been obtained about you before your withdrawal may have been modified or used in reports or publications. These cannot be removed any more. However I promised to make a good faith an effort to comply with your wishes as much as is practicable.

Statement of person obtaining informed consent:

I have fully explained this research to_____
and have given sufficient information, including about risks and benefits, to make an informed decision.
DATE_____ SIGNATURE_____

NAME_____

Statement of person giving consent

I have read the description of the research .I have also talked it over with my physiotherapist to my satisfaction. I understand that my participation is voluntary. I know enough about the purpose, methods, risks and benefit of the research study to judge that I want to take part in it I understand that I may freely stop being part of this study at any time. I have received a copy of this consent form and additional information to keep for myself.

DATE_____ SIGNATURE_____

NAME_____

WITNESS' SIGNATURE_____

WITNESS NAME_____

This research has been approved by the ethics committee of the University of Ibadan and the Chairman of this committee can be contacted at Biode Building, Room T10, 2nd floor, Institute for Advanced Medical Research and Training, College of Medicine, University of Ibadan. In addition, if you have any question about your participation in this research you can contact the principal investigator, Ogunlana Michael. O Department of Physiotherapy, College of Medicine, University of Ibadan, 08034659378, opeoluwamic@yahoo.com.

*PLEASE KEEP A COPY OF THE SIGNED INFORMED CONSENT.

APPENDIX VIII

Screening for Red flags in Low Back Pain

Red Flags

Possible fracture	Possible tumour or infection	Possible significant neurological deficit
From history		
• Major trauma • Minor trauma in elderly or osteoporotic	• Age >50 or <20 years • History of cancer • Constitutional symptoms (fever, chills, weight loss) • Recent bacterial infection • IV drug use • Immunosuppression • Pain worsening at night or when supine	• Severe or progressive sensory alteration or weakness • Bladder or bowel dysfunction
From physical examination		
		• Evidence of neurological deficit (in legs or perineum in the case of low back pain)

The presence of red flags in acute low back pain suggests the need for further investigation and possible specialist referral as part of the overall strategy. If there are no red flags present in this situation it is safe to reassure the patient and move ahead with a multimodal management approach.

APPENDIX IX

Activity Log

(Sullivan 2010)

Name:		Week 1 2 3 4 5 6 7 8 9			
	Day 1 :	Day 2 :	Day 3 :	Day 4 :	Day 5 :
6:00					
7:00					
8:00					
9:00					
10:00					
11:00					
12:00					
1:00					
2:00					
3:00					
4:00					
5:00					
6:00					
7:00					
8:00					
9:00					

APPENDIX X

ÀWÓN OHUN TÍ Ó JẸMỌ́ BÍ OLÙKÓPA SE JẸ́

Ìrírí àti ìsètójú olùkópa wádìí Déètì òní: _____

Ẹ jọ̀wọ́ ẹ dáhùn àwọn ìbèèrè wọ̀nyí nípa dídí àwọn àlàfo tí ó ṣófo tàbí kí ẹ yí òdò sí ìdáhùn tí ó tọ̀nà. Ìdáhùn yín sí àwọn ìbéèrè wọ̀nyín ni a ó pamọ́, a kì yóò sì jẹ́ kí ẹnikẹ́ni mọ̀ nípa rẹ̀.

Se ọkùnrin niyín tàbi obìrin 1. ọkùnrin { } 2. obìrin { }

Kí ni déètì ojóibí yín (Tí ẹ bá mọ̀)? _____ _____ _____

Ọjọ́ Osù Ọdún

Ọjọ́ orí:_____ Ìwòn:_____ Gíga:_____

Báwo ni e ṣe kàwé tó?

(a) N kò kàwé [ ] (b) Ilé ìwé Alákọ́bèrè [ ] (d) Ilé-Ìwé Gírámà [ ]

(e) Ilé-ìwé gíga [ ] (ẹ) Àwọn mìíràn (Ṣọ ní pàtó):_____

_____

Kí ní iṣẹ́ rẹ? _____

Ǹjẹ́ ẹ ti gbéyàwó tàbí lọ́kọ?

(a) Àpọ́n/ omidan [ ] (b) Lọ́kọ/Gbéyàwó [ ]

(d) Kọ̀sílẹ̀ [ ] (e) Opó [ ]

Kí ni èyà rẹ? (a) Yorùbá [ ] (b) Hausa [ ] (d) Igbo [ ]

(e) èyà mìíràn (so ni pàtó): _____

Irú ẹ̀sìn wo ni ẹ ń ṣe?

