THE RELATIONSHIP BETWEEN LUMBAR MOBILITY AND HAMSTRING FLEXIBILITY IN PADDLERS WITH AND WITHOUT LOW BACK PAIN

ROBYN JOHN

JHNROB005

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SUPERVISORS

THERESA BURGESS PhD(Exercise Science); BSc(Phys)

ROMY PARKER MSc(Pain); BSc(Med)(Hons)Ex Sci(Phys); BSc(Phys)

DEPARTMENT OF HEALTH AND REHABILITATION SCIENCES

GROOTE SCHUUR HOSPITAL

ANZIO ROAD; OBSERVATORY

SOUTH AFRICA

DECLARATION

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LIST OF ABBREVIATIONS

AKE	Active knee extension
CSA	Cross-sectional area
EMG	Electromyographic
KEA	Knee extension angle
L1	First lumbar vertebra
LBP	Low back pain
PKE	Passive knee extension
ROM	Range of motion
SBL	Superficial back line
SIJ	Sacroiliac joint
SLR	Straight leg raise
T12	Twelfth thoracic vertebra
VAS	Visual analogue scale

GLOSSARY OF TERMS

Flexibility:	Defined as 'the ability of a muscle to lengthen, allowing one joint (or more than one joint in a series) to move through a ROM' (Decoster *et al*, 2004; Frontera *et al*, 2006). Flexibility has also been described as a change in muscle length or a change in the ratio of length to tension (Frontera *et al,* 2006).
Low back pain:	Described as 'pain originating from the back and defined in an area bounded by the 12th thoracic vertebra and 12th ribs superiorly, the gluteal folds, inferiorly, and the contours of the trunk laterally' (Cleeland and Ryan, 1994).
Paddling:	A contemporary term used to describe 'a sport that involves manually propelling a small boat forward though water using a paddle' (http://en.wikipedia.org/wiki/Paddling). This is a general term which incorporates various styles, including canoeing, kayaking, dragon boating, surf skiing and, white water boating. Kayakers use a double-bladed paddle, whereas canoeists use a single-blade paddle (Kameyama *et al,* 1999). The kayaker sits in the boat while a canoeist kneels with their legs held far apart. In addition, a kayak has a closed cockpit and a canoe has an open cockpit (Shepard, 1987). In South Africa, the term 'canoeing' is used to refer to kayaking and 'Canadian canoe' is used to refer to canoeing.

ABSTRACT

Background: Low back pain (LBP) is a significant cause of morbidity in the athletic population. However, there is currently a lack of evidence regarding the mechanisms underlying and the factors contributing to LBP in paddlers. Paddling requires repetitive trunk flexion and rotation, and these athletes may therefore have an increased risk of developing LBP. It has also been postulated that a reduction in hamstring flexibility may be associated with LBP or altered lumbar range of motion (ROM). However, this relationship has not been systematically examined in paddlers that participate in sporting disciplines that report a prevalence of LBP.

Aim: To investigate the relationship between lumbar mobility and hamstring flexibility in paddlers with and without LBP.

Methods: Thirty endurance paddlers were recruited through advertisements and word of mouth. Fifteen participants (13 males, two females) who presented with paddling-associated LBP in the six months prior to testing formed the case group. The control group consisted of 15 participants (12 males, three females) with no history of LBP. Participants completed an informed consent form, a baseline questionnaire and a LBP questionnaire which included a visual analogue scale (VAS). An inclinometer was used to measure spinal flexion and extension ROM. The inclinometer was positioned on the base of the sacrum to record sacral flexion, extension and composite sacral ROM. The inclinometer was then positioned over the T12 and L1 spinous processes to measure T12/L1 flexion, extension and composite ROM. True lumbar flexion, extension and composite ROM were calculated as the difference between T12/L1 and sacral flexion, extension and composite ROM readings respectively. Hamstring flexibility was assessed using the active knee extension (AKE) test. The knee extension angle (KEA) represents hamstring flexibility and was measured using a Leighton’s flexometer. Average hamstring flexibility was calculated by averaging the sum of the left KEA and the right KEA.

Results: There were no significant differences in lumbar or sacral ROM between the case and control groups. Data were analysed through the use of Student's independent t-test and Mann-Whitney U tests. Relationships between variables were determined using a Pearson's product-moment correlation coefficient. Statistical significance was set at $p < 0.05$. A significant negative correlation was observed between age and composite T12/L1 ROM for the total group ($r = -0.39$; $p = 0.03$) as well as for the case group ($r = -0.59$; $p = 0.02$). There were also significant negative correlations between age and composite true lumbar ROM for the total group ($r = -0.53$; $p = 0.003$) and the control group ($r = -0.60$; $p = 0.02$). There were no significant correlations between age and hamstring flexibility. Left hamstring flexibility was significantly reduced in the case group compared to the control group (37.07 ± 9.12 vs. 29.60 ± 10.15 respectively, $p = 0.043$). There were no significant correlations between true lumbar ROM and hamstring flexibility, or sacral ROM and hamstring flexibility for the total group, case group or control group.

Discussion and conclusion: In conclusion, there were no significant differences in lumbar ROM between paddlers with and without a history of LBP. Although there were also no significant differences in average hamstring flexibility in the case and control groups, there was a significant reduction in unilateral hamstring flexibility in the case group. Although there were no significant relationships between average hamstring flexibility and lumbar ROM, there was a tendency for a negative relationship between average hamstring flexibility and lumbar ROM in the case group, which differed from the positive relationship in the control group. Without longitudinal studies, it is not possible to determine whether this relationship is causative of LBP, or as a consequence of the condition. Current clinical guidelines for the management of LBP, which include addressing lumbar mobility and hamstring flexibility, should remain until further evidence is forthcoming.

1. INTRODUCTION

Low back pain (LBP) may account for 5% to 8% of athletic injuries and is a significant cause of morbidity in the athletic population (Marais and Vlok, 2000). Certain athletic populations may be at risk of LBP due to increased loading of the spine, repetitive lumbar flexion and rotation, or hyperextension (Trainor and Wiesel, 2002). Elite, or high-level, sporting activity is considered a risk factor for LBP or the early development of degenerative disc disease (Barile *et al,* 2007; Heneweer *et al,* 2009; Ong *et al,* 2003).

Previous studies have determined a prevalence of LBP in paddlers, with reported rates varying between 15% and 52% (Fiore and Houston, 2001; Kameyama *et al*, 1999; Schoen and Stano, 2000). It is evident that endurance paddling requires repetitive motion that may overload the lumbar spine (Kameyama *et al*, 1999; Mann and Kearney, 1980). Endurance paddling also requires prolonged periods of unsupported sitting, which may increase the risk of overloading the lumbar spine (Deykin, 2006 as cited in Edge, 2006), and the development of LBP.

Although there is a relatively high prevalence of LBP in paddlers (Kameyama *et al*, 1999), there is currently a lack of evidence regarding the underlying mechanisms and the factors contributing to the development of LBP. Lumbar flexion and rotation have been identified as risk factors for LBP (Andersson, 1981; Esola *et al*, 1996), and lumbar range of motion (ROM) may also be altered in the presence of LBP (Andersson, 1981; Brukner and Kahn, 2001; Esola *et al*, 1996; Li *et al*, 1996). Paddling requires repetitive trunk flexion and rotation, and these athletes may therefore have an increased risk of developing LBP (Mann and Kearny, 1980; Shepard, 1987).

It has also been postulated that a reduction in hamstring flexibility may be associated with LBP or altered lumbar ROM (Decoster *et al*, 2004; Göeken and Hof, 1993; Esola *et al*, 1996; Halbertsma *et al*, 2001; Li *et al*, 1996). However, this relationship has not been systematically examined in athletes that participate in sporting disciplines that report a high prevalence of LBP. Accordingly, the aim of this study was to investigate the relationship between lumbar mobility and hamstring flexibility in paddlers with and without LBP.

In preparation for the experimental section of the thesis, a comprehensive review of the literature on the epidemiology of LBP in sports, paddling action, the biomechanics of the lumbar spine, the lumbar ROM, hamstring flexibility and the factors contributing to the development of LBP in paddlers will be discussed. This will be followed by a description of the study including methods, results, and discussion designed to investigate the relationship outlined above. The summary and conclusion will complete the thesis.

2. LITERATURE REVIEW

2.1 Epidemiology of low back pain in sports

Low back pain is a common and disabling musculoskeletal symptom that affects both the general population and athletes (Bahr *et al,* 2004; Brukner and Khan, 2001; Frymoyer *et al,* 1983; Twomey and Taylor, 1994). Sixty percent to 90% of people will suffer from LBP at some time in their lives (Brukner and Khan, 2001; Nadler *et al,* 1998; Trainor and Wiesel, 2002; Twomey and Taylor, 1994). A combination of prolonged bending and twisting activities may often contribute to the development of work-related LBP (Andersson, 1981; Heneweer *et al,* 2009). In a prospective cohort study conducted over a three year period, Hoogendoorn *et al* (2000) established that flexion and rotation, as well as lifting were moderate risk factors for LBP. Frymoyer *et al* (1983) also observed that office workers who complained of mild LBP tended to participate in more recreational sporting activities. It may be theorised that the same stresses that cause pain in the workplace can be related to the stresses experienced during endurance sports such as paddling. Sports which require extensive rotational motions are commonly associated with LBP (Trainor and Wiesel, 2002).

Various sporting activities may, due to the repetitive high loading of the spine, place the athletic population at an increased risk of LBP compared to the non-athletic population (Trainor and Wiesel, 2002). Elite, or high-level, sporting activity is considered a risk factor for LBP and the early development of degenerative disc disease (Barile *et al,* 2007; Heneweer *et al,* 2009; Ong *et al,* 2003). A high incidence of LBP has been reported in Norwegian elite rowers and cross-country skiers compared to age-matched non-athletes, especially during periods of higher training loads or competition (Bahr *et al,* 2004). LBP may account for 5% to 8% of athletic injuries and is a significant cause of morbidity in the athletic population (Marais and Vlok, 2000). Further, Nadler *et al* (1998) found that 9.3% of college athletes in New Jersey, USA required treatment for LBP during a one-year period.

The prevalence of LBP in sports may also vary according to the type of sport. For example, 15% of competitive male rowers and 20% of competitive female rowers have reported LBP respectively (Bono, 2004). In addition, during the 12 months prior to questioning, the prevalence of LBP was reported to be 63% amongst cross-country skiers, 49.8% amongst orienteerers and 55% amongst rowers (Bahr *et al*, 2004). Other reports have mentioned a 29% incidence of LBP in professional golfers, only 9% in runners and 12% in racquet sports (consisting of squash, tennis and badminton) (Trainor and Wiesel, 2002). However, there is a lack of evidence for the incidence of LBP in endurance sports (Bahr *et al,* 2004).

Approximately 80% of back injuries occur during training. Twelve percent are overuse injuries and 29% are a recurrence of a previous injury (Marais and Vlok, 2000). In addition, 40% to 60% of back pain recurs (Trainor and Wiesel, 2002). A previous history of LBP is a common risk factor for LBP (Greene *et al*, 2001). Predisposing factors that may also contribute to the development of LBP in sport include: poor lumbar biomechanics, inadequate conditioning of the lumbar spine, muscular inflexibility, leg length discrepancy, sacroiliac motion, sacroiliac dysfunction, lower extremity overuse injury and preseason training (Marais and Vlok, 2000; Nadler *et al,* 1998; Trainor and Wiesel, 2002). Further, Nadler *et al* (1998) was unable to determine an association between LBP and hip flexor tightness or leg length discrepancy in college athletes. However, there was a relationship between LBP and ligamentous and overuse injury in the lower limb. It was therefore proposed that there may be a kinetic link between the lower limb and the low back, where abnormal distal forces are transmitted proximally to the low back. These factors may provide insight into the development of LBP in paddlers, as well as the relationship between LBP and hamstring muscle flexibility in paddlers.

2.2 *Epidemiology of low back pain in paddling*

The spinal region was reported as a common site of injury in paddlers and has been shown to account for just fewer than 15% of injuries in kayaking. Injuries to the back/chest/hip area appeared to have the longest effect on paddling. Females, reported more chronic back/chest/hip injuries than males. However, competitive male white-water paddlers reported more acute back injuries than female white-water paddlers. The majority (72%) of the respondents to the study were male, and there was also a general increase in freestyle competition involving rapid rotation movements which may lead to an increase in acute injuries (Krupnick *et al*, 1998; Schoen and Stano, 2000). A 31% incidence of low back strain was observed in white water kayakers (Fiore and Houston, 2001).

Kameyama *et al* (1999) conducted an epidemiological questionnaire-based survey of 417 paddlers from the Japan Canoe Association that included Canadian canoe, slalom and kayak styles of paddling. It was established that 22.5% of the paddlers experienced LBP, limited back movement or numbness in the back. The majority of paddlers (n = 324) used a kayak style of paddling. 22.8% of kayak style paddlers had experienced low back injury. In addition, a medical examination of elite, competitive paddlers (n = 63) found that 52.3% (n = 33) reported low back problems, including spondylosis, Schmorl's nodes, myofascial pain syndrome and disc herniation. The elite paddlers underwent a radiological examination, which showed that 17.5% had spondylolysis and 85.7% had ballooning discs, primarily of the third, fourth and fifth lumbar discs.

It has therefore been established that LBP is a common occurrence in sports and may develop particularly in activities that require repetitive lumbar flexion, lateral flexion and rotation (Bahr *et al,* 2004; Hoogendoorn *et al,* 2000; Trainor and Wiesel, 2002). The next sections of the literature review will describe normal lumbar biomechanics and lumbar and pelvic motion in an attempt to discern the normal biomechanics of the functional movements required for sporting activities. The anatomy of the hamstring muscle group and the factors affecting hamstring muscle flexibility will be discussed. The current evidence for the relationship between hamstring flexibility and lumbar ROM will be reviewed. These factors may provide insight into the development of LBP in paddlers, as well as the relationship between LBP and hamstring muscle flexibility in paddlers.

2.3 *Factors contributing to the development of low back pain in paddlers*

2.3.1 Biomechanics of the lumbar spine

There is a paucity of evidence regarding lumbar spine biomechanics, as well as muscle activity and function of the trunk and lower limb during paddling. A brief review of the biomechanics of lumbar spine with particular reference to the movements of flexion, side flexion and rotation as performed in the paddling stroke is provided below to facilitate an understanding of the effects of repetitive loading associated with paddling.

A vertebral motion segment is central to the function of the lumbar spine (Lundon and Bolton, 2001). It is defined by two adjacent vertebrae separated by an intervertebral disc, and includes the surrounding ligaments (Adams *et al,* 2006; Hamill and Knutzen, 1995; Lundon and Bolton, 2001). The primary function of the intervertebral disc is the transmission and absorption of forces. The zygapophyseal joint maintains stability and mobility around a motion segment, and protects the disc, nerves and ligaments from abnormal loads (Twomey and Taylor, 1994). The main movements that occur at the lumbar spine are flexion and extension, whereas lateral flexion and rotation movements are limited (Greenman, 2003). Lateral flexion and rotation of the lumbar spine usually occur together as a coupled motion (Greenman, 2003; Lee, 2004).

During lumbar flexion, the coronal axis moves forward and there is a small degree of anterior translation coupled with anterior sagittal rotation of each lumbar vertebrae (Hamill and Knutzen, 1995; Lee, 2004; Norris, 1995). During sagittal rotation, the facets move apart and allow anterior translation, which involves the inferior articular process of the superior vertebrae gliding forward over the superior articular process of the inferior vertebrae (Lee, 2004). Flexion range of motion (ROM) is limited by apposition of the zygapophyseal joint surfaces, the tension developed in the joint capsule, and the posterior spinal ligaments (Norris, 1995).

Lumbar flexion increases the pressure on the anterior aspect of the intervertebral disc, which compresses the anterior annulus and stretches the posterior annulus, increasing the tension in the annulus fibrosis. The nucleus pulposus is forced posteriorly, which may result in bulging of the nucleus pulposus (Hamill and Knutzen, 1995; Jensen, 1980; Lundon and Bolton, 2001; Norris, 1995).

During lumbar flexion, the total intervertebral disc pressure remains unchanged, however there is an increased soft tissue tension (Norris, 1995). Lumbar flexion also increases tensile load on the erector spinae musculature, the zygapophyseal joint capsule, and the posterior spinal ligaments (Esola *et al*, 1996). Twomey and Taylor (1983) found that apposition of the zygapophyseal joint surfaces was the greatest non-contractile restraint of lumbar flexion ROM.

Lateral flexion of the lumbar spine is generally coupled with some degree of rotation towards the opposite side (except for L5/S1 where there is no associated rotation) (Lee, 2004; Norris, 1995; Sahrmann, 2002). The superior vertebrae tilts towards the side of lateral flexion, which compresses the fibres of the annulus on the side of the concavity, and increases tension on the opposite fibres (Hamill and Knutzen, 1995; Norris, 1995).

Only 2° to 3° of pure lumbar rotation is possible, which places great stress on the annulus and may lead to microtrauma (Lee, 2004; Norris, 1995; Sahrmann, 2002). Lumbar rotation ROM is limited by the orientation of the zygapophyseal joints and the outer layers of the annulus fibrosis (Lundon and Bolton, 2001; Sahrmann, 2002). As rotation ROM increases, the contralateral zygapophyseal joint is compressed and the ipsilateral joint will be tensioned (Lee, 2004; Norris, 1995). Increased tension also develops in the supraspinous ligaments and interspinous ligaments as the spinous processes move apart (Norris, 1995).

If spinal rotation continues beyond 3°, the impacted zygapophyseal joint becomes the new axis of rotation and the upper vertebral body will pivot posterolaterally around this new axis. Therefore, further rotation becomes a coupled motion around an oblique axis, which may increase stress on the zygapophyseal joints and intervertebral discs (Lee, 2004; Norris, 1995). This may have implications for paddlers, as it is theorised that rotation ROM is increased in sitting with the trunk slightly flexed and the supporting soft tissue structures are relaxed (Sahrmann, 2002). However, although functional ROM may be increased, the implications for normal lumbar biomechanics and loading of structures are unclear. These factors may contribute to the development of low back pain in paddler.

