MODERATE EXERCISE AND THE PSYCHO-NEURAL AND BIO-MECHANICAL NATURE OF CHRONIC LOW BACK PAIN

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Table of Contents 1

Abstract

Low back pain (LBP) is one of the most common health problems, which in 10-15% of patients progresses to develop into a chronic low back pain (CLBP) condition, bringing enormous psychological and financial costs to the affected individuals, their families and society as a whole. There are many theories – pure bio-medical, bio-mechanical, psychological, as well as poly-modal ones, attempting to explain the pathology and aetiology of CLBP and guide its treatment . Although there is a general consensus that CLBP, as many other chronic pain conditions, is heterogeneous in nature, involving both organic (bio-mechanical and physiological) and psycho-social factors, there is still not a definite answer to the exact interactions and causative links betweens these factors.

The present study employed a 15 minutes moderate treadmill exercise intervention in 10 CLBP sufferers and 10 healthy controls in order to further clarify the interactions between these physiological, bio-mechanical and psychological factors. The results from the present study, in accordance with previous research confirmed that CLBP participants are clearly identifiable according to their affective and cognitive profile - CLBP participants reported significantly higher levels of Pain Catastrophising ($t = 3.81$, $p = .003$), as well as Depression ($t = 2.35$, $p = .03$). Although both Pain Catastrophising and Depression were significantly correlated with the background pain ($r = .755$, $p < .001$ and $r = .559$, $p = .010$ respectively) in the CLBP group alone, contrary to previous CLBP studies, there were no significant associations between the background pain level and duration, Depression, Anxiety, Stress and even Pain Catastrophising. The Negative affective (NA) state was measured dynamically throughout the experiment and was reduced non-significantly by the exercise in both groups. Surprisingly, The NA was very low – 18 to 20 on a scale of 15 to 75, in both groups (even lower in the CLBP) and there was no significant difference between the NA of the CLBP and Control groups at any time-point of the experiment.

The ES and MF back muscles activity was measured using a static standing and fully bent forward ratio, as well as flexion-relaxation ratio (FRR) pre- and post-exercise. The present study's results, although less robust than the results from previous research, confirmed that both standing and fully bent forward ES and MF measures, as well as FRR distinguished CLBP from control participants – CLBP had a higher level of static muscle activity (significant for the pre-exercise right MF in fully bent down position $t = -2.48$, $p = .$

044) and reduced FRR (significant for the pre-exercise left ES – t = 3.09, p = .013 and post-exercise left ES and right MF – t = 2.33, p = .038; t = 2.73, p = .032 respectively).

Although the exercise produced non significant improvement of the ES and MF muscle activity, it also produced a divergent pain intensity (PPI) response between different CLBP participants – 5 (55.56%) had a decrease in PPI, while 4 (44.44%) had an increase. The pain decrease sub-group exhibited only a mild back muscles dysfunction, characterised by increased activity of the ES and MF in resting state (muscle tension), combined with a higher negative cognitive and affective mental set. The main characteristics of the pain increase sub-group was abnormal ES and MF flexion-relaxation, which pointed to compensatory increased activity of the superficial back muscles due to possible intrinsic spinal instability.

In conclusion, the moderate treadmill exercise, utilised in the present study, was capable of identifying the existence of CLBP sub-groups, which were otherwise undistinguishable by the rest of their background pain, psychological or muscle activity characteristics. The low back pain of one of the sub-groups was associated with primary psychological top-down dysfunction of the 'active system' (muscles), while in the second sub-group the principal underlying factor identified was primary organic bio-mechanical dysfunction of the 'passive system'. The possible mechanisms and implications of this dichotomous nature of CLBP in respect to its aetiology, diagnosis, treatment and further research are discussed.

Section I – Background

Low back pain (LBP) is one of the most common health problems, affecting each year more than 20% of the adult population in the west (Balague et al., 2007; Savigny et al., 2008; Negrini et al., 2008). Fortunately, in the majority of cases it is a self-limiting condition, which resolves spontaneously requiring only minimal painkilling and anti-inflammatory medication and reassurance. Out of all LBP patients, 10-15% progress to develop a chronic low back pain (CLBP) condition, which brings enormous psychological and financial costs to the affected individuals, their families and society as a whole (Balague et al., 2007; Savigny et al., 2008). Despite all recent developments in understanding the underlying mechanisms of chronic pain, there are no established diagnostic criteria, clinical tests and effective treatment for CLBP. There is a plethora of theories – pure bio-medical, bio-mechanical, psychological, as well as poly-modal ones, attempting to explain the pathology and aetiology of CLBP and guide its treatment (Panjabi, 1992; Panjabi, 2003; Bousema et al., 2007; Verbunt et al., 2003; Hodges & Moseley, 2003; Turk & Okifuji, 2002; Waddell, 1992; Waddell, 1996). Although there is a general consensus that CLBP, as many other chronic pain conditions, is heterogeneous in nature, involving both organic (bio-mechanical and physiological) and psycho-social factors, there is still not a definite answer to the exact interactions and causative links betweens these factors. As exercise therapy has shown some encouraging results in the treatment of CLBP, as well as other chronic pain conditions (Hayden et al., 2005; Busch et al., 2007; Larun et al., 2004), the present study utilised a moderate treadmill exercise intervention in CLBP sufferers in order to shed further light on the interactions between these different factors.

1. Chronic Low Back Pain (CLBP)

NICE in their 2008 draft guideline (Savigny et al., 2008) defined chronic non-specific low back pain as ‘’pain, muscle tension or stiffness affecting the low back for which there is not a recognised patho-anatomical cause’’. This definition reflects the complexity, variability and multidimensional aspects of CLBP. To fully appreciate the difficulties involved in CLBP diagnosis and treatment it is necessary to first understand the anatomy and functionality of the lower back and especially the lumbar spine.

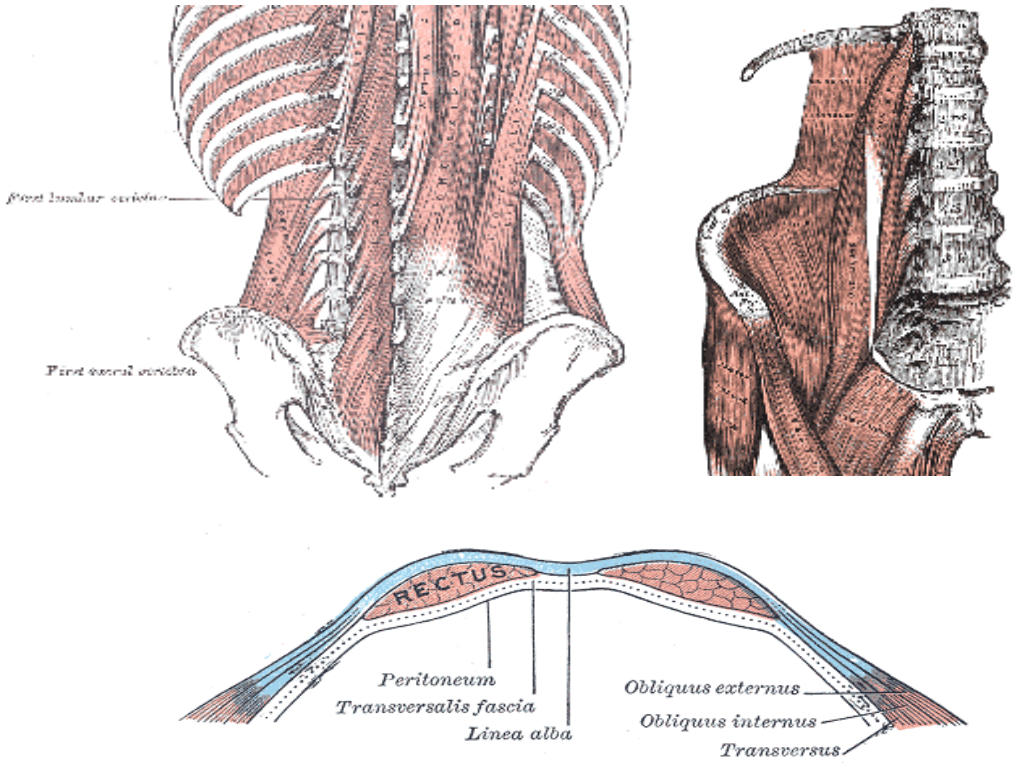


Figure 1. Low body 'passive and active systems' – Lumbar spine and back and abdominal muscles

The back area between the lower ribs and the buttock creases is commonly referred to as low back (Figure 1, above). Functionally, the anatomical structures of the low back can be divided into two principal types – passive and active ones (Panjabi, 1992). The lower torso skeleton forms the passive structures, which include the lumbar and sacral spinal vertebrae and the pelvic bones with all their intervertebral discs, joint articulating surfaces, menisci, joint capsules and many ligaments. The lower torso muscles form the active

structures of the low back, which include the Multifudi (MF), Erector Spinae (ES), Latissimus Dorsi LD), Quadratus Lumborum (QL), Psoas (Ps), the hip flexors and extensors, as well as the different abdominal muscles – Rectus Abdominis (RA), External and Internal Obliques and Transversus Abdominis (TrAb), the diaphragm and other thoracic muscles (Dutton, 2008). The principle function of the passive and active structures is to simultaneously provide stability to the body while allowing for a free expression of movements within a biomechanically determined physiological range.

The passive structures' design is such that it only permits limited segmental forward, backward and side bending, as well as rotation, preventing vertebral sliding and separation (Dutton, 2008). The passive structures are incapable of producing any stability by themselves – the spine buckles under its own weight (Cholewicki & McGill, 1996). The functional stability of the lumbar spine and low back relies on the co-ordinated activation of the many different muscles of the area – different activation patterns produce different types of stability. Segmental stability is achieved by contraction of predominantly deep axial muscles – deep MF, Ps, parts of the QL and TrAb, as well as the diaphragm. Global stability is achieved by co-contraction of the antagonistic multi-segmental superficial back and abdominal muscles – ES, superficial MF, RA, External and Internal Obliques (Cholewicki et al., 1997; Dutton, 2008; Hodges & Richardson, 1999b). Thus, optimal spinal and low back stability is the result of precise muscle activation patterns, which are generated by the third element of the system – the motor control executed by the Central Nervous System (CNS) (Panjabi, 2003; Panjabi 1992). Alteration of muscle activity in the low back can dramatically change the bio-mechanical forces on the passive structures of the spinal stability system. This in turn can produce supra-physiological loading and soft tissue damage (ligament and muscle attachment tears, disc deformity, joint surfaces damage) and lead to tissue deterioration and dysfunction. Panjabi (2003) proposed that

this tissue damage leads to a decrease of the intrinsic stability of the passive structures (increase in the neutral zone of the spine), which might be the principle cause of non-specific back pain. It is worth pointing that all the structures of both the passive and active systems are extensively enervated by mechanoreceptors involved in proprioception, which can also act as nociceptors (Khalsa, 2004), so any abnormal loading of the passive structures, which threatens the systems integrity is experienced as painful and alarming and can in turn alter motor control (Zedka et al., 1999; Prochazka & Yakovenko, 2002). As the active parts of the stabilising system are under the intimate control of the CNS, at both the spinal cord reflex level as well as higher brain centres, it is tempting to conclude that low back problems and especially non-specific CLBP are direct results of inadequate or incorrect motor control and in turn pain from the structures in the low back can alter CNS motor control. What the exact relationship between pain and neural muscle control is and how pain alters muscle activity and general motor strategy are questions awaiting a definite answer (Zedka et al., 1999; Hodges, 2001; Hodges & Moseley, 2003; Van Dieen et al., 2003).

To summarise, it appears that different patterns of activity of the low back and abdominal muscles are both the cause and a product of CLBP and these patterns can distinguish low back pain sufferers from healthy individuals. If that was the case, diagnosing back pain would be simple and straightforward. Unfortunately, that is not the case. Clinically and experimentally muscle activity patterns are almost impossible to determine objectively – many tissue stiffness and tension devices have been tried, but none has been accepted (Arokoski, 2005; Bizzini & Mannion, 2003). The principal established investigative tool used to examine muscle activity is measuring the electrical signal generated by a contracting muscle – electromyography (EMG). The electrical muscle activity can be recorded using invasive needle and wire electrodes and non-invasive surface electrodes.

Each of these methods has its advantages and disadvantages. The invasive electrodes give precise information about the activity of the individual muscle fibres close to them, but do not capture the general activity of the whole muscle (De Luca, 2006) and are more expensive and complex to use, requiring higher level of skill and expertise. On the other hand surface electrodes are easy to use and can pick up the electrical activity of the entire muscle, but also any nearby muscle – the so-called crosstalk. Normally, muscle contraction generates only micro volts of electrical current and the EMG signal can be very easily contaminated by the heart electrical signal, as well as any other electrical source, including the EMG equipment itself (Hu et al., 2009).

Setting the technical difficulties in accurately capturing and recording the EMG signal aside, another factor further complicating surface electromyography (sEMG) is the variability of the skin and subcutaneous tissues electrical resistance. The electrical resistance of the skin and subcutaneous tissue varies largely from person to person, as well as changes in accordance with the physiological status of the body (De Luca, 2006), for which reason raw sEMG signals are not representative of true muscle activity and need to be normalised. It is also worth pointing that the muscles, both posterior and anterior ones, acting on the lumbar spine are positioned at different depths, often overlapping each other. SEMG can reliably be used only to assess the surface muscles of the low back and abdomen. This is one of the principle reasons why with all the advances of modern medical technology, investigating the low back muscle activity patterns in their entirety remains problematic. None the less, sEMG has helped to provide some insights into the low back muscles activity in CLBP, which will be covered in greater detail in the following paragraphs.

2. Muscle activity aspects of CLBP

A Pub Med central database of controlled trials literature search of articles on muscle activity, EMG, and back pain returned over 200 results. The vast majority of papers used EMG experimental protocols to study different aspects of the low back and abdominal muscle patterns, assumed to take part in spinal stabilisation and back pain. The experimental protocols used by different researchers varied widely to the type, number, state, timing of activation, movement, perturbation and fatigability of muscles monitored, as well as the particular EMG measures used. It is not surprising that the results were often difficult to reconcile and interpret from a singular theoretical perspective (for a review see Van Dieen et al., 2003; Geisser et al., 2005). Here will be covered only the methods, which provide a more clear-cut distinction between CLBP sufferers and healthy controls – the flexion relaxation phenomenon of the para-spinal muscles (MF and ES) and the anticipatory contraction of the deep spinal (deep Multifidi - MF) and deep abdominal (Transverse Abdominis - TrAb) muscles.

Most studies of static back muscles monitored the para-spinal Multifidi (MF) and Erector Spinae (ES), and most specifically the Longissimus Lumborum, in standing, prone and fully flexed position using surface EMG (sEMG). The only EMG measure, which was highly reliable and reproducible (Chronbach's $\alpha > 0.80$), sensitive (93%) and specific (75%) in distinguishing CLBP sufferers was the flexion-relaxation ratio (Watson et al., 1997; Watson et al., 1995; Geisser et al., 2005). In brief, the flexion-relaxation ratio is a measure, which reflects the reflexive silencing of the para-spinal muscles at fully bent forward position (Watson et al., 1997; Watson et al., 1995).The measure is the ratio between the EMG of ES and MF activity (Root Mean Square -RMS) of the forward bending and the EMG of the same muscles while at fully bent position. In these studies (Watson et al., 1997; Watson et al., 1995) CLBP sufferers demonstrated much lower ratio, mainly due to increased muscle

activity in the fully bent position, but also due to the subdued muscle activity in the active phase of bending down. There are few possible mechanisms of muscle control, which can explain the flexion-relaxation phenomenon. Local spinal reflexes are likely to play a role originating from antagonistic muscles as well as spinal ligaments' and the para-spinal muscles' receptors themselves (Colloca & Hinrichs, 2005; Brumagne et al., 2000). There is also a very strong likelihood of involvement of higher CNS motor planing of guarding against forward bending due to fear of re-injury – repeated forward bending increases the range of both the bent down position and the flexion-relation ratio in healthy subjects (Dickey et al., 2003).

The second distinctive finding in EMG studies of muscle activation patterns in CLBP is the delayed contraction of deep abdominal and para-spinal muscles – the TrAb and the deep fibres of the MF in active limb movements. Hodges and Richardson (1999a; 1999b) used wire electrode EMG recordings of multiple abdominal and limb muscles to determine the precise activation timing of each of the muscles in respect to limb perturbations. Their findings demonstrated that in healthy subjects the different abdominal muscles are activated independently of each other when the limbs are moved – TrAb activation precedes the perturbation, while the other abdominal muscles activity coincides with the limb muscles activity. They interpreted this 'anticipatory' phenomenon to be a product of 'feed-forward' strategy of the motor control system, in order to stabilise the torso and spine prior to limb movement. In fast and intermediate speeds of limb movements CLBP patients demonstrated distinctive lack of the TrAb 'anticipatory' phenomenon in comparison to healthy controls (Hodges & Richardson, 1999a). Interestingly, experimental muscle pain, induced by hyper-saline injection in the ES also abolished the 'anticipatory' TrAb contraction in healthy volunteers (Hodges et al., 2001; Moseley et al., 2001), pointing to the complex relationship, which exists between pain and motor control.

Similar lack of 'anticipatory' activation of the deep fibres of the MF compared to the activation of the superficial fibres of the MF during arm perturbation were observed by MacDonald et al. (2009). In their study of asymptomatic CLBP sufferers, the EMG timing measurements were obtained via wire electrodes inserted under ultrasound deferentially in both the deep and the superficial fibres of the MF. The 'anticipatory' deep MF activation was present in both sides of the healthy controls, but only on the non-painful side of the CLBP patients. These results point to the independent motor control of the deep MF, which was altered in the CLBP participants even in the absence of pain. MacDonald et al. (2009) interpreted the results as a dysfunctionality of motor control, which caused inadequate spinal stabilisation and was a likely cause for the recurrent nature of CLBP. Results from both ultrasound and MRI imaging studies further confirmed these findings – low back injury produced a marked wasting of MF, which was limited to only the injured side and segment (Hides et al., 1994; Kader et al., 2000). Furthermore, the MF deterioration did not resolve spontaneously even after the acute low back pain episode had subsided (Hides et al., 1996) and involved both quantitative and qualitative organic changes to the muscle fibres composition (Demoulin et al., 2007a).