(a) Onígbàgbọ́ [ ] (b) Ẹlẹ́sìn Ísíláàmù [ ]

(d) Ẹlẹ́sìn Ìbílẹ̀ [ ] (e) N kò lẹ́sìn [ ]

(ẹ) Ẹ̀sìn mìíràn (sọ ni pàtó) _____

_____

Ó tó ìgbàwo tí ìsàlẹ̀ èyìn tí ó ń dùn yín lọ́wọ́lọ́wọ́ yìí ti bẹ̀rẹ̀?

(a) Ó dín lọ́sẹ̀ méfà [ ]

(b) Láàárin ọ̀sẹ̀ méfà sí méjìlá [ ]

(d) Ó ju ọ̀sẹ̀ méjìlá [ ]

APPENDIX XI

ÀSỌDÙN BÍ ÌRORA ṢE BURÚ TÓ

Sullinan MJL, Bishop S. Pivik J. (1995)

Orúkọ Ọjọ́ orí Ọkùnrin/Obìrin Déètì

_____ _____ [ ] Ọkùnrin [ ] Obìrin _____

Gbogbo ènìyàn ni ó máa ń ní ìrírí ìrora ní ìgbà kan tàbí òmíràn ní ìgbà ayé wọn. Irú àwọn ìrírí bẹ́ ẹ̀ lè jẹ́ ẹ̀fọ́rí, ẹ̀yin dídùn, ìrora oríkèéríkèé ara tàbí ti iṣan ara. Ní ọ̀pọ̀lọpọ̀ ìgbà ní àwọn ènìyàn máa ń dojúkọ àwọn ohun ti ó lè fà ìrora bíi àìsàn, ọgbẹ́, ìtójú eyín àti ti iṣẹ́ abẹ.

<u>Àkíyèsí</u>:
Ohun tí ó jẹ́wálógún jù ní èrò àti ìrírí yín nígbà tí ẹ bá wà nínú ìrora. Ní ìsàlẹ̀ ìwé yìí, a ti ṣe ìtòlẹ́ṣẹẹṣẹ àwọn nǹkan mẹ́tàlá tí ó ń ṣàpèjúwe orísiríṣi èrò àti ìrírí tí ó lè bá ìrora kọ́wọ̀ọ́. Nípa ṣíṣe àmúnlò òdiwọ̀n ìsàlẹ̀ yìí, jọ̀wọ́ ṣe àlàyé àfihàn ipò tí ẹ tí ni àwọn ìrírí àti èrò yìí nígbà tí ẹbá ń ní ìrírí ìrora.

Òṣùnwọ̀n	0	1	2	3	4
Ìtumọ̀	Kò sí rárá	Ó wà díẹ̀díẹ̀	Ó mọ níwọ̀n tunwọ̀nsì	Ó pọ̀ gan	Ní gbogbo ìgbà

Nígbà tí mo bá wà nínú ìrora …

Nọ́mbà	**Gbólóhùn**	**Òṣùwọ̀n**
1.	Mo máa ń ṣe àníyàn ní gbogbo ìgbà lórí bóyá ìrora náà yóò dópin.	
2.	Mó ní ìmọ̀lára pé ń kò ní lè tẹ̀síwájú báyìí.	
3.	Ó lè púpọ̀, mo sì rò pé kò le è dáwọ́dúró.	
4.	Ó burújù, ó dàbí ẹni pé ó ti ńkọjá agbára mi.	
5.	N kò lérò pé mo leè faradàá mọ́	
6.	Ẹ̀rù a máa bà mí pé ìrora náà yóò burú sí.	
7.	Mo ń ronú àwọn ìsẹ̀lẹ̀ ìrora mìíràn ní ìgbà gbogbo.	
8.	Mo ń ní ìtara pé mo fé kí ìrora náà pòórá lógán.	
9.	Ó dàbí ẹni pé ń kò le è gbé e kúrò lọ́kàn	
10.	Mo máa ń ronú lórí bí ó ṣe ń dùn mí tó.	
11.	Mó ń ronú lórí bí mo tí fẹ́ kí ìrora náà dópín ní gbogbo ọ́nà.	