2.3.2 Lumbar range of motion

There is a lack of scientific evidence regarding the ROM of the lumbar spine required for participation in paddling. There is also limited biomechanical analysis of the ROM utilised in the boat while seated with the legs extended in front. However, this study will not be assessing the ROM necessary for paddling, but rather assessing the relationship of lumbar spine ROM to hamstring flexibility in paddlers.

In healthy individuals, the lumbar spine accounts for 63% of gross flexion ROM and pelvic motion accounts for 37% of the flexion ROM up to 90°. A mean lumbar flexion ROM of 55° ± 9.2° and a mean extension ROM of 27° ± 12.8° was measured in 13 healthy male and female participants between the ages of 19 and 51 years (Mayer *et al*, 1984). Chiarello and Savidge (1993) assessed lumbar ROM in pain-free male (n = 4) and female (n = 8) participants between the ages of 23 and 35 years. Lumbar flexion ROM was measured as 59° ± 4.8° and lumbar extension ROM was measured as 32° ± 9.6° using a two-inclinometer technique.

i. Factors affecting lumbar range of motion

Due to the paucity of evidence regarding lumbar ROM and biomechanics in paddlers, the literature relating to LBP in the general population will be discussed. It has been suggested that LBP may influence lumbar ROM (Wong and Lee, 2004). Mayer *et al* (1984) established that gross flexion ROM was reduced in participants with LBP associated with chronic spine dysfunction, compared to participants without LBP.

Esola *et al* (1996) observed that participants with a history of LBP tended to expend their available lumbar ROM earlier, during forward flexion, before utilising the available motion of the pelvis compared to participants without a history of LBP. However, the LBP group did not have less lumbar ROM. This early lumbar motion may place additional tensile stress on the posterior elements of the lumbar spine and may contribute to a high recurrence of LBP.

The underlying mechanisms for the reduction in ROM associated with LBP are unclear and may be attributed to articular or myofascial structures (Halbertsma *et al*, 2001; Lee, 2004). Excessive lumbar mobility may also increase the tensile loading of the lumbar spine and may be associated with tissue overload, microtrauma and the development of degenerative joint and disc disease (Esola *et al*, 1996). Other factors associated with influencing lumbar ROM include pathological condition, age related spinal changes, gender, time of day and functional activities. It is acknowledged that there may be other factors which exist that could influence lumbar ROM, but research to establish these other factors is limited and is beyond the scope of this literature review.

Moll and Wright (1971) observed age-related changes in spinal ROM in pain-free participants. Twomey (1979) also observed a progressive decrease in cadaver lumbar ROM with increasing age. In addition, Fitzgerald *et al* (1983) determined a similar age-related reduction in spinal mobility and suggested that the reduction in ROM, particularly extension and lateral flexion ROM, may occur in 20-year intervals. Further, it has been proposed that there may be an age-related alteration in the instantaneous axis of rotation for spinal motion, which may lead to excessive posteroanterior and lateral translation during physiological movement and subsequent degenerative changes (Hamill and Knutzen, 1995; Lee, 2004).

Moll and Wright (1971) also observed that male participants had increased spinal flexion and extension ROM compared to female participants. In contrast, Twomey (1979) demonstrated that female participants had increased sagittal plane ROM compared to male participants. These differences were only evident in participants up to 35 years of age; thereafter, spinal ROM was similar in male and female participants. However, Esola *et al* (1996) found that the characteristics of forward flexion motion between men and women were similar.

Further, diurnal variations in lumbar flexion ROM have been observed in the early morning and late afternoon. Adams *et al* (1987) assessed a group of healthy men and women and found that participants could flex further from the lumbar spine in the late afternoon compared to early morning. This may be associated with the fact that lumbar discs imbibe fluid from the surrounding tissues during recumbency, causing changes in disc hydration and height (Porter and Trailescu, 1990; Reid and Mcnair, 2000).

In addition, functional movements of the trunk required during different sporting activities require a complex interaction between the lumbar spine, pelvis, and hip joints. Co-ordination of trunk motion around the pelvis and pelvic motion around the hips during lumbar flexion and extension is referred to as the lumbopelvic rhythm and this may vary under conditions such as LBP (McClure *et al,* 1997; McGorry *et al*, 2001).

2.3.3 Functional spinal movements

In many sports the lumbar spine is required to perform various athletic manoeuvres which place unique demands on the intervertebral discs (Bono, 2004). This occurs in paddling where the lumbar spine is 'exposed to repetitive heavy shear loads' (Kameyama *et al,* 1999). Frequent forward flexion of the lumbar spine has been identified as a risk factor for the development of LBP (Esola *et al*, 1996). Many activities of daily living also require a combination of lumbar and hip motion to flex the trunk (Esola *et al*, 1996; Li *et al*, 1996). Excessive loading of lumbar tissues may occur if there is altered co-ordination of movement between the lumbar spine and the hip joints, which may lead to LBP (Esola *et al*, 1996; Norris and Matthews, 2006). In addition, an extension torque combined with trunk rotation, for example, when performing an action to the side of the body in sitting, may be a further risk factor for the development of LBP (Van Dieën, 1996).

The torsional activities in golf and rowing are associated with large compressive forces across the lumbar vertebra, particularly at the levels of L3 and L4 (Bono, 2004). In the paddle stroke, the main trunk motion is a combination of flexion, rotation and lateral flexion, where asymmetrical forces will act through the spine (Kameyama *et al*, 1999).

In paddling the point of contact with the water is lateral and anterior to the base of support. The trunk pivots around the lumbar spine which acts as a fulcrum to generate forward propulsion. A full description of the paddle stroke is presented in Appendix I. Intervertebral disc injury and pain may be associated with torsional stress or compressive forces on the annulus fibrosis. Excessive or repetitive torsional stress may lead to damage of both the zygapophyseal joint and the annulus fibrosis. Tearing of the annulus fibrosis may lead to an inflammatory reaction that stimulates a chemical irritation of the nociceptors in the outer third of the posterior annulus fibrosis, resulting in the development of LBP (Bono, 2004; Brukner and Kahn, 2001).

In addition, the erector spinae muscles appear to function asymmetrically during combined extension and rotation, with an increase in muscle activity on the contra-lateral side to the direction of rotation as the torque and the angle of rotation increases. This asymmetrical force leads to a gradient of stress in the vertebrae, with the stress concentrated on the contra-lateral side to the direction of rotation. If the intervertebral disc does not distribute the stress evenly, the concentration of stress will accumulate at the lower vertebrae. This stress concentration may be further increased by fibre strain that results from intervertebral rotation during rotated postures (Van Dieën, 1996). Further, the hamstrings muscles attach to the pelvis, and may therefore influence lumbar ROM and function. The contribution of hamstring flexibility to lumbopelvic motion, and to the development of LBP will be discussed in the following section.

2.3.4 Hamstring flexibility

i. Anatomy of the hamstring muscle group

The hamstrings are a biarthrodial muscle group (Gajdosik, 2001; Hertling and Kessler, 1996) and consist of a medial and lateral component with fascial connections to the pelvis (Lee, 2004). The medial component consists of the semimembranosus and the semitendinosus muscles and the lateral component consists of the biceps femoris muscle, which has a long head and a short head (Myers, 2001). Semitendinosus and the biceps femoris long head share a common tendon which arises from the ischial tuberosity and the distal part of the sacrotuberous ligament. The biceps femoris fibres tend to attach more laterally on the ischial tuberosity (Romanes, 1986; Vleeming *et al,* 1996).

Semimembranosus also arises from the ischial tuberosity distal to the common attachment of the semitendinous and biceps femoris long head. The biceps femoris short head arises from the linea aspera and the supracondylar line of the femur (Kendall *et al,* 2005; Romanes, 1986). The biceps femoris long head and short muscles form a common tendon and pass laterally over the posterior surface of the knee joint to insert into the head of the fibula. The semimembranosus muscle passes medially across the posterior knee to insert into the groove on the posteromedial surface of the medial condyle of the knee. The semitendinosus muscle passes across the medial side of the knee to insert into the upper part of the medial surface of the medial condyle of the tibia. This insertion will be posterior to the tendons of the gracilis and sartorius muscles (Romanes, 1986).

The hamstrings have a combined action of hip extension and knee flexion (Kolber and Zepeda, 2004; Romanes, 1986). Since the hamstring muscle group also attaches below the knee, it will also function to flex the knee with some rotation at this joint (Hamill and Knutzen, 1995). If the pelvis is moved into a position of anterior rotation, the distance between the origin and insertion of the hamstrings may be increased, resulting in tension of the muscles (Kolber and Zepeda, 2004). In the case of unilateral action of the hamstring muscle group, there may be potential for posterior torsion of one ilium (Hertling and Kessler, 1996). The hamstrings also assist in maintaining an upright posture by pulling down on the ischial tuberosity to create a posterior pelvic tilt (Hamill and Knutzen, 1995).

ii. Factors influencing hamstring flexibility

Numerous factors may result in a reduction in flexibility of the hamstring muscle group, including joint capsule or other soft tissue restrictions (Decoster *et al*, 2004; Frontera *et al*, 2006). Flexibility may also be affected by gender, age, temperature, reflex activity, central nervous system disease processes and the strength of the antagonists. The influence of gender and age on hamstring flexibility will be discussed in more detail; however, further discussion of other factors that may influence hamstring flexibility is beyond the scope of this review.

Gender:

Krivickas and Fienberg (1996) assessed muscle tightness in college athletes with a mean age of 19.8 ± 1.5 years in males and 19.6 ± 1.2 years in females. The iliotibial band, iliopsoas, rectus femoris, hamstrings and gastrocsoleus muscles were measured using ‘standard physical examination techniques’. A modified Obers test, Thomas test, quadriceps-inhibited knee flexion angle, popliteal angle, and ankle dorsiflexion ROM was used to test the flexibility of these muscles respectively. There was a reduction in flexibility of the iliopsoas, hamstring and gastrocsoleus muscles in males compared to females. The reduction in flexibility was also associated with an increased injury risk in males. Keeley *et al* (1996) noted a difference in hamstring flexibility and pelvic flexion ROM between males and females, where females demonstrated a greater straight leg raise (SLR) measurement than men. Further, Biering-Sørensen (1984) also showed that females had ‘more elastic’ hamstrings than males.

Age:

There is an age-related decline in muscle strength and power that may be associated with a loss of functional motor units and a decrease in the cross-sectional area (CSA) of the individual fibres, or a loss in the number of muscle fibres (Buckwalter *et al,* 1993; Faulkner *et al,* 2007; Feland *et al,* 2001; Gajdosik, 2001; Hopp, 1993; Nair, 2005. It has been observed that age-related deletions and mutations of mitochondrial DNA occur, which may be as a result of constant exposure to free oxygen radicals after a long period of time (Balagopal *et al,* 2001; Nair, 2005. The CSA of the fast type 2 fibres tend to decrease with age, but the slow type 1 fibres tend to maintain their CSA (Balagopal *et al,* 2001; Faulkner *et al,* 2007; Nair, 2005. These changes may also contribute to an age-related reduction in muscle length (Buckwalter *et al,* 1993; Feland *et al,* 2001; Gajdosik, 2001).

Both decreased CSA and fibre number may contribute to a decrease in muscle mass with increasing age. A 30% to 50% decrease in muscle mass is generally noticed between 40 and 80 years of age (Faulkner *et al,* 2007). With aging there tends to be an increase in the fat and connective tissue content of muscle tissue, especially in the more sedentary individual (Gajdosik, 2001; Hopp, 1993). There may be an associated decrease in the strength of the soft tissue matrices and a reduction in mesenchymal stem cells with aging (Buckwalter *et al,* 1993; Feland *et al,* 2001).

iii. The relationship between the hamstring muscle group and lumbopelvic motion

'The mechanics of the low back are inseparable from that of the pelvis and lower extremities' (Kendall *et al,* 2005). Therefore, muscle imbalances in one part of the body may lead to changes in other parts of the body. The lumbar-pelvic-hip complex includes the L4 and L5 vertebrae and associated discs, the sacrum and the coccyx, the two innominates and the two femora (Greenman, 2003; Hertling and Kessler, 1996; Lee, 2004). The lumbopelvic region is of importance for static weight bearing, normal biomechanics and posture. 'The forward flexion of the trunk involves a combined motion of lumbar flexion and pelvic rotation' (Esola *et al*, 1996; Sihvonen, 1997).

During lumbopelvic flexion there is increased tension in the iliolumbar, interosseous and sacrotuberous ligaments (Hertling and Kessler, 1996). Motion which occurs at the sacrum is associated with the spine, while the motion of the innominates is associated with the hip (Hertling and Kessler, 1996). Stability across the sacroiliac joint (SIJ) is necessary for the effective transfer of load from the trunk to the legs. It has been shown that there is co-activation of the erector spinae during activation of the latissimus dorsi, gluteus maximus or biceps femoris (Van Wingerden *et al*, 2004). The muscles around the lumbopelvic area contribute to the strength of the ligaments of the SIJ by their fibrous expansions which blend with the ligaments and thereby provide dynamic ligamentous stability. Adaptive shortening or neuromuscular imbalances may be associated with a reduction in muscle length around the pelvis and secondary alterations in pelvic mechanics (Hertling and Kessler, 1996).

Anatomically, there are interconnections between ligaments and fascial structures within the lumbopelvic region (Brolinson *et al,* 2003). The superficial back line (SBL) is strongly involved with mediating posture in the sagittal plane. It assists with limiting flexion or exaggerating extension. This myofascial line runs along the posterior surface of the body, from the scalp fascia along the erector spinae and attaches via the lumbosacral fascia to the sacrum. The SBL continues down the sacrotuberous ligament to the ischial tuberosity where it has fibrous connections to the hamstrings. Further, it continues down the hamstrings to the lower limb. The fascial line has a constant postural function therefore it has thickened fascial sheaths along this line (for example, the hamstrings, sacrotuberous ligament and thoracolumbar fascia) (Lee, 2004; Myers, 2001). Anecdotally, it is theorised that these fascial connections link the trunk and the hamstrings in a sagittal plane.

There are also fascial connections which run in an oblique direction called functional lines. It is theorised that these lines connect the trunk to the lower limb and run from the shoulder to the opposite hip. These fascial connections may facilitate functional activities and may also enhance distal muscle strength (Myers, 2001). Functional lines provide compression across the SIJ in combination with forces in the ligaments and fascia (Lee, 2004; Snijders *et al,* 1995). The back functional line runs from the attachment of latissimus dorsi on the humerus to the thoracolumbar fascia. It crosses the midline of the body via fibrous connections to the gluteus maximus on the opposite side and runs under the iliotibial band to attach onto the linea alba of the femur (Lee, 2004; Myers, 2001; Snijders *et al,* 1995).

There are also fascial connections across the SIJ connecting the thoracolumbar fascia and the hamstrings through the sacrotuberous ligament (Brolinson *et al,* 2003). Anecdotally, this functional line would be important to paddlers in the power phase where there is a powerful use of scapular retraction, trunk rotation and pressure through the opposite lower limb into the foot board. The hamstring muscles provide a link between the boat and the opposite shoulder (Deykin, 2006 as cited in Edge, 2006).

It is difficult in a clinical setting, with manually performed tests, to determine whether a limitation in lumbar flexion ROM is due to an increase in muscle stiffness or a decrease in the extensibility of the hip or back muscles (Göeken and Hof, 1994). Flexibility is the ability to move a single joint or series of joints through an unrestricted, pain-free ROM and this depends on the extensibility of the connective tissues or muscles crossing the involved joints (Frontera *et al,* 2006; Göeken and Hof, 1994; Kolber and Zepeda, 2004). Göeken and Hof (1993) observed poor extensibility of the hamstrings in participants that were unable to bend forward from a standing position and touch the ground with their fingertips while keeping their knees in full extension compared to participants that could touch the ground. Halbertsma *et al* (2001) also established that participants with non-specific LBP, with a limited SLR test and an inability to touch the ground with their fingertips while keeping their knees in full extension had poor extensibility of the hamstring muscle group.

Göeken and Hof (1994) suggested that ‘short hamstrings’ were probably not related to mechanical overloading of the low back, as the moment that can be exerted by ‘short hamstrings’ is lower than in hamstrings with good flexibility. In contrast, Esola *et al* (1996) demonstrated a tendency for hamstring flexibility to be reduced in participants with LBP compared to healthy, pain-free participants. Interestingly, there were no differences in total lumbar spine ROM and hip flexion ROM between the groups. A larger sample size was recommended for future studies. Furthermore, Halbertsma *et al* (2001) concluded that an inability to bend forward and touch the ground and a reduction in SLR ROM were primarily due to a decreased stretch tolerance of the hamstring muscle group.

Li *et al* (1996) examined the influence of hamstring muscle stretching on lumbar and hip ROM during forward bending in pain-free participants with a SLR of less than 70°. Participants were either allocated to a stretching group, which underwent a 3-week stretching programme, or a control group. Post-intervention increases in hamstring length and hip ROM during the later phase of forward flexion were observed in the stretching group compared to the control group. However, there were no differences in lumbar ROM between the groups (Li *et al,* 1996).