At first glance these two principal phenomena of CLBP muscle activity appear to contradict each other. The 'anticipatory' phenomenon EMG studies, supported by the findings of the imaging studies, draw a picture of CLBP to be a condition of diminished spinal stability, caused by weakened and under-active deep MF and TrAb muscles. On the other hand, the flexion-relaxation phenomenon studies demonstrate lower relaxation ratio with increased para-spinal, including MF muscle activity at the fully bent forward position, which is biomechanically most challenging for spinal stability. In other words, what characterises MF muscles activity in CLBP is a pattern where they are simultaneously less active when they should be active and more active when they should be switched off. The most likely

explanation of this discrepancy lies in the method by which MF activity is measured in the different studies. The deep MF' s 'anticipatory' activation is measured by precisely inserted wire electrodes, while the flexion-relaxation of MF relies on surface EMG measurement. In sEMG, MF are most commonly measured by placing bipolar electrodes on the skin 15-25 mm beside L5 vertebrae (Hekmens et al., 1999). This placement is more likely to capture signal coming only from the most surface MF fibres, which co-contract with the ES (MacDonald et al., 2009) as well as possible cross-talk from other muscles in the area. Interestingly, a wire EMG study (Andersson et al., 1996) found flexion-relaxation silencing of ES in healthy volunteers to be associated with concomitant contraction of Quadratus Lumborum (QL), which is a deep lumbar muscle, assumed to play a stabilising role in combination with TrAb for the low body and lumbar spine (Dutton, 2008). The lack of the flexion-relaxation phenomenon in CLBP sufferers is likely to reflect a muscle activation pattern, where the lack of intrinsic spinal stability, which is caused by dysfunction and under-activity of the deep spinal stabilisers, as well as possible intrinsic instability of the passive spinal structures, leads to compensatory activation of the surface back muscles – ES and the superficial fibres of MF.

The spinal instability model, developed by Panjabi (1992; 2003) to explain CLBP from bio-mechanical perspective, stipulates that spinal stability depends on the activity of the low trunk muscles and specifically on simultaneous co-activation of the antagonistic muscles (muscles whose action produces movements in opposite direction) of the low torso. This model has been extensively studied using EMG measurements of low body muscles in both static and dynamic experimental paradigms (for review see Demoulin et al., 2007b). The most striking feature of these studies is the choice of measured muscles – the back Erector Spinae (ES) and Latissimus Dorsi (LD) and the front Rectus Abdominis (RA), External Oblique (EO) and Internal Oblique (IO) (Cholevwicki & Van Vliet, 2002;

Cholewicki et al., 1997; Gardner-Morse & Stokes, 1998), without the inclusion of any of the deep low body muscles. The results of these studies confirmed that co-activation of antagonistic muscles is required for the stability of the spine, but they also pointed, that this co-activation increases the compressive loading of the spine (Gardner-Morse & Stokes, 1998). The results of two studies (Marras et al., 2000; Davis et al., 2002) of the role of psychological stress and spinal loading shed further light on the effects of co-activation of the same external back and front muscles – ES, LD, RA, EO and IO. Both psychological stress and mental processing increased the co-activation of these antagonistic muscles, which translated into higher compression forces on the passive structures of the spine and increased the risk of low back injury (Marras et al., 2000; Davis et al., 2002).

To summarise, both co-activation of external low torso as well as activation of the deep para-spinal MF and deep abdominal TrAb muscles increase spinal stability. The difference between these two distinct stabilisation patterns lies in the bio-mechanical and functional effects they produce on the spine and low body movements. External muscle co-activation increases spinal compressive loading and restricts body movements, while internal deep axial back and abdominal muscles contraction stabilises the spine segmentally, allowing for precise control and free expression of movements (Hodges & Moseley, 2003). This is obvious in the rigid and dysfunctional posture control and body movements, observed in sufferers of low back pain (Brumagne et al., 2008). In order to produce realistic and accurate biomechanical models of spinal stability in health and low back pain it is essential to include the deep back and abdominal muscles as part of these models. As at present the capacity to study the functioning of the deep torso muscles is very limited, a lot of the questions surrounding spinal stability and back pain will have to wait for a definite answer, which will only come after the development of new methods and technologies to study

dynamically muscle activation patterns.

The preceding paragraphs illustrate the inherent difficulties of studying and diagnosing the precise pathology of non-specific CLBP – it is virtually impossibility to determine experimentally, let alone clinically, the exact low back muscle activation patterns in each individual case and the specific structures, which give rise to the pain sensation. Not surprisingly, none of the many clinical examination and investigation techniques used to diagnose CLBP yields consistently satisfactory results (Rubinstein, 2008). The diagnostic difficulties limit the diagnosis accuracy, treatment specificity and effectiveness and make the prediction of treatment outcomes impossible and unreliable (Kumar & Clark,1994; Savigny et al., 2008). Hence, the purely bio-medical approach to CLBP has been superseded by the bio-psycho-social model, which views CLBP as a result of general psycho-neural dysfunction, caused by the interplay of psychological emotional and cognitive processes with chronic pain (Waddell, 1996; Turk & Okifuji, 2002; Gatchel et al., 2007).

3. Psycho-neural aspects of CLBP

When analysing CLBP it is very easy to forget, that this condition detrimentally affects the daily lives of real people on every possible level – physical, mental, emotional as well as on family and wider social level. A qualitative study (Corbett et al., 2007) of six patients living with CLBP clearly illustrated the emotional and mental turmoil of hope and despair that characterised their lives. The study also highlighted uncertainty, worry and fear of the future, social context of living with pain and impact on self as being the most prominent psychological aspects of CLBP. The realisation of the un-separability of the mental, emotional and bio-physiological factors in chronic pain, as well as other chronic health conditions, has led to the development of the bio-psycho-social model of chronic pain

(Waddell, 1996).

In the past twenty years psychological cognitive-behavioural theories of fear avoidance and coping as well as stress processing have been adapted to study chronic pain conditions in an attempt to explain how and why some individuals develop such conditions (Vlaeyen & Linton 2000; Vlaeyen et al., 1995; Turk & Okifuji, 2002). Central concept in these theories is the fear of pain and re-injury, further developed by Waddell et al. (1993) into the fear-avoidance beliefs about physical activity and work, which the authors found to be strongly related to disability and loss days of work, above other biomedical specifics of the pain conditions. The results of a number of independent studies consistently demonstrated the close relationship between fear of movement, pain catastrophising, depression/ negative affective state and disability (Leeuw et al., 2007; Vlaeyen et al., 1995). The most commonly used reliable and valid psychometric tools for measuring fear of movement and pain are the Tampa Scale for Kinesiophobia (TSK) (French et al., 2007; Sweenkels-Meewisse et al., 2003) and the Pain Catastrophising Scale (PCS) (Sullivan et al., 1995; Picavet et al., 2002).

Pain catastrophising is a negative cognitive and emotional mental orientation set, which is characterised by excessive focussing on pain and potential pain-inducing events and is closely connected with hyper-vigilance, rumination, depression and other negative affective states (Sullivan et al., 2001; Richardson et al., 2009). Pain catastrophising leads to withdrawal from many rewarding daily activities and especially avoidance of physical activity, which severely restricts chronic pain sufferers' behaviour. It is closely related to self-reported depressive symptoms and is associated with increase of sensory perception of induced pain in CLBP patients (Richardson et al., 2009). As a measure, it is consistently a good predictor of both back pain recurrence and persistence of pain in back pain

sufferers, as well as emergence of back pain in pain free individuals, even after adjustments for pain duration, pain severity and disability (Picavet et al., 2002). These findings further confirm the multi-factorial nature of CLBP, where cognitive and emotional factors are inseparable part of the clinical condition, and possibly play a crucial role in its aetiology.

Co-morbidity of chronic pain, depression and anxiety spectrum disorders is very high in both developed and developing countries (Tsang et al., 2008). Miller and Cano (2009) in a study of 1179 Michigan, USA residents found 21.9% prevalence of chronic pain out of which 35% had a co-morbid depression. Although their initial results pointed towards older females to be more likely to suffer either chronic pain or depression, further regression analysis revealed that co-morbidity is more common in younger sufferers compared to chronic pain alone.

A review (Edwards et al., 2006) of pain catastrophising in arthritis, fibromyalgia and other rheumatic conditions analysed data from both cross-sectional and longitudinal studies. Their results further confirmed that catastrophising is positively related across different musculoskeletal conditions with pain severity, affective distress, pain experience, poor outcome of pain treatment and pain-related disability, even after controlling for depression. They hypothesised that catastrophising exerts its harmful effects via multiple mechanisms - detrimental pain-coping, increased attention to pain, alteration of pain processing and social maladaptive alterations.

Besides the similarities of psychological factors involved, chronic pain conditions (Rheumatoid and other auto-immune conditions, Fibromyalgia, Chronic fatigue syndrome and other non-specific pain conditions) share many other physiological and neuro-

endocrine similarities - lower energy status, abnormal function of the sympathetic nervous system and Hypothalamus-Pituitary-Adrenal function with ensuing neurological, endocrine, immunological and other general metabolic disturbances (Sudhaus et al., 2008; Fishbain et al., 2004; Demitrak, 1997; McBeth et al., 2007; Gaab et al., 2005; Geiss, 1997; Clauw & Chrousos, 1997; Goldenberg, 2009; Bruehl & Chung, 2004). The similarity in pathology (more functional, rather than organic abnormality) between these conditions points to the existence of overlap between pain, emotion/cognition and neuro-endocrine regulation in the central nervous system (CNS) (Campbell & Edwards, 2009; Chapman et al., 2008).

This inter-disciplinary psycho-neuro-endocrine approach has been predominantly applied in the study of stress, depression and mood disorders. Neuro-imaging studies of fear, stress and anxiety disorders have pointed to the central role played by the Amg in these conditions (Shin & Liberzon, 2010). Further research in the area of emotive and mood disorders has identified the role played by the extended Amygdala (Amg) in connection with the prefrontal cortex (PFC) in 'central regulation' – to direct the orchestrated response of the CNS via modulation of Hypothalamic metabolic and endocrine output, mid-brain and brainstem autonomic balance, Peri-Aquaductal Grey (PAG) and the descending pain inhibitory system (Gold & Chrousos, 2002; Cardinal et al., 2003; Phelps & leDoux, 2005; Neugebauer et al., 2004; Walker et al., 2009; Price & Drevets, 2010; Apkarian et al., 2005).

The extended Amygdala (Amg) is a functional concept of structurally and circuitry similar and anatomically close nuclei in the basal forebrain, proposed by Heimer (Heimer & Alheid, 1991) to play a major role as interface between the limbic and motor systems, which influences both approach (reward) and avoidance (punishment) behaviour. The extended Amg includes the medial part of NAc (shell), the central nucleus of the Amg

(cAmg) and the bed nucleus of the stria terminalis (BNST). Two different MRI studies shed further light on the patterns of Amg activation in response to induced pain in fibromialgya (Gracely et al., 2004) and healthy individuals (Semionowicz & Davis, 2006) – Amg activation increases were closely related to catastrophising, which coincided with reduction of activity of the PFC, a cortical area implicated in moderating the pain response (Semionowicz & Davis, 2006).

The extended Amg's close anatomical and functional connection with the striatum (Haber, 2003; Haber et al., 2000; Ikemoto, 2007) and reciprocal connections with the Parabrachial nuclear complex (PBN), led Balaban (Balaban & Thayer, 2001; Balaban, 2002) to propose PBN-Amg substrate to be the principle link between anxiety and balance control. Over the past few years the effects of emotional factors on posture and motor control have been studied extensively. Different studies employed various experimental protocols to create anxiety and measure sensory and postural changes with consistently similar results – anxiety and alteration of posture control are equally linked in younger and older adults (Brown et al, 2006), children (Erez et al, 2004) as well as mice models (Lepicard et al, 2003).

Hillman et al. (2004) examined the effects of emotive picture viewing on postural control in healthy under-graduate students. Their results confirmed that both positive and negative emotion pictures increased arousal (Galvanic skin response) and only negative pictures increased the Startle reflex (alarm). Interestingly, only negative emotion pictures were also associated with changes in posture – female participants leant more backwards, while male ones leant more forward.

Another comprehensive study (Bolmont et al, 2002) established that the postural changes,

associated with negative mood states (tension, depression, hostility, fatigue) and anxiety in healthy subjects, resulted from changes in sensory information processing and motor control. The negative mood states and anxiety impaired the ability of the participants to adequately utilise postural sensory information (visual, somato-sensory and vestibular) as well as increased the latency of their motor response (correcting posture after unbalancing perturbation). Interestingly, similar rigid and dysfunctional balance strategy was observed in CLBP patients (Brumagne et al., 2008). In this study CLBP participants exhibited significantly different posture control strategy, favouring ankle muscles', rather than para-spinal muscles' proprioceptive control, even when this strategy was inappropriate (standing on unstable surface).

All the previous paragraphs clearly illustrate the existence of overlapping CNS neural regulatory circuits, centred around the extended Amg. This 'central regulating system' (Figure 2, below) simultaneously co-ordinates the metabolic state, emotional state, attention, cognition as well as modulating motor patterns, in order to generate coherent behavioural response of the organism in relation to its needs . Pain usually signals tissue damage or potential for tissue damage, which raises an alarm response and evokes negative emotional experience (Merskey et al., 1979). So it is not surprising that nociception can exert a potent modulation of the 'central regulating system' – the ascending nociceptive pathways terminate preferentially in the Thalamus and PBN (Siegel & Sapru, 2006), which are closely connected with the Amg (Balaban, 2002; Balaban & Thayer, 2001). To summarise, on the one hand emotional factors can affect 'central regulation', which alters motor control and on the other hand pain can directly modulate the 'central regulating system' and alter emotional state.

The precise interactions between pain, emotional/cognitive state and motor control are

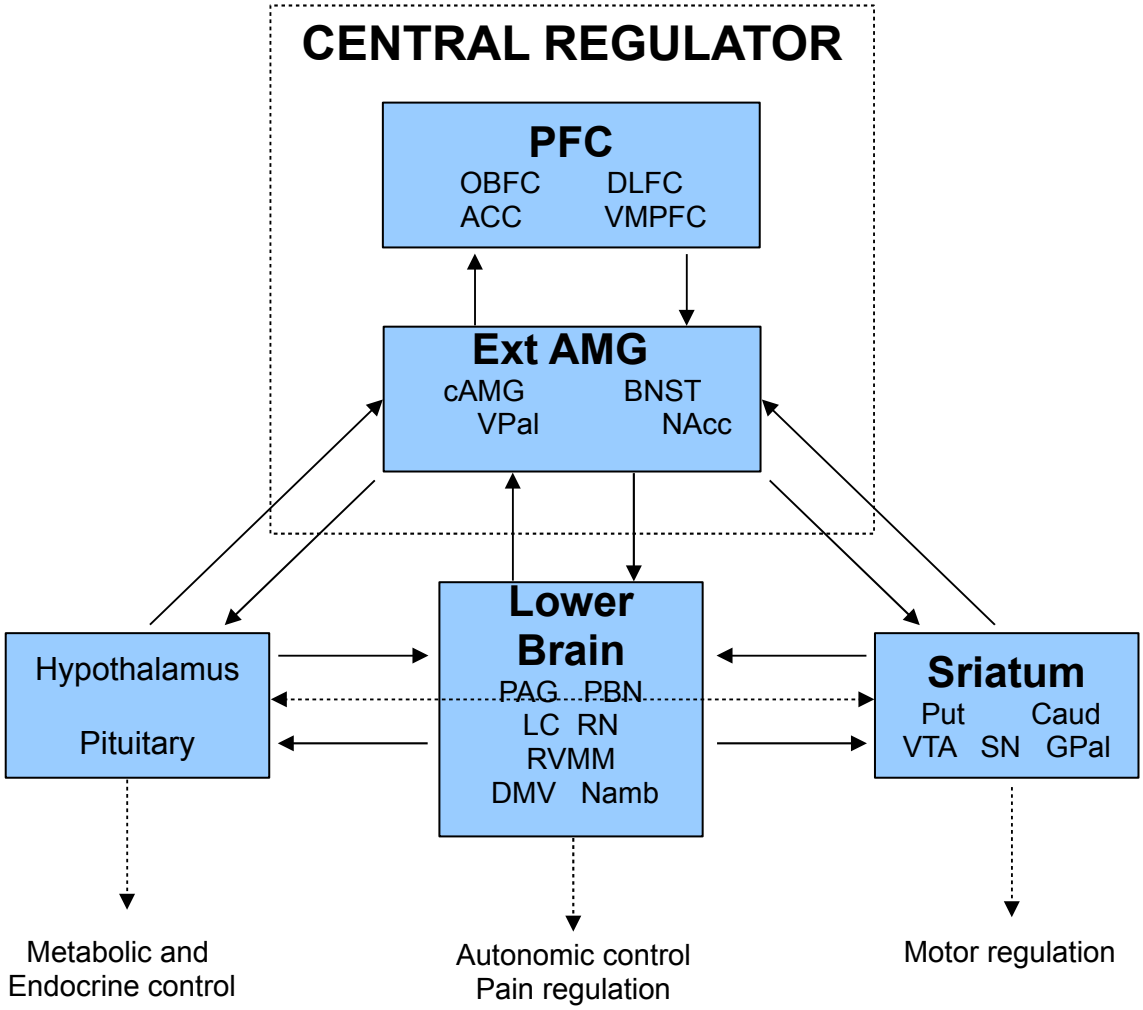


Figure 2. The 'Central regulator". PFC – Pre-Frontal Cortex; OBFC – Orbito-frontal Cortex; ACC – Anterior Cingulate Cortex; DLFC – Dorso-lateral frontal Cortex; VMPFC – Ventr-medial Pre-frontal Cortex; AMG – Amygdala; cAMg – Central nucleus of Amg; VPal – Ventral Pallidium; BNST – Bed nucleus of Stria Terminalis; NAcc – Nucleus Accumbens; PAG – Peri-Aqueductal Grey; PBN – Parabrachial Nuclei; LC – Locus Coeruleus; RN – Rafae Nuclei; RVMM – Rostro-ventral medial Medulla; DMV – Dorsal Motor of Vagus; NAmb – Nucleus Ambiguus; Put – Putamen; VTA – Ventral Tegmental Area; SN – Substancia Niagra; Caud – Caudate; GPal – Globus Pallidus.

difficult to determine in the aetiology and pathology of CLBP (for an insightful review see Hodges & Moseley, 2003). Are emotional and personality aspects the ones which predispose some individuals to injure their low back by adopting inappropriate postures (spinal stabilisation strategies), which lead to uncontrolled loading of the spine and injury, or on the long run produce a slow accumulative damage to the structures of the passive system, prior to the injurious event? Or are the negative emotional and cognitive states, and the altered low body muscle activity in CLBP only a consequence of the persistent pain itself? Is it more important to treat the pain, the spinal muscle dysfunction, or address

the emotional and cognitive sides of CLBP? The answers of these questions are paramount, if treatment and prevention of CLBP are to be successful.

4. The present study

The present study employed moderate treadmill exercise as an intervention to test the psycho-neural and motor control aspects of CLBP. Exercise is well accepted as an activator of 'central function' and treadmill exercise has been shown to raise mildly both cortisol and ACTH in healthy adults (Luger et al., 1987). As a behaviour, moderate exercise or general physical activity require a synchronised and co-ordinated control of the body on every possible level – physiological (to meet metabolic demands), motor control (for the planning and execution of movements), as well as attention and cognition (to effectively obtain and process an increase of external and internal information, associated with movement). In this respect exercise can be viewed as a modulator of 'central function', which is capable of rectifying any internal dysfunctions of the 'central regulating system' and is paramount for the health of both the body and 'mind'.