12.	Kò sí ohun tí mo lè ṣe láti dín bí ìrora náà ṣe pọ̀tó kù.	
13.	ẹ̀rù tilẹ̀ ń bà mí pé nǹkan tó burú yóò ṣẹlẹ̀.	

APPENDIX XII

ÒSÙWÒN *TÁMPÀ* FÚN ÌBẸ̀RÙ ÀTI GBÉRA (KINẸSIÓFÓBÍÀ)

(Miller Kori àti Todld 1991)

1. N kò faramọ́ ọ rárá
2. N kò faramọ́ ọ
3. Mo faramọ́ ọ
4. Mofaramọ́ ọ pátápátá

Ònkà		**1**	**2**	**3**	**4**
1.	Ẹ̀rù ńbàmí pé mo lè ṣe ara mi léṣe tí mo bá ṣe eré ìdárayá.				
2.	Ìrora mi yóò tún pọ̀ sí tí mo bá fẹ́ gbìyànjú láti borí rẹ̀.				
3.	Ara mí ń sọ fún mi pé ewu wà.				
4.	Bóyá ìrora mí lè dínkù tí mo bá ṣe eré ìdárayá.				
5.	Àwọn ènìyàn kò kọ ibi ara sí ìlera mi bí ó ti yẹ.				
6.	Ìjàmbá ti sọ aramí di ewu fún gbogbo ìgbésí ayé mi.				
7.	Ìrora máa ń túmọ̀ sí pé mo tí ṣe ara mi lése				
8.	Pé nǹkan mú kí ìrora mi pọ̀ si kò túmọ̀ sí pé ó léwú.				
9.	Ẹ̀rù ń bà mí pé mo lè ṣèṣì ṣe ara mi léṣe.				
10.	Ṣíṣọ́ra láti máà gbé ara mi lọ́nà òdì ni ọ̀nà àìléwu tí mo lè gbà láti máá jẹ́ kí ìrora mi pọ̀si.				
11.	Mi ò lè ní ìrora tó pọ̀ tó yìí tí kò bá ṣe wípé ohun kan tí ó léwu ńṣẹlẹ̀ lágọ̀ọ́ ara mi.				
12.	Bí ó tílè jẹ́ pé mo ní ìrora yóò dára fún mi tí mo bá lè gbéra kánkán/tàbí ṣeré ìdárayá.				
13.	Ìrora jẹ́ kí ń mo àkókò tí mo gbọ́dọ̀ dá eré ìdárayá dúró láì ṣe ara mi léṣe				
14.	Ó léwu fún ẹni tí ó wà nínú ìrora bí i tèmi láti gbé ara kánkán/ tàbí ṣe eré ìdárayá.				
15.	N kò le è ṣe gbogbo nǹkan tí àwọn tí ara wọ́n yá ń ṣe nítorí pé mo tètè máa ń farapá.				
16.	Bí ó tílẹ̀ jẹ́ pé nǹkan kan ń fà ìrora púpọ̀ fún mi, ń kò rò pé ó léwu lóótọ́.				
17.	Kò nílò kí ẹnikẹ́ni ṣe eré ìdárayá nígbà tí irú ẹni bẹ̀ẹ̀ bá wà nínú ìrora.				

APPENDIX XIII
ÀTÚNSE ÀWỌN ÌBÉÈRÈ ÌṢẸ́-ÌWÁDÌÍ TI *OSWESTRY* NÍPA ÌSÀLẸ̀ ẸYÌN DÍDÙN

Orúkọ ẹni tí ó ń gba ìwòsàn: _____ Déètì: _____

Jọ̀wọ́ kà àkíyèsí yìí: Àwọn ìbéèrè ìṣẹ́ ìwádìí yìí ni a ṣètò láti fún wa ni ànfààní láti mọ bí ìrora ìsàlẹ̀ ẹ̀yìn rẹ ti ṣe ìdíwó tàbí àbùkú fún àwọn ojúṣe ojoojúmọ́ rẹ. Jọ̀wọ́ dáhùn ìsọ̀rí kọ̀ọ̀kan nípa yíyí òdo sí èyí tí ó kàn ọ gbọ̀ngbọ̀n. A sàkìyèsí pé ó ṣeéṣe kí èyí tí ó ṣẹ́wo mọ́ọ yín lára ju ọ̀kan lọ, sùgbọ́n ẸYỌ KAN ṢOṢO TÍ Ó ṢÀPÈJÚWE ÌṢÒRO YÍN NI KÍ Ẹ YÍ ÒDO SÍ BÁYÌÍ