Anecdotally, in the clinical setting, a reduction in hamstring flexibility is often thought to be associated with a posterior pelvic tilt due to the attachment of the hamstrings to the ischial tuberosity. However, Norris and Matthews (2006) observed that the ischial tuberosity is positioned slightly posterior to the femoral head and the line of pull of the hamstrings is almost vertical. It may also be theorised that any posterior rotational force due to decreased hamstring flexibility would potentially be outweighed by the anterior force of the hip flexors. A reduction in hamstring flexibility may therefore not influence the total range of pelvic tilt. Further studies are required to investigate the relationship between pelvic movement and hamstring flexibility.

iv. The relationship between hamstring flexibility and the development of low back pain

Poor hamstring flexibility has often been associated with injuries to the low back and lower extremities. It is also postulated that increasing muscle flexibility could prevent muscular strain and overuse injuries in sport, but evidence to support this theory is limited (Decoster *et al*, 2004; Krivickas and Fienberg, 1996). During paddling, there is often increased resting tension in the hamstring muscle group (Deykin, 2006 as cited in Edge, 2006) (Appendix I). It is theorised that postural or movement faults of the hip and pelvis may be associated with a reduction in hamstring flexibility and the development of LBP (Halbertsma *et al*, 2001; Li *et al*, 1996). Hip dysfunction may be related to LBP due to the close proximity of the hip to the lumbosacral region and the combined movements of the lumbar-pelvic-hip complex (Harris-Hayes *et al,* 2009). It has been suggested that lumbar disc pain may be associated with alterations in lumbar and lower limb ROM and that decreased flexibility of extra-spinal muscles should be addressed in a treatment programme (Bono, 2004).

Lumbar spine disorders and chronic LBP are frequently associated with hamstring muscle tightness (Biering-Sørensen, 1984; Tafazzoli and Lamontagne, 1996). It has been proposed that a restriction in hip ROM may be associated with a compensatory increase in lumbar ROM (Li *et al*, 1996). The excessive lumbar ROM may lead to increased tensile forces on the spine, thereby resulting in the development of LBP (Esola *et al*, 1996; Li *et al*, 1996). Hip and lumbar mobility may also be limited by hamstring spasm in cases of mild L4/L5 and L5/S1 disc problems, spondylolysis or facet joint arthrosis (Muckle, 1982).

Previous studies have demonstrated that participants with a prior history of LBP presented with a tendency toward decreased hamstring muscle flexibility (Biering-Sørensen, 1984; Esola *et al,* 1996; Li et al, 1996). Biering-Sørensen (1984) assessed various physical measurements to identify risk indicators for LBP and identified that females with decreased hamstring flexibility were more likely to experience LBP. It may be proposed that a decrease in hamstring flexibility could be a predisposing factor for LBP. However, it has not yet been determined if LBP may result in a reduction in hamstring flexibility. The cause and effect of this potential relationship is therefore unclear (Esola *et al,* 1996). It is possible that muscle activation patterns of the lumbar spine and hamstring muscles may contribute to this relationship.

2.3.5 Muscle activation patterns

During forward flexion of the spine, with the knees in extension, the eccentric activity of erector spinae initially increases until it reaches a certain critical point, where after it rapidly decreases as full lumbar flexion is approached (McGorry *et al*, 2001; Neblett *et al*, 2003; Sihvonen, 1997). As full spinal flexion is approached, the erector spinae muscles become inactive and lumbar flexion is counteracted by the passive tension of the muscles, as well as the posterior spinal ligaments (Esola *et al*, 1996). This pattern of muscle activity during trunk flexion is known as the flexion-relaxation phenomenon (McGorry *et al*, 2001; Neblett *et al*, 2003; Sihvonen, 1997). The flexion-relaxation phenomenon of the lumbar muscles has been shown to be a consistent and predictable pattern in participants without LBP (Neblett *et al*, 2003). Erector spinae muscle activity is greater during the return to neutral from lumbar flexion, compared to lumbar flexion (Sihvonen, 1997). The flexion-relaxation phenomenon may be associated with a stretch inhibition reflex, which allows the erector spinae to relax while the passive elements provide an extension moment (McGorry *et al*, 2001).

Forward flexion of the trunk is a combination of lumbar flexion and pelvic rotation (lumbar-pelvic rhythm) and requires careful co-ordination of activity of the back muscles and hamstrings (Sihvonen, 1997). The posterior hip muscles are also active during trunk flexion to control the flexion motion of the pelvis on the hips (Esola *et al*, 1996). Electromyographic (EMG) studies have demonstrated reductions in hamstring muscle activity with progressive lumbar flexion ROM to a point where hamstring muscle activity ceases before full lumbar flexion is reached. Although the hamstrings become inactive during forward flexion, they remain active for a longer period of time than the erector spinae muscles (McGorry *et al,* 2001; Sihvonen, 1997). Therefore, the last part of lumbar flexion occurs without back muscle activity and the last part of pelvic rotation occurs without hamstring bracing (Sihvonen, 1997).

Flexion-relaxation of the erector spinae may be disrupted in participants with LBP, with elevated activity during full voluntary trunk flexion and a lack of relaxation in the flexed position (McGorry *et al*, 2001; Neblett *et al*, 2003; Sihvonen, 1997). Sihvonen (1997) observed that the flexion-relaxation phenomenon was present only in the hamstrings, but not in the erector spinae, and that lumbar flexion ROM was decreased in a patient with ankylosing spondylitis. It may be suggested that there may not be a direct relationship between flexion-relaxation of the erector spinae and the hamstring muscle group. A lack of flexion-relaxation may therefore alter lumbar spine ROM or hamstring muscle tightness. However, further studies are required to fully understand muscle activation during functional and sporting activities. In addition, due to the endurance nature of paddling, the influence of muscle fatigue in the development of LBP in paddlers will be discussed.

2.3.6 Muscle fatigue

Paddling uses sustained repetitive motion while maintaining an upright posture for long periods (Kameyama *et al,* 1999), which may lead to fatigue of the lumbar spine musculature. Fatigue of the low back musculature may be a contributing factor to the development of LBP. Fatigue may be associated with the recruitment of antagonistic muscle groups and muscle insufficiency, which may shift spinal loading to passive tissues, thereby increasing the risk of injury. A previous study demonstrated significant increases in internal oblique and latissimus dorsi muscle activity as the erector spinae became fatigued during repetitive isometric trunk extension (Sparto and Parnianpour, 1998).

Other studies have identified a high association between poor endurance of the back extensor muscles and LBP (Biering-Sørensen, 1984; Nourbakhsh and Arab, 2002). In addition, the ligaments and muscles in the trunk provide neurological feedback that mediates vertebral segment joint position sensibility and muscular reflex stabilisation. Mechanical damage or injury may occur when the control system is unable to adapt to a change in loading (Taimela *et al*, 1999).

Hodges and Richardson (1996) observed that participants with LBP had insufficient stabilisation of the trunk during arm and leg movements due to delayed activation of the transverses abdominus muscle. Taimela *et al* (1999) demonstrated that fatigue of the lumbar paraspinal muscles impaired the ability to sense a change in lumbar position in participants with and without LBP. However, there was a greater reduction in proprioception in the LBP group compared to the control group. It was therefore theorised that there may be a period immediately after a fatiguing task during which proprioception may be affected, thereby increasing the risk of injury to the lumbar spine, particularly in individuals with LBP (Taimela *et al*, 1999). Fatigued lumbar muscles also have slower reaction times for the deceleration of the lumbar spine during flexion tasks. There may therefore be abnormally high ranges of lumbar motion during repetitive tasks or activities due to dampened reflexive proprioceptive inputs (Dolan and Adams, 2001). Further, it is recognised that sustained sitting postures required for paddling may be one of the factors influencing the development of LBP in paddlers.

2.3.7 Adaptations associated with prolonged sitting postures

Postural adaptations may be associated with the development of pain through the constant or repeated application of relatively low loads or sustained loading over a prolonged period of time (Kendall *et al*, 2005; McGill, 1997). Habitual posture may lead to adaptive shortening of muscle and connective tissue, which may result in decreased flexibility and ROM. The muscles which tend to shorten generally perform postural functions (Ekstrand *et al*, 1982). This may be evident in endurance sports such as paddling, where habitual sitting postures are often sustained for prolonged time periods. A paddler is required to maintain an anterior pelvic tilt to retain a slight lumbar lordosis. If this is not maintained and the athlete acquires a flexed lumbar posture, this may increase the activation of the back muscles (Deykin, 2006 as cited in Edge, 2006).

In sitting, the pelvis is the primary base of support with the ischial tuberosities as the main points of support (Harrison *et al,* 1999; Myers, 2001). The normal sitting posture tends to rotate the pelvis posteriorly in comparison to the anatomical position. This may lead to a reduced lumbar lordosis, with the centre of gravity anterior to the spine. Disc shear forces tend to dissipate to zero in an erect or slightly extended sitting posture (Hedman and Fernie, 1997).

The slightly flexed posture that is often found in unsupported sitting may be associated with a reduction in the ability of the facet joints to transmit compressive loads. This may result in increased pressure on the intervertebral discs (Adams and Hutton, 1980; Harrison *et al,* 1999; Sahrmann, 2002) and an increased contribution of posterior passive tissues to maintain the sitting posture (Beach *et* al, 2005). The increased contribution of the passive tissues over a sustained period of time may result in accumulated trauma and therefore increase the chance of injury to these tissues due to failure (McGill, 1997). The greatest lumbar disc pressure has been observed in sitting while leaning forward 20° (Kayis and Hoang, 1999; Nachemson, 1966 as cited in Jensen*,* 1980). The intervertebral disc pressure in unsupported sitting may be up to one third greater than in standing (Adams and Hutton, 1980; Nachemson, 1966 as cited in Jensen*,* 1980). A lordotic lumbar curve should be maintained during sitting to minimise intervertebral disc pressure (Harrison *et al,* 1999).

The further forward the centre of gravity moves in relation to the lumbar vertebrae, the greater the load on the lumbar intervertebral discs and muscles. During unsupported sitting with a flexed posture, there may also be increased activity of the psoas muscle (and other muscles attaching directly to the spine) to stabilise the lumbar spine and pelvis, which may contribute to increased loading and shear forces on the intervertebral disc (Andersson and Ortengren, 1974 as cited in Jensen, 1980; Hedman and Fernie, 1997; Sahrmann, 2002). Unsupported sitting may also be associated with an increased risk of sclerosis of the facet joints (Fujiwara *et al,* 2000). If flexed lumbar postures are sustained for prolonged periods during sitting, viscoelastic creep deformation may occur in the posterior ligaments and the posterior fibres of the intervertebral disc. This may result in increased stress to the outer annulus fibrosis and the collapse of the inner annulus fibrosis into the nucleus pulposus, leading to micro-failure of the intervertebral disc (Beach *et al,* 2005; Harrison *et al,* 1999; McGill, 1997).

In addition, Beach *et al* (2005) established that sustained sitting was associated with increased intervertebral joint stiffness and a reduction in lumbar flexion ROM. It was theorised that these changes may be related to increased intervertebral disc height, secondary to disc swelling. Further, it was proposed that these changes may result in an increased risk of LBP when combined with full lumbar flexion tasks that are performed after an hour of more of sustained seated postures (Beach *et al,* 2005).

It is hypothesised that an anterior pelvic tilt is an important component of sitting posture during the paddle stroke as it facilitates the maintenance of a lumbar lordosis, which decreases loading of the intervertebral discs and muscles (Deykin, 2006 as cited in Edge, 2006; Harrison *et al,* 1999). It is recognised that endurance paddlers may be subjected to prolonged periods of unsupported sitting, which may increase the risk of overloading the lumbar spine and the development of LBP. However, further studies are required to investigate the relationship between the seated paddling position and LBP in endurance paddlers. The next section will briefly review pathological conditions that may lead to the development of LBP.

2.3.8 Pathological conditions of the lumbar spine

It is difficult to identify the exact structures involved in causing LBP as any structure which receives nervous innervation in the lumbar spine has the potential to cause pain. These include, but are not limited to, the intervertebral disc, the zygapophyseal joints, the interspinous ligaments, and the lumbar musculature (Adams *et al*, 2006).

i. Disc herniation

The nucleus pulposus may be expelled though the annulus fibrosis into the vertebral canal where it may compress the nerve roots. In some cases, the nuclear material does not penetrate the annulus, but instead bulges and stretches the annulus (Bogduk and Twomey, 1991). Nerve root irritation or compression caused by intervertebral disc protrusion is considered to be a common dysfunction which may be a cause of LBP (Van Den Hoogen *et al*, 1996).

Disc herniation may also occur secondary to the aging process or mechanical trauma. End-plate fractures may occur as a result of compressive loads, as the vertebral end-plate is weaker than the annulus. This will allow nuclear material to be exposed to the circulation which may result in an inflammatory response. Disc herniation or degradation differs from disc degeneration in that it tends to occur in focal areas that have sustained injury, such as repeated torsional injury to the annulus fibrosis causing radial fissures (Bogduk and Twomey, 1991).

ii. Spondylolysis

Spondylolysis has been reported in 17.5% of elite paddlers in Japan (Kameyama *et al,* 1999). Spondylolysis is a defect in the bone of the pars interarticularis that commonly occurs at the L4 and L5 vertebrae and may be due to the repetitive loading of the vertebral segments causing a stress fracture of the neural arch (Adams *et al,* 2006; Bono, 2004). The pars interarticularis is particularly vulnerable in situations of alternating flexion and extension motions due to the pivoting of the inferior articular process about the pars. This condition is common in athletes due to repetitive flexion or extension motions and, occasionally, repetitive rotation motions (Adams *et al,* 2006). The sports with the highest prevalence of spondylolysis are diving, wrestling, weightlifting, sports that involve throwing, gymnastics and rowing (Bono, 2004).

iii. Spondylolisthesis

Spondylolisthesis may occur after spondylolysis due to the loss of resistance to shear forces by the motion segment, which is usually provided by the zygapophyseal joint. There is a forward slip of the superior vertebra on the inferior vertebra, usually at the L4 or L5 vertebrae (Adams *et al,* 2006).

iv. Zygapophyseal joint dysfunction

The zygapophyseal joint is a non-osseous cause of LBP. The pain from this joint may be due to subchondral fractures, capsular tears, capsular avulsion and haemorrhage into the joint space. Generally, zygapophyseal injuries are associated with unilateral pain and the pain is commonly reproduced on lumbar extension (Brukner and Kahn, 2001). Studies have not been able to identify clinical tests that could be used to isolate pain coming from the zygapophyseal joints and forced extension may result in damage of the joint and joint capsule (Adams *et al,* 2006).

Acute locked back is a sudden onset of pain and limited movement which occurs when returning to neutral from a flexed position. It is often associated with lifting. The underlying mechanisms are unclear, with possible mechanisms including meniscal entrapment in the subcapsular recess of the zygapophyseal joint and the shearing of fragments of articular cartilage. A further theory suggests that satellites of nuclear material escape through radial fissures in the lumbar disc during forward flexion and are then compressed on returning from lumbar flexion by the outer annulus fibrosis causing pain (Bogduk and Twomey, 1991).

v. Soft tissue dysfunction

Lumbar joint sprains may occur when one or more spinal ligaments are disrupted, but the prevalence of this injury has not been reported (Bono, 2004). LBP may develop over time from microtrauma and the failure of tissues in the lumbopelvic region to resist and adapt to repeated stress (McGill, 1997). Muscular strains are one of the most common causes of LBP in college athletes (Bono, 2004). Erector spinae dysfunction may contribute to the development of LBP. Erector spinae muscle activity is one of the main determinants of the forces acting on the spine. These muscles are required to produce relatively high muscle contractions (because of the small lever arm) to counterbalance the predominantly flexing gravitational forces acting on the trunk. This results in high compression and shear forces on the spine, which may lead to damage and pain (Briggs *et al,* 2004; Van Dieën, 1996). The large contraction forces needed to resist the flexion moment may also result in muscle strains (Briggs *et al,* 2004). Without muscle, the human spine is unable to withstand the physiological loads placed on it and it buckles easily under relatively low loads of approximately 20 N (Panjabi *et al*, 1988; Van Dieën, 1996).

vi. Degeneration of the lumbar spine

Degenerative changes of the lumbar spine occur mainly in the facet joints and the intervertebral discs. In elite Japanese paddlers, 24.5% were found to have osteophyte formation, ballooning discs and joint space narrowing (Kameyama *et al,* 1999). The facet joints and intervertebral discs are important structures for maintaining the stability of the motion segment and disc degeneration has been found to greatly affect the motion segment (Fujiwara *et al*, 2000). Mechanical loading affects degenerative changes in the lumbar spine. It is proposed that the prolonged loading of tissues in certain postures may contribute to spinal degenerative changes due to excessive load transmission and tissue deformation (Hedman and Fernie, 1997).

Disc degeneration is largely associated with aging and trauma. In disc degeneration, there is a decrease in hydraulic or elastic properties due to decreased water content, decreased elastic collagen tissue (which is replaced by non-elastic fibrous tissue) and cartilage degeneration of the end-plates (Jensen, 1980; Lundon and Bolton, 2001). The intervertebral disc becomes more fibrous with increasing age and the nucleus pulposus becomes more like a solid dry mass (Lundon and Bolton, 2001). Fujiwara *et al (*2000) found that both disc degeneration and, to a lesser extent, cartilage degeneration lead to an increase in axial rotation, flexion and extension.

Degenerative changes due to creep loading may also lead to increased lumbar flexion and lateral bending ROM, but does not affect extension ROM. This is particularly evident in cases of vertebral end-plate fracture and disc injury. These injuries, together with the associated instability may lead to pain (Adams *et al,* 2006). Subchondral sclerosis is also observed in the degeneration of motion segments, and occurs predominantly on the medial part of the superior facet. There may also be associated degenerative changes of the ligamentum flavum and capsular ligaments due to sclerosis of the facet joint, which may limit flexion and axial rotation. Fujiwara *et al (*2000) agreed with the concept of ‘three stages of spinal degeneration, namely dysfunction, instability and stabilisation’.