Nowadays exercises of many types are widely used by millions of people to keep fit, de-stress and for general well-being. As a therapeutic intervention, exercise is used most commonly in the rehabilitation of musculo-skeletal, but also in cardiac, respiratory, as well as obesity, diabetes and many more health conditions . Therapeutically, exercise has shown promising results not just for the treatment of CLBP (Hayden et al., 2005), but also for Fibromyalgia (Busch et al., 2007), Chronic Fatigue Syndrome (Larun et al., 2004) and depression (Mead et al., 2008). As a therapy for CLBP, exercise is significantly more effective than no treatment and is as effective as cognitive behaviour therapy (CBT) in reducing pain catastrophising, pain and disability perception and depression (Smeets et al., 2006). Moderate exercise is very safe and it was not associated with any adverse

effects by any of the above-mentioned Cochrane reviews (Hayden et al., 2005; Busch et al., 2007; Larun et al., 2004; Mead et al., 2008).

Exercise is extensively used in the treatment of CLBP and there are many different approaches and opinions to what constitutes an effective exercise program (Hayden et al., 2005; Keller, 2006; Standaert et al., 2008; Arokoski et al., 2004; Hides et al, 2001; O'Sullivan et al., 1998). The purpose of the present study was not to examine and determine how effective moderate treadmill walking exercise is in the treatment of CLBP – for this purpose a much different, longitudinal trial design would be necessary. This study employed exercise as a mild and safe perturbation for the system, which modulates 'central function' with corresponding physiological, emotional/cognitive and motor control alterations. The analysis of the system's reaction to the perturbation was used to clarify the interaction between the different factors involved in CLBP.

From a CLBP model of complex interaction between psycho-neural and bio-mechanical factors involved in motor control, the following hypothesises were formulated:

1. Both emotional/cognitive and back muscles function measures distinguish CLBP participants from healthy controls
2. Moderate exercise produces the same neuro-psychological effects in both CLBP participants and healthy controls, which results in reduction of negative emotional states
3. Alteration of the negative emotional state leads to reduction of pain experience in the CLBP group
4. The reduction of negative emotional state and pain in CLBP participants is associated with alteration of back muscles activity

Section II - Method

The present experiment was designed as a between group comparison with repeated measurements. The two groups, patients suffering chronic low back pain (CLBP) and healthy volunteers (Control), performed a moderate physical activity – 15 minutes walking on a treadmill (Woodway, Germany) at 55% of maximal effort, which was calculated using the HR reverse method – [HR max (220 minus age) minus resting HR] multiplied by 55% plus resting HR (Armstrong et al., 2005). The physiological parameters, which were measured pre- and post- intervention were: resting heart rate (HR) and blood pressure (BP); present pain intensity (PPI); negative affect (NA); static sEMG of ES and MF para-spinal muscles. Heart rate and BP were used as indicators of physiological activation (Armstrong et al., 2005). Information on possible demographic co-founding factors: age; ethnic origin; exercise level; pain duration and general pain level; and psychological emotional and cognitive factors (depression, anxiety, stress and pain catastrophising), was also collected with the use of a questionnaire.

1. Ethics approval

Ethics application (Appendix 1) was submitted in accordance with Roehampton University (RU) guidelines and ethics approval was given by the School of Human & Life Sciences Ethics Committee. As part of the ethics application, the principal risks associated with the experiment were assessed and found to be very low for each of the activities and procedures involved (Appendix 2).

Health and safety of the participants and university students and staff were paramount at all times and health and safety rules and procedures were strictly adhere to at all stages of the experiment.

2. Participants

Participants were recruited predominantly from personal contacts and Roehampton University (RU) student volunteers. Due to time restrains (NHS lengthy ethics application process), NHS ethics approval was not applied for, which prevented the recruitment of NHS CLBP patients. After RU Ethics approval was obtained, recruitment posters (Appendix 3) and participant's information sheets (Appendix 4) were distributed in many suitable locations around Roehampton University. Back pain charities, as well as private physiotherapy, osteopathic and chiropractic clinics were contacted with very minimal response. The original aim was to recruit between 20 and 30 CLBP patients and to randomly choose 10 to take part in the study. Unfortunately, only 10 CLBP volunteers came forward, so all of them were included in the study. This fact made the study sample more of an opportunistic one, which poses serious questions about the general validity of the study's results – is the sample truly representative of CLBP.

All volunteers, that came forward, were screened for suitability. All the suitable participants were debriefed about the procedures involved in the experiment and reassured that their participation is fully voluntary and they can terminate their participation at any time of the experiment, without giving any reasons for it.

The main inclusion criteria for participation were as follows:

Study group (CLBP): 10 chronic low back pain male participants

Inclusion criteria:

- Age – 24 to 64
- BMI – 18 to 25
- Low back pain localisation – predominantly concentrated over the lumbar and

back of the hips area, but also radiating to the legs

- Low back pain duration – a minimum of 12 consecutive weeks in the past 12 months
- Pain severity – any level of pain for which medical treatment has been sought
- Ability to walk briskly for 15 minutes
- Good command of written and spoken English

Exclusion criteria:

- Inflammatory rheumatic conditions: Rheumatoid arthritis, ankylating spondylitis, psoriatic arthritis, SLE and other auto-immune conditions
- Spinal stenosis, severe discopathy, spondylolisthesis and other back deformities
- Spinal dislocations, fractures and operations
- Other recent unresolved traumatic injuries
- Mental illness or clinical depression, requiring medication
- Cardiac and pulmonary diseases, requiring treatment
- Steroid, β-blockers or other metabolicly active medication
- Mouth and gum diseases

Control group (Control): 10 healthy male volunteers, who hadn't suffered low back pain in the preceding 6 months and had similar vital characteristics: 24-64 years old and BMI of 18 to 25, with good command of written and spoken English.

3. Measurements

As the principal aim of the study was to investigate the interaction between psycho-neural and back muscles activity factors in CLBP, the main measurements, which were taken

repeatedly before and after the exercise intervention were: the Present Pain Intensity (PPI), the Negative affect (NA) and the standing and fully bent forward sEMG of Erectus Spinae (ES) and Multifidi (MF) back muscles and their Flexion-relaxation Ratio (FRR). Resting heart rate (HR) and blood pressure (BP) were measured to assess the state of general physiological activation. Demographic characteristics like age, ethnicity, level of exercising, as well as pain specifics and psychological emotional/cognitive inclination were also assessed for possible co-founding effects, with the help of a questionnaire - Back Pain questionnaire (BPQ - Appendix 6).

Present Pain Intensity (PPI)

In order to capture the subjectively experienced by the participants pain at different stages of the experiment, Present Pain Intensity (PPI) was measured using a 100mm visual analogue scale (VAS). VAS is a horizontal line of 100mm, whose ends are anchored by 'No pain' at one end and 'Worst possible pain' at the other end. VAS is well accepted and extensively used in pain research, consistently demonstrating good reliability (Wewers & Lowe, 1990).

As exercise's effects as central activator alter neuro-endocrine physiological state of the body, they can potentially alter the subjective pain experience, as well as the affective state. For this reason PPI was measured at three points in the experiment – after the initial rest period (PPI 1), immediately after the end of the exercise (PPI 2) and after the second rest period (PPI 3). The participants were asked to place a mark on the line at a place, which they felt reflected their pain at that moment. The distance from the beginning of the scale ('No Pain") were later on measured using a ruler and then recorded in millimetres. Scores possible were between 0 and 100 and higher scores indicated stronger pain.

Negative affect (NA)

Negative emotional and mood states are intimately connected with the pain experience, physiological state and motor control (as illustrated in Section 1, above). For this reason Negative Affect (NA) was measured at three time points - after the initial rest period (NA 1), immediately after the end of the exercise (NA 2) and after the second rest period (NA 3) with the use of a self-report questionnaire (Emotional State Questionnaire – Appendix 7).

The ESQ was based on the Positive and Negative Affect Schedule (PANAS) with the inclusion of further 9 items - 4 positive and 5 negative. The PANAS is a 20-item self-report measure assessing the frequency of experiencing positive affect (PA) and negative affect (NA), which can be used to measure both state and trait affect (Watson et al., 1988). PANAS is composed of two independent scales for PA and NA with high cross-cultural, ethnic and age internal consistency and reliability (Watson & Clark, 1994).

For the current study, only the results from the NA sub-scale from PANAS were used in combination with 5 extra items, in order to determine the NA state. NA was measured by asking participants to indicate what best described the way they felt at that particular moment of time. Each of the 15 items (descriptive words) was scored between 1 and 5, where 1 was “Very slightly or not at all” and 5 was “Extremely”. The sum of all the 15 scores of the scale was used as the NA measure and higher values indicated a more negative affect state.

Back muscles sEMG, Static ratio and Flexion-relaxation ratio (FRR)

The static sEMG of bilateral ES and MF were obtained in three different positions: prone, standing and fully bent forward at two time points in the experiment – after the initial resting period and after the second resting period, which followed the treadmill exercise.

SEMG was recorded using bi-polar active (x 1000 gain) electrodes (10mm in diameter and 10mm inter-electrode distance) and Biometrics sEMG system (DataLog P3X8) sampling at 1000Hz, the bandwidth was 20 - 450 Hz with a notch filter at 48.5 - 51.5 Hz. The electrodes were placed bilaterally on pre-prepared (shaved and cleaned with alcohol wipes) locations on the back, as recommended by the SENIAM protocol (Hekmens et al., 1999) – ES location was 40mm beside the mid dorsal line on the level of the first lumbar vertebrae; MF location was on a line linking the intervertebral space between the first and second lumbar vertebrae and the superior iliac spine, approximately 15-25 mm from the mid dorsal line beside the fifth lumbar vertebrae.

For reasons explained in the first section, for the EMG signal to reflect the actual muscle activity, it has to be normalised first. Commonly the EMG is normalised as a percentage of the RMS of maximum voluntary contraction, but this can be misleading as back pain patients may be reluctant (consciously or sub-consciously) to exert a full maximum contraction of their back muscles. In the present study the EMG normalisation was achieved by using RMS of the quiet lying down EMG to calculate two ratios reflecting the ES and MF muscle activity in the standing and fully bent forward position at two time points – the standing one was the ratio between the RMS of the standing EMG and the RMS of the lying prone EMG (Standing 1 and Standing 2); the bent forward one was the ratio between the RMS of the EMG at fully bent position and the RMS of the lying prone EMG (Bent forward 1 and Bent forward 2).

The flexion-relaxation ratio (FRR) is the principle measure used to study the flexion-relaxation phenomenon, which is consistently found to be absent in CLBP sufferers. FRR is calculated by dividing the EMG's Root Mean Square (RMS) of the back muscles activity during the downward forward bending and the EMG's RMS of the back muscles while fully

bent down (Watson et al.,1997). This method of calculating FRR relies on specific precise timing (1-5 seconds) for each of the stages of bending forward, holding the bent down posture and raising up, which poses practical problems. This study employed a slightly different method of calculation the FRR – only the holding of the fully bent forward position was timed (5 seconds) and the participants were instructed to bend forward and raise up in a relaxed manner as quickly as they could, but not timed. The FRR was calculated by dividing the averaged sum of the bending down and raising up EMG's RMS (1-2 seconds) and the RMS of the EMG of the fully bent position (5 seconds). The flexion-relaxation ratio (FRR) was then calculated for each of the bilateral ES and MF muscles at two time points - after the initial rest and after the post-exercise rest periods - (FRR 1 and FRR 2).

Heart rate (HR) and blood pressure (BP)

Heart rate and BP were measured as an indicator of the level of general physiological activation. For this reason only the systolic element of the BP was utilised. The resting heart rate and BP were measured using chest sensors (Polar, model RS800) and arm cuff (BOSO-Medicus, Germany) at three time-points of the experiment - after the initial rest period (HR 1; BP 1), immediately after the end of the exercise (HR 2; BP 2) and after the second rest period (HR 3; BP 3).

Demographic and psychological characteristics

As illustrated in the preceding section, the affective/emotional and cognitive aspects are a major factor in CLBP. A Back Pain questionnaire (BPQ – Appendix 6) was designed to measure participants' pain experience, attitude and general emotional and cognitive orientation (affect and trait) including established psychometric scales. It also included questions about general demographic characteristics, which had the potential to co-found the results: age, ethnic origin, exercising level, low back pain duration and overall intensity.

The psychological measurements were based on two well established parametric tools – the Pain Catastrophising Scale (Sullivan et al., 1995; Picavet et al., 2002) and Depression, Anxiety and Stress Scale-21 (Norton, 2007).

Pain Catastrophising Scale (PCS)

Pain Catastrophising Scale (PCS) was used to measure the negative mental orientation and attitude to pain and pain-inducing events (pain catastrophising), which is a crucial factor in CLBP and other chronic pain conditions (Sullivan et al., 2001; Leeuw et al., 2007; Richardson et al., 2009). The PCS is a 13 item scale of descriptions of pain experiences with high internal consistency and reliability (Sullivan et al., 1995; Picavet et al., 2002).

Participants were asked to score each of the 13 descriptive statements in respect of the degree to which each statement applied to them between 0 and 4, where 0 was “not at all” and 4 was “all the time”. The Pain Catastrophising was the sum of the scores of all the items in the PCS and higher scores indicated greater pain catastrophising.

Depression, Anxiety and Stress Scale-21 (DASS-21)

DASS-21 was used to measure the underlying general negative emotional and mood state. DASS-21 is a shortened version of the main 42 item Depression, Anxiety and Stress self report scale, which measures these three emotional and mood states.

DASS-21 is composed of three sub-scales for each of the three emotional and mood states, which have 7 items each (Lovidond & Lovibond, 1995)**.** The psychometric properties of the scale were tested in several studies (Brown et al, 1997; Lovibond and Lovibond, 1995; Henry & Crawford, 2005; Norton, 2007) and found to possess a high internal consistency and reliability across different cultural and ethnic groups.

DASS-21 was used only to measure the general background level of mood and affect. Each of the three sub-scales 7 items is a statement reflecting perceived experience, which was scored between 0 and 3, where 0 was “Did not apply to me at all' and 3 was “Applied to me very much, or most of the time”. The sum of the 7 scores of each sub-scale was used as the Depression, Anxiety and Stress measurement, where higher values indicated more perceived depression, anxiety and stress.

4. Experimental protocol

All the participants were debriefed about the procedures involved in the experiment, advised to abstain from alcohol for 24 hours and avoid eating and smoking for 2 hours prior to the test. To ensure as constant as possible a level of physiological status and address natural diurnal fluctuations of cortisol and DHEA, the time of the experiment was set for mid afternoon – 2-5pm.

The experiment took place in the biomechanics laboratory of the School of Human & Life Sciences, Whitelands College, Roehampton University, London SW15 on different days between March and July 2010. To insure that there were no variations or omissions in the experimental procedure, a Protocol sheet (Appendix 8) was created and used to record each participant's experiment.

On the day of the experiment, participants were familiarised with all the procedures and equipment involved in the experiment. After signed informed consent (Appendix 5) was obtained, the participants were given to fill the Back Pain Questionnaire.

Participants' backs and chests were then examined and body hair was shaven from the areas for electrodes attachment. A strap, which housed the ECG electrodes and transmitter (Polar, RS800) was fitted comfortably to the chest below the sternum.

Participants were then asked to lie down on a treatment bench, made comfortable and left to relax for 15 minutes in a quiet room at a constant temperature of 23ºC.

At the end of the 15 minutes rest period, the initial measurements (test 1) were taken:

1. Heart rate and BP – HR 1, BP 1
2. Saliva sample for cortisol and DHEA analysis – saliva sample 1
3. Present Pain Intensity (PPI) – PPI 1
4. Participants filled the Emotional State Questionnaire (ESQ) – ESQ 1
5. sEMG measurements of ES and MF in three positions – prone, standing and fully bent forward as follows:

Participants were asked to lie back on the bench in prone position and the locations for the sEMG electrodes were determined (as described above), verified by a second investigator and then marked. The electrodes were then placed (the reference electrode was attached to the olecranon) and the participants were left to relax for 30 seconds, then a 20 sec sEMG were recorded (EMG prone 1). The participants were then asked to stand up and stay still in a relaxed manner with feet shoulder apart and eyes fixed directly ahead at eye level on a mark on the wall. After a 30 seconds calming period, a 20 seconds sEMG were recorded (EMG standing 1). The participants were then asked to bend forward in a relaxed manner as fast as they could and as far as they were comfortable and stay in that position until they were given a signal (5 seconds) to raise up in a relaxed manner as fast as they could. After making sure that none of the electrodes were felt abnormally pulling, a second run of the same bending forward procedure was performed and the sEMG recorded (EMG bent 1).

The electrodes were removed from the participants' back and the treadmill exercise was initiated at 17.5 degree inclination. The speed of the treadmill was gradually increased until

the heart rate (HR) reached 55% of participants' max effort. Participants maintained walking for 15 min adjusting the speed of the treadmill in order to stay at 55% max effort level. After the 15 minutes treadmill walk, participants were sat down and a second set of measurements (test 2) were taken:

6. Heart rate and BP – HR 2, BP 2
7. Saliva sample for cortisol and DHEA analysis – saliva sample 2
8. Present Pain Intensity (PPI) – PPI 2
9. Participants filled the Emotional State Questionnaire (ESQ) – ESQ 2

The participants were then asked to lie back on the bench and relax for 15 minutes as at the beginning of the procedure. At the end of the 15 minutes rest a third set of measurements (test 3) were taken:

10. Heart rate and BP – HR 3, BP 3
11. Saliva sample for cortisol and DHEA analysis – saliva sample 3
12. Present Pain Intensity (PPI) – PPI 3
13. Participants filled the Emotional State Questionnaire (ESQ) – ESQ 3
14. sEMG measurements of ES and MF in three positions – prone, standing and fully bent forward as follows:

Participants were asked to lie back on the bench in prone position and the electrodes were then placed on the previously marked places (the reference electrode was attached to the olecranon) and the participants were left to relax for 30 seconds, then a 20 sec sEMG were recorded (EMG prone 2). The participants were then asked to stand up and stay still in a relaxed manner with feet shoulder apart and eyes fixed directly ahead at eye level on a mark on the wall. After a 30 seconds calming period a 20 seconds sEMG were recorded (EMG standing 2). The participants were then asked to bend forward in a relaxed manner as fast as they could and as far as they were comfortable and stay in that position until

they were given a signal (5 seconds) to raise up in a relaxed manner as fast as they could. After making sure that none of the electrodes were felt abnormally pulling, a second run of the same bending forward procedure was performed and the sEMG recorded (EMG bent 2).

The collected saliva samples were frozen at -20º C and later analysed (Salimetrics salivary cortisol ELISA) for cortisol and DHEA content (as part of a parallel running project by a fellow investigator).

The EMG recordings were uploaded from the DataLog storage card onto a computer, later analysed using Biometrics software and the Root Mean Square (RMS) of each EMG recording was determined.

The questionnaires, consent forms and protocol sheets were collated and placed in individual folders and later on entered into a computer and analysed using SPSS 17 program.

5. Data analysis

Descriptive statistics of the principal demographic variables were obtained (frequency, percentage, mean, SD). Independent samples t-tests were used to determine the baseline affective differences between CLBP and controls, as well as different psychological and muscle activity measurements. Repeated measures t-tests and ANOVAs, as well as two-way ANOVAs were used to compare the PPI, NA and the measurements of the ES and MF muscles at the different time points between CLBP and Controls. Correlations were performed on the variables, which showed statistical difference in order to establish what the relationship between them was. All results were considered significant at .05 level.