Abala Kinni – Bí írora ṣe pọ tó

(1) Ìrora náà ń wá ó sì ń lọ láti ìgbà dé ìgbà ṣùgbọ́n kò pọ̀ rárá.
(2) Ìrora náà kò pọ́ bẹ́ẹ̀ ni kò si yàtọ̀ púpọ̀.
(3) Ìrora náà ń wá, ó ńlọ́, ó sì mọ níwọ̀n.
(4) Ìrora náà mọ ní ìwọ̀ntúnwọ̀nsí kò sìyàtọ̀ púpọ̀.
(5) Ìrora náà ń wá, ó si ń lọ, ó sì lágbára gan.
(6) Ìrora náà pọ gan-an bẹ́ẹ̀ ni kò sì yàtọ̀ púpọ̀.

Abala Kefa – Ìdúró

(1) Mo lè dúró bí ó tí wùmí fún ìgbà pípé láìsí ìrora
(2) Mo máà ń ni ìrora ní orí ìdúró, ṣùgbọ́n kìí pọ̀ síi bí mo bá dúró pẹ́.
(3) N kò lè dúró ju wákàtí kan lọ láìsí ni ìrora
(4) N kò lè dúró jú ọgbọ̀n ìṣẹ́jú lọ láìsí ìrora.
(5) N kò lè dúró ju ìṣẹ́jú mẹ́wàá lọ lái sí ìrora.
(6) Mo máa ń yàgò fún ìdúró nítorí pé ó máa ń fikùn ìrora mi lẹ́ṣẹ̀kẹ́ṣẹ̀.

Abala Kejì – Ṣíṣe Ìtọ́jú ara ẹni

(1) N kò ní láti yí ọ̀nà ìmúra tàbí ìfọṣọ mi padà láti mú ìrora kúrò.
(2) N kò sáábà yí ọ̀nà ìmúra tàbí ìfọṣọ mi padà bí ó tilẹ̀ jẹ́ pé ó máa fa ìrora.
(3) Fífọ̀ àti ìmúra máa ń mú kí ìrora mi pọ̀ síi ṣùgbọ́n nkò yí ọ̀nà ìgbésẹ̀ wón padà.
(4) Mimúra àti fífò ọ máa ń mú kí ìrora mi pọ̀ síi, mo sì ríi bí ohun tí ó ṣe kókó láti yí bí mo ṣe ń ṣe wọ́n padà.
(5) Nítorí ìrora, ó ṣòro fún mi láti fọṣọ tàbí wọṣọ láìsí ẹni tí yóò ràn mí lọ́wọ́.
(6) Nítorí ìrora ń kò lè wọṣọ tàbí fọṣọ rara láìsí ẹni tí yóò ràn mí lọ́wọ́.

Abala Kejì – Oorun sísùn

(1) N kìí ní ìrora lórí ibùsùn.
(2) Mo máà ń mó ìrora lórí ibùsùn ṣùgbọ́n kìí dí mi lówó láti sùn dáadáa.
(3). Nítorí ìrora, oorun alaalẹ́ mi ti dínkù, bí ìdá kan nínú mérin
(4) Nítorí ìrora, oorun alaalé mi ti dínkù, bí ìdá kan nínú méjì.
(5). Nítorí ìrora, oorun alaale mi ti dínkù, bí ìdá méta nínú mérin
(6) Ìrora kìí jẹ́ kí n lè sùn rárá.

.

Abala Kẹta – Gbígbé Nnkan

(1) Mo lè gbé nǹkan tó wúwo láìsí ìrora.
(2) Mo lè gbé nnkan tó wúwo ṣùgbọ́n ó máa ń mú ìrora lọ́wọ́.
(3) Ìrora ń dí mi lọ́wọ́ láti gbé nǹkan tó wúwo láti ilẹ̀.
(4) Ìrora máa ń dí mi lọ́wọ́ láti gbé nǹkan tí ó wúwo nílẹ̀. Ṣùgbọ́n, mo lè rọ́jú gbé e tí a bá gbé e sí orí tábîlì.
(5) Ìrora máa ń di mi lọ́wọ́ láti gbé nǹkan tí ó wúwo ṣùgbọ́n, mo lè gbíyànjú gbé nǹkan tí kò wúwo púpọ̀ tí wọ́n bá gbé wọ́n ka ibi tí ó ga.
(6) Nǹkan tí kò wúwo rárá ni mo lè gbé.