2.3.9 Summary of factors contributing to the development of low back pain in paddlers

It is evident that endurance paddling requires repetitive motion that may overload the lumbar spine (Kameyama et al, 1999; Mann and Kearney, 1980). Endurance paddling also requires prolonged periods of unsupported sitting, which may increase the risk of the development of LBP. Lumbar flexion and rotation have been identified as risk factors for LBP (Andersson, 1981; Esola *et al*, 1996). Paddling requires repetitive trunk flexion and rotation, and these athletes may therefore have an increased risk of developing LBP (Mann and Kearny, 1980; Shepard, 1987). A reduction in hamstring flexibility may be associated with LBP or altered lumbar ROM (Decoster *et al*, 2004; Göeken and Hof, 1993; Esola *et al*, 1996; Halbertsma *et al*, 2001; Li *et al*, 1996). However, this relationship has not been systematically examined in athletes that participate in sporting disciplines that report a high prevalence of LBP.

2.4 Instrumentation

2.4.1 Measurement of lumbar range of motion

The measurement of lumbar ROM is important in the assessment of LBP, as it is used to determine the extent of impairment associated with LBP and to establish the response to treatment interventions and the restoration of function (Chiarello and Savidge, 1993; Fitzgerald *et al*, 1983; Keeley *et al,* 1986; Shirley *et al*, 1994). However, the measurement of spinal ROM is complex, due to the multiaxial motion of intersegmental articulations and the combined movement of the lumbar-pelvic-hip complex (Keeley *et al,* 1986; Mayer *et al*, 1984).

Radiographic examination is considered the gold standard for the measurement of spinal ROM (Shirley *et al*, 1994). Although the examination is accurate, repeated evaluation is expensive, radiation exposure is harmful to the patient and it may not be readily accessible to physiotherapists (Fitzgerald *et al*, 1983; Mayer *et al*, 1984). A universal goniometer measures uni-axial motion and is therefore considered unsuitable to measure lumbar ROM (Chiarello and Savidge, 1993). Moll and Wright (1971) used distraction and plumb-line techniques to measure lumbar flexion and extension ROM respectively. The spine was marked in two places and the distance between the two marks was measured before and after lumbar flexion and extension ROM. Frost *et al* (1982) stated that the distraction and plumb-line techniques for the measurement of lumbar ROM were generally inaccurate.

The use of a tape measure is a reliable method for the measurement of trunk flexion and lateral flexion ROM, but the measurement of trunk extension and rotation ROM showed poor reliability (Frost *et al*, 1982). This technique determines gross ROM and is unable to differentiate between movement occurring at the thoracic or lumbar spine, pelvis or hip joints (Chiarello and Savidge, 1993). This method of measurement may also not be appropriate for comparisons between participants due to differences in upper and lower limb lengths (Burdett *et al*, 1986).

An inclinometer measures the absolute orientation of a line in a vertical plane and provides an objective measurement of spinal inclination. An inclinometer may also be used to measure sagittal and coronal movement of the spine and is a simple, inexpensive measurement instrument for assessing spinal ROM (Mayer *et al*, 1984). The inclinometer is also able to differentiate between lumbar, pelvic and hip movement (Chiarello and Savidge, 1993). This is an important consideration for this study, as this allows for the differentiation between lumbar and sacral ROM.

Lumbar ROM may be measured using the single inclinometer or two-inclinometer technique (Gill *et al*, 1987; Mayer *et al*, 1984; Neblett, 2003). Mayer *et al* (1984) compared the single inclinometer and the two-inclinometer techniques and found no difference between the two techniques. The two-inclinometer technique measurements of spinal ROM were also within 10% of radiographic measurements of spinal ROM (Mayer *et al*, 1984). The two-inclinometer technique is therefore a reliable, valid and reproducible technique for the measurement of lumbar ROM (Chiarello and Savidge, 1993; Keeley *et al,* 1986; Gill *et al*, 1987; Mayer *et al*, 1984; Saur *et al,* 1996). The reliability of the inclinometer also depends on accurate palpation of bony landmarks for correct placement and measurement of ROM (Chiarello and Savidge, 1993; Mayer *et al*, 1984).

2.4.2 Measurement of hamstring flexibility

Various tests have been used to indirectly determine hamstring muscle length by measuring the hip flexion angle during a SLR test (Bohannon, 1982; Bohannon *et al*, 1985; Ekstrand *et al,* 1982; Gajdosik and Lusin, 1983; Gajdosik *et al,* 1993; Li *et al,* 1996) or by measuring the knee flexion angle after either active knee extension (AKE) or passive knee extension (PKE) with the hip fixed at 90° flexion (Decoster *et al*, 2004; Gajdosik and Lusin, 1983; Gajdosik *et al,* 1993). In most cases, the pelvis and the contralateral hip are stabilised to prevent excessive pelvic motion (Gajdosik *et al,* 1993).

The SLR test may be influenced by the peripheral nerves and fascia. It has also been used to test for neurogenic pathology or normality of the sciatic nerve roots (Bohannon *et al*, 1985; Gajdosik and Lusin, 1983; Gajdosik *et al,* 1993; Göeken and Hof, 1993; Göeken and Hof, 1994; Hsieh *et al*, 1983). If sciatic pain extending beyond the knee is experienced during an SLR test, it may indicate the presence of nerve root irritation or sciatic pain known as Lasègué's sign (Van Den Hoogen *et al*, 1996). Therefore, the SLR test should be used with caution when assessing hamstring muscle length (Gajdosik and Lusin, 1983) as it may be unclear whether the test is being limited by muscular or neurological tissue (Cameron and Bohannon, 1993).

In addition, the SLR test has been questioned as a valid measurement of hamstring musculotendinous unit length due to the extent of pelvic motion that occurs during the test (Bohannon, 1982; Cameron and Bohannon, 1993; Gajdosik and Bohannon, 1987). Bohannon (1982) performed cinematography analyses of the passive SLR test and found that different methods of stabilising the pelvis did not prevent pelvic rotation during the test. The SLR test may therefore not provide an accurate reflection of hamstring muscle length, but measures a combination of femoral and pelvic motion (Bohannon, 1982; Gajdosik and Lusin, 1983).

Further, Bohannon *et al* (1985) demonstrated that pelvic motion occurred at 9° of hip flexion during a SLR test and suggested that pelvic motion may provide a substantial contribution to the ROM achieved during a SLR test. During the testing, the pelvis was also not secured to the padded table, but the tester used one hand to firmly hold down the leg not being tested (Bohannon *et al,* 1985).

The AKE and PKE tests may provide more specific measurements of hamstring length, as the hip is stabilised in 90° flexion (Gajdosik *et al,* 1993). In the AKE test, participants are positioned supine with one strap over the pelvis and another strap over the thigh of the leg not being tested. The hip is flexed to 90° and the participants actively extend the knee to a point of mild resistance without forcing the movement. This ensures that there is no associated movement in the hip, SIJ and lumbar spine (Decoster *et al*, 2004; Esola *et al*, 1996; Gajdosik and Lusin, 1983). The degree of knee flexion that remains from terminal knee extension is known as the knee extension angle (KEA) (Davis *et al,* 2008). This test may be used to represent hamstring muscle tightness. Previous studies have used either a flexometer or an inclinometer to measure the KEA (Decoster *et al*, 2004; Gajdosik and Lusin, 1983; Li *et al*, 1996).

The AKE test has a high test-retest reliability with intratester and intertester correlation coefficients of ≥ 0.98 (Decoster *et al,* 2004; Draper *et al*, 2004; Gajdosik and Lusin, 1983; Gillette *et al*, 1991; Li *et al,* 1996; Webright *et al*, 1997). The testing procedure for the PKE test is very similar to that of the AKE test. The test is carried out by the examiner passively extending the lower limb at the knee to a point of strong resistance, which is felt by either the examiner or the patient (Davis *et al,* 2008). Davis *et al* (2004) found the intratester reliability of the PKE test to be 0.94.

The measurement of the KEA is considered the gold standard for the measurement of hamstring flexibility (Davis *et al,* 2008). In this study, the KEA was measured using a Leighton's flexometer. The Leighton's flexometer is a highly reliable measurement tool for the assessment of joint ROM, with correlation coefficients of between 0.91 - 0.99 (Hseih *et al*, 1983; Leighton, 1955). The Leighton's flexometer has the advantage of responding to gravity, thereby allowing a more reliable recording of the axis of rotation of a segment (Misner *et al*, 1992).

Figure 2.1. *Leighton Flexometer (taken from http://www.bionetics.ca/exercise/range.htm)*

In this study, a universal goniometer was also used to ensure an accurate starting position of 90º of hip and knee flexion for the test leg. The universal goniometer is generally accepted as a valid clinical tool, even though small errors may exist in the construction of the goniometer. Reliability of goniometric measurement is dependent on careful measurement technique, accurate identification of bony landmarks and the accurate alignment of the goniometer (Gajdosik and Bohannon, 1987). The goniometric measurements of six different joint motions in the upper and lower body showed intratester reliability of 0.85 and intertester reliability of 0.72 (Boone *et al,* 1978). In addition, goniometric measurements of the hip joint during the SLR test established intrasession reliability of 0.99 and intersession reliability of 0.84 (Hseih *et al,* 1983).

2.5 Summary

Although there is a relatively high incidence of low back injury in paddling (Kameyama *et al*, 1999), there is currently a lack of evidence regarding the underlying mechanisms and the factors contributing to LBP in paddlers. Lumbar flexion and rotation have been identified as risk factors for LBP (Andersson, 1981; Esola *et al*, 1996) and lumbar ROM may also be altered in the presence of LBP (Andersson, 1981; Brukner and Kahn, 2001; Esola *et al*, 1996; Li *et al*, 1996). Paddling requires repetitive trunk flexion and rotation and these athletes may therefore have an increased risk of developing LBP. In addition, alterations in lumbar ROM may affect the paddling stroke and exercise performance (Mann and Kearny, 1980; Shepard, 1987). It has also been postulated that a reduction in hamstring flexibility may be associated with LBP or altered lumbar ROM (Decoster *et al*, 2004; Göeken and Hof, 1993; Esola *et al*, 1996; Halbertsma *et al*, 2001; Li *et al*, 1996). However, this relationship has not been systematically examined in athletes that participate in sporting disciplines that report a high prevalence of LBP. Accordingly, the aim of this study is to investigate the relationship between lumbar mobility and hamstring flexibility in paddlers with and without LBP.

2.6 Significance of the study

Low back pain is a frequent occurrence in the general and sporting population, and is the most common condition treated by physiotherapists (Parker, 2007). Although there is a relatively high prevalence of LBP in paddlers (Kameyama *et al*, 1999), there is currently a lack of evidence regarding the underlying mechanisms and factors contributing to the development of LBP in this population. Contemporary physiotherapy practice emphasises addressing hamstring length and spinal mobility in the management of LBP (NICE Clinical Guideline 88, 2009). However, there is a paucity of evidence to support this approach. This study will add to the body of evidence on this topic with particular relevance to the prevention and management of LBP in paddlers.

3. METHODOLOGY

3.1 Aims and objectives

The aim of this study was to determine the relationship between lumbar mobility and hamstring flexibility in paddlers with and without low back pain.

Specific objectives included:

- To determine whether the training and racing history in paddlers with LBP
- To determine whether the training and racing history in paddlers without LBP
- To determine lumbar ROM and hamstring flexibility in paddlers with LBP
- To determine lumbar ROM and hamstring flexibility in paddlers without LBP
- To determine whether there were any relationships between lumbar ROM and hamstring flexibility, and lumbar ROM and age, in paddlers with LBP
- To determine whether there were any relationships between lumbar ROM and hamstring flexibility, and lumbar ROM and age, in paddlers without LBP
- To determine whether there were any differences in training and racing history between paddlers with and without LBP
- To determine whether there were any differences in lumbar ROM and hamstring flexibility between paddlers with and without LBP
- To determine whether there were any relationships between lumbar ROM and hamstring flexibility, and lumbar ROM and age, in paddlers with and without LBP

3.2 Participants and study design

Thirty male and female participants were recruited for the study, which had a descriptive, cross-sectional correlational design. The study was advertised through electronic mail groups and bulletin boards. Participants were also recruited for the study at marathon canoe race events. Fifteen participants (13 males, two females) who presented with paddling-associated LBP in the six months prior to testing formed the case group. The control group consisted of 15 participants (12 males, three females) with no history of LBP. The groups were matched according to age and sex.

3.2.1 Inclusion criteria

Participants were included in the study if they were under the age of 65 years and had completed a two-day canoe marathon in the last six months.

3.2.2 Exclusion criteria

Participants were excluded from the study if they reported any relevant medical or surgical history, including lumbar disc lesions, neurological dysfunction, trauma involving the spine, pelvis or lower limbs, or injury involving the hamstring muscle group. Participants with birth defects or genetic deformities of the spine and lower limbs, spondylolysis, spondylolisthesis or ankylosing spondylitis were also excluded from the study.

3.2.3 Sample size calculation

Data from a previous study which measured lumbar ROM in participants with and without LBP was used to ensure that the sample size would provide sufficient statistical power (Chiarello and Savidge, 1993). Lumbar ROM was selected to determine the required sample size, as it is one of the main outcome measures of this study. Required sample size for lumbar ROM was calculated using a small meaningful difference of 7º, and a standard deviation of 4º. With statistical significance accepted as $p < 0.05$, groups of 11, 15 and 18 participants will provide 80%, 90% and 95% statistical power for muscle soreness respectively. Therefore 30 male and female participants were recruited for this study, to ensure sufficient statistical power if some participants were unable to complete the study.

3.3 Instrumentation

3.3.1 Baseline questionnaire

The participants completed a baseline questionnaire to establish age, body mass, stature, medical and surgical history, injury history, training and competition history, and physical activity levels *(Appendix III)*. The baseline questionnaire was adapted from a previously validated questionnaire (Buchholtz *et al*, 2005; Micklesfield *et al*, 2005). The adaptation of the questionnaire for this study involved the exclusion of a single section, which was aimed at collecting data from female participants regarding risk factors for osteoporosis.

3.3.2 Low back pain

Low back pain was assessed subjectively in the case group using a visual analogue scale (VAS) for pain based on the Brief Pain Inventory (Cleeland and Ryan, 1994)(*Appendix IV*). The control group reported no pain. Case participants were required to mark the area of pain on a body chart and rate LBP experienced during the preceding week according to ‘worst pain’, ‘least pain’ and ‘average pain’. Case participants were also required to rate ‘present pain’ immediately prior to testing. Case participants were asked to rate the pain in each of the aforementioned categories by drawing a vertical line on a 100 mm pain rating scale, where 0 mm represents ‘no pain’, and 100 mm represents ‘unbearable pain’ (Esola *et al*, 1996; Lund *et al*, 2005). The distance along the pain rating scale to the vertical line drawn by the case participant was measured in millimetres and the pain score for each category was recorded.

3.3.3 Lumbar range of motion

Lumbar ROM was measured using an inclinometer in a modification of the technique described by Mayer *et al* (1984). Mayer *et* al (1984) used a two-inclinometer technique to measure lumbar ROM. In this study, a single two-point base inclinometer was used to measure lumbar ROM, as modern inclinometers can be zeroed at a starting position allowing a single ROM measurement to be obtained. For all physical measurements, participants wore shorts, with the lumbar spine and sacrum exposed. With the participants lying prone, the base of the sacrum, the T12 spinous process and the L1 spinous process were palpated and marked. Participants were positioned standing with their feet shoulder-width apart and their arms relaxed at their sides. The participants were instructed to maintain full knee extension during all movement tests (Li *et al*, 1996; Mayer *et al*, 1984; Saur *et al*, 1996).

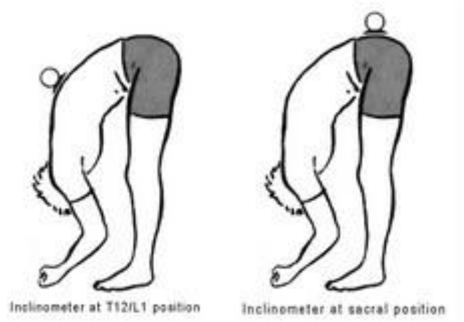

Figure 3.1 *Schematic representation of end range lumbar flexion ROM testing. The T12/L1 and sacral inclinometer positions are demonstrated separately.*

The top edge of the base of the inclinometer was positioned over the base of the sacrum. The inclinometer position was manually maintained during all testing. The inclinometer was zeroed in the starting position. The participants performed lumbar flexion and the angle of sacral flexion was recorded at the limit of pain-free active ROM. The participants then returned to the starting position, as indicated by the inclinometer reading zero. The participants performed lumbar extension and the angle of sacral extension was recorded at the limit of pain-free active ROM (Keeley *et al*, 1986; Mayer *et al*, 1984; Saur *et al*, 1996). The participants again returned to the starting position, as indicated by the inclinometer reading zero. Sacral flexion and extension ROM readings were combined to get a composite sacral ROM value (Mayer *et al*, 1984).

The top edge of the base of the inclinometer was then positioned over the T12 spinous process, with the centre of the inclinometer over the L1 spinous process. Lumbar flexion and extension were repeated as described above, to determine T12/L1 flexion and extension ROM. Composite T12/L1 ROM was also calculated by combining the T12/L1 flexion and extension ROM readings (Mayer *et al*, 1984). This testing procedure was performed three times and an average for flexion and extension ROM was recorded for both the sacral and T12/L1 ROM measurements.

T12/L1 ROM measurement represents gross spinal flexion ROM, which includes both the lumbar ROM and the sacral ROM measurements. True lumbar flexion and lumbar extension ROM were calculated by subtracting sacral flexion and extension ROM from T12/L1 flexion and extension ROM measurements respectively. Composite true lumbar ROM was calculated as the difference between composite T12/L1 ROM and composite sacral ROM (Mayer *et al*, 1984; Saur *et al*, 1996).

3.3.4 Hamstring flexibility

Hamstring flexibility was assessed using the AKE test (Decoster *et al*, 2004; Esola *et al,* 1996; Gajdosik and Lusin, 1983; Li *et al,* 1996; Willy *et al*, 2001). With the participants positioned standing, the lateral femoral epicondyle, fibular head and greater trochanter of both legs were palpated and marked. Participants were positioned lying supine with one flat pillow placed behind their head. Participants were instructed to allow the ankle to plantarflex during testing to limit the effect of potential increased neural tension that may occur with ankle dorsiflexion (Davis *et al,* 2008; Gajdosik and Lusin, 1983; Gajdosik *et al,* 1985; Gajdosik *et al*, 1993; Polachini *et al*, 2005; Webright *et al*, 1997).