Section III – Results

This section covers the principle results from the study – the Present Pain Intensity (PPI), Negative Affect (NA) and static activity of the ES and MF back muscles in relaxed standing and fully bent forward position as well as their Flexion-relaxation ratio (FRR). It also presents the background demographic as well as psychological aspects of the studied CLBP and control groups.

1. Demographic statistics

Originally, the experiment was planned with equal groups of participants – 10 CLBP and 10 healthy controls. After the careful examination of the filled questionnaires, it was found that one of the CLBP (ID 9) participants did not fulfil the precise inclusion criteria, as he had only experienced an average pain of 10 (100mm VAS) for the preceding week and longer (personally confirmed after the experiment – D.L.), and our inclusion pre-requisite was at least an average pain of 20 (similar to many other studies). There were two further control participants (ID 1 and 13) who also reported an average preceding pain of respectively 10 and 12. For this reason he was transferred into the Control group and the two groups became slightly uneven – 11 controls and 9 CLBP participants.

Both groups were composed of almost equal numbers of white european and asian participants – CLBP group had 4 (44.4%) white and 5 (55.6%) asian participants; control group had 4 (36.4%) white and 7 (63.6%) asian participants. The differences were not significant – Chi-square test for independence revealed no association between ethnicity and CLBP status, Chi-square (1, n = 20) = .00, p = .71 (Table 1, Appendix 9).

The two experimental groups also had non significant age (controls - 31.55, SD = 6; CLBP

– 36, SD = 10) differences (Table 2, Appendix 9) – t = -1.24, p = 0.24, as well as level of exercising – Chi-square (3, n = 20) = 4.463, p = .22 (Table. 3, Appendix 9).

2. Background affective and cognitive characteristics

Depression, Anxiety and Stress. The self-reported Depression, Anxiety and Stress levels were measured by scales (DASS-21) with scores from 0 to 21. Although they were higher in the CLBP group (Tables 4, below), it was form a generally very low level , so there was significant difference only for Depression – t = -2.35, p = .03 (Tables 5, Appendix 9). Reliability analysis of the separate sub-scales of DASS-21 confirmed their internal consistency – respective Chronbach's α for Depression, Anxiety and Stress of .79, .67 and .76.

Table 4. Depression, Anxiety and Stress group Statistics

	group	N	Mean	Std. Deviation	Std. Error Mean
Depression	1 control	**11**	**2.36**	**2.461**	**.742**
	2 CLBP	**9**	**5.67**	**3.808**	**1.269**
Anxiety	1 control	**11**	**1.64**	**2.292**	**.691**
	2 CLBP	**9**	**3.44**	**2.555**	**.852**
Stress	1 control	**11**	**4.91**	**4.415**	**1.331**
	2 CLBP	**9**	**7.44**	**3.245**	**1.082**

Pain catastrophisig was measured using Pain Catastrophising Scale (PCS) with scores ranging from 0 to 52. As expected pain catastrophisig was much higher in the CLBP group 22 (SD=12.66) versus 4.55 (SD=5.87) in the control group – t = 3.81, p = .003 (Table 7, Appendix 9). Pain Catastrophising also strongly correlated with the self reported pain level, r =.79, n = 20, p < .001, where higher pain catastrophising was associated with higher level of pain (Table 8, Appendix 9).

Table 6. Pain Catastrophising Group Statistics

	group	N	Mean	Std. Deviation	Std. Error Mean
Pain Catastrophisisng	1 control	**11**	**4.55**	**5.871**	**1.770**
	2 CLBP	**9**	**22.00**	**12.659**	**4.220**

Although the links between pain, pain catastrophising and negative affective and mood

states are intuitive – pain is capable of inducing negative emotional states, and chronic pain alters both emotional and cognitive perceptions, correlation analysis between Pain Catastrophising, Depression, Anxiety and Stress revealed only a moderate non-significant positive association between Pain Catastrophising and Anxiety, r = .41, n =20 , p = .07 (Table 9, Appendix 9).

Furthermore, when only the CLBP group psychological characteristics were analysed, it became apparent, contrary to expectation, that there was no significant relationship between any of the affect and mood measures as well as the perceived pain level and Pain Catastrophising (Table 10, below). It appeared that in our CLBP sample there was no connection between chronic pain and mental/ emotional state, or in other words some of the CLBP participants, although reporting high levels of pain also reported low levels of distress. In comparison, in the control group the emotion and mood measurements of Depression, Anxiety and Stress correlated significantly with each other (Table. 11, below). These results pointed to great variations of the emotional/ cognitive state in the CLBP group, which was indicative of the heterogeneity of the group and possibly the nature of chronic low back pain in general.

Table 10. Correlations between Pain, Pain Catastrophising, Depression, Anxiety and Stress in CLBP

		pain level	Pain Catastrophisisng	Depression	Anxiety	Stress
pain level	Pearson Corr	**1**	**.478**	**.466**	**.215**	**-.039**
	Sig. (2-tailed)		**.193**	**.207**	**.578**	**.921**
	N	**9**	**9**	**9**	**9**	**9**
Pain	Pearson Corr	**.478**	**1**	**-.104**	**.182**	**-.061**
Catastrophisisng	Sig. (2-tailed)	**.193**		**.791**	**.640**	**.876**
	N	**9**	**9**	**9**	**9**	**9**

Table. 11 Correlations between Depression, Anxiety and Stress in Control

		Depression	Anxiety	Stress
Depression	Pearson Correlation	**1**	**.735****	**.905****
	Sig. (2-tailed)		**.010**	**.000**
	N	**11**	**11**	**11**
Anxiety	Pearson Correlation	**.735****	**1**	**.619***
	Sig. (2-tailed)	**.010**		**.042**

	N	**11**	**11**	**11**
Stress	Pearson Correlation	**.905****	**.619***	**1**
	Sig. (2-tailed)	**.000**	**.042**	
	N	**11**	**11**	**11**

**. Correlation is significant at the 0.01 level (2-tailed) *. Correlation is significant at the 0.05 level (2-tailed)

3. Heart rate (HR) and blood pressure (BP) effects of exercise

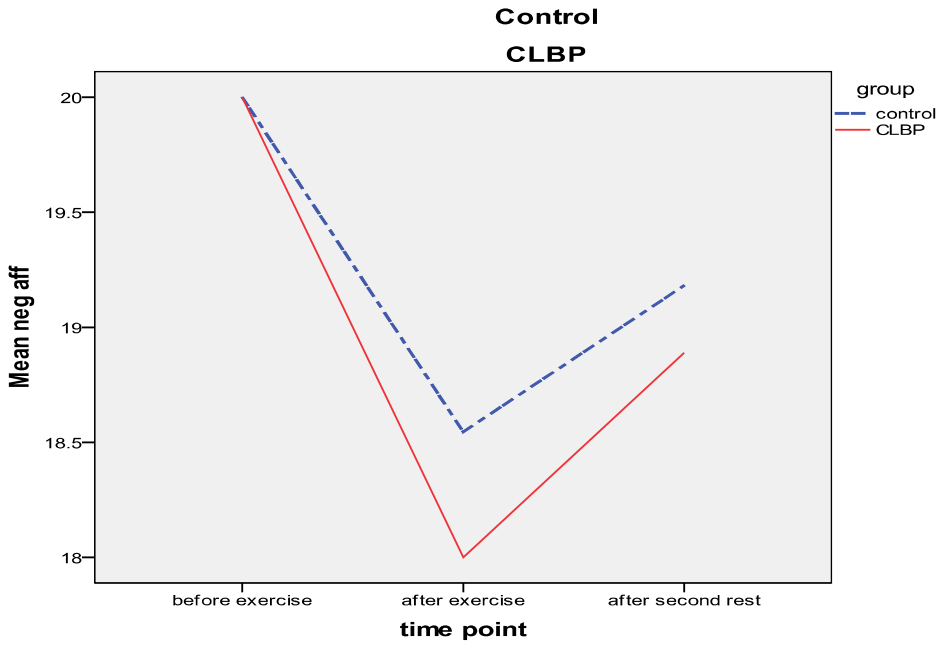
The moderate treadmill exercise produced, as expected, a generalised physiological activation in both the control and CLBP groups, which was reflected in the changes of the participants heart rate (HR) and systolic blood pressure (BP). A one-way repeated measures ANOVAs were conducted to compare HR and BP at three time-points - after the initial 15 minutes rest period, immediately after the 15 minutes treadmill exercise and at the end of the second 15 minutes rest period, in both controls and CLBP groups. The means and standard deviations are presented in Table 12, below. The exercise produced a significant effect on HR in both groups – Control Wilk's Lambda = .22, F = 13.91, p = .002, multivariate partial eta squared = .78; CLBP Wilk's Lambda = .09, F = 34.55, p < .001, multivariate partial eta squared = .91. Similar effect was observed also for BP - Control Wilk's Lambda = .22, F = 14.50, p = .002, multivariate partial eta squared = .78; CLBP Wilk's Lambda = .22, F = 12.14, p = .005, multivariate partial eta squared = .78. Two-way ANOVA confirmed that there were no differences between Control and CLBP groups HR change at these time points F = .35, p = .70, as well as BP changes F = .30, p = .74.

Table 12. Descriptive statistics for HR and BP for Controls and CLBP at the three time-points

Group	measure	Time point	Number	Mean	SD
Control	HR1	after rest 1	10	66.2	13.27
	HR2	after exercise	10	97.2	16.48
	HR3	after rest 2	10	76.8	10.86
CLBP	HR1	after rest 1	9	67.22	7.92
	HR2	after exercise	9	91.67	12.48
	HR3	after rest 2	9	71.89	8.71
Control	BP1	after rest 1	10	121	8.12
	BP2	after exercise	10	144.4	15.12
	BP3	after rest 2	10	123.2	11.52
CLBP	BP1	after rest 1	9	122.11	8.8
	BP2	after exercise	9	144.89	16.42
	BP3	after rest 2	9	129	9.38

4. Negative Affect (NA)

The NA was the principal measure of emotional and mood change. NA was repeatedly measured throughout the experiment – after the initial 15 minutes rest period, immediately after the 15 minutes treadmill exercise and at the end of the second 15 minutes rest period – NA 1, NA 2 and NA 3 respectively. It was hypothesised that the exercise would improve the mood and decrease the NA in both Control and CLBP groups. Repeated measures ANOVA for both groups revealed a non-significant decrease of NA after the exercise and subsequent slight non significant increase of NA after the second rest period (Graph 1, below) – Control Wilk's Lambda = .878, F = .624, p = .557; CLBP Wilk's Lambda = .772, F = 1.036, p = .404. The means and standard deviations are presented in Table 13, below.



Graph !. Negative Affect of Control and CLBP groups at the three time-points

Table 13.CLBP and Control Group Statistics for Negative Affect at three time points

	group	N	Mean	Std. Deviation	Std. Error Mean
Negative Affect 1	1 control	11	20.00	5.882	1.774
	2 CLBP	9	20.00	5.454	1.818
Negative Affect 2	1 control	11	18.55	6.919	2.086
	2 CLBP	9	18.00	4.153	1.384
Negative Affect 3	1 control	11	19.18	10.971	3.308
	2 CLBP	9	18.89	8.521	2.84

Furthermore, there was no significant difference of the reported Negative Affect (NA) at

any time point between the CLBP and controls – NA 1 t = .00, p – 1.00; NA 2 t = .207, p = .84; NA 3 t = .07, p = .95 (Table 14, Appendix 9). It is worth pointing that both groups reported only very low mean levels of Negative Affect (NA) – between 18 and 20 on a scale of 15 to 75, which further confirmed that contrary to expectations, our CLBP group had a very low level of NA and was indistinguishable from the controls in respect to their emotional and mood state.

5. ES and MF muscles activity – Static ratio and Flexion-relaxation ratio

The bilateral back muscles ES and MF sEMG was recorded in three positions (prone, standing and bending forward) at the end of the first rest period and at the end of the post-exercise rest period. From the raw EMG recordings the RMS of each muscle at each position and time-point was obtained and used to calculate ratios, which reflected the activity of ES and MF in both the Control and CLBP participants. The two measures were the static standing and fully bent ratio, and the Flexion-relaxation ratio for each of the muscles at the two time points.

Static ES and MF activity

Two static muscle activity ratios were obtained – a standing one and a fully bent forward one. The standing ratio was calculated by dividing the RMS of the standing EMG by the RMS of the lying prone EMG, while the fully bent ratio was calculated by dividing the RMS of the fully bent EMG by the RMS of the lying prone EMG. Independent samples t-tests were performed on all of the static muscles activity measures, revealing that there were higher levels of static activity (for means and SD, see Table 15 below) of most of the muscles in the CLBP group, which reached statistical significance only for the fully bent forward position of the right MF at both time-points – MF(R) Bent 1 t = -2.65, p = .04; MF(R) Bent 2 t = -2.57, p = .05 (Table 16, Appendix 9).

Table 15. Group Statistics of the Static ES and MF muscles activity measures

	group	N	Mean	Std. Deviation	Std. Error Mean
ES(L) standing 1	1 control	8	1.3950	.65012	.22985
	2 CLBP	7	1.6943	1.03194	.39004
ES(L) standing 2	1 control	8	1.2487	.31147	.11012
	2 CLBP	7	1.4929	.80647	.30482
ES(R) standing 1	1 control	8	1.9400	2.05554	.72674
	2 CLBP	7	2.2714	1.52925	.57800
ES(R) standing 2	1 control	8	2.6850	3.44037	1.21635
	2 CLBP	7	1.5529	.50454	.19070
ES(L) bent forward1	1 control	8	1.5850	1.14034	.40317
	2 CLBP	7	33.6429	56.36198	21.30283
ES(L) bent forward 2	1 control	8	1.4000	.58175	.20568
	2 CLBP	7	15.4300	32.91936	12.44235
ES(R) bent forward 1	1 control	8	2.0025	1.28812	.45542
	2 CLBP	7	3.2329	2.14925	.81234
ES(R) bent forward 2	1 control	8	1.5775	.94004	.33235
	2 CLBP	7	3.1614	2.06838	.78178
MF(L) standing 1	1 control	8	3.3288	3.74082	1.32258
	2 CLBP	7	2.4157	1.74931	.66118
MF(L) standing 2	1 control	8	1.5838	.96769	.34213
	2 CLBP	7	1.7929	1.05248	.39780
MF(L) bent forward 1	1 control	8	2.7638	3.85652	1.36349
	2 CLBP	7	8.0257	7.44446	2.81374
MF(L) bent forward 2	1 control	8	2.3400	3.21608	1.13706
	2 CLBP	7	10.2914	9.68534	3.66071
MF(R) bent forward 1	1 control	8	2.1263	1.76256	.62316
	2 CLBP	7	8.8000	6.93846	2.62249
MF(R) bent forward 2	1 control	8	1.5138	.87484	.30930
	2 CLBP	7	12.9000	12.57246	4.75194
MF(R) standing 1	1 control	8	2.5588	2.75752	.97493
	2 CLBP	7	3.4943	4.35693	1.64676
MF(R) standing 2	1 control	8	3.5125	2.96146	1.04703
	2 CLBP	7	2.2871	1.43016	.54055

Paired-samples t-test was performed on the CLBP group to compare the static ES and MF muscle activity before and after the exercise. Most of the muscle activity was reduced, without reaching statistical significance (for details see Table 17, Appendix 9).

Flexion-relaxation ratio (FRR)

FRR was obtained for the bilateral ES and MF at the two time-points. To calculate the FRR the sEMG signal of the bending forward activity was visually inspected and subdivided into three sections – the bending down movement (1-2 seconds), the fully bent forward static state (5 seconds) and the raising up movement (1-2 seconds). The RMS of these separate sections were then obtained. The FRR was calculated by dividing the averaged sum of the RMS of the active down and up movements by the RMS of the static fully bent forward position. Independent samples t-test revealed, as expected, a reduced FRR in the CLBP

group (for group statistics see Table 18, below), which was statistically significant for the left ES at both time-points, as well as for right MF at time-point 2 – ES(L) FRR 1 t = 3.09, p = .01; ES(L) FRR 2 t = 2.33, p = .047; MF(R) FRR 2 t = 2.73, p = .03 (Table 19, Appendix 9).

Table 18. Group Statistics of Flexion-relaxation ratios

	group	N	Mean	Std. Deviation	Std. Error Mean
ES(L) FRR1	1 control	7	3.5737	1.63717	.61879
	2 CLBP	7	1.4362	.82165	.31055
ES(L) FRR2	1 control	7	3.8208	2.34223	.88528
	2 CLBP	7	1.5597	1.04505	.39499
ES(R) FRR1	1 control	7	4.3529	5.00714	1.89252
	2 CLBP	7	1.7725	.76266	.28826
ES(R) FRR2	1 control	7	2.3725	1.67665	.63371
	2 CLBP	7	3.5688	3.48435	1.31696
MF(R) FRR1	1 control	7	9.1822	11.81136	4.46427
	2 CLBP	7	5.9539	12.40404	4.68829
MF(R) FRR2	1 control	7	7.7917	5.96388	2.25414
	2 CLBP	7	1.5437	1.03704	.39197
MF(L) FRR1	1 control	7	16.1999	26.99273	10.20229
	2 CLBP	7	12.2255	28.71846	10.85456
MF(L) FRR2	1 control	7	10.9586	13.55724	5.12416
	2 CLBP	7	1.9773	1.89771	.71727

Paired-samples t-test was performed on the CLBP group to compare the FRR of ES and MF muscle before and after the exercise. Most of the muscles FRR was increased, but some of the muscles had a decrease of FRR. There were very large differences within the CLBP group, which was reflected in the large SD, so none of the changes of FRR reached statistical significance (for details see Table 20, Appendix 9).

6. Baseline Pain Level (BPL) and Present Pain Intensity (PPI)

The Baseline Pain Level (BPL) and the Present Pain Intensity (PPI) were the principal measure reflecting the perceived pain experience, which was assumed to indicate dynamically the underlying factors involved in the CLBP pathology. BPL and PPI were self-reported on a 100mm VAS scale at baseline (Back Pain Questionnaire) and at three time-points of the experiment – after the initial 15 minutes rest period, immediately after the 15 minutes treadmill exercise and at the end of the second 15 minutes rest period – PPI 1, PPI 2 and PPI 3 respectively. The BPL was unsurprisingly significantly higher in the CLBP

group (43.78, SD = 19.06) than the Control group (3.27, SD = 4.92) t = -6.21, p < .001.

The principle employed dynamic measure of the present pain sensory experience was the Present Pain Intensity (PPI). PPI was expected to be affected by both psychological and bio-mechanical factors and to be associated with change of muscle activity patterns before and after the exercise. It was measured at three time-points of the experiment – after the initial rest period (PPI 1), immediately after the exercise (PPI 2) and after the post-exercise rest period (PPI 3) using a 100mm VAS. As expected PPI was significantly higher in the CLBP group at all three time-points (Table 21, below) – PPI 1 t = 3.94, p = .004; PPI 2 t = 4.27, p = .003; PPI 3 t = 3.87, p = .005 (Table 22, Appendix 9).