Abala Kẹjọ – Ìgbé-ayé Láwùjọ

(1) Ìgbé-ayé ìbákẹ́gbépọ̀ mi bá ìlànà mu kìí sì fún mi ni ìrora.
(2) Ìgbé-ayé mi láwùjọ dán mọ́rán, ṣùgbọ́n ó máa ń fikùn ìrora mi.
(3) Ìrora kò ní ipà tí ó lápẹ̀rẹ lórí ìgbé-ayé mi láwùjọ sùngbọ́n kìí jẹ́ kí n lè kópa nínú áwọn nǹkan tí ó lágbára bíí àpẹẹrẹ: ijó jíjó, abbl.
(4) Ìrora ti dín ìgbé ayé àwùjọ mi kù, kìí jẹ́ kí n lè jáde ní gbogbo ìgbà.
(5) Ìrora tí sé mi mọ́ ilé pátápátá.
(6) Agbára káká ni mo fi ń kópa nńú ìgbé ayé láwùjọ nítorí ìrora.

Abala Kẹrin – Ìrìn Rínrìn

(1) Mí kìí ní ìrora lórí ìrìn.
(2) Mo máa ní ìrora tí mo bá ń rìn Sùgbọ́n, kìí pọ̀ sí bí ọ̀nà bá jìn.
(3) Bi mo ba rin kọja ibùsọ̀ kan, irora mi yóò pọ̀ si
(4) Bí mo bá rìn kọjá ààbọ̀ ibùsọ̀, ìrora mi yóò pọ̀ sí.
(5) Bí mo bá rìn kọjá ìdámẹ́rin ibùsò ìrora mi yóò pò sí.
(6) N kò lè rìn rárá kí ìrora mi má pọ̀ sí.

Abala Karùn – Ìjókòó

(1) Mo lè jókòó lórí àgakága pẹ́ bí mo bá ti fẹ́
(2) Mo lè jókòó lórí àga tí ó wù mí pẹ́ bí mo bá ti fẹ́
(3) Ìrora máa ń dí mi lọ́wọ́ láti jókòó jú wákàtí kan lọ.
(4) Ìrora máa ń dí mi lọ́wọ́ láti jókòó jú ààbọ̀ wákàtí lọ.
(5) Ìrora máa ń dí mi lọ́wọ́ láti jókòó jú ìṣẹ́jú mẹ́wàá lọ.
(6) Mo máa ń yàgò fún ìjókòó nítorí ó máa ń ṣe àlékùn ìrora lẹ́ṣẹ̀kẹṣẹ̀.

Abala Kẹsàn-án – Ìrìnàjò

(1) Mi kìí ní ìrora nígbà tí mo bá ń rìn ìrìnàjò.
(2) Mo máa ń ni ìrora nígbà tí mo ba ń rìn ìrìnàjò, ṣùgbọ́n kò si ọ̀kan ninu ọ̀nà ìrìnàjò mi tó mú un pọ̀ si
(3) Mo máa ń ní àlékún ìrora nígbà tí mo bá ń rìn ìrìnàjò ṣùgbọ́n kò sọ ọ́ di ọ̀ranyàn fún mi láti wa ọ̀nà ìrìnàjò mìíràn
(4) Mo máa ń ní àlékún ìrora nígbàtí mo báń rìn ìrìnàjò èyí tí ó sọ́ ọ di ọ̀ranyàn fún mi láti wá ọ̀nà ìrìnàjà mìíràn.`
(5) Ìrora dí ìrìnàjò mi kù pátápátá.
(6) Ìrora ń ṣàkóbá fún ìrìnàjò mi àyàfi èyí tí mo Bá dùbúlẹ̀

Abala Kẹwàá – Bí Ìrora Ṣe ń Yípadà

(1) Ìrora mi ti dínkù gan-an.
(2) Ìrora mi ń lọ sókè sódò ṣùgbọ́n ó ń dínkù.
(3) Ó dàbí ẹni pé ìrora mi ń dínkù ṣùgbọ́n, ìpadàbọ̀ sípò ń lọ tintin lọ́wọ́ báyìí.
(4) Ìrora mi kò dínkù bẹ́ẹ̀ni kò pọ sí i.
(5) Ìrora mi ń pọ̀ si díẹ̀díẹ̀.
(6) Ìrora mi ń pọ̀ sí gidigidi.