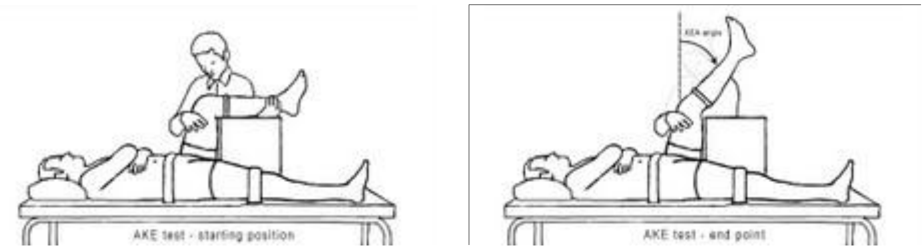

Figure 3.2 *Schematic representation of the AKE test (pelvic and knee straps are in place, the flexometer is strapped just below the knee over the head of the fibular)*

An adjustable strap was placed over the anterior superior iliac spines to limit pelvic movement during testing. An additional strap was placed over the thigh of the leg not being tested to maintain hip extension (Decoster *et al*, 2004; Gajdosik and Lusin, 1983; Gajdosik *et al*, 1993; Gillette *et al*, 1991; Taylor *et al*, 1995). The leg being tested was placed on a wooden platform which was used to maintain 90º of hip and knee flexion. These positions were established using a universal goniometer. A Leighton's flexometer was strapped in position over the neck of the fibula and zeroed. The participants were instructed to extend the knee actively at a slow rate to avoid hamstring muscle spindle excitation (Chalmers, 2004 as cited in Sharman *et al*, 2006) until the first onset of a stretch sensation (Cameron and Bohannon, 1993; Taylor *et al*, 1995; Turl and George, 1998), as opposed to discomfort (Behm *et al*, 2006), was perceived. At this point, the angle on the Leighton flexometer was recorded and then subtracted from 90° to give the KEA. .The KEA is used to indicate "hamstring flexibility". Therefore a higher KEA indicates reduced hamstring flexibility.

For consistency, the participants' right leg was tested first. A single measurement of the KEA was recorded for each leg. Previous studies have demonstrated that there is no significant variation in ROM when three repeated measurements are performed (Boone *et al*, 1978; Coppeiters *et al,* 2001; Rothstein *et al*, 1983). Average hamstring flexibility was calculated by averaging the sum of the left KEA and the right KEA (Nourbakhsh and Arab, 2002).

3.4 Testing procedure

Participants were required to complete the informed consent form *(Appendix II)* prior to the commencement of testing. Participants subsequently completed the baseline questionnaire *(Appendix III)* and rated their LBP (*Appendix IV)*. The lumbar ROM testing was performed, followed by hamstring flexibility testing. On completion of the testing, all participants received a pamphlet containing relevant stretching and strengthening exercises (*Appendix V).*

3.5 Statistical analyses

Statistical analyses were performed using the Statistica software (StatSoft Inc. 2008 STATISTICA (data analysis software system), version 8, www.statsoft.com). Student's independent t-tests were performed to analyse descriptive characteristics (height, body mass and body mass index), time trial speed, ROM measurements, and average hamstring flexibility. Non-parametric data were analysed using the Mann-Whitney U test. These data included age, training and racing history, and other sporting involvement. A Pearson's product-moment correlation coefficient determined the relationships between the variables (true lumbar ROM and average hamstring flexibility, and sacral ROM and average hamstring flexibility). All data are presented as the mean ± standard deviation. Box and whisker plots represent the median and the 5th and 95th percentile. Statistical significance was set as $p < 0.05$.

3.6 Ethical considerations

The study was granted ethical clearance by the Ethics and Research Committee of the Faculty of Health Sciences, University of Cape Town (REC REF 175/2006) (*Appendix VI*). Participants were informed about the purpose of the study, the testing to be undertaken, the possible risks related to the study and the right to withdraw from the study. All participants gave written informed consent prior to the commencement of testing. Participants under the age of 18 years provided written assent to participate in the study and parents or guardians gave written informed consent *(Appendix II).*

3.6.1 Risks to participants

There were no potential risks in completing the questionnaire. Although the measurement of lumbar ROM may have aggravated LBP in the case group, participants were instructed to only move to the limit of their pain-free ROM. Similarly, the assessment of hamstring flexibility was at the first onset of a stretch sensation. These precautions were adopted to minimise risk of injury or exacerbation of symptoms.

3.6.2 Benefits to participants

Participants were provided with a pamphlet that included low back and hamstring stretches and exercises for the prevention and management of LBP (*Appendix V*). Participants in the case group were also referred for appropriate management and rehabilitation of their condition.

4. RESULTS

4.1 Descriptive characteristics

The descriptive characteristics of the participants are shown in Table 4.1. There were no significant differences between groups for age (U = 97.00; p = 0.52), height (t = 1.53; p = 0.14), body mass (t = 1.50; p = 0.14) and body mass index (BMI) (t = 0.90; p = 0.38).

Table 4.1 *Descriptive characteristics of participants in the case (n=15) and control (n=15) groups. Data are expressed as mean ± standard deviation.*

	Case group	**Control group**
Age (years)	30.27 ± 9.65	30.60 ± 14.84
Height (m)	1.79 ± 0.07	1.75 ± 0.08
Body mass (kg)	78.55 ± 10.19	72.55 ± 11.63
BMI	24.48 ± 2.30	23.67 ± 2.61

4.2 Training characteristics

There were no significant differences between the groups in average weekly training hours or maximum weekly training hours during off-season training. Similarly, there were no significant differences between the groups in average weekly training hours or maximum weekly training hours during pre-competition training (three months prior to an event) (Table 4.2). There were also no significant differences between the groups in time trial performance [case group 3.3 ± 0.37 m.s^{-1}; control group 3.52 ± 0.47 m.s^{-1} (t = -1.24; p = 0.23)].

Table 4.2 *Training characteristics of participants in the case (n=15) and control (n=15) groups. Data are expressed as mean ± standard deviation.*

	Case group	**Control group**
Off-Season Training		
Average training (hr.wk^{-1})	8.20 ± 3.97	6.87 ± 4.00
Maximum training (hr.wk^{-1})	12.13 ± 4.36	12.00 ± 6.09
Pre-competition training		
Average training (hr.wk^{-1})	10.13 ± 3.29	8.53 ± 4.07
Maximum training (hr.wk^{-1})	14.27 ± 3.81	13.00 ± 6.14

There were no significant differences between the groups in the total number of years of paddling experience [case group 6.50 ± 4.36 years; control group 9.90 ± 8.49 years (U = 84; p = 0.25). Training and racing history of the case and control groups is shown in Figure 4.1. Although there was a tendency towards a greater number of Berg River Canoe Marathons completed in the control group (case group 0.67 ± 0.90; control group 4.80 ± 8.53), there were no significant differences between the groups (U = 83.50; p = 0.23). There were also no significant differences between the groups in the number of other multi-day paddling marathons completed [case group 9.47 ± 9.75; control group 13.33 ± 12.59 (U = 91.50; p = 0.38)].

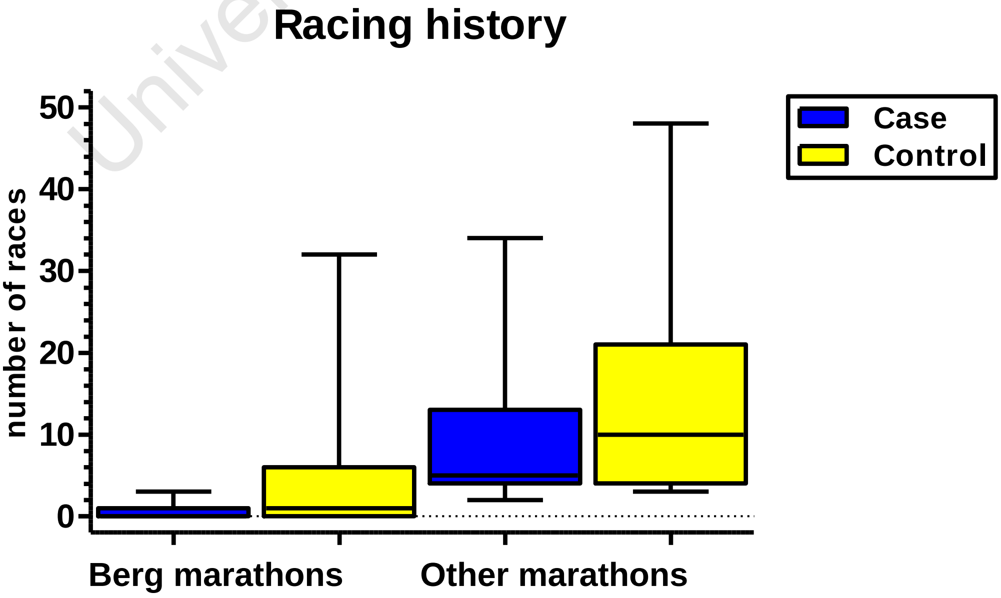

Figure 4.1 *Racing history of participants in the case (n = 15) and control (n = 15) groups. Data are expressed as median ± 5th and 95th percentile.*

4.3 Other sport involvement

With regard to other sport involvement, there were no significant differences between the groups [case group 2.53 ± 1.36; control group 1.93 ± 1.16 (U = 86.00; p = 0.281)]. There were also no significant differences in the total training hours for other sports during the 12-month period preceding the study [case group 230.16 ± 165.45 $h.yr^{-1}$ control group 174.58 ± 154.93 $h.yr^{-1}$(U = 92.00, p = 0.407)].

4.4 Low back pain

Pain scores of ‘worst pain’, ‘least pain’, ‘average pain’ and ‘present pain’ of participants in the case group are reflected in Table 4.3.

Table 4.3 *Low back pain scores of participants in the case group (n=15). Data are expressed as mean ± standard deviation.*

Pain scores (mm)	Case group
Worst pain	37.9 ± 32.8
Least pain	4.1 ± 5.9
Average pain	16.1 ± 18.0
Present pain	12.1 ± 16.7

4.5 Lumbar range of motion

4.5.1 T12/L1 range of motion

The differences in T12/L1 ROM between participants in the case and control groups are shown in Figure 4.2. There were no significant differences between the groups in T12/L1 flexion ROM [case group 112.20° ± 10.35°; control group 117.60° ± 13.32° (t = -1.24; p = 0.23)] or extension ROM [case group 33.24° ± 9.20°; control group 36.58° ± 13.07° (t = -0.81; p = 0.43)]. In addition, although there was a tendency for composite T12/L1 ROM to be lower in the case group, there were no significant differences between groups [case group 145.44° ± 15.93°; control group 153.91°± 18.29° (t = -1.35; p = 0.19)].

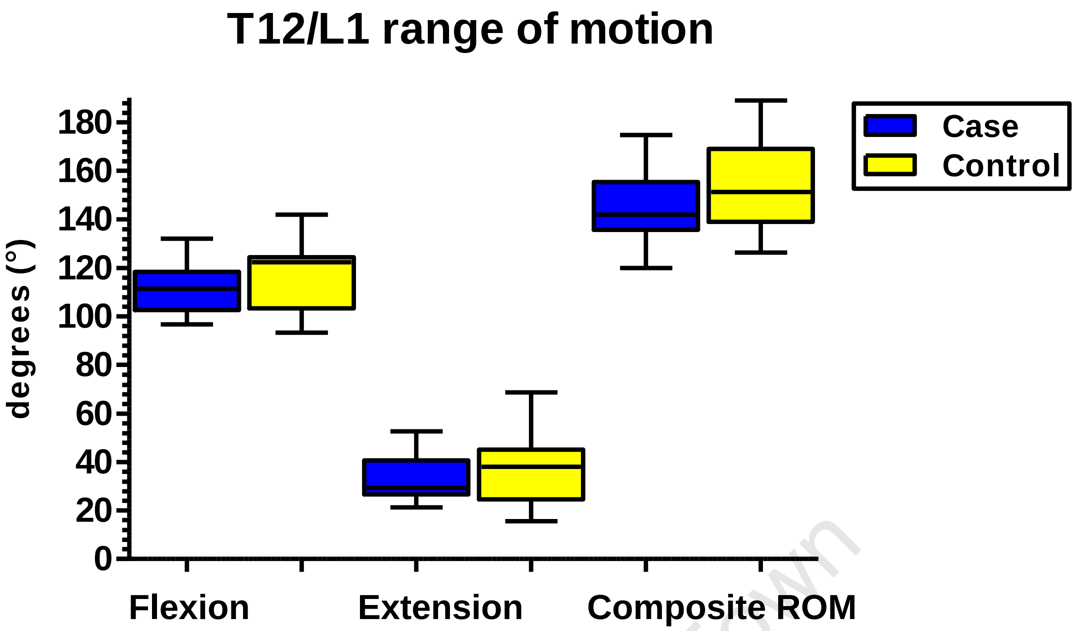

Figure 4.2 *T12/L1 ROM measurements for the case (n = 15) and control (n = 15) groups. Data are expressed as median ± 5th and 95th percentile.*

4.5.2 Sacral range of motion

The differences in sacral ROM between participants in the case and control groups are shown in Figure 4.3. Although there was a tendency for the sacral flexion ROM to be less in the case group, there were no significant differences between groups in sacral flexion ROM [case group 57.60° ± 10.09°; control group 64.09° ± 12.94° (t = -1.53; p = 0.14)] or sacral extension ROM [case group 15.00° ± 4.98°; control group 13.87° ± 5.69° (t = 0.58; p = 0.57)]. There were no significant differences between groups for composite sacral ROM [case group 72.60° ± 12.41°; control group 77.95° ± 13.66° (t = -1.12; p = 0.27)].

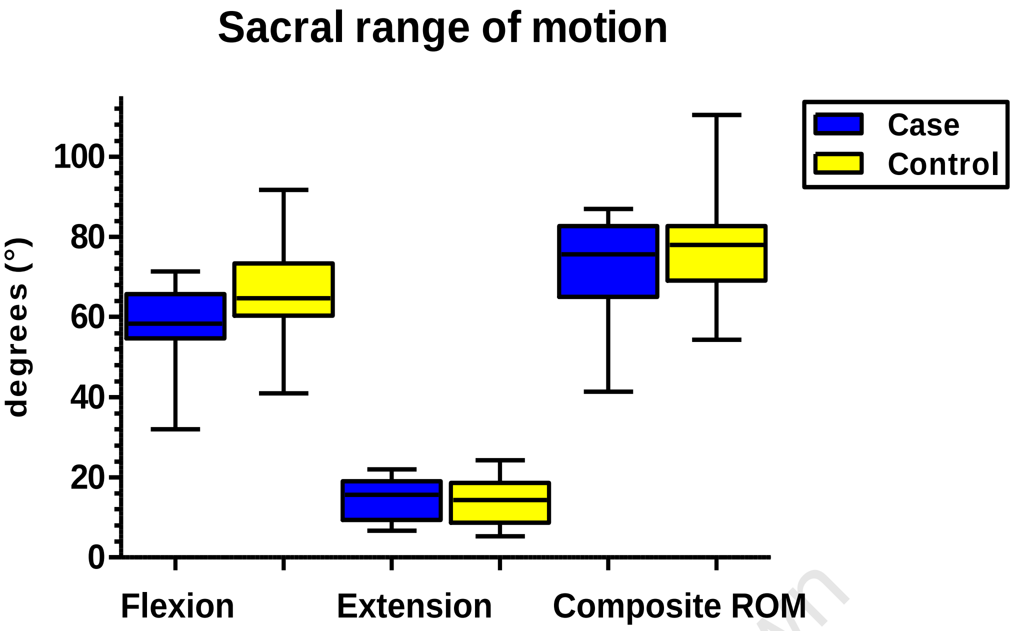

Figure 4.3 *Sacral ROM measurements for the case (n = 15) and control (n = 15) groups. Data are expressed as median ± 5th and 95th percentile*

4.5.3 True lumbar range of motion

The differences in true lumbar ROM between participants in the case and control groups are demonstrated in Figure 4.4. There were no significant differences between groups for lumbar flexion ROM [case group 54.6° ± 8.39°; control group 53.51° ± 5.98°(t = 0.41; p = 0.69)]; lumbar extension ROM [case group 18.24° ± 8.5°; control group 22.71° ± 10.89° (t = -1.25; p = 0.22)] or composite true lumbar ROM [case group 72.84° ± 14.31°; control group 75.05° ± 13.93° (t = -0.43; p = 0.67)].