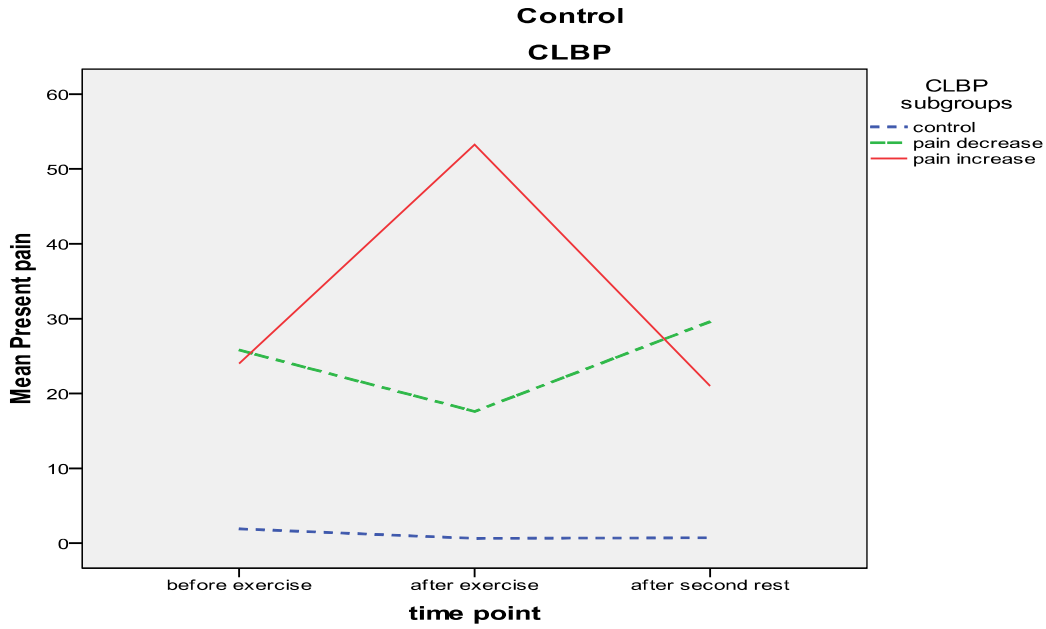
Table 21. Present Pain Intensity Group Statistics

	group	N	Mean	Std. Deviation	Std. Error Mean
pain1	1 control	**11**	**1.91**	**3.081**	**.929**
	2 CLBP	**9**	**25.00**	**17.328**	**5.776**
Pain 2	1 control	**11**	**.64**	**1.120**	**.338**
	2 CLBP	**9**	**33.44**	**23.017**	**7.672**
pain 3	1 control	**11**	**.73**	**1.679**	**.506**
	2 CLBP	**9**	**25.78**	**19.338**	**6.446**

The results from a repeated measures ANOVA confirmed that exercise reduced pain in the Control group, albeit non-significantly and from a very low level (for group statistics see table 21, above) – Wilks' Lambda = .84, F = .852, p = .458, partial eta squared = .159 (Table 23, Appendix 9). In the CLBP group the results were at first glance different than expected – PPI increased immediately after the treadmill exercise, followed by a reduction after the second rest period (Table 21, above), without reaching statistical significance – Wilks' Lambda = .84, F (2, 7) = .861, p = .592, partial eta squared = .139 (Table 23, Appendix 9).

On a closer inspection, the treadmill exercise produced both a decrease of PPI and an

increase in PPI in different participants with CLBP (Graph 2, below) - 5 of the CLBP participants had a decrease in PPI (mean 17.6, SD = 15.77), while 4 had an increase (mean 53.25, SD = 11.82). Furthermore, the PPI at the post-exercise time-point was significant between these two sub-groups of CLBP participants t = -3.739, p = .007 (Table 24. Appendix 9).



Graph 2. Present pain Intensity of the different sub-groups of CLBP at three time-points

Further analysis between the CLBP participants' PPI at the three time-points and baseline pain level, Pain Catastrophisig, Depression, Anxiety, Stress, as well as the three time-points Negative Affects produced only significant positive correlation results for Depression and PPI 1 and PPI 3 (PPI 1 r = .824. N = 9, p = .005; PPI 2 r = .827, N = 9, p = .006), apart from the expected strong correlation between the baseline pain level and the PPI 1 (before exercise) and PPI 3 (after post-exercise rest), but not PPI 2 (immediately after exercise) (Table 25, Appendix 9). PPI did not correlate significantly with any of the muscle activity measures, which had shown a significant between group difference – the static right MF at fully bent forward position (both time 1 and 2), the Flexion-relation ratio of the left ES (both time 1 and 2) and right MF (time 2) (Table 25, Appendix 9).

Section IV – Discussion

1. Summary of the results

Chronic low back pain (CLBP) is a complex clinical condition, which is not characterised by a clear singular pathology. Both psychological and bio-mechjanical factors have been identified as possible mechanisms responsible for the development and persistence of CLBP. The aim of the present study was to clarify the respective roles played by these different factors by investigating the affective and muscle activity responses of CLBP and healthy control participants to a moderate physical activity (treadmill walking exercise) perturbation.

The results from the present study, in accordance with previous studies (Leeuw et al., 2007; Picavet et al., 2002; Vlaeyen et al., 1995; Richardson et al., 2009) confirmed the hypothesis that CLBP participants are clearly identifiable according to their affective and cognitive profile. In the present study CLBP participants reported significantly higher levels of Pain Catastrophising 22 (SD = 12.66) versus 4.55 (SD = 5.87) in the control group – $t = 3.81$, $p = .003$, as well as Depression 5.67 (SD = 3.81) versus 2.36 (SD = 2.46) – $t = 2.35$, $p = .03$. Perceived Anxiety and Stress were also higher in the CLBP group 3.44 (SD = 2.55) versus 1.94 (SD = 2.92) and 7.44 (SD = 3.25) versus 4.91 (SD = 4.42) respectively.

Both self-reported Pain Catastrophising and Depression were significantly correlated with the perceived pain (Baseline Pain Level) $r = .755$, $n = 20$, $p < .001$ and $r = .559$, $n = 20$, $p = .010$ respectively (Table 26, below), but not with each other or the self-reported Anxiety and Stress. Further analysis of the same psychological factors in the CLBP group alone revealed that, contrary to the above-mentioned CLBP studies, there were no significant associations between the perceived Pain, Depression, Anxiety, Stress and even Pain

Catastrophising. The most likely explanation for this discrepancy is the present study's much smaller sample size – only 9 CLBP participants. Another possible contributing factor is the composition of the CLBP group – predominantly personal contacts, which might have influenced the honesty of the self-reporting – our CLBP participants reported similar levels of Pain Catastrophising, but lower levels of Depression.

Table 26. Correlations between Baseline Pain Level, Pain Catastrophising and Depression

		Depression	pain level	Pain Catastrophisisng
Depression	Pearson Correlation	**1**	**.559***	**.374**
	Sig. (2-tailed)		**.010**	**.104**
	N	**20**	**20**	**20**
pain level	Pearson Correlation	**.559***	**1**	**.755****
	Sig. (2-tailed)	**.010**		**.000**
	N	**20**	**20**	**20**
Pain Catastrophisisng	Pearson Correlation	**.374**	**.755****	**1**
	Sig. (2-tailed)	**.104**	**.000**	
	N	**20**	**20**	**20**

*. Correlation is significant at the 0.05 level (2-tailed) **. Correlation is significant at the 0.01 level (2-tailed)

The negative affective state (NA) was measured dynamically throughout the experiment (NA 1, NA 2, NA 3) and was expected to be closely related to the pain experience and back muscles activity, as well as background psychological traits. Surprisingly The NA was very low – 18 to 20 on a scale of 15 to 75, in both groups (even lower in the CLBP) and there was no significant difference between the NA of the CLBP and Control groups at any time-point of the experiment - NA 1 $t = .00$, $p – 1.00$; NA 2 $t = .207$, $p = .84$; NA 3 $t = .07$, $p = .95$. Furthermore, there was no significant correlation between the NA and background Depression, Anxiety, Stress, Pain Catastrophising or Perceived Pain, as well as the Present Pain at any time-point. This apparent contradiction could be explained by either assuming dishonesty in self-reporting the present affective state by the CLBP group, or by genuine discrepancy of the perceived general pain and the actual momentary pain experience. Similar results of dissociative links between background emotional and cognitive mental set and affective and intensity experience of immediate pain in CLBP

were observed in a study, which used induced (ischemic) pain paradigm (Richardson et al., 2009). Their results pointed that only the sensory and intensity, but not the affective aspects of the induced pain were related to the background Pain Catastrophising, which they attributed to the difference of the type and location of the induced acute pain (ischemic pressure pain to the upper arm) from the background chronic low back pain. Our results, although derived from much smaller sample, further confirmed that even acute low back pain induced by exercise is experienced more sensory (increased pain intensity), without corresponding increase of the Negative Affect. These combined results might be pointing to a potential general characteristic of CLBP and possibly other chronic pain conditions – the existence of mismatch of the internal cognitive perception of the pain (the imaginary pain threat) and the reality of the pain experience, and explain the beneficial effects of Cognitive-Behavior Therapy for CLBP (Smeets et al., 2006).

The present study employed moderate treadmill walking exercise in order to produce a mild physiological as well as bio-mechanical perturbation. Exercise has been shown to improve both chronic pain and negative affect and mood conditions (Hayden et al., 2005; Keller, 2006; Standaert et al., 2008; Arokoski et al., 2004; Hides et al, 2001). Although exercise has been linked to a decrease in depression and neurogenesis in animal studies (Cotman & Berchtold, 2002), the precise neural and humoral mechanisms are not well established and potentially involve alteration of the 'central regulator' function with ensuing changes of neural circuits activity and release of signalling molecules like endorphins, Vascular endothelial growth factors, Brain-derived neurotrophic factors, Serotonin, Dopamine, Orexins, Glucocorticoids, just to mention a few (Ernst et al., 2006; Siegel, 2004). We hypothesised that moderate exercise would lead to similar physiological and psychological responses in both groups, which would result in a decrease of Negative

Affect with subsequent reduction of experienced pain and modulation of the ES and MF back muscles activity. The non-significant post-exercise reduction of NA in both groups, as described above, confirms that. Physiologically, exercise produced similar response of the Hypothalamus-Pituitary-Adrenal axis with no-significantly different increase of Cortisol and DHEA between CLBP and Control participants (unpublished results from a parallel Masters project of a fellow researcher - F.M.). The dynamically measured present pain intensity (PPI – 1, 2, 3) and back muscles activity patterns, however told a different story.

The bilateral ES and MF activity was measured in order to compare the back muscles activity between the two groups at two time points of the experiment – after the initial rest period and after the post-exercise rest period. Two ratios, a static standing and fully bent forward ratio (ES and MF standing 1 and 2; ES and MF fully bent 1 and 2) as well as Flexion-relaxation ratio (ES and MF FRR 1 and 2) were calculated on the base of the ES and MF sEMG in static lying prone and quiet standing, as well as active bending forward, holding the bent down posture and raising up at the two time-points. The results of the initial standing and fully bent forward ES and MF measures indicated a higher level of static muscle activity in the CLBP group, which only reached statistical significance for the right MF in fully bent down position $t = -2.48$, $p = .044$. The initial flexion-relaxation ratios (FRR) of the ES and MF further confirmed that CLBP participants differed from healthy controls in regard to their back muscles activity – CLBP group had, as expected, a lower FRR of all four muscles, which reached significance only for the left ES – $t = 3.09$, $p = .013$. The present study's results were less robust than the results from previous studies (Watson et al., 1997; Watson et al., 1995), which might be due to the difference of calculation of the FRR – we employed the averaged RMS of the entire bending down plus raising up sEMG (1.5-2 seconds each), while Watson et al. (1997) only utilised the RMS of 1 second peak of the bending down sEMG of ES at two locations on the back divided by

the RMS of 1 second of the fully bend down sEMG. Our method reflected the ratio of the entire ES and MF muscle activity during the down and up active phases divided by the static fully bent muscle activity, which we reasoned would better reflect the reflexive silencing of the back muscles at fully flexed position, avoiding potential co-founding of higher motor control psychological factors (Dickey et al., 2003).

The second post-exercise rest muscle activity measurements showed similar patterns in the two groups. The static ES and MF muscle activity was again, in general, higher in the CLBP group, almost reaching significance for the right MF at fully bent forward position $t = -3.92$, $p = .054$. The second FRR were also lower in the CLBP group, which was significant for the left ES and right MF – $t = 2.33$, $p = .038$; $t = 2.73$, $p = .032$ respectively.

There was no significant change of any of the back muscles activity measures in either group produced by the exercise, although there was a tendency to reduction of the static muscles activity and increase of the FRR in the CLBP participants. As muscle activity is modulated by both psychological and bio-mechanical factors, as well as pain, it is necessary to analyse it in context with the corresponding experienced pain (Hodges & Moseley, 2003).

The principal employed dynamic measure of the present pain experience was the Present Pain Intensity (PPI). PPI reflected predominantly the sensory-discriminative, rather than the affective characteristics of the pain experience. It was measured at three time-points of the experiment – after the initial rest period (PPI 1), immediately after the exercise (PPI 2) and after the post-exercise rest period (PPI 3) using a 100mm VAS. As expected PPI was significantly higher in the CLBP group – PPI 1 $t = 3.94$, $p = .004$; PPI 2 $t = 4.27$, $p = .003$; PPI 3 $t = 3.87$, $p = .005$. Interestingly a repeated measures ANOVA revealed that in the

CLBP group PPI increased immediately after the treadmill exercise, followed by a reduction after the second rest period, without reaching statistical significance – Wilks' Lambda = .84, F (2, 7) = .861, p = .592, partial eta squared = .139. This result was opposite to the predicted outcome – exercise increased PPI, despite decreasing the Negative Affect. This contradiction was clarified by the discovery of dual response in regard to the exercise in the CLBP group - 5 of the CLBP participants (55.56%) had a decrease in PPI (mean 17.6, SD = 15.77), while 4 (44.44%) had an increase (mean 53.25, SD = 11.82). furthermore these two groups had a significantly different post-exercise PPI t = -3.739, p = .007, but not statistically different PPI at time-point 1 or 3.

To clarify what characterised this two sub-groups of CLBP participants, further analyses were performed. The Pain Decrease sub-group had slightly higher Background pain level

Table 27. CLBP Sub-Groups Statistics

	CLBP sub-group	N	Mean	Std. Deviation	Std. Error Mean
Age	1 pain decrease	**5**	**36.00**	**7.714**	**3.450**
	2 pain increase	**4**	**36.50**	**13.626**	**6.813**
Pain Length	1 pain decrease	**5**	**2.80**	**1.643**	**.735**
	2 pain increase	**4**	**2.50**	**1.000**	**.500**
Background pain Level	1 pain decrease	**5**	**46.40**	**22.131**	**9.897**
	2 pain increase	**4**	**40.50**	**17.020**	**8.510**
Pain Catastrophisig	1 pain decrease	**5**	**23.20**	**10.085**	**4.510**
	2 pain increase	**4**	**20.50**	**16.921**	**8.461**
Depression	1 pain decrease	**5**	**5.00**	**5.000**	**2.236**
	2 pain increase	**4**	**6.50**	**1.915**	**.957**
Anxiety	1 pain decrease	**5**	**3.60**	**2.302**	**1.030**
	2 pain increase	**4**	**3.25**	**3.202**	**1.601**
Stress	1 pain decrease	**5**	**7.60**	**3.362**	**1.503**
	2 pain increase	**4**	**7.25**	**3.594**	**1.797**
Negative Affect 1	1 pain decrease	**5**	**22.80**	**5.933**	**2.653**
	2 pain increase	**4**	**16.50**	**1.732**	**.866**
Negative Affect 2	1 pain decrease	**5**	**19.60**	**4.930**	**2.205**
	2 pain increase	**4**	**16.00**	**2.000**	**1.000**
Negative Affect 3	1 pain decrease	**5**	**21.80**	**11.009**	**4.923**
	2 pain increase	**4**	**15.25**	**.500**	**.250**

and Pain catastrophisig, Anxiety, Stress as well as NA at all three time-points and reported

less Depression (Table 27, above), but none of these measures reached significance.

The static muscles activity of the two groups also differed – the standing ES and MF ratios were lower in the Pain Decrease sub-group (smaller than 1), indicating higher prone compared to standing back muscles activity and tension, while the Pain Increase sub-group had a higher static fully bent forward activity, indicating a decrease of the flexion-relaxation phenomenon (Table 28, below). Despite the small size of the subgroups these differences reached significance for the initial left ES standing and left MF standing – t = -3.4, p = .033; t = -3.43, p = .027 respectively (Table 29, Appendix 9).

Table 28. CLBP Sub-groups Static ES and MF muscle activity Statistics

	CLBP sub-group	N	Mean	Std. Deviation	Std. Error Mean
ES(L) standing 1	1 pain decrease	**3**	**.8167**	**.22898**	**.13220**
	2 pain increase	**4**	**2.3525**	**.86427**	**.43213**
ES(L) standing 2	1 pain decrease	**3**	**1.4033**	**1.18568**	**.68455**
	2 pain increase	**4**	**1.5600**	**.59121**	**.29561**
ES(R) standing 1	1 pain decrease	**3**	**1.8900**	**1.89850**	**1.09610**
	2 pain increase	**4**	**2.5575**	**1.42118**	**.71059**
ES(R) standing 2	1 pain decrease	**3**	**1.1900**	**.45508**	**.26274**
	2 pain increase	**4**	**1.8250**	**.37501**	**.18751**
ES(L) bent forward 1	1 pain decrease	**3**	**28.0533**	**45.01887**	**25.99165**
	2 pain increase	**4**	**37.8350**	**70.33875**	**35.16937**
ES(L) bent forward 2	1 pain decrease	**3**	**2.8400**	**2.39056**	**1.38019**
	2 pain increase	**4**	**24.8725**	**43.43005**	**21.71503**
ES(R) bent forward 1	1 pain decrease	**3**	**2.7333**	**1.89374**	**1.09335**
	2 pain increase	**4**	**3.6075**	**2.53201**	**1.26601**
ES(R) bent forward 2	1 pain decrease	**3**	**2.5733**	**1.62161**	**.93624**
	2 pain increase	**4**	**3.6025**	**2.48960**	**1.24480**
MF(L) standing 1	1 pain decrease	**3**	**.9133**	**.52272**	**.30179**
	2 pain increase	**4**	**3.5425**	**1.40997**	**.70499**
MF(L) standing 2	1 pain decrease	**3**	**.9600**	**.78077**	**.45078**
	2 pain increase	**4**	**2.4175**	**.77147**	**.38573**
MF(L) bent forward 1	1 pain decrease	**3**	**7.9067**	**7.21092**	**4.16323**
	2 pain increase	**4**	**8.1150**	**8.72641**	**4.36320**
MF(L) bent forward 2	1 pain decrease	**3**	**12.1633**	**10.35204**	**5.97675**
	2 pain increase	**4**	**8.8875**	**10.48983**	**5.24491**
MF(R) bent forward 1	1 pain decrease	**3**	**10.5800**	**9.81220**	**5.66508**
	2 pain increase	**4**	**7.4650**	**5.15302**	**2.57651**

MF(R) bent forward 2	1 pain decrease	**3**	**18.0100**	**17.75909**	**10.25322**
	2 pain increase	**4**	**9.0675**	**7.75760**	**3.87880**
MF(R) standing 1	1 pain decrease	**3**	**1.4433**	**.75831**	**.43781**
	2 pain increase	**4**	**5.0325**	**5.49737**	**2.74869**
MF(R) standing 2	1 pain decrease	**3**	**1.3867**	**.49743**	**.28719**
	2 pain increase	**4**	**2.9625**	**1.58327**	**.79164**

FRR analysis further confirmed that the Pain Increase group tended to have a diminished Flexion-relaxation ratio for most of the back muscles (Table 30, below), although none was significant, possibly due to the small size of the two sub-groups combined with large variability.