APPENDIX XIV

ÒṢÙNWỌN MÍMỌ ÀYỌRÍSÍ MÍMÚNÁDÓKO FÚN ÌMÚPADÀBÒ SÍPÒ

(Swinkels-Meewisse et al 2003)

Àkíyèsí: Òṣùwọn àkítiyàn ara ẹni fún àyọrísí ìbọ́sípò pèsè ọ̀rọ̀ méjìlá tí ó kádìí gbólóhùn "Ní àkókò inú ìpadàbọ̀sípò mi, mo gbàgbọ́ pé mo lè ṣe…" Jọ̀wọ́ parí ìwádìí yìí nípa yíyan nọ́mbà tí ó yẹ fún ọ̀rọ̀ kọ̀ọ̀kan. A gbé òṣùwọn yìí lé orí òṣùwọn olójú mọ́kànlá tí ó bẹ̀rẹ̀ láti orí "0" (N kò le è ṣe) dé orí "10" (Dájúdájú mo lè ṣe é). Jọ̀wọ́ béèrè ìbéèrè tí ó bá ní fún àrídájú.

0 1 2 3 4 5 6 7 8 9 10

N kò lè ṣe é Dájúdájú mo lè ṣe é

Ní àkókò tí mo ń gba ìtọ́jú mo gbàgbọ́ pé mo lè ṣé e…

1. Gbígba ìtọ́jú tí ó jẹ mọ́ kí ń naa èyìn mi _____
2. Gbígba ìtọ́jú tí ó jẹ mọ́ kí ń gbé èyìn mi _____
3. Gbígba ìtọ́jú tí ó jẹ mọ́ kí ń ṣé èyìn mi _____
4. Gbígba ìtọ́jú tí ó jẹ mọ́ kí ń dìdé _____
5. Gbígba ìtọ́jú tí ó jẹ mọ́ pé kí ń rìn _____
6. Gbígba gbogbo ìtọ́jú tí ó jẹ mọ́ eré dárayá_____
7. Bí a ṣe ṣe ìlànà gbígba ìtọ́jú mi lójoojúmọ́ _____
8. Àwọn ìdárayá tí olùtọ́jú mi sọ wípé mo gbọdọ̀ ṣe, kódà, bí ń kò tílẹ̀ lóye ore rẹ̀
9. Gbígba ìtọ́jú mí pẹ̀lú ẹ̀dùn okan ki ẹ̀dùn okan tí mo lè ni _____
10. Gbígba ìtọ́jú mi bí ó ti wù kí ó rẹ̀ mí tó _____
11. Gbígba ìtọ́jú mi bí mo tílẹ̀ ti ní àwọn àìléra mìrán tí ó burú jàyì _____
12. Gbígba ìtọ́jú mi bí ó ti wù kí ìrora mí pọ̀ tó_____

APPENDIX XV

FEDERAL MEDICAL CENTRE

Bisi Onabanjo Way, Idi-Aba, P. M. B. 3031 (Sapon Post Office), Abeokuta, Nigeria.
07056790001 - 3
e-mail: fmcabk@yahoo.com

Medical Director
Dr. O. S. Sotiloye
MBBS, FWACS, FICS, Dip. Reproductive Med & Biology (Geneva) D MAS

Head of Clinical Services
Dr. C. O. Akisanya
MB; BS, FWACS FMCR

Director of Admin.& Sec. Board of Mgt.
Dr. O. A. Oyeku
Bsc, MPA, AMNIM, AIPM, AHAN

14th June, 2013.

Our Ref: _____ Your Ref: _____ Date: _____

NAME OF PRINCIPAL INVESTIGATOR: OGUNLANA, MICHAEL OPEOLUWA

TITLE OF STUDY: EFFECTS OF PROGRESSIVE GOAL ATTAINMENT PROGRAMME ON SELECTED PAIN CHARACTERISTICS AND PYSCHOSOCIAL FACTORS IN PATIENTS WITH MECHANICAL LOW BACK PAIN

RESEARCH LOCATION: FEDERAL MEDICAL CENTRE (FMC), ABEOKUTA

PROTOCOL NUMBER: FMCA/238/HREC/12/2013

NREC REG. NUMBER: NREC/08/04/2010

NOTIFICATION OF FULL MEMBER APPROVAL OF RESEARCH PROTOCOL

This is to inform you that the Federal Medical Centre, Abeokuta Health Research Ethics Committee (HREC) at its sitting on 13th June, 2013 decided to give full membership approval to your research proposal, after necessary reviews and corrections, under the regulations guiding experiments in human subjects.