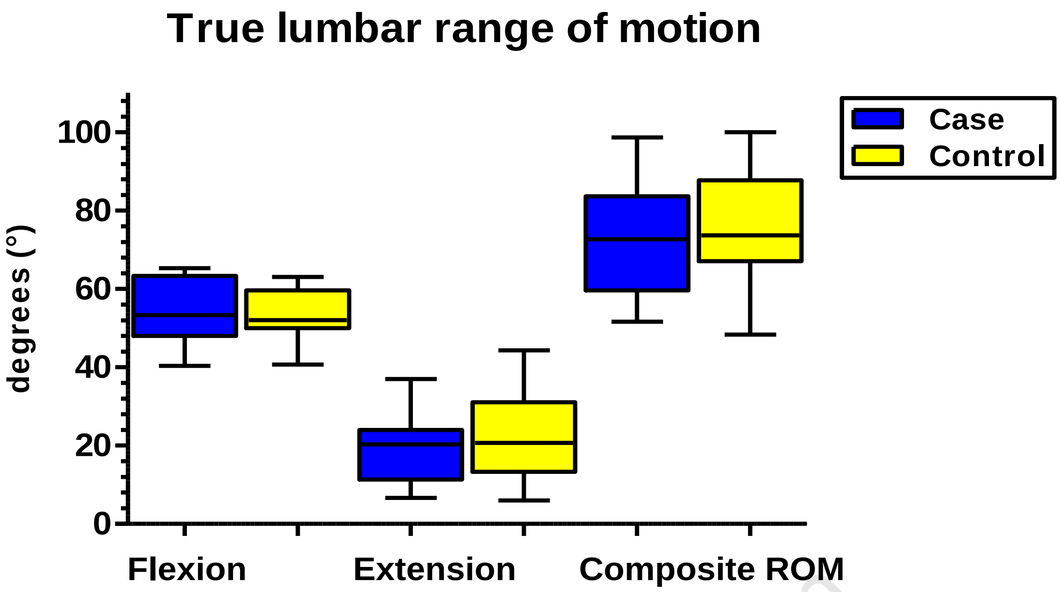

Figure 4.4 *True lumbar ROM measurements for the case (n = 15) and control (n = 15) groups. Data are expressed as median ± 5th and 95th percentile.*

4.6 Range of motion and age

4.6.1 T12/L1 range of motion and age

There were significant correlations between age and composite T12/L1 ROM for the total group (r = -0.39; p = 0.03; CI: -0.66 to -0.04) (Figure 4.5) and the case group (r = -0.59; p = 0.02; CI: -0.85 to -0.11). There was no significant correlation between age and control group composite T12/L1 ROM (r = -0.31; p = 0.26; CI: -0.71 to 0.24) when data from this group were analysed separately.

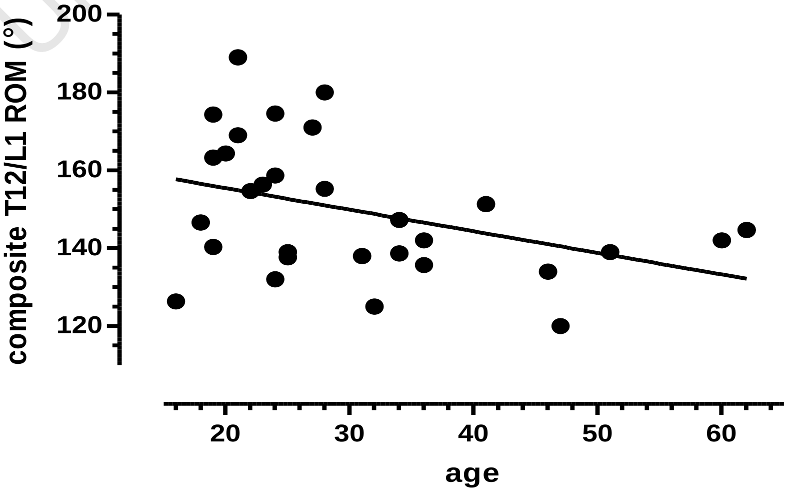

Figure 4.5 *Relationship between age and composite T12/L1 ROM for the total group (n = 30).*

4.6.2 Sacral range of motion and age

There were no significant correlations between age and composite sacral ROM for the total group (r = -0.04; p = 0.83; CI: -0.40 to 0.32), the case group (r = -0.21; p = 0.45; CI: -0.65 to 0.34) or the control group (r = 0.05; p = 0.86; CI: -0.47 to 0.55). Similarly, there were no significant correlations between age and average hamstring flexibility for the total group (r = -0.05; p = 0.79; CI: -0.40 to 0.32), the case group (r = -0.04; p = 0.88; CI: -0.54 to 0.48) or the control group (r = -0.06; p = 0.85; CI: -0.55 to 0.47).

4.6.3 True lumbar range of motion and age

There were also significant correlations between age and composite true lumbar ROM for the total group (r = -0.53; p = 0.003; CI: -0.75 to -0.21) (Figure 4.6) and the control group (r = -0.60; p = 0.02; CI: -0.85 to -0.12). There was no significant correlation between age and case group composite true lumbar ROM (r = -0.48; p = 0.07; CI: -0.79 to 0.05) when data from this group were analysed separately.

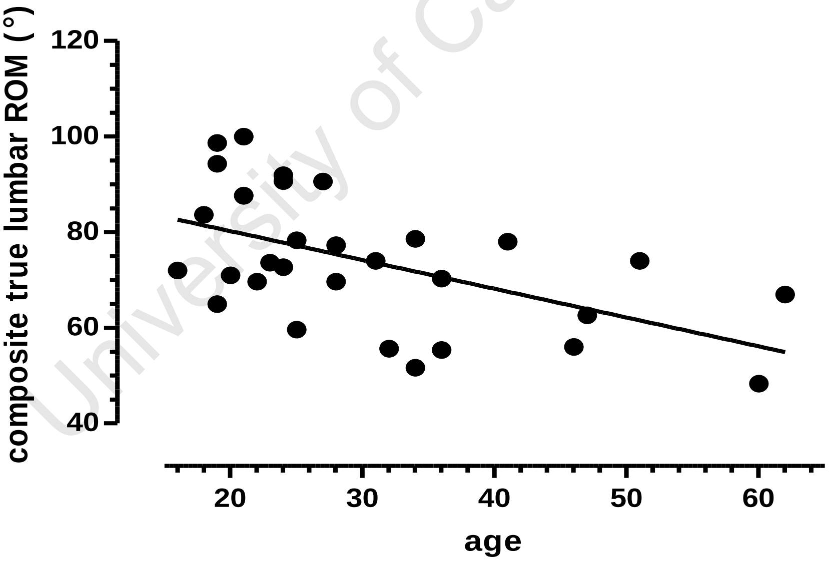

Figure 4.6 *Relationship between age and composite true lumbar ROM for the total group (n = 30).*

4.7 Hamstring flexibility

The left hamstring, right hamstring and average hamstring flexibility measurements of participants in the case and control groups are shown in Table 4.4. These measurements are reflected as the KEA. Although there was a tendency for average hamstring flexibility to be reduced in the case group, there were no significant differences between groups in average hamstring flexibility (t = 1.74; p = 0.094) or right hamstring flexibility (t = 1.23; p = 0.229). However, left hamstring flexibility was significantly reduced in the case group compared to the control group (t = 2.12; p = 0.043).

Table 4.4 *Knee extension angles (°) of participants in the case (n=15) and control (n=15) groups. Data are expressed as mean ± standard deviation.*

Knee extension angle (KEA)	Case group	Control group
Left KEA (°)	37.07 ± 9.12	29.60 ± 10.15
Right KEA (°)	33.40 ± 11.23	29.00 ± 8.13
Average KEA (°)	35.23 ± 9.88	29.30 ± 8.81

4.8 True lumbar range of motion and average hamstring flexibility

There were no significant correlations between true lumbar flexion ROM and average hamstring flexibility for the total group (r = 0.29; p = 0.12; CI: -0.09- 0.60), the case group (r = 0.29; p = 0.31; CI: -0.29- 0.70) or the control group (r = 0.22; p = 0.43; CI: -0.35- 0.67).

There were no significant correlations between true lumbar extension ROM and average hamstring flexibility for the total group (r = -0.073; p = 0.70; CI: -0.43 - 0.31), the case group (r = 0.44; p = 0.11; CI: -0.12- 0.78) or the control group (r = -0.37; p = 0.18; CI: -0.75- 0.20).

There were no significant correlations between true composite lumbar ROM and average hamstring flexibility for the total group (r = 0.11; p = 0.55; 95 % confidence intervals (CI): -0.27- 0.46), the case group (r = 0.46; p = 0.08; CI: -0.08 - 0.79) or the control group (r = -0.25; p = 0.36; CI: -0.69- 0.31).

4.9 Sacral range of motion and average hamstring flexibility

There were no significant correlations between sacral flexion ROM and average hamstring flexibility for the total group (r = -0.31; p = 0.09; CI: -0.61 - 0.06), the case group (r = -0.28; p = 0.30; CI: -0.70 - 0.28) or the control group (r = -0.21; p = 0.45; CI: -0.66 to 0.35).

There were no significant correlations between sacral extension ROM and average hamstring flexibility for the total group (r = -0.17; p = 0.38; CI = -0.50 to 0.22), the case group (r = -0.40; p = 0.14; CI: -0.76 to 0.16) or the control group (r = 0.02; p = 0.94; CI: -0.51 to 0.54).

There were no significant correlations between composite sacral ROM and average hamstring flexibility for the total group (r =-0.36; p = 0.05; CI: -0.65- 0.01), the case group (r = -0.42; p = 0.12; CI: -0.78- 0.13) or the control group (r = -0.30; p = 0.29; CI:-0.71- 0.27).

In summary; a greater KEA indicates a reduction in hamstring flexibility. A negative correlation indicates that as average hamstring flexibility decreases, lumbar or sacral ROM also decreases. A positive correlation indicates that as hamstring flexibility decreases, lumbar or sacral ROM increases. A summary of the relationships between true lumbar and sacral ROM and average hamstring flexibility is provided in Table 4.5.

Table 4.5 *Relationships between true lumbar and sacral ROM and average hamstring flexibility. Note '+' indicates a positive correlation, and '-' indicates a negative correlation.*

	Case group			Control group		
	Relationship	**r**	**p**	**Relationship**	**r**	**p**
Lumbar flexion	+	0.29	0.31	+	0.22	0.43
Lumbar extension	+	0.44	0.11	-	-0.37	0.18
True composite lumbar ROM	+	0.46	0.08	-	-0.25	0.36
Sacral flexion	-	-0.28	0.30	-	-0.21	0.45
Sacral extension	-	-0.40	0.14	+	0.02	0.94
True composite sacral ROM	-	-0.42	0.12	-	-0.30	0.29

4.10 Summary of results

There were no significant differences in lumbar or sacral ROM between the case and control groups. A significant negative correlation was observed between age and composite T12/L1 ROM for the total group (r = -0.39; p = 0.03) as well as for the case group (r = -0.59; p = 0.02). There were also significant negative correlations between age and composite true lumbar ROM for the total group (r = -0.53; p = 0.003) and the control group (r = -0.60; p = 0.02). There were no significant correlations between age and hamstring flexibility. Left hamstring flexibility was significantly reduced in the case group compared to the control group (37.07 ± 9.12 vs. 29.60 ± 10.15 respectively, p = 0.043). There were no significant correlations between true lumbar ROM and hamstring flexibility, or sacral ROM and hamstring flexibility for the total group, case group or control group.

5. DISCUSSION

The purpose of this study was to investigate the relationship between lumbar mobility and hamstring flexibility in paddlers with and without LBP. As was elucidated in the literature review, there is a paucity of evidence regarding the mechanisms contributing to LBP in paddlers. Despite anecdotal evidence and common clinical practice, there is a lack of scientific evidence for the relationship between lumbar ROM and hamstring flexibility, and also for the relationship between LBP and hamstring flexibility. Consequently, the critical discussion of the results of this study will include reference to the available literature pertaining to lumbar ROM, hamstring flexibility, and LBP. It is noted that the majority of this evidence refer to the general, non-athletic population. Where possible, inference has been made to paddlers.

5.1 Descriptive characteristics

In this study, there were no significant differences between the groups in age and gender (Section 4.1, page 37). There are age-related morphological changes in the size and shape of lumbar vertebral bodies and intervertebral discs, which may result in reductions in the flexibility and compliance of the lumbar spine, as well as decreased reaction times to sustained loading with increasing age (Twomey and Taylor, 1994). A cadaver study showed a decrease in lumbar ROM with increasing age across genders. It was theorised that the age-related reduction in ROM may be associated with increased lumbar disc stiffness and alterations in the shape of the vertebral end plates (Twomey and Taylor, 1980 as cited in Twomey and Taylor, 1994).

This study did not investigate relationships between gender and LBP or average hamstring flexibility in paddlers. The case and control groups consisted of similar numbers of male and female participants. However, Schoen and Stano (2000) reported a higher incidence of acute low back injuries in male compared to female white-water athletes. Keeley *et al* (1996) noted a difference in hamstring flexibility and pelvic flexion ROM between males and females, where females demonstrated a greater straight leg raise (SLR) measurement than men. Biering-Sørensen (1984) also established that females tend to have more ‘elastic hamstring muscles’ than males, and that the KEA was a predictor of LBP in females.

In contrast, Nourbakhsh and Arab (2002) were unable to determine significant associations between hamstring length and LBP when data from male and female participants were analysed separately or combined in a pooled group. This equivocal evidence suggests that further investigation of the relationship between gender and flexibility is needed.

There were also no significant differences in BMI between the case and control groups (Section 4.1, page 37). Youdas *et al* (2006) observed a weak positive relationship between BMI and standing lumbar curve in female participants without LBP. In addition, a number of studies have identified positive relationships between body weight or percentage body fat and LBP (Croft *et al,* 1999; Leboeuf-Yde, 2000; Toda *et al,* 2000). However, Toda *et al* (2000) were unable to determine differences in BMI between participants with and without LBP. It is noted that BMI does not differentiate fat mass from lean body mass and it may therefore be necessary for further studies to investigate more specific anthropometric measurements.

5.2 Training characteristics

The current study showed no significant differences between groups in off-season and pre-competition training, time trial performance and racing history (Section 4.2, pages 37 & 38). Hagemann *et al* (2004) were unable to demonstrate correlations between training history and soft tissue symptoms or abnormalities, and concluded that the number of years paddling and the number of endurance events completed may not be related to an increased risk of injury in paddling (Hagemann *et al*, 2004). Training adaptations may also allow the body to accommodate to the prolonged repetitive loading associated with endurance paddling. A paddler who is 'fitter' may therefore be able to maintain improved concentration levels and sustain an ideal paddling posture for longer periods of time, thereby reducing the incidence of injury (Du Toit *et al*, 1999). Although no differences in training history were observed in this study, a possible limitation may have been the method of recording training history. Training history was documented as the weekly number of hours of training, which does not reflect training intensity or frequency. Future studies should consider other training variables so that they may comprehensively evaluate endurance training loads.

5.3 Other sporting involvement

In this study, the case group tended both to be involved in a greater number of different sporting activities and to have increased training hours for other sporting activities compared to the control group; although these differences were not significant (Section 4.3, page 39). It may be proposed that LBP caused by other inherent dysfunction or overuse and perhaps related to participation in other sports, may be aggravated by endurance paddling training (Bahr *et al,* 2004). However, this study did not examine the cause of LBP in the study cohort, but rather it was specified that the case group experience LBP related to paddling.

In this study, paddlers reported the number of hours they spent participating in other sporting activities in the 12-month period preceding the study. Therefore, although physical activity levels were recorded as a point prevalence, it is not possible to accurately quantify the spinal loading that may have occurred due to participation in other sports. The cumulative effect of a broad range of activities in relation to the development of LBP is unclear. The self-reported nature of these data may influence the interpretation of activity levels and spinal loading. It may therefore be necessary to conduct longitudinal studies to determine specific relationships between spinal loading and the development of LBP based on the timing of onset of LBP during paddling training (Heneweer *et al*, 2009).

5.4 Pain characteristics

In this study, there were no differences in lumbar ROM between the case and control groups, and lumbar ROM testing was not limited by pain in the case group (Section 4.4, page 39).

Previous studies that have investigated the relationship between lumbar ROM and LBP have different inclusion criteria. For example, Esola *et al* (1996) and McClure *et al* (1997) specified that participants should have no pain at the time of testing or any pain in the two weeks prior testing. Participants were included in these studies if they had experienced pain at some point within the last five years. In contrast, Mayer *et al* (1984) and Keeley *et al* (1986) included participants with chronic spinal dysfunction, participants that had extended periods off work due to LBP and post-operative participants. Yet Mayer *et al* (1984) and Keeley *et al* (1986) did not document the pain characteristics of their participants.

However, Wong and Lee (2004) included participants in their study with current LBP which rated six on a pain analogue scale (PAS), but they excluded participants with a post-operative history, spinal trauma or neurological fall out. It is however difficult to know if the LBP participants are comparable, as a VAS score was used in this study to demonstrate ‘present pain’ which was 12.1 ± 16.7 mm and considered to be mild pain (Collins *et al*, 1997).

These factors limit the comparisons between studies as some of the studies included participants with no current LBP but documented the pain characteristics of the pain experienced two weeks or more before the study (Esola *et al,* 1996; McClure *et al,* 1997). However, other studies included chronic pain and dysfunction patients but did not document pain characteristics, and did not determine the presence or absence of pain during testing (Mayer *et al,* 1984; Keeley *et al,* 1986). Moreover, another study included participants that had pain at the time of testing, which is similar to the current study, but the pain scale used is slightly different (Wong and Lee, 2004). It is therefore recommended that future studies of LBP should document pain characteristics using standardised pain assessment instruments.

5.5 Lumbar range of motion

In this study, there were no significant differences in T12/L1, lumbar or sacral ROM between the groups (Section 4.5, page 39 to 42). There is a paucity of literature on the relationship between LBP and ROM in paddlers, therefore, the literature relating to ROM and LBP in the general population will be discussed. There is equivocal evidence for changes in spinal ROM with LBP. Some studies report no change in lumbar ROM in participants with LBP (Esola *et al,* 1996; Li *et al,* 1996; McClure *et al,* 1997), while other studies demonstrated a reduction in lumbar ROM in participants with LBP (Biering-Sørensen, 1984; Mayer *et al,* 1984; Keeley *et al,* 1986; Wong and Lee, 2004). It is possible that the absence of a warm-up prior to the measurement of spinal ROM may influence findings. Mayer *et al* (1984) observed a lumbar flexion ROM of 55° ± 9.2° in pain-free participants, which is comparable to the control group true lumbar flexion ROM observed in this study (Section 4.5.3, page 41 & 42). In both Mayer *et al* (1984) and this study, warm-up movements prior to testing where not performed. However, Keeley *et al* (1986) recorded a slightly greater lumbar flexion ROM of 65.0° ± 8.2° in pain-free male participants and 64.4° ± 8.2° in pain-free female participants following repeated lumbar flexion and extension warm-up movements prior to testing.