Table 30. CLBP Sub-groups ES and MF Flexion-realaxation Ratios Statistics

	CLBP sub-group	N	Mean	Std. Deviation	Std. Error Mean
ES(L) FRR 1	1 pain decrease	**3**	**1.8845**	**1.19911**	**.69230**
	2 pain increase	**4**	**1.1000**	**.20000**	**.10000**
ES(L) FRR 2	1 pain decrease	**3**	**2.1727**	**1.49333**	**.86218**
	2 pain increase	**4**	**1.1000**	**.20000**	**.10000**
ES(R) FRR1	1 pain decrease	**3**	**2.0894**	**.95595**	**.55192**
	2 pain increase	**4**	**1.5348**	**.61507**	**.30753**
ES(L) FRR 2	1 pain decrease	**3**	**3.5440**	**3.36529**	**1.94295**
	2 pain increase	**4**	**3.5873**	**4.09026**	**2.04513**
MF(R) FRR 1	1 pain decrease	**3**	**11.9734**	**19.12989**	**11.04465**
	2 pain increase	**4**	**1.4393**	**.58435**	**.29217**
MF(R) FRR 2	1 pain decrease	**3**	**1.9778**	**1.61007**	**.92957**
	2 pain increase	**4**	**1.2182**	**.30498**	**.15249**
MF(L) FRR 1	1 pain decrease	**3**	**26.3827**	**44.12463**	**25.47537**
	2 pain increase	**4**	**1.6076**	**.87489**	**.43744**
MF(L) FRR 2	1 pain decrease	**3**	**2.7459**	**3.00373**	**1.73421**
	2 pain increase	**4**	**1.4009**	**.39246**	**.19623**

Although the results of the CLBP sub-groups analyses are difficult to generalise, due to their small size, they point to consistent tendencies and trends of their psychological and muscle activity characteristics. On the one hand were the CLBP participants, whose PPI was higher after the resting periods and decreased after the moderate treadmill walking

exercise. In this sub-group back pain was associated with higher levels of Pain catastrophisig and negative affect. These participants also tended to have a higher levels of static lying muscle activity (reflected in standing ratios of less than 1) and relatively preserved FRR. On the other hand were the CLBP participants, whose pain was lower while resting and dramatically increased after the moderate treadmill walking exercise. This sub-group of CLBP participants generally had lower levels of Pain Catastrophisig and Negative Affect and scored only marginally higher on the Depression measurement. Their back muscles activity demonstrated an opposite tendency towards higher muscle activity in the fully flexed forward static position, as well as lower FRR.

The most striking difference between the two groups was the response to the moderate treadmill walking, which could possibly hold the key to the differences in the specifics of their back pain. It is worth pointing that the treadmill walking was performed at slightly steep 17.5 degrees of inclination, which is likely to have posed a challenge not just physiologically but also mechanically, altering the angle of the hips and pelvis/lumbar spine. In participants, whose back pain was associated with dysfunction of the passive structures of the spine stabilising system and intrinsically decreased spinal stability (increase of the 'neutral zone', Panjabi, 2003) this mechanical challenge possibly caused abnormal vertebral movements with ensuing compression of the local soft tissues, which led to increase in their pain intensity. In CLBP participants whose pain was associated more with dysfunction of the 'active' spinal stabilising system (abnormal static muscle activity) due to possible psychological and cognitive factors, but relatively normal intrinsic spinal stability, the treadmill walking did not pose a mechanical challenge. On the contrary, the exercise improved their affective state as well as muscle function, which led to the decrease of their pain intensity.

2. Study limitations

The present study was a masters project, which had many foreseen and unforeseen limitations, dictated by time and resources restrains. By far the biggest limitation was associated with the size and composition of the CLBP participants group. The sample was not a random one and furthermore, most of the participants in this group were personal contacts, which could have added further bias, as a great deal of the measurements were based on self-report scales.

The size of the sample was deemed adequate when the study was first designed, but the discovery of sub-groups of CLBP patients rendered it far too small to perform meaningful correlation and regression analyses and establish causative links between the different factors involved in CLBP. It is estimated that at least 60 CLBP participants would be necessary, provided the composition of the sample has similar equal proportions of the two sub-groups as it was the case in the present study, in order to have sufficient power.

Another limitation of the study is the lack of background disability measurement, which is one of the principal characteristics of CLBP. The present study was more focused on the underlying psychological and biological measurements of CLBP and due to time restrains (participants' as well as researchers') only the minimal number of demographic measurements deemed essential were included.

A major unforeseen methodological shortcoming of the study, which limited the establishment of stronger associations between psychological factors and spinal instability in low back pain, was the omission of back muscles measurements immediately after the treadmill exercise, when the differences in the present pain intensity (PPI) distinguished the two CLBP sub-groups. Originally the project's main emphasis had been on

psychological and physiological measurements, connected with the negative affective states associated with chronic pain. For this reason back muscles measurements were secondary and were only measured at the initial and end stages of the experiment. Future CLBP studies employing exercise as a perturbation, should incorporate measuring of the back muscles immediately after the exercise in order to better capture possible differences in their activity between sub-groups of CLBP patients.

The present study was a pilot project, aiming to explore the relationship of the different psychological, physiological and motor control factors involved in CLBP, using moderate exercise as a safe perturbation. Any further research studies, employing similar experimental methodology, should aim to avoid the shortcomings of the present study in order to obtain more accurate and valid results.

3. Conclusions

Despite all its sample limitations, the results of the present study were in line with findings from previous research in CLBP and confirmed that measures of both psychological and back muscles activity were altered in the CLBP group. Furthermore, the present study results indicated that the CLBP group was heterogenous - different factors were found to determined the nature of the back pain in two sub-sets of the CLBP group. On one hand was the sub-group, which responded with a post-exercise pain decrease. Participants of this sub-group exhibited only a mild back muscles dysfunction, characterised by increased activity of the ES and MF in resting state (muscle tension), combined with a higher negative cognitive and affective mental set. On the other hand was the CLBP sub-group, whose pain increased immediately after the exercise. The main characteristics of the participants of this sub-group was abnormal ES and MF flexion-relaxation, which pointed to compensatory increased activity of the superficial back muscles due to possible intrinsic

spinal instability.

Due to technical limitations associated with sEMG, the present study monitored bilaterally the activity of only two of the many low body muscles – the superficial back extensors ES and MF. The activity of the deep low back (deep fibres of MF, Quadratus Lumborum, Psoas) and abdominal (TrAb) was inferred only indirectly from the behaviour of the ES and MF and the back pain intensity after a physiological and biomechanical exercise challenge. Following the spinal stability model (Panjabi, 1992; Panjabi 2003; Cholewicki & McGill, 1996; Cholewicki et al., 1997) normal torso muscles (both deep and superficial) function is essential for spinal stability and movement, which relies on the generation of correct muscle activation patterns by the third element – the motor neural control system. Previous research has pointed to functional and structural abnormalities of the deep MF and TrAb muscles (Hides et al., 1994; Hides et al., 1996; Hodges & Richardson, 1999a; MacDonald et al, 2009), as well as increased compensatory activity of the superficial back extensors (Watson et al., 1995; Watson et al., 1997; Cholevwicki & Van Vliet, 2002; Cholewicki et al., 1997; Gardner-Morse & Stokes, 1998) in patients with low back pain, without clarifying the role played by the neural motor control in this abnormal muscle activity. There are many interacting factors (physiological, psychological, cognitive, sensory), which affect and modulate motor control (Hodges & Moseley, 2003), but broadly they can be divided into top-down CNS driven (primary neuro-psychological and cognitive dysfunction) and periphery driven (primary organic and structural dysfunction). The two sub-groups of CLBP patients identified in the present study fell broadly in this top-down and periphery-up driven model of CLBP, providing initial evidence to the dichotomous nature of CLBP, which warrants further vigorous experimental confirmation.

The real challenge, both experimentally and clinically, is how to establish the precise top-

down or periphery-up mechanism in the pathology of each individual CLBP patient, as both alter the activity of the muscles involved in spinal stabilisation in a similar way – shifting stabilisation from the deep axial and abdominal muscles to co-contraction of superficial antagonistic muscles. The results of our study confirmed that this pattern of dysfunctional stabilisation characterised both CLBP sub-groups, although it manifested in a subtly different way in each sub-group. This difference only became distinguishable after the moderate exercise perturbation - the back pain of one of the sub-groups was associated with primary top-down dysfunction of the 'active system', while in the second sub-group the principal underlying factor was primary organic dysfunction of the 'passive system'.

It is interesting that in the present study the moderate treadmill walking at 17.5 degrees of inclination was capable of identifying the existence of CLBP sub-groups, which were otherwise undistinguishable by the rest of their background pain, psychological or muscle activity characteristics. As treadmill exercise is a safe perturbation, which produced only temporary increase of pain intensity in one of the sub-groups (the PPI at the post-exercise rest period had decreased even below the initial pain in that sub-group) it offers a possible method of better identification of the bio-mechanical and psycho-neural factors involved in CLBP. Further cross-sectional and longitudinal studies using adequate in size and composition CLBP samples are necessary to confirm the validity of this methodology as well as the results of the present pilot study.

The ability to differentiate between sub-groups of CLBP patients and establish the precise interactions between the psycho-neural and biomechanical factors in each low back pain patient would tremendously improve both diagnosis and treatment as well as prevention of CLBP. It will allow the appropriate (in respect to modality and timing) application of

pharmacological, physical therapy, surgical, psychological / cognitive / educational and various types of aerobic, mobilising, stretching and core stability exercise approaches to be optimally utilised in the treatment of CLBP, as well as low back pain in the acute and sub-acute stages. Adequate diagnosis and treatment of low back pain will in turn lead to prompt resolution and prevention of further recurrence and chronification of low back problems, which will save both the individuals affected and society as a whole personal suffering and precious financial resources.

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APPENDICES

APPENDIX 1. Ethics Application

Roehampton University London

ETHICS APPLICATION FORM 2009

Please read the Notes for Applicants before completing this form

The form should be completed electronically using black size 11 or 12 font.

PLEASE TICK THE RELEVANT BOX			
STUDENT (MSc)	x	RESEARCH STUDENT (MPhil, PhD, EdD, PsychD)	
EXTERNAL RESEARCHER		MEMBER OF STAFF	

SECTION 1: PERSONAL DETAILS *Please complete the header with your name and School*	
Student name:	Dragomir P Lubomirov
Student no:	LUB08275895
Other researchers:	
Correspondence address (term time):	4 Chelmsford Close Hammersmith London W6 8HU
Telephone (home & mobile):	07976 253682
Email: *(all correspondence will be sent by email unless otherwise requested)*	dragomirl@btinternet.com
FOR STUDENTS ONLY:	
Programme of study:	MSc Clinical Neuroscience
Mode of study (full-time/part-time)	Part-time
Programme tutor:	Jolanta Opacka-Juffrey

SECTION 2: RESEARCH PROJECT	
Title of project:	How does Chronic Back Pain affect Psycho-Physiological Responses of the body to Moderate Exercise?
Proposed start date (e.g. recruiting participants, data collection):	February 2010 (?)
Duration of project:	6 – 8 weeks
Source of funds (private, external funding):	
Purpose of the proposed investigation (max 100 words):	
The aims of the present study are to examine the immediate physiological effects of treadmill exercise in CLBP patients and to investigate whether these responses are moderated/mediated by individual levels of: fear of injury, pain catastrophising and depression. Its purpose is to provide a snapshot of the effects of exercise in CLBP patients and further the understanding of their neuroendocrine mechanisms, through simultaneous HPA axis, autonomic nervous system and sensory-motor activity measurements.	
Outline plan of research (2,000 words; headings should follow guidelines provided – introduction, method including subheadings such as design etc, benefits and limitations of study; references need to be included but are not included in word count):	

Background

Low back pain (LBP) is one of the most common health problems, affecting each year more than 20% of the adult population in the west (Balague et al., 2007; Savigny et al., 2008; Negrini et al., 2008). Fortunately, in the majority of cases it is a self-limiting condition, which resolves spontaneously requiring only minimal painkilling and anti-inflammatory medication and reassurance. Out of all LBP patients, 10-15% progress to develop a chronic low back pain (CLBP) condition, which brings enormous psychological and financial costs to the affected individuals, their family and society as a whole (Balague et al., 2007; Savigny et al., 2008). Despite all recent developments in understanding the underlying mechanisms of chronic pain, there is no established effective treatment for CLBP. Exercise therapy has shown some encouraging results in that respect (Hayden et al., 2005).

NICE in their 2008 draft guideline (Savigny et al., 2008) define chronic non-specific low back pain as ‘’pain, muscle tension or stiffness affecting the low back for which there is not a recognised patho-anatomical cause’’. Over the past few decades many theories to the exact mechanism of CLBP have evolved, looking beyond the simple patho-anatomical model: the spinal functional stability and control model (Panjabi, 1992), the disuse and de-conditioning model (Hasenbring et al., 1994; Bousema et al., 2007) and the bio-psycho-social model (Waddell, 1992; Waddell, 1996; Gatchel et al., 2007).

As most chronic pain conditions, CLBP is a multidimensional problem affecting not just the physical, but also the mental (emotional and cognitive) state, as well as social behavior. Apart from the persistent pain and physical dysfunction, the other main clinical symptoms in CLBP patients are fatigue and depression, which are very similar to other chronic conditions with unclear

pathology and multi-system involvement - Chronic Fatigue Syndrome (CFS) and fibromyalgia (FM) (Claw and Chrousos, 1997; Fishbain et al., 2004). Although there is no complete consensus, the evidence points to central dysregulation of the stress neuro-matrix and the hypothalamo-pituitary-adrenal (HPA) axis as their main underlying cause, with hypocortisolism and ensuing autonomic, endocrine, immunological and sensory-motor dysfunctions (Claw and Chrousos, 1997; Griep et al., 1993; Griep et al., 1998; Geiss et al., 1997; Chapman et al., 2008; Fries et al., 2005; Hodges and Mosley, 2003; Flor et al., 1992).
These multidimensional aspects and unclear pathology of CLBP are reflected in the variety of approaches to its research. Recent CLBP studies have followed broadly two main lines of enquiry. On one hand are the studies, which investigate the psycho-neural interactions and mechanisms and on the other hand are the back muscles control and activity studies.
In CLBP, as in other chronic pain conditions, fear from injury/re-injury and pain catastrophising (maladaptive negative emotional/cognitive state, connected with activating the stress response) have consistently been associated with patients' experience of pain intensity, disability and pain behavior (McBeth et al., 2007; Leeuw et al., 2007; Edwards et al., 2006; Turk and Okifuji, 2002; Thieme et al., 2005; Gracely et al., 2004; Vlaeyen et al., 1995; Burns, 2003). Similar to atypical depression, in CLBP pain catastrophising is associated with hypocortisolism and altered morning cortisol response (Campbel and Edwards, 2009; McBeth et al., 2007; Sudhaus et al., 2008; Johansen et al., 2008).
Regarding low back muscle function, there is mounting evidence that CLBP is associated with abnormal muscle activity: deactivation and delayed activation of the deep stabilising spinal muscles - multifidi lumborum (MF) and transverse abdominal (TrA); and abnormal flexion-relaxation ratio of the superficial back extensors - longissimus and iliocostalis lumborum (ES) (Hodges, 2001; Hodges et al., 2001; Hodges and Moseley, 2003; Hodges and Richardson, 1996; Brumagne et al., 2000; Hides et al., 1996; Hides et al., 1993; Zedka et al., 1999; Watson et al., 1997). Electromyography (EMG) and especially its non-invasive version – surface EMG (sEMG), have been used extensively for the last few decades to study muscle function in LBP. Despite its spatial and other limitations, sEMG studies have provided consistent evidence for two main abnormalities of function of the low back muscles in LBP: the flexion-relaxation ratio of the ES muscles (Watson et al., 1997; Geisser et al., 2005), delayed activation of TrA muscles (Hodges, 2001; Hodges and Richardson, 1996) and dysfunction of the MF (Hides et al., 1996; Hides et al., 1993).
As a therapeutic intervention, exercise has shown promising results not just for the treatment of CLBP (Hayden et al., 2005), but also for FM (Busch et al., 2007), CFS (Larun et al., 2004) and depression (Mead et al., 2008). As a therapy for CLBP, exercise is significantly more effective than no treatment and is as effective as cognitive behavior therapy (CBT) in reducing pain catastrophising, pain and disability perception and depression (Smeets et al., 2006). CBT on the other hand has been linked (one limited quality study – Roberts et al., 2009) with raising the salivary cortisol level after 6 months of treatment.
Exercise is well accepted as an activator of central function and treadmill exercise has shown to raise mildly both cortisol and ACTH in healthy adults (Luger et al., 1987). On the other hand, recent studies in rodents, have demonstrated positive plastic changes and neurogenesis in the hippocampus (Cotman and Berchtold, 2002; Earnst et al., 2006) in response to exercise. Normally stress and elevation of cortisol are associated with hippocampal deterioration, so this apparent contradiction might indicate the possibility that in chronic hypocortisolim (as well as in normal and hypercortisolism) conditions, exercise 'stress' and subsequent elevation of cortisol relate to normalization of the HPA axis and central function and ensuing regulation of autonomic, endocrine, immune and sensory-motor systems.

The aims of the present study are to examine the immediate physiological effects of treadmill exercise in CLBP patients and to investigate whether these responses are moderated/mediated by individual levels of: fear of injury, pain catastrophising and depression. As far as I am aware no such study has been done before.

Method
The present experiment is designed as a between group comparison with repeated measurements. The two groups, patients suffering chronic low back pain (CLBP) and healthy volunteers (CONT), are to perform a moderate physical activity – 15 minutes walking on a treadmill. The physiological parameters, which are being measured pre- and post- intervention are: resting heart rate and BP; saliva cortisol level; static sEMG of paraspinal muscles and present pain intensity (PPI). Heart rate and BP are well accepted as indicators for the activity of the autonomic (Sympathetic and Parasympathetic) nervous system (Armstrong et al., 2005), while salivary cortisol is good representor of free cortisol (Kirchbaum and Hellhammer, 1994) and the HPA axis activity. Data on demographic co-founding factors: age; ethnic origin; pain duration and experience; positive and negative affectivity (trait and state) and exercise level, is also collected with the use of a questionnaire.