This approval is for a period of one year from 17th June, 2013 to 16th June, 2014. If there is delay in starting this research, please inform the HREC so that dates of approval can be adjusted accordingly. Note that no activity related to this research may be conducted outside these dates. No changes are permitted in the research without prior approval by HREC.

All forms and questionnaires used in this study must carry the HREC assigned number and the duration of HREC approval.

You are to note further that, the National Code of Health Research Ethics requires you to comply with all institutional guidelines, rules and regulations, to follow trends of the code. Please ensure that any adverse effect from your study is promptly reported to the HREC Federal Medical Centre, Abeokuta.

You are expected to submit a progress report to this Committee every three (3) months from the date of approval. The HREC reserves the right to conduct compliance visits on your research sites without previous notification.

Thank you.

Dr. (Mrs.) T. O. Akinremi
Chairman, Hospital Research Ethics Committee

APPENDIX XVI

INSTITUTE FOR ADVANCED MEDICAL RESEARCH AND TRAINING (IAMRAT)
COLLEGE OF MEDICINE, UNIVERSITY OF IBADAN. IBADAN, NIGERIA.
Director: Prof. A. Ogunniyi, B.Sc(Hons), MBChB, FMCP, FWACP, FRCP (Edin), FRCP (Lond)
Tel: 08023038583, 08038094173
E-mail: aogunniyi@comui.edu.ng

UI/UCH EC Registration Number: **NHREC/05/01/2008a**

NOTICE OF FULL APPROVAL AFTER FULL COMMITTEE REVIEW

Re: Effects of Progressive Attainment Programme on Selected Pain Characteristics and Psychosocial Factors in Patients with Mechanical low back Pain

UI/UCH Ethics Committee assigned number: UI/EC/12/0360

Name of Principal Investigator: **Michael O. Ogunlana**

Address of Principal Investigator: Department of Physiotherapy,
College of Medicine,
University of Ibadan, Ibadan

Date of receipt of valid application: 23/10/2012

Date of meeting when final determination on ethical approval was made: **N/A**

This is to inform you that the research described in the submitted protocol, the consent forms, and other participant information materials have been reviewed and *given full approval by the UI/UCH Ethics Committee.*

This approval dates from 05/03/2013 to 04/03/2014. If there is delay in starting the research, please inform the UI/UCH Ethics Committee so that the dates of approval can be adjusted accordingly. Note that no participant accrual or activity related to this research may be conducted outside of these dates. *All informed consent forms used in this study must carry the* UI/UCH EC *assigned number and duration of* UI/UCH EC *approval of the study.* It is expected that you submit your annual report as well as an annual request for the project renewal to the UI/UCH EC early in order to obtain renewal of your approval to avoid disruption of your research.

The National Code for Health Research Ethics requires you to comply with all institutional guidelines, rules and regulations and with the tenets of the Code including ensuring that all adverse events are reported promptly to the UI/UCH EC. *No changes are permitted in the research without prior approval by the* UI/UCH EC *except in circumstances outlined in the Code. The* UI/UCH EC *reserves the right to conduct compliance visit to your research site without previous notification.*

UI/UCH EC
05 MAR 2013
APPROVED
COLLEGE OF MEDICINE, U.I.

Professor A. Ogunniyi
Director, IAMRAT
Chairman, UI/UCH Ethics Committee
E-mail: uiuchirc@yahoo.com

APPENDIX XVII

PAIN MANAGEMENT PHYSIOTHERAPY GROUP

A special interest Group of the South African Society of Physiotherapy

CERTIFICATE

This is to certify that

Michael Ogunlana

HPCSA Number

MRTBN 0888

Successfully completed the exam for

Progressive Goal Attainment Program

Module 4 of The National Pain Course

Presented by

Dr. Michael Sullivan

June 2011

CEUs: 12 APDL Level 2

ACCREDITATION NUMBER: PPB007/PT013/2011/011

Beverley Bolton
PMPG Chair