It is possible that the variation in findings of these studies may be related to the differences in pain characteristics of the studies as described in Section 5.4 (page 49). In contrast to the current study, Mayer *et al* (1984) and Keeley *et al* (1986) demonstrated a reduction in spinal ROM associated with a history of LBP, but the pain groups consisted of chronic pain and post-surgical participants in contrast to the healthy athletic population experiencing episodic LBP investigated in the current study. Mayer *et al* (1984) recruited participants from a pain management programme, and observed reductions in true lumbar ROM (37° ± 21.6°), lumbar flexion ROM (28° ± 14.1°) and lumbar extension ROM (9° ± 9.5°). Keeley *et al* (1986) also reported reductions in mean lumbar ROM in participants with chronic pain compared to a control group. It was hypothesised that the reduction in lumbar ROM might be related to either the sensation of pain, or a fear of causing pain. Alternatively, the decreased ROM may be caused by facet joint stiffness or myofascial tightness as result of lengthy periods of wearing a lumbar corset or maintaining a rigid spine due to pain avoidance strategies (Mayer *et al*, 1984). It is therefore evident that there may be differences in lumbar ROM in participants with chronic LBP compared to the paddlers with episodes of LBP in the six-month period prior to testing who participated in the current study.

The findings of this study are similar to previous studies which have been unable to establish differences in lumbar ROM between participants with and without LBP. Esola *et al* (1996) used 3-D optoelectric motion analysis and observed similar patterns of lumbar flexion ROM in a LBP group (43.0° ± 10.3°) and a control group (40.3° ± 14.1°). The LBP group investigated by Esola *et al* (1996), were pain-free when lumbar ROM was assessed and had been pain-free for at least two weeks prior to testing, which differs to the current study as the case group had LBP at the time of testing.

In contrast, Wong and Lee (2004) used 3SPACE fastrak 3-D magnetic resonance imaging to compare lumbar ROM between groups with isolated LBP, LBP with a restricted SLR and a control group. Participants performed a warm-up prior to testing and were instructed to move to the limit of available ROM, or to the point at which pain or other symptoms became intolerable. Both LBP groups showed significantly decreased lumbar ROM compared to the control group. In addition, the LBP participants required more time to complete tasks involving trunk movements. It was therefore concluded that subacute LBP may be associated with altered movement characteristics of the lumbar spine and hips, as decreases in lumbar spine ROM were found in all directions, as well as a decrease in hip flexion ROM during forward bending (Wong and Lee, 2004).

In addition, differences in intersegmental motion of the lumbar spine were not determined in this study. There is a high degree of measurement error for intersegmental motion due to skin movement. Although poor intersegmental motion may not necessarily cause LBP, it may be an indication of compensatory changes occurring within the body as a result of pain or dysfunction (Adams *et al,* 2006). Wong and Lee (2004) showed altered movement characteristics in participants with subacute LBP.

It may be proposed that paddlers develop a loss of intersegmental motion as a consequence of the particular seating position required in the activity. Habitual posture may lead to adaptive shortening of muscle and connective tissue, which may result in decreased flexibility and ROM (Ekstrand *et al*, 1982). Beach *et al* (2005) established that sustained sitting was associated with increased intervertebral joint stiffness and a reduction in lumbar flexion ROM. These changes may result in an increased risk of LBP. Future studies should therefore investigate changes in sequencing of both intersegmental motion and gross spinal motion as potential causative or contributing factors to the development of LBP in athletes.

Lumbar ROM studies have varied in regards to pain characteristics of the participants, ROM assessment techniques and population groups. There is also limited evidence to support normative values for spinal ROM in healthy and athletic individuals such as paddlers. This confounds the interpretation of lumbar ROM in participants with LBP. Regular monitoring of athletes may be required to accurately determine changes in lumbar ROM associated with the development of LBP (Lehman, 2004).

5.6 Range of motion and age

This study established a significant negative correlation between age and composite T12/L1 ROM for the total and the case groups. There were also significant negative correlations between age and true composite lumbar ROM for the total and the control groups. This finding is in agreement with other studies (Fitzgerald *et al,* 1983; Moll and Wright, 1971; Twomey 1979) which have shown a progressive decrease in spinal ROM with increasing age and suggests that the sport of paddling does not protect from age related changes in the lumbar spine.

Adams *et al* (2006) suggested that although aging is associated with biomechanical and cellular adaptations of the spine, it may not be predictive of the development of LBP. Further, Boden *et al* (1990) demonstrated that approximately 30% of asymptomatic participants had abnormalities of the lumbar spine magnetic resonance image (MRI) studies. Unfortunately no literature reporting on lumbar spine MRI studies in paddlers could be found. In addition, there were a higher percentage of abnormal MRI scans in asymptomatic participants over the age of sixty years. Therefore, although there may be a progressive increase in degenerative changes of the lumbar spine with age in both the general population and in paddlers, these changes may not be associated with LBP.

5.7 Hamstring flexibility

In this study, there were no differences in average hamstring flexibility between groups (Section 4.7, page 44). However, there was a significant reduction in left hamstring flexibility in the case group (37.07° ± 9.12°) compared to the control group (29.60° ± 10.15°). Various studies have combined the values of the AKE test for the left and right lower limbs and have not compared the lower limbs separately between groups (Davis *et al*, 2005; Decoster *et al*, 2004; Nourbaksh and Arab, 2002). Other studies have only assessed unilateral hamstring flexibility (Esola *et al,* 1996; McClure *et al,* 1997; Norris and Matthews, 2006). The underlying mechanisms for the unilateral difference in flexibility are unclear and may possibly be related to LBP or compensatory mechanisms to decrease pain or protect injured tissues (Van Wingerden *et al,* 1997; Wong and Lee, 2004).

Wong and Lee (2004) determined that participants with a decreased SLR (which is also used to measure hamstring flexibility) and LBP had a greater reduction in lumbar ROM compared to participants with LBP alone and a control group. It was proposed that the difference in ROM may be associated with mechanical changes of the posterior hip tissues or due to changes in the activity of the hamstring muscles. Increased hamstring muscle electrical activity has been associated with facet joint irritation, caused by hypertonic saline solution stimulation of the L4/5 and L5/S1 facet joints. The facet joint irritation was associated with a decreased SLR on the same side of the affected facet joint (Mooney and Robertson, 1976).

In this study, the case group had a significant reduction in unilateral hamstring flexibility. However, it is unclear whether this was related to the presence of a unilateral LBP, and possible underlying facet joint involvement. Future studies should categorise LBP into sub-groups according to the distribution of symptoms (unilateral or bilateral).

In studies that assess hamstring stretching as an intervention, a KEA of greater than 20° has been used to indicate hamstring muscle tightness (Bandy and Irion, 1994; Bandy *et al*, 1998; Davis *et al*, 2005; Davis *et al*, 2008; Decoster *et al,* 2004). Accordingly, the participants in the current study would all be classified as having hamstring muscle tightness. However, the aforementioned studies used the PKE test to measure hamstring muscle length, whereas this study used the AKE test. Gajdosik *et al* (1993) observed significant differences in hamstring muscle length with the AKE test, compared to the PKE test. It was postulated that there may be greater posterior rotation of the pelvis during the PKE test, which may lead to apparent increased hamstring flexibility.

In addition, previous studies lacked reporting of physical activity levels, and also had incomplete descriptions of testing procedures (for example, method of stabilisation of the pelvis and position of the opposite leg during testing) (Esola *et al,* 1996; McClure *et al,* 1997). These factors limit the comparison of the hamstring flexibility of the paddlers in this study and the potentially sedentary participants in previous studies.

5.8 Lumbar and sacral range of motion and average hamstring flexibility

In this study, there were no significant correlations between lumbar or sacral ROM and average hamstring flexibility (Sections 4.8 and 4.9, pages 44 & 45). Current literature provides equivocal evidence for the relationship between lumbar ROM and average hamstring flexibility, and the relationship between LBP and average hamstring flexibility. Previous studies (Gajdosik *et al,* 1994; Li *et al,* 1996; Norris and Matthews, 2006) have been unable to establish a relationship between pelvic tilt and hamstring muscle length in healthy active participants. Further, there was no significant relationship between standing lumbar posture and hamstring muscle length (Gajdosik *et al,* 1994; Li *et al,* 1996). However, in one isolated study, a negative correlation was found between hamstring length and lumbar lordosis, suggesting that shorter hamstrings were associated with an increased lumbar curve. This study assessed standing lumbar curve and SLR in pain free adolescent females which limits the interpretation of this study (Toppenberg and Bullock, 1986).

Li *et al* (1996) assessed pain-free participants who had been involved in a hamstring stretching programme. Although hamstring muscle length was increased in the stretching group, there was no change in standing lumbar posture. It was suggested that in the erect standing posture the hamstrings are 'slackened', therefore 'short hamstrings' would not affect lumbar angle or pelvic inclination. The increased hamstring flexibility allowed for increased hip joint motion during the early phase of forward bending (Li *et al*, 1996). However, Gajdosik *et al* (1994) found that 'short hamstrings' were associated with decrease lumbar and pelvic ROM.

Numerous studies have identified increased hamstring muscle stiffness in participants with a history of LBP (Biering-Sørensen, 1984; Halbertsma *et al*, 2001; Li *et al,* 1996; McClure *et al,* 1997). Nourbakhsh and Arab (2002) examined 600 male and female participants that were divided into symptomatic and asymptomatic groups. The majority of LBP participants (68%) had experienced pain for a period of over six months prior to testing. There were significant differences in hamstring flexibility between the LBP groups and the control group, with the LBP groups showing increased hamstring tightness. However, physical activity levels and pain ratings were not reported which limits the interpretation of these findings (Nourbakhsh and Arab, 2002).

In contrast, Esola *et al* (1996) found that participants with a history of LBP did not show a significant decrease in hamstring flexibility compared to a pain-free group, although the LBP participants showed a tendency towards tighter hamstrings.

Further, Esola *et al* (1996) found a strong, positive correlation of hamstring flexibility to total forward bending ROM in LBP participants. It was theorised that the hamstrings may be involved in controlling forward bending in LBP participants. In contrast, Li *et al* (1996) demonstrated that a reduction in hamstring muscle length was associated with a greater relative lumbar ROM during the late phase of forward bending. Although speculative, it may be theorised that a reduction in hamstring flexibility might develop as a consequence of LBP to generate control of movement, rather than a causative factor leading to LBP. However, this theory requires further investigation through prospective, longitudinal studies.

It was interesting to observe the relationships between average hamstring flexibility and lumbar ROM in this study. Although there were no significant relationships, there was a tendency for a negative relationship between average hamstring flexibility and lumbar ROM in the case group, which differed from the positive relationship in the control group. Without longitudinal studies, it is not possible to determine whether this relationship is causative of LBP, or as a consequence of the condition. However, these results may indicate that in the clinical management of LBP, addressing hamstring flexibility may facilitate rehabilitation of LBP. An alternative interpretation is that LBP may be a result of excessive lumbar ROM, and management thereof should not include rehabilitation techniques that are aimed at increasing spinal ROM. Further longitudinal studies are required to confirm these proposed theories. It is therefore not possible to make clinical recommendations based on the findings of this study.

6. CONCLUSION

In conclusion, there were no significant differences in lumbar ROM between paddlers with and without a history of LBP. Although there were also no significant differences in average hamstring flexibility in the case and control groups, there was a significant reduction in unilateral hamstring flexibility in the case group. In addition, this study was unable to establish a relationship between LBP and hamstring flexibility in paddlers.

This study did not identify significant relationships between hamstring flexibility and lumbar ROM. Clinically, it is important to establish the role of cause and effect between biomechanical abnormalities identified in populations with LBP. This study was unable to establish any cause and effect relationship between hamstring flexibility and LBP in paddlers. However, this study investigated this relationship in a sport-specific population only. Therefore, the current clinical guidelines for the management of LBP in a general population, which includes addressing lumbar mobility and hamstring flexibility, should remain until further evidence is forthcoming.

6.1 Recommendations

This study only examined lumbar ROM in a sagittal plane and did not assess differences in lumbar rotation ROM. Therefore, further studies should investigate differences in lumbar ROM in the frontal and horizontal planes. Further studies should determine the relationship between muscle fatigue and the development of LBP. In addition, studies are warranted to determine the influence of cumulative training loads on the development of LBP and lumbar ROM. Future research should also investigate changes in sequencing of both intersegmental motion and gross spinal motion as potential causative or contributing factors to the development of LBP in athletes.

6.2 Limitations of the study

There are some limitations associated with the design of this study. A possible limitation of this study may have been the method of recording training history. Training history was documented as the weekly number of hours of training, which does not reflect training intensity or frequency. Future studies should consider other training variables so that they may comprehensively evaluate endurance training loads.

It is possible that the composite ROM readings may have been affected by repeated measures of flexion and extension, as it is proposed that this may increase ROM. In addition, paddlers reported the number of hours they spent participating in other sporting activities in the 12-month period preceding the study. The self-reported nature of these data may influence the interpretation of activity levels and spinal loading.

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APPENDIX I- Overview of the Paddling Action

The paddling action is a continuous motion of the upper body performed on both sides of the boat. The paddler is required to execute a repetitive, complex action of powerful horizontal strokes to generate the forward motion of the boat for prolonged periods of time (Kameyama *et al*, 1999; Mann and Kearney, 1980). If paddling is repeated too frequently, it may lead to the overuse of, or injury to, the upper body or trunk (Kameyama *et al*, 1999). There is little scientific evidence demonstrating the biomechanics of the paddle stroke.

When sitting in the boat, the trunk should be flexed 5° to 10° forward at the hips. There should be a mild thoracic kyphosis and a shallow lumbar lordosis, with either a neutral or anterior pelvic tilt. The anterior pelvic tilt will maintain the lumbar lordosis; however, tight and short hamstring muscles may prevent the paddler from achieving an anterior pelvic tilt, as will weakness of the stability muscles of the lumbopelvic region. Further, it is reported that if a lumbar lordosis is not maintained and the athlete acquires a flexed lumbar posture, the activation of the back muscles may increase, leading to a reversal of the spinal curves. This may lead to trunk rotation and affect the paddle stroke (Deykin, 2006 as cited in Edge, 2006).

The hips will be in slight flexion to assist with maintaining the unsupported sitting posture. The feet should be resting on the footboard which should be angled at approximately 40° to 50° in the boat, this will allow a popliteal angle of 120° to 130° at the knee (Figure 1) (Campbell, 2006).

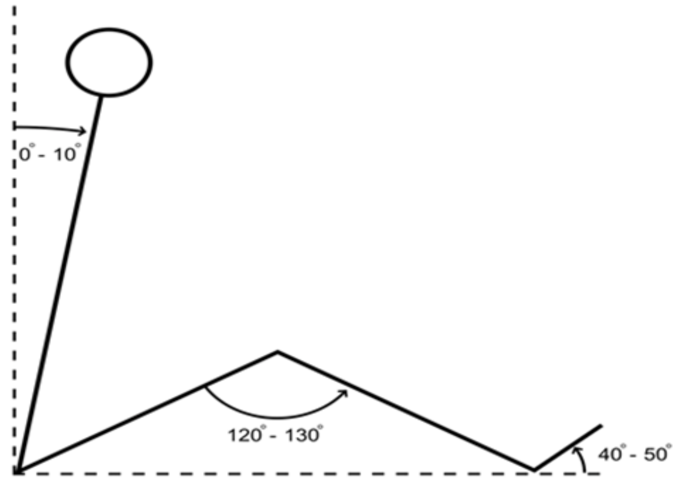

Figure 1 *Posture in the boat*

PADDLE STROKE

The paddle stroke consists of three phases: namely, the entry and catch phase, the power phase, and the exit and recovery phase.

The entry and catch phase

During this phase of the paddle stroke, the paddler drives the blade into the water as far towards the front of the boat as possible. The entry is facilitated by using trunk rotation to extend the reach, thereby placing large muscle groups in the most advantageous position without compromising good posture. This movement is facilitated by increased hip and knee flexion on the paddle entry side of the boat (Campbell, 2006; Mann and Kearny, 1980; Shepard, 1987).

At the time of paddle entry, the trunk is flexed forward between 0° to 10° at the hips and the trunk is rotated approximately 70° away from the side of paddle entry, with a small amount of lateral flexion (Figure 2) (Campbell, 2006; Plagenhoef, 1979; Shepard, 1987). The shoulder on the paddle entry side is therefore positioned well ahead of, and lower than, the opposite shoulder, which allows for optimal force generation during the paddle stroke (Mann and Kearny, 1980; Shepard, 1987).

Figure 2 *Entry and catch position (picture used with permission from Viviers, 2009)*

The catch is the point at which the blade enters the water and becomes a fixed point in the water from which the boat is propelled forward (Almasi, 2004; Campbell, 2006; Kameyama *et al*, 1999). Stability is created by pressure through the legs into the foot rest while maintaining a strong upright posture (Campbell, 2006). The optimum paddle angle at entry is approximately 35° to 40° to the water surface, with the blade kept close to the boat (Plagenhoef, 1979). Tension through the leg into the footrest occurs just prior to paddle entry, in preparation for the 'catch'. The blade must be 'locked' and a good connection must be maintained between the footrest, hip, trunk and shoulder as the force is transmitted to the blade (Campbell, 2006).

The power phase

The paddler uses simultaneous trunk rotation and arm pull to propel the boat forward around the ‘catch’ point (Figure 3). The trunk reverses the rotation achieved for the ‘catch’ phase and continues to rotate while a downward pressure is maintained on the blade to accelerate the boat past the blade (Campbell, 2006). The knees will extend about 10° due to the pressure exerted through the legs into the footboard for stability. The knee on the paddle entry side is further extended as the foot is pushed into the footboard to maximise the pulling power transferred to the blade and there is a simultaneous counter pressure directed backward, though the buttock, to the seat (Almasi, 2004; Campbell, 2006; Shepard, 1987). The whole trunk rotates on a firm base of support created by the legs and the pelvis (Campbell, 2006).