Participants
Participants are to be recruited from members of the public (personal and colleague's contacts, back pain charities and support groups) and Roehampton University students and staff volunteers. Twenty participants per group are randomly selected from a pre-consented group of fifty CLBP patients and fifty healthy volunteers. The main participation criteria are as follows:

- **Study group (CLBP):** 20 chronic low back pain male patients

  Inclusion criteria:
  - Age – 35 to 60
  - BMI – 18 to 27
  - Low back pain localisation – predominantly concentrated over the lumbar and back of the hips area, but also radiating to the legs
  - Low back pain duration – a minimum of 12 consecutive weeks in the past 6 months
  - Pain severity – any level of pain for which medical treatment has been sought
  - Ability to walk briskly for 15 minutes
  - Good command of written and spoken English

  Exclusion criteria:
  - Inflammatory rheumatic conditions: Rheumatoid arthritis, ankylating spondylitis, psoriatic arthritis, SLE and other auto-immune conditions
  - Spinal stenosis, severe discopathy, spondylolisthesis and other spinal deformities
  - Spinal dislocations, fractures and operations
  - Other recent unresolved traumatic injuries
  - Mental illness or clinical depression, requiring medication
  - Cardiac and pulmonary diseases, requiring treatment
  - Steroid, β-blockers or other serious medication

- Mouth and gum diseases

- **Control group (CONT):** 20 healthy sedentary (exercising less than twice of 1 hour per week), male volunteers, who haven't suffered low back pain in the last 6 months and have similar vital characteristics: 35-60 years old and BMI of 18 – 27, with good command of written and spoken English.

Measurements

As the principal aim of the study is to investigate physiological changes, the main measurements are: resting heart rate and BP (HR&BP); salivary cortisol level (SCL); static sEMG of deep multifidi (MF) and superficial erector spinae (ES) mm and present pain intensity (PPI). The resting heart rate and BP are measured using chest sensors and arm cuff (Polar, model S625X) and standardised protocol in supine position. The saliva cortisol level is measured with DRG Salivary cortisol ELISA (SLV-2930). Static sEMG of MF and ES is measured in three different positions: prone, standing and fully bent down, using Biometrics sEMG system, model Datalog P3X8. The electrodes are placed on pre-prepared (shaved and cleaned with alcohol wipes) locations on the back, as recommended by the SENIAM protocol (Hekmens et al., 1999). The present pain intensity (PPI) is measured using standard 100 points visual analogue scale (VAS).
As illustrated in the preceding paragraphs, the affective/emotional and cognitive aspects are a major factor in CLBP. A Back Pain questionnaire (BPQ – Appendix 1) was designed to measure participants' pain experience, attitude and general emotional status (affect and trait). It incorporates three well-established scales: short-form McGill Pain Questionnaire (Melzack, 2005); Pain Catastrophising Scale (Sullivan et al., 1995; Picavet et al., 2002) and DASS-21 scale (Norton, 2007). It also includes questions about general demographic characteristics, which might co-found the results: age, ethnic origin, low back pain duration and exercising level.

Experiment protocol

The 20 CLBP and 20 CONT randomly selected participants are debriefed about the procedures involved in the experiment, demonstrated walking on a treadmill and advised to abstain from alcohol for 24 hours and avoid eating for 2 hours prior to the test. Participants are randomly allocated to experiment dates in groups of 2 CLBP and 2 CONT. To ensure as constant as possible level and exclude natural diurnal fluctuations of cortisol, the time of the experiment is set for mid afternoon – 3-5pm.
On the day of the experiment, participants are given to fill the Back Pain questionnaire. After resting (lying down, or if lying is uncomfortable – sitting down) in a quiet room for 15 min, initial measurements (test 1) are taken:
Heart rate and BP

Saliva sample for cortisol analysis

sEMG of MF and ES mm in 3 static positions: prone lying down, standing and fully bent down

Present Pain Intensity (PPI)

After gradual increase of the speed of the treadmill to 60% of max effort (heart rate reverse method – Armstrong et al., 2005), participants maintain walking for 15 min at that level, than slow down gradually.
After the treadmill walk, participants rest (lying down, or if lying is uncomfortable – sitting down) again for 15 min in a quiet room and second set of measurements (test 2) are taken:
Heart rate and BP

Saliva sample for cortisol analysis

sEMG of MF and ES mm in 3 static positions: lying down, standing and fully bent down

Present Pain Intensity (PPI)

The collected saliva samples are frozen and later analysed (DRG salivary cortisol ELISA) for cortisol content.

Data analysis
The results are recorded in a logbook and entered into computer for analysis using SPSS 16.0 program. Descriptive statistics of the independent variables and the difference (test 2 minus test 1) of the dependent variables are obtained (mean, SD) and analysed for normality of distribution. Two-way ANOVA is performed on the mean differences (test 2 minus test 1) of all dependent variables, comparing CLBP against CONT. Correlations are performed on all the variables in order to establish what the relationship between them is. Regression analysis is later performed, controlling for emotional/affective measurements, pain intensity, ethnic origin, age and other variables, which show strong correlation, in order to clarify the relationship between them.

Benefits and limitations
Most of the exercise studies in CLBP are longitudinal, concentrating on functionality and pain outcomes. This study, in comparison, measures short-term immediate physiological changes. Its purpose is to provide a snapshot of the effects of exercise in CLBP patients and further the understanding of their neuroendocrine mechanisms, through simultaneous HPA axis, autonomic nervous system and sensory-motor activity measurements.
There are few intrinsic limitations to the design of our experiment – the size and composition of the study sample; one rather than three control groups (two more groups of CLBP and CONT not performing moderate exercise, would strengthen the validity of the results). Exercise produces its beneficial effects through slow, accumulative changes, so to investigate its long-term physiological effects in CLBP, a different, longitudinal experiment is required.
In this study the psychological/affective component, which is a major feature in CLBP, is used only as an independent variable. A longitudinal study would have also allowed for the examination of the dynamic and complex interactions between exercise, physiological and psychological aspects of CLBP.
This is a pilot, MSc project, which is restricted both financially and time-wise. It is hoped that the results it produces will lay the foundations for larger, better-controlled and more comprehensive studies.

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Zedka M, Prochazka A, Knight B, Gillard D, Gauthier M. Voluntary and reflex control of human back muscles during induced pain. Journal of Physiology 1999; 520(2):591-604.

SECTION 3: SOURCE OF DATA INCLUDING USE OF PARTICIPANTS

What is the nature of the data you use? Please state clearly:

Human participants YES If yes, please answer questions below

Archival data NO If yes, describe whether confidentiality and data protection are issues and they will be addressed

Other data sources (provide details)

…… …….

- You should use the attached consent form and adapt it as required
- You should also attach any other information to be given to participants (information sheet, questionnaires, debriefing form)
- You should consider carefully what information you provide to participants, e.g. scope of study, number of participants, duration of study, risks/benefits of the project
- If images or anything else which might allow the identification of participants is to be publicly accessible (e.g. on the web), further written consent must be secured

Give details of the method of recruitment, and potential benefits to participants if any:

10. Personal contacts
11. Fellow therapists
12. Advertising with back pain charities and back pain support groups – placing a recruitment poster on websites and forums
13. Roehampton University staff and students – placing recruitment posters and email drops

Will you be using participants who are aged under 18? NO

If you have answered Yes please highlight the particular issues raised by working with these participants and how these issues have been addressed.

SECTION 4: HEALTH AND SAFETY

1. You should download and complete the Risk Assessment Form
2. You should be able to demonstrate that appropriate mechanisms are in place for the research to be carried out safely
3. If necessary the University's Health, Safety & Environment Manager should be consulted before the application is submitted

Has a Risk Assessment been carried out for this research?

YES

If a Risk Assessment has not yet been carried out please explain why:

Is this a clinical trial or a project which may involve abnormal risk to participants? NO

If you have answered Yes please refer to Sections 3.5 and 4.2 of the Ethical Guidelines

SECTION 5: PUBLICATION OF RESULTS

How will you publish your results?

MSc Dissertation

How will you ensure the anonymity of your participants?
(If your participants do not wish to remain anonymous you must obtain their written consent.)

Participants are ascribed ID numbers, which are used in all the computer data entry and analysis. The participants names are present only on the original signed consent forms and paper questionnaires, which a re stored safely in a locked cabinet.

SECTION 6: STORAGE OF DATA

Section 2.7 of *Roehampton University Code of Good Research Practice* states the following 'research data must normally be retained intact for a period of at least six years from the date of any publication which is based upon it. Researchers should be aware that specific professional bodies and research councils may require a longer period of data retention.'

Describe how and where the following data will be stored and how they will be kept secure:

Raw and processed data
Raw data is kept filed in a secure filing cabinet and computer.

Documents containing personal details of any volunteers

Questionnaires with personal information are kept filed in a secure filing cabinet.

SECTION 7: EXTERNAL GUIDELINES AND APPROVAL

Are there any relevant subject-specific ethical guidelines (e.g. from a professional society)? NO

If so how will these inform your research process?

Has/will the project be submitted for approval to the ethical committee of any other organisation? NO

What is the outcome of this?

SECTION 8: APPLICANT'S SIGNATURE

Applicant's signature:	Dragomir P Lubomirov
Date:	18 November 2009

PLEASE NOTE: YOU MUST NOT BEGIN YOUR RESEARCH UNTIL YOUR ETHICS APPLICATION HAS BEEN APPROVED BY 2 ACADEMIC MEMBERS OF STAFF.

APPENDIX 2. Risk Assessment

	Event / Activity:				Date Assessed: Review Date:				Assessor's Name: Assessor's Signature:
		Uncontrolled Risk Severity x Likelihood = Risk Rating				Residual Risk Severity x Likelihood = Risk Rating			
Hazard	To Whom	S	L	R	Control Risk by	S	L	R	Further Action Needed
treadmill walking	participant	1	1	1				0	
HR and BP measuring	participant	1	1	1				0	
saliva sample taking	participant/ investigator	1	1	1				0	
sEMG electrodes	participant	1	1	1				0	
saliva cortisol ELISA	investigator	1	1	1				0	
computer data processing	investigator	1	1	1				0	

Severity

HIGH	3	Fatality or major injury causing long-term disability
MEDIUM	2	Injury or illness causing short-term disability
LOW	1	Other injury or illness

Likelihood

HIGH	3	Certain or near certain
MEDIUM	2	Reasonably likely
LOW	1	Very seldom or never

Risk Matrix

		Likelihood		
Severity		H	M	L
	H	9	6	3
	M	6	4	2
	L	3	2	1

Risk Rating

6 - 9 HIGH RISK Immediate action required to reduce risk

APPENIX 3. Recruitment Poster

Roehampton University London

VOLUNTEERS NEEDED!!!

We are looking to recruit chronic low back pain (CLBP) male sufferers, as well as healthy male volunteers, 24-64 years old, to take part in an exciting MSc research project. Our study will involve one-off 15 minutes moderate treadmill exercise (walking) and will examine the immediate physiological effects that it produces. Resting heart rate and blood pressure, salivary cortisol level, back muscles activity and pain intensity will be measured by non-invasive means pre and post exercise.

The aim of our study is to provide a snap-shot of the physiological mechanisms, which underlie the effects of exercise in CLBP sufferers. With your indispensable participation and help we hope the results of our study will further CLBP and exercise knowledge and contribute to the treatment and management of this chronic debilitating condition.

For further information or to register your interest, please contact Dragomir: Telephone - **07976 253682,** or email - dragomirl@btinternet.com

THANK YOU

APPENDIX 4. Participant Information Sheet

Roehampton University London

PARTICIPANT INVITATION LETTER

Research project: *How does Chronic Back Pain affect Psycho-Physiological Responses of the body to Moderate Exercise?*

Dear participant,

We invite you to take part in a research study at the School of Human and Life Sciences, Roehampton University. First, we wish you to know that taking part in this research is entirely voluntary. You may choose not to take part, or you may withdraw from the study at any time. Before you decide to take part, please take as much time as you need to ask any questions.

Purpose and Procedures

The aims of this study are to further our understanding of how the body responds to treadmill exercise in patients suffering Chronic Low Back Pain (CLBP) and to investigate whether these responses are associated with psychological and emotional factors.

At the beginning, you will be given a short written questionnaire to complete. Please note that the questionnaire is designed simply to look at normal variation in human emotional and physical experiences of pain.

Prior to being accepted to the study, we will need to ask some questions about your general health and lifestyle. All this information will be available only to the research team and treated in the strictest confidence. Participation will involve one afternoon lab session, lasting about 1.5 hour – please bring comfortable cloths and walking shoes/trainers. You will also be asked to complete a short health questionnaire before taking part, in order for the experimenter to judge that it is safe for you to take part and will be explained and demonstrated walking on treadmill.

On the day of the experiment, you will be given to fill a Back Pain questionnaire and will perform treadmill walking at 60% of your maximal effort. Two sets of non-invasive body measurements – one before and one after the treadmill walking - will be taken: Heart rate and BP; saliva sample for cortisol analysis; sEMG of lower back muscles in 3 static positions: prone lying down, standing and fully bent down; Present Pain Intensity. There will be a resting period (lying down, or if lying is uncomfortable – sitting down in a quiet room) of 15 min before each set of measurements is taken.

Possible Side Effects and Hazards

Treadmill walking, Heart rate, blood pressure and sEMG measuring are very safe and harmless procedures. Saliva will be collected to learn about your levels of the stress

hormone called cortisol.

Benefits

Information learned from this study will be used to help our understanding of our bodily and psychological reactions to Chronic Low Back Pain (CLBP), as well as the effects of exercise for this chronic pain condition. This knowledge may expand the scope of application of exercise as a therapeutic intervention. Furthermore, this study may lead to advances in the prevention and treatment of CLBP and other chronic pain conditions.

Thank you for considering your contribution to this research project.

1 November, 2009

Principal Investigator (signed)

Dragomir P Lubomirov

APPENDIX 5. Participant Consent Form

Roehampton University London

PARTICIPANT CONSENT FORM

Chronic Low Back Pain Study

This study will be performed under the supervision of Dragomir Lubomirov at the School of Human and Life Sciences, Roehampton University. It has been approved by Roehampton University's Ethics Board. The study will involve 15 minutes moderate treadmill exercise (walking) and will examine the immediate physiological effects it produces by measuring resting heart rate and blood pressure, salivary cortisol level, back muscles activity, emotional state and pain intensity.

Consent Statement:

a. I have read and received a copy of this consent form and have been given the opportunity to ask questions. You have given me: (i) an explanation of the procedures to be followed in the project, including an identification of those, which are experimental; and (ii) answers to inquiries I have made.

b. I understand that there may be no direct benefit to me from my participation in this study as described above.

c. I understand that my participation will not cost me anything other than the time and effort involved.

d. I understand that all personal data relating to volunteers are held and processed in the strictest confidence, in accordance with the Data Protection Act (1998).

e. I understand that by signing this agreement, I do not waive any legal rights or release Roehampton University, its agents, or you from liability for negligence.

f. I understand that I am free to withdraw from the study at any time without needing to justify my decision.

g. I understand that this study is entirely anonymous. My identity will not be recorded or passed on to anyone not involved in this study, and will be protected in the publication of any findings. Researchers involved in the study will be unaware of any links between my identity and the data collected and accordingly no individual feedback will be given.

h. I understand that it is envisaged that the results – which will be entirely anonymous – will be submitted for publication or conference presentations

i. I confirm that I have read and understood the above and freely consent to participating in this study. I have been given adequate time to consider my participation and agree to comply with the instructions and restrictions of the study.

Name ……
.

Signature ……

Date ……

If you require advice, information or reassurance about a technical or health related matter, or have a concern about any other aspect of your participation, please raise this with the principal investigator, Dragomir Lubomirov:
email: dragomirl@btinternet.com, Telephone: 07976 253682.

Alternatively please contact Dr Jolanta Opacka-Juffry:
email: j.opacka_juffry@roehampton.ac.uk, Telephone: 020-8392 3563

or

Dean of the School of Human and Life Sciences, Mr Michael Barham:
email: M.Barham@roehampton.ac.uk, Telephone: 020-8392 3617

If you are a student and feel that you need counselling support after this experiment, please contact the student counselling service, Telephone: 020 8392 3636.

APPENDIX 6. Back Pain Questionnaire

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. This questionnaire will ask you a series of questions about your personal experience of low back pain and how it affects you physically and emotionally in your everyday life.

We would be very grateful if you could spare 5 to 10 minutes to complete this short questionnaire. You will find that some aspects will be addressed by more than one question. Please ensure that you answer all questions - this will enable us to develop a shorter questionnaire for future studies. There are no right or wrong answers. All information you give will be treated in confidence.

For each question please circle the appropriate answer.

1. Name ……..

2. What is your age? ..... years

3. Are you?	White	1
	Black Afro-Caribbean	2
	African	3
	Asian	4
	Other	5

4. If you suffered low back pain, how long have you suffered for?

Do not suffer back pain	0
Less than 1 year	1
1 – 3 years	2
4 – 5 years	3
6 – 10 years	4
more than 10 years	5

5. Do you currently engage in any exercise/physical activity?
(By exercise or physical activity we mean any planned physical exertion aimed at improving or maintaining physical fitness and health, lasting more than 20-30 minutes. This includes aerobics, brisk walking, jogging, swimming, biking, rowing etc. However, lawn bowling, snooker, casual walking and similar activities are not included.)

No at all	1
Less than once per week	2
At least once per week	3
More than once per week	4

6. From the list bellow, please circle what best describes your pain in general.
0 = none
1 = mild
2 = moderate
3 = severe

Throbbing	0	1	2	3
Shooting	0	1	2	3
Stabbing	0	1	2	3
Sharp	0	1	2	3
Cramping	0	1	2	3
Gnawing	0	1	2	3
Hot-burning	0	1	2	3
Aching	0	1	2	3
Heavy	0	1	2	3
Tender	0	1	2	3
Splitting	0	1	2	3
Tiring-exhausting	0	1	2	3
Sickening	0	1	2	3
Fearful	0	1	2	3
Punishing-cruel	0	1	2	3

7. How strong was your pain on average over the past week?

No pain I----------I Worst possible pain

8. What thoughts and feelings do you experience when you are in pain?
0 = not at all
1 = to a slight degree
2 = to a moderate degree
3 = to a great degree
4 = all the time

I worry all the time about whether the pain will end	0	1	2	3	4
I feel I can't go on	0	1	2	3	4
It's terrible and I think it's never going to get any better	0	1	2	3	4
It's awful and I feel that it overwhelms me	0	1	2	3	4
I feel I can't stand it anymore	0	1	2	3	4
I become afraid that the pain will get worst	0	1	2	3	4
I keep thinking of other painful events	0	1	2	3	4
I anxiously want the pain to go away	0	1	2	3	4

I can't seem to keep it off my mind	0	1	2	3	4
I keep thinking how much it hurts	0	1	2	3	4
I keep thinking about how badly I want the pain to stop	0	1	2	3	4
There's nothing I can do to reduce the intensity of the pain	0	1	2	3	4
I wonder whether something serious may happen	0	1	2	3	4

9. How much do the statements from the list below applied to you over the past week?
0 = Did not apply to me at all
1 = Applied to me to some degree, or some of the time
2 = Applied to me to a considerable degree, or a good part of time
3 = Applied to me very much, or most of the time

I found it hard to wind down	0	1	2	3
I was aware of dryness of my mouth	0	1	2	3
I couldn't seem to experience any positive feeling at all	0	1	2	3
I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
I found it difficult to work up the initiative to do things	0	1	2	3
I tended to over-react to situations	0	1	2	3
I experienced trembling (eg. in the hands)	0	1	2	3
I felt I was using a lot of nervous energy	0	1	2	3
I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
I felt I had nothing to look forward to	0	1	2	3
I found myself getting agitated	0	1	2	3
I found it difficult to relax	0	1	2	3
I felt down-hearted and blue	0	1	2	3
I was intolerant of anything that kept me fro getting on with what I was doing	0	1	2	3
I felt I was close to panic	0	1	2	3
I was unable to become enthusiastic about anything	0	1	2	3
I felt I wasn't worth much as a person	0	1	2	3
I felt that I was rather touchy	0	1	2	3
I was aware of the action of my heart in the absence of physical exertion (eg. Sense of heart rate increase, heart missing a beat)	0	1	2	3
I felt scared without any good reason	0	1	2	3
I felt that life was meaningless	0	1	2	3

Thank You

APPENDIX 7. Emotional State Questionnaire

This questionnaire consists of a number of words that describe different feelings and emotional states. Please read each item and then circle the appropriate answer which best describes the way you feel at this moment. You will find that some aspects will be addressed by more than one question. Please ensure that you answer all questions - this will enable us to develop a shorter questionnaire for future studies. There are no right or wrong answers. All information you give will be treated in confidence.

For each question please circle the appropriate answer

	Very slightly or not at all	A little	Moderately	Quite a bit	Extremely
1. Interested	1	2	3	4	5
2. Distressed	1	2	3	4	5
3.Excited	1	2	3	4	5
4.Upset	1	2	3	4	5
5.Strong	1	2	3	4	5
6.Guilty	1	2	3	4	5
7.Scared	1	2	3	4	5
8.Hostile	1	2	3	4	5
9.Enthusiastic	1	2	3	4	5
10.Proud	1	2	3	4	5
11.Irritable	1	2	3	4	5
12.Alert	1	2	3	4	5
13.Ashamed	1	2	3	4	5
14.Inspired	1	2	3	4	5
15.Nervous	1	2	3	4	5
16.Determined	1	2	3	4	5
17.Attentive	1	2	3	4	5
18.Jittery	1	2	3	4	5
19.Active	1	2	3	4	5
20.Afraid	1	2	3	4	5
21.Amused	1	2	3	4	5
22.Content	1	2	3	4	5
23.Angry	1	2	3	4	5
24.Disgusted	1	2	3	4	5
25.Fearful	1	2	3	4	5
26.Sad	1	2	3	4	5
27.Surprised	1	2	3	4	5
28.Happy	1	2	3	4	5
29.Anxious	1	2	3	4	5

If you are in pain, how strong is your pain at present?

No pain I----------I Worst possible pain

APPENDIX 8. Research protocol sheet

Roehampton University London

PROTOCOL SHEET

ID Number:

Date: Time:

2. Explain the protocol to the participant and obtain signed consent	
3. Ask the participant to fill in the Back Pain Questionnaire (BPQ)	
4. Inspect the back and chest and prepare the areas for electrode attachment	
5. Fit the HR monitor	
6. Ask the participant to lie down (supine) and relax for 15 min	
7. Measure HR and BP at the end of rest period	
8. Take first saliva sample	
9. Ask participant to fill in the Emotional State Questionnaire (ESQ)	
10. Ask the participant to turn over and lie on the front, mark the locations for MF and ES measurements and fix sEMG sensors (low body slightly flexed)	
11. After lying for 30 sec take a 20 sec EMG in prone position	
12. After standing up slowly, adjust posture at feet shoulder apart and eyes straight ahead. Allow for 30 sec settling time and take 20 sec standing EMG	
13. Ask the participant to bend forward to reach his toes at normal speed, remain fully flexed for 5 seconds and straighten up at normal speed. Rehearse the sequence once and then take EMG of the entire sequence	
14. Remove the electrodes from the participant's back	
15. Start the 15 min treadmill walk (treadmill at 17.5º incline) and increase the speed gradually, aiming for 55% of max effort	
16. After the treadmill walk, measure second BP and HR	
17. Take a second saliva sample	
18. Ask participant to fill in a second ESQ	
19. Allow the participant to cool down and then ask them to lie down and relax for 15 minutes as in 5	
20. Measure third HR and BP at the end of rest period	
21. Take third saliva sample	
21. Ask participant to fill in a third ESQ	
22. Ask the participant to turn over and lie on the front and attach the sensors on the marked places as in 9	
23. After relaxing for 30 sec take a second 20 sec EMG in prone position	
24. After standing up slowly and adjusting posture as in 11 allow 30 sec settling time and take a second 20 sec standing EMG measurement	
25. Ask the participant to bend forward to reach his toes at normal speed, remain fully flexed for 5 seconds and straighten up at normal speed. Take EMG recording of the entire sequence	

APPENDIX 9. Tables

Table 1. Experimental groups / Ethnicity Chi-Square Test

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	**.135[a]**	**1**	**.714**		
Continuity Correction	**.000**	**1**	**1.000**		
N of Valid Cases	**20**				

a. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 3.60.

Table 2. Independent Samples t-Test of Age between CLBP and Control

		Levene's Test for Eq of Variances		t-test for Equality of Means					
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff.	Std. Error Difference	95% CI of the Difference
									Lower
age	Eq var assumed	**2.381**	**.140**	**-1.302**	**18**	**.209**	**-4.677**	**3.593**	**-12.225**
	Eq var not assumed			**-1.238**	**12.499**	**.239**	**-4.677**	**3.778**	**-12.872**

Table 3. CLBP and Control groups Exercise frequency Chi-Square Test

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	**4.463[a]**	**3**	**.216**
N of Valid Cases	**20**		

a. 7 cells (87.5%) have expected count less than 5. The minimum expected count is .90.

Table 5. Depression, Anxiety and Stress independent Samples t-Test between CLBP and Control

		Levene's Test for Eq of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Diff	
									Lower	Upper
Depression	Eq var assumed	**.882**	**.360**	**-2.347**	**18**	**.031**	**-3.303**	**1.408**	**-6.260**	**-.346**
	Eq var not assumed			**-2.247**	**13.170**	**.042**	**-3.303**	**1.470**	**-6.475**	**-.131**
Anxiety	Eq var assumed	**.072**	**.791**	**-1.667**	**18**	**.113**	**-1.808**	**1.084**	**-4.086**	**.470**
	Eq var not assumed			**-1.648**	**16.338**	**.118**	**-1.808**	**1.097**	**-4.129**	**.513**

Stress	Eq var assumed	**2.561**	**.127**	**-1.432**	**18**	**.169**	**-2.535**	**1.770**	**-6.254**	**1.183**
	Eq var not assumed			**-1.478**	**17.842**	**.157**	**-2.535**	**1.715**	**-6.141**	**1.070**

Table 7. Pain Catastrophising Independent Samples t-Test between CLBP and Control

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Difference	
									Lower	Upper
Pain Cata-strophisising	Eq var assumed	**5.110**	**.036**	**-4.085**	**18**	**.001**	**-17.455**	**4.273**	**-26.431**	**-8.478**
	Eq var not assumed			**-3.814**	**10.796**	**.003**	**-17.455**	**4.576**	**-27.55**	**-7.360**

Table 8. Correlations between general pain level and Pain Catastrophising

		pain level	Pain Catastrophisisng
pain level	Pearson Correlation	**1**	**.755****
	Sig. (2-tailed)		**.000**
	N	**20**	**20**
Pain Catastrophisisng	Pearson Correlation	**.755****	**1**
	Sig. (2-tailed)	**.000**	
	N	**20**	**20**

**. Correlation is significant at the 0.01 level (2-tailed).

Table 9. Correlations between Pain Catastrophising, Depression, Anxiety and Stress

		Pain Catastrophisisng	Depression	Anxiety	Stress
Pain Catastrophisisng	Pearson Correlation	**1**	**.374**	**.410**	**.258**
	Sig. (2-tailed)		**.104**	**.073**	**.272**
	N	**20**	**20**	**20**	**20**
Depression	Pearson Correlation	**.374**	**1**	**.571****	**.654****
	Sig. (2-tailed)	**.104**		**.009**	**.002**
	N	**20**	**20**	**20**	**20**
Anxiety	Pearson Correlation	**.410**	**.571****	**1**	**.658****

	Sig. (2-tailed)	.073	.009		.002
	N	20	20	20	20
Stress	Pearson Correlation	.258	.654**	.658**	1
	Sig. (2-tailed)	.272	.002	.002	
	N	20	20	20	20

**. Correlation is significant at the 0.01 level (2-tailed).

Table 14. Control and CLBP Negative Affect Independent Samples t-Test at three time-points

		Levene's Test for Eq of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Difference	
									Lower	Upper
NA 1	Eq var assumed	.005	.944	.000	18	1.000	.000	2.560	-5.379	5.379
	Eq var not assumed			.000	17.669	1.000	.000	2.540	-5.343	5.343
NA 2	Eq var assumed	.572	.459	.207	18	.838	.545	2.631	-4.982	6.073
	Eq var not assumed			.218	16.699	.830	.545	2.504	-4.744	5.835
NA 3	Eq var assumed	.188	.669	.065	18	.949	.293	4.475	-9.109	9.695
	Eq var not assumed			.067	17.971	.947	.293	4.360	-8.868	9.454

Table 16. Control and CLBP Independent Samples t-Test of fully bent forward right MF at 2 time-points

		Levene's Test for Eq of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Difference	
									Lower	Upper
MF(R) bent forward 1	Eq var assumed	8.201	.013	-2.638	13	.020	-6.67375	2.52977	-12.13898	-1.20852
	Eq var not assumed			-2.476	6.678	.044	-6.67375	2.69551	-13.11037	-.23713
MF(R) bent forward 2	Eq var assumed	11.416	.005	-2.569	13	.023	-11.38625	4.43301	-20.96319	-1.80931
	Eq var not assumed			-2.391	6.051	.054	-11.38625	4.76200	-23.01474	.24224

Table 17. Paired Samples Statistics of static muscle activity in the CLBP group

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	ES(L) standing 1	**1.6943**	**7**	**1.03194**	**.39004**
	ES(L) standing 2	**1.4929**	**7**	**.80647**	**.30482**
Pair 2	ES(R) standing 1	**2.2714**	**7**	**1.52925**	**.57800**
	ES(R) standing 2	**1.5529**	**7**	**.50454**	**.19070**
Pair 3	ES(L) bent forward1	**33.6429**	**7**	**56.36198**	**21.30283**
	ES(L) bent forward 2	**15.4300**	**7**	**32.91936**	**12.44235**
Pair 4	ES(R) bent forward 1	**3.2329**	**7**	**2.14925**	**.81234**
	ES(R) bent forward 2	**3.1614**	**7**	**2.06838**	**.78178**
Pair 5	MF(L) standing 1	**2.4157**	**7**	**1.74931**	**.66118**
	MF(L) standing 2	**1.7929**	**7**	**1.05248**	**.39780**
Pair 6	MF(L) bent forward 1	**8.0257**	**7**	**7.44446**	**2.81374**
	MF(L) bent forward 2	**10.2914**	**7**	**9.68534**	**3.66071**
Pair 7	MF(R) bent forward 1	**8.8000**	**7**	**6.93846**	**2.62249**
	MF(R) bent forward 2	**12.9000**	**7**	**12.57246**	**4.75194**
Pair 8	MF(R) standing 1	**3.4943**	**7**	**4.35693**	**1.64676**
	MF(R) standing 2	**2.2871**	**7**	**1.43016**	**.54055**

Table 19 Control and CLBP Independent Samples t-test of the FRR of left ES and right MF muscles

		Levene's Test		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Difference	
									Lower	Upper
ES(L) FRR1	Eq var assumed	**8.835**	**.012**	**3.087**	**12**	**.009**	**2.13752**	**.69235**	**.62902**	**3.64602**
	Eq var not assumed			**3.087**	**8.842**	**.013**	**2.13752**	**.69235**	**.56705**	**3.70799**
ES(L) FRR2	Eq var assumed	**4.034**	**.068**	**2.332**	**12**	**.038**	**2.26106**	**.96940**	**.14892**	**4.37321**
	Eq var not assumed			**2.332**	**8.298**	**.047**	**2.26106**	**.96940**	**.03951**	**4.48262**
MF(R) FRR2	Eq var assumed	**17.038**	**.001**	**2.731**	**12**	**.018**	**6.24801**	**2.28796**	**1.26297**	**11.23305**
	Eq var not assumed			**2.731**	**6.363**	**.032**	**6.24801**	**2.28796**	**.72599**	**11.77003**

Table 20. Paired Samples Statistics of FRR of ES and MF of CLBP participants

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	ES(L) FRR1	**1.4362**	**7**	**.82165**	**.31055**
	ES(L) FRR2	**1.5597**	**7**	**1.04505**	**.39499**
Pair 2	ES(R) FRR1	**1.7725**	**7**	**.76266**	**.28826**
	ES(R) FRR2	**3.5688**	**7**	**3.48435**	**1.31696**
Pair 3	MF(R) FRR1	**5.9539**	**7**	**12.40404**	**4.68829**
	MF(R) FRR2	**1.5437**	**7**	**1.03704**	**.39197**
Pair 4	MF(L) FRR1	**12.2255**	**7**	**28.71846**	**10.85456**
	MF(L) FRR2	**1.9773**	**7**	**1.89771**	**.71727**

Table 22. Control and CLBP Present Pain Intensity Independent Samples t-Test

		Levene's Test for Eq of Variance		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Diff	
									Lower	Upper
Pain1	Eq var assumed	**26.670**	**.000**	**-4.362**	**18**	**.000**	**-23.091**	**5.294**	**-34.213**	**-11.969**
	Eq var not assumed			**-3.947**	**8.415**	**.004**	**-23.091**	**5.850**	**-36.466**	**-9.715**
Pain 2	Eq var assumed	**33.571**	**.000**	**-4.750**	**18**	**.000**	**-32.808**	**6.907**	**-47.319**	**-18.297**
	Eq var not assumed			**-4.272**	**8.031**	**.003**	**-32.808**	**7.680**	**-50.506**	**-15.110**
Pain 3	Eq var assumed	**10.004**	**.005**	**-4.303**	**18**	**.000**	**-25.051**	**5.822**	**-37.281**	**-12.820**
	Eq var not assumed			**-3.874**	**8.099**	**.005**	**-25.051**	**6.466**	**-39.929**	**-10.172**

Table 23. Multivariate Tests of PPI at three time-points in Control and CLBP

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared
Control PPI	Pillai's Trace	**.159**	**.852[a]**	**2.000**	**9.000**	**.458**	**.159**
	Wilks' Lambda	**.841**	**.852[a]**	**2.000**	**9.000**	**.458**	**.159**
	Hotelling's Trace	**.189**	**.852[a]**	**2.000**	**9.000**	**.458**	**.159**
	Roy's Largest Root	**.189**	**.852[a]**	**2.000**	**9.000**	**.458**	**.159**
CLBP PPI	Pillai's Trace	**.139**	**.566[a]**	**2.000**	**7.000**	**.592**	**.139**
	Wilks' Lambda	**.861**	**.566[a]**	**2.000**	**7.000**	**.592**	**.139**
	Hotelling's Trace	**.162**	**.566[a]**	**2.000**	**7.000**	**.592**	**.139**
	Roy's Largest Root	**.162**	**.566[a]**	**2.000**	**7.000**	**.592**	**.139**

Table 24. Independent Samples t-Test of Present Pain after exercise between CLBP sub-groups

		Levene's Test		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Diff	
									Lower	Upper
Pain after exercise	Eq var assumed	**1.203**	**.309**	**-3.739**	**7**	**.007**	**-35.650**	**9.534**	**-58.194**	**-13.106**
	Eq var not assumed			**-3.875**	**6.992**	**.006**	**-35.650**	**9.201**	**-57.411**	**-13.889**

Table 25. Correlations between PPI and Psychological and muscle activity measures in CLBP

		pain1	Pain 2	pain 3
pain level	Pearson Correlation	**.835****	**.083**	**.711***
	Sig. (2-tailed)	**.005**	**.832**	**.032**
	N	**9**	**9**	**9**
Depression	Pearson Correlation	**.824****	**.648**	**.827****
	Sig. (2-tailed)	**.006**	**.059**	**.006**
	N	**9**	**9**	**9**
Anxiety	Pearson Correlation	**.167**	**.300**	**.230**
	Sig. (2-tailed)	**.668**	**.433**	**.552**
	N	**9**	**9**	**9**
Stress	Pearson Correlation	**.147**	**.268**	**.129**
	Sig. (2-tailed)	**.706**	**.485**	**.740**
	N	**9**	**9**	**9**
Pain Catastrophisisng	Pearson Correlation	**.134**	**-.010**	**.212**
	Sig. (2-tailed)	**.730**	**.979**	**.583**
	N	**9**	**9**	**9**
Negative Affect 1	Pearson Correlation	**.169**	**-.471**	**.198**
	Sig. (2-tailed)	**.663**	**.201**	**.610**
	N	**9**	**9**	**9**
Negative Affect 2	Pearson Correlation	**.170**	**-.564**	**.223**
	Sig. (2-tailed)	**.662**	**.114**	**.565**
	N	**9**	**9**	**9**
Negative Affect 3	Pearson Correlation	**.316**	**-.519**	**.205**
	Sig. (2-tailed)	**.408**	**.152**	**.597**
	N	**9**	**9**	**9**
MF(R) bent forward 1	Pearson Correlation	**.031**	**-.096**	**-.158**
	Sig. (2-tailed)	**.947**	**.837**	**.735**
	N	**7**	**7**	**7**
MF(R) bent forward 2	Pearson Correlation	**.092**	**-.237**	**.005**
	Sig. (2-tailed)	**.844**	**.609**	**.991**
	N	**7**	**7**	**7**

ES(L) FRR1	Pearson Correlation	**-.281**	**-.507**	**-.178**
	Sig. (2-tailed)	**.541**	**.245**	**.703**
	N	**7**	**7**	**7**
ES(L) FRR2	Pearson Correlation	**-.245**	**-.541**	**-.156**
	Sig. (2-tailed)	**.596**	**.210**	**.738**
	N	**7**	**7**	**7**
MF(R) FRR2	Pearson Correlation	**-.398**	**-.363**	**-.328**
	Sig. (2-tailed)	**.377**	**.424**	**.473**
	N	**7**	**7**	**7**

**. Correlation is significant at the 0.01 level (2-tailed). *. Correlation is significant at the 0.05 level (2-tailed).

Table 29. ES(L) and MF(L) Standing 1 Independent Samples t-Test between sub-groups of CLBP

		Levene's Test for Eq of Var		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Diff	Std. Error Diff	95% CI of the Difference	
									Lower	Upper
ES(L) standing 1	Eq var assumed	**11.054**	**.021**	**-2.936**	**5**	**.032**	**-1.53583**	**.52313**	**-2.88059**	**-.19107**
	Eq var not assumed			**-3.399**	**3.541**	**.033**	**-1.53583**	**.45190**	**-2.85729**	**-.21438**
MF(L) standing 1	Eq var assumed	**8.483**	**.033**	**-3.017**	**5**	**.030**	**-2.62917**	**.87153**	**-4.86950**	**-.38883**
	Eq var not assumed			**-3.428**	**3.999**	**.027**	**-2.62917**	**.76687**	**-4.75857**	**-.49976**