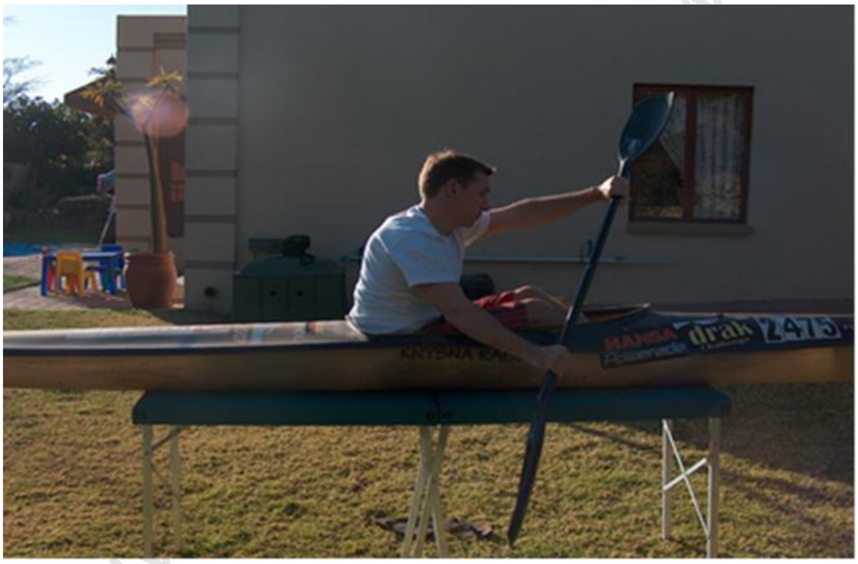

Figure 3 *Power phase* *(photo used with permission from Viviers, 2009)*

Maximum horizontal boat acceleration occurs at the vertical paddle position. A horizontal arm action of ‘push-then-pull’ is coupled with trunk rotation to achieve horizontal acceleration of the boat. The arm on the side opposite to paddle entry facilitates the ‘push’ and the ‘pull’ is facilitated by the lower arm on the side of paddle entry (Mann and Kearney, 1980). As the stroke approaches ‘paddle exit’, the trunk rotates approximately 60° towards the paddle entry side of the boat (Shepard, 1987). The whole stroke of the power phase occurs in front of the body and the blade should be out of the water by the time it reaches the level of the hip (Almasi, 2004; Campbell, 2006).

The exit and recovery phase

During the recovery phase, the paddle travels from the exit point of the water to the new catch position on the opposite side of the boat (Figure 4) (Campbell, 2006). The blade is vertically withdrawn from the water when it is level with the hip (Almasi, 2004). Anecdotally, as the blade exits the water, the trunk will continue to rotate another 10° to achieve the new start position of approximately 70° away from the 'catch' point on the opposite side of the boat. There will be simultaneous hip flexion and knee flexion on the opposite side of the body in preparation for the entry and catch phase.

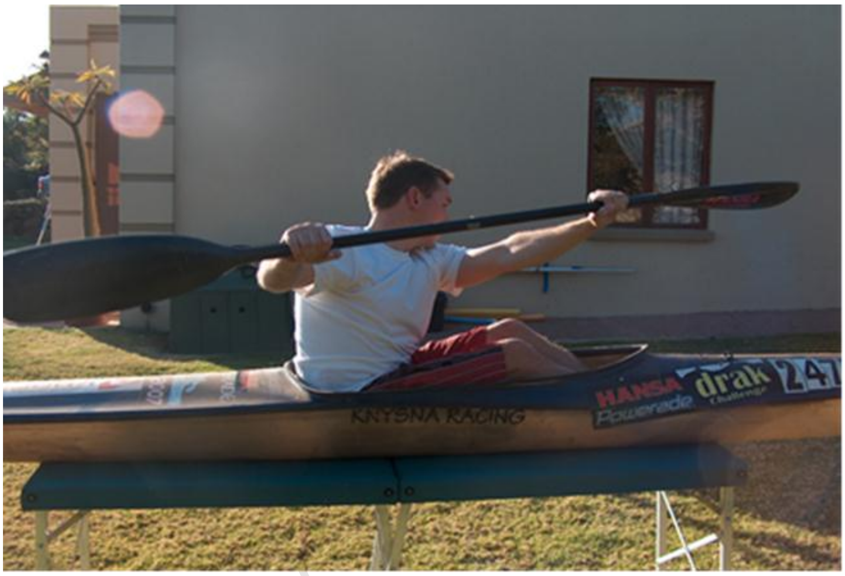

Figure 4 *Exit and recovery phase* (picture used with permission from Viviers, 2009)

A late exit from the water will delay the motion through the air to the stroke on the opposite side of the boat, which may compromise the next stroke (Campbell, 2006). Mann and Kearney (1980) suggested that paddle/water contact should be terminated as quickly as possible to avoid 'paddle drag'.

It is therefore evident that endurance paddling requires repetitive motion that may overload the lumbar spine. Propulsive forces are primarily generated through trunk rotation and shoulder extension. These areas may therefore be prone to developing overuse injuries due to their rigorous contribution to force generation during the paddling stroke (Kameyama *et al,* 1999).

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APPENDIX II – Informed consent

UNIVERSITY OF CAPE TOWN

DIVISION OF PHYSIOTHERAPY

INFORMED CONSENT FORM

Dear Canoeist

The UCT Division of Physiotherapy and the MRC/UCT Research Unit for Exercise Science and Sports Medicine will be conducting a study to investigate the following:

- the incidence of injuries among marathon canoeists
- the relationship between paddle-shaft diameter and individual handgrip size and the development of tennis elbow and wrist pain in marathon canoeists
- the relationship between the lower back pain, lumbar range of movement and hamstring flexibility in marathon canoeists

The study will provide further insight into training, performance and injuries among marathon canoeists. In addition, it will provide a basis for future studies that aim to decrease the risk of injuries and improve performance in marathon canoeists.

The study will involve the following tests:

1. A baseline questionnaire

- this will include questions regarding demographic information, medical and surgical history, training history, paddling history, boat information, and injury history
- this questionnaire may be completed electronically before the 2006 Isuzu Berg River Canoe Marathon, or before the race at registration

2. At race registration (before the race) the following tests will be performed:

2.1 the completion of the baseline questionnaire (if not completed prior to registration)
2.2 body mass and stature measurements
2.3 anthropometric assessment of body composition involving the measurement of skinfold thicknesses using skinfold callipers

2.4 the diameter of your paddle and the size of your handgrip will be recorded using a tape measure

2.5 the following tests will be performed to assess wrist function:

- your elbow will be straightened and your wrist will be flexed to determine the presence of any pain
- you will then close your hand around your thumb, your elbow will be straightened and your hand stretched towards the little finger side of your hand again to determine the presence of any pain
- the maximal grip strength for each hand will be measured using a hand dynamometer. A dynamometer records the force produced by muscles

2.6 the following tests will be performed to assess lower back function:

- a questionnaire to determine the presence of any lower back pain and contributing factors
- the range of lower back movement will be measured using an inclinometer. This is a device that will be positioned over certain points on the spine. This device records degrees of movement. The range of flexion (bending forwards) and extension (bending backwards) will be recorded three times in order to obtain an average measurement
- the inclinometer will also be used to determine the degree of hamstring (back of thigh muscle) flexibility in both legs. This test will be performed in lying, with a strap over your pelvis to prevent movement during the test. A box will be placed under your thigh to maintain the hip at right angles during testing. The inclinometer will be held over a point on your knee. The knee will then be straightened as much as possible to determine the flexibility of the hamstring muscle. This test will be performed once on each leg. There may be slight discomfort (a sensation of a strong muscle stretch) during this test

3. At the end of each stage of the race, the following tests will be performed:
    - 3.1 should any injury occur during any stage of the race, you will be asked to complete a questionnaire to determine the type of injury, the mechanism of injury, and any associated factors that may have contributed to the injury
    - 3.2 you will be required to complete a daily “rating of pain” scale to monitor the injury during the race
    - 3.3 if you develop wrist or elbow pain during any stage of the race, the wrist function tests (described above) will be completed
    - 3.4 if you develop lower back pain during any stage of the race, the lower back function tests (described above) will be completed

- ***Possible risks to subjects***

There are no potential risks that may be associated with completing the questionnaire, mass, stature, skinfold measurements, muscle pain measurements, paddle size and handgrip measurements. During the wrist function tests, the only possible risk is the potential to cause muscle injury during the maximal grip strength test. However, this risk will be greatly minimised by thorough explanation of procedures, familiarisation of equipment and careful control of all testing procedures by an experienced investigator. During the lower back function tests, there is the risk of discomfort during the range of movement tests, and the hamstring flexibility test. This risk of discomfort will be minimised through thorough explanations, familiarisation, and control of the testing procedures by an experienced investigator.

- ***Anticipated benefits to subjects***

Subjects will receive a full summary of their individual results, as well as the overall findings from this study. Should subjects present with, or develop an injury during the 2006 Isuzu Berg River Canoe Marathon, advice and an exercise sheet will be given to assist with rehabilitation and to prevent further injuries from occurring.

- ***Privacy and confidentiality***

All records and results generated within this study will be stored in a computer database in a secure facility, and in a manner that maintains subject confidentiality. All participants will remain anonymous in any ensuing publication.

- ***Contact Information***

Investigator Name	Telephone	Email
Richard Feher	082 781 4403	richphysio1@worldonline.co.za
Wendy Viviers	082 466 8468	wkviviers@absamail.co.za
Robyn John	083 236 8017	robjon@discoverymail.co.za
Theresa Burgess	021 406 6171	tburgess@uctgsh1.uct.ac.za
Romy Parker	021 406 6571	rparker@uctgsh1.uct.ac.za

I confirm, if I complete the questionnaire electronically, that I have read and understood the informed consent form. I have contacted one of the investigators to have any questions explained to me. I understand that, if I complete the informed consent form and questionnaire electronically, that I will be required to sign a hard copy of the consent form at the 2006 Isuzu Berg River Canoe Marathon.

I confirm that the exact procedures and possible complications of the above tests have been explained to me. I understand that I may ask questions at any time during the testing procedures. I realise that I am free to withdraw from the study without prejudice at any time, should I choose to do so. I have been informed that the personal information required by the researchers will be held in strict confidentiality. In addition, I know that the information derived from the testing procedures will remain confidential and will be revealed only as a number in statistical analyses.

I have carefully read this form. I understand the nature, purpose and procedure of this study. I agree to participate in this research project of the UCT Division of Physiotherapy and the MRC/UCT Research Unit for Exercise Science and Sports Medicine.

Name (in full) of volunteer:

____

Signature of volunteer:

____

Signature of parent/ guardian:

____

Name (in full) of witness:

____

Signature of witness:

____

Date:

____

APPENDIX III- Baseline Questionnaire

UNIVERSITY OF CAPE TOWN
BERG RIVER CANOE MARATHON STUDY

INSTRUCTIONS:

- This questionnaire is 6 pages long and consists of 7 sections
- Please read each question carefully, as it is important that we obtain accurate information.
- Please place information in the appropriate text box
  e.g. Date of Birth: **12/03/1976** Day/Month/Year
- If a question is asked, please place an 'x' in the appropriate text box
  e.g. To which ethnic group do you belong?
  ☐ Black ☐ White ☒ Coloured ☐ Asian ☐ Indian ☐ Other
- Please answer all questions as truthfully as possible. All personal information will be kept strictly confidential
- If you have any questions, do not hesitate to contact us on:

Richard Feher	082 781 4403
Theresa Burgess	083 300 7763
Romy Parker	072 658 6836

SECTION ONE: PERSONAL DETAILS

a. Name: ______

b. Staff number: ______

c. Contact number: ______

d. Home address: ______

e. Gender ☐ Male ☐ Female

f. To which ethnic group do you belong?

☐ Black ☐ White ☐ Coloured ☐ Asian ☐ Indian ☐ Other

g. Date of Birth ______ Day/Month/Year

h. Height ______ (cm)

i. Weight ______ (kg)

j. Dominant hand ☐ Left ☐ Right

SECTION TWO: MEDICAL HISTORY

a. Have you ever been diagnosed with any of the following diseases?

☐ Asthma
☐ Diabetes
☐ Inflammatory Arthritis
☐ Thyroid disease
☐ Osteoarthritis

☐ Renal disease
☐ High blood pressure
☐ Tuberculosis
☐ Osteoporosis
☐ Cancer

☐ [illegible] disease
☐ High cholesterol
☐ Coronary artery disease
☐ Bilharzia/Schistosomiasis
☐ Other

If other, please specify: ______

b. What medications did you or do you take to treat these conditions?

Date	Disease	Medication

c. In the last 6 months have you taken any medication, such as:

☐ Non Steroidal Anti Inflammatory Drugs (NSAIDs)

☐ Pain killers/analgesics

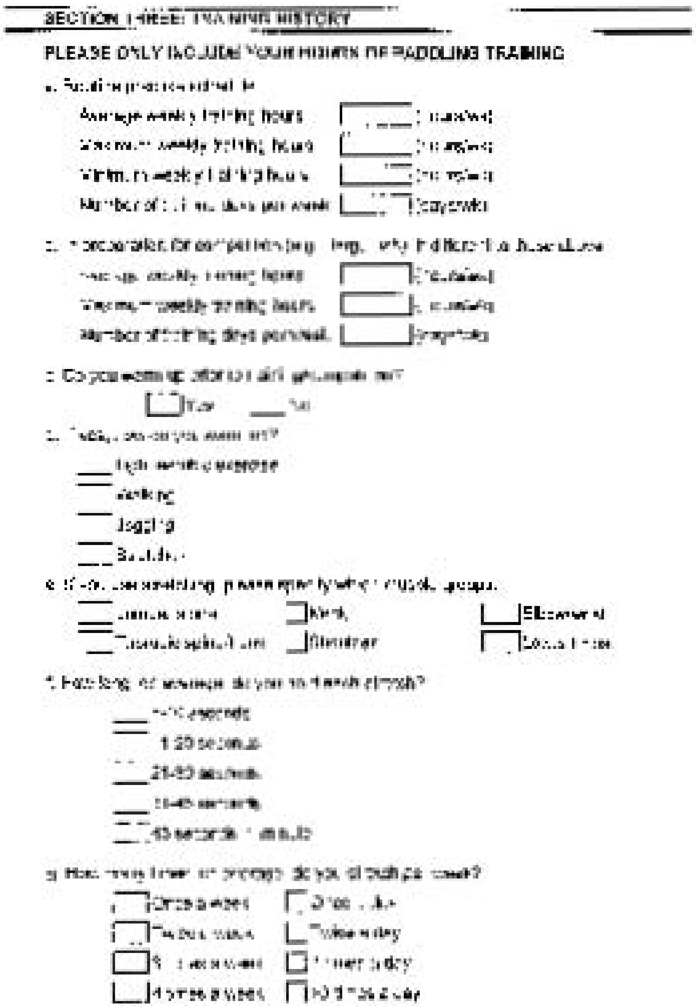
Please specify ______

d. Have you ever had surgery to any of the following:

☐ Lumbar spine ☐ Shoulder ☐ Wrist/hand

☐ Neck ☐ Elbow ☐ Lower limbs

Please specify ______



SECTION THREE: TRAINING HISTORY

PLEASE ONLY INCLUDE YOUR HOURS OF PADDLING TRAINING

a. Routine practice schedule

Average weekly training hours ☐ (hours/wk)
Maximum weekly training hours ☐ (hours/wk)
Minimum weekly training hours ☐ (hours/wk)
Number of training days per week ☐ (days/wk)

b. In preparation for competitions (e.g. [illegible] only if different to those above)

Average weekly training hours ☐ (hours/wk)
Maximum weekly training hours ☐ (hours/wk)
Number of training days per week ☐ (days/wk)

c. Do you warm up before training/competition?

☐ Yes ___ No

d. If yes, how do you warm up?

___ Light aerobic exercise
___ Walking
___ Jogging
___ [illegible]

e. [illegible] stretching [illegible] muscle groups.

___ Lumbar spine ☐ Neck ☐ Elbow/wrist
___ Thoracic spine/[illegible] ☐ Shoulder ☐ Lower limbs

f. How long (on average) do you hold each stretch?

___ <10 seconds
___ 11-20 seconds
___ 21-30 seconds
___ 31-45 seconds
___ 45 seconds - 1 minute

g. How many times (on average) do you stretch per week?

☐ Once a week ☐ Once a day
☐ Twice a week ☐ Twice a day
☐ 3 times a week ☐ 3 times a day
☐ 4 times a week ☐ >3 times a day

SECTION FOUR: PADDLING HISTORY

a. Number of years paddling ☐

b. PADDLING HISTORY
Please complete the following table.

Race	Number completed	PB			Most recent performance		
		Year	Position	Time	Year	Position	Time
Berg							
Breede							
Dusi							
Fish							
Umko							
[illegible]							
Vaal							
Lowveld Croc							
Other K1 marathon							
10 km K1 time trial							

c. OTHER EVENTS
Please complete the following table.

Event	Number completed	PB		Most recent performance	
		Year	Time	Year	Time
Two Oceans					
Cape Argus Cycle Tour					
Freedom Challenge					

SECTION FIVE: BOAT INFORMATION

a. Type of personal flotation device
☐ Polystyrene sheets ☐ [illegible] ☐ Polystyrene strips

b. What boat are you using in the [illegible] Berg River Canoe Marathon?
☐

c. Is this boat [illegible]? ☐ Yes ☐ No

d. [illegible]?
☐

e. Paddle type: ☐

f. Paddle length: ☐ cm

g. Hand protection used:
☐ Gloves ☐ Vaseline ☐ [illegible] ☐ Not applicable

h. Head protection used:
☐ None ☐ Cap ☐ [illegible]

SECTION 5A: PHYSICAL ACTIVITY PARTICIPATION

a. We would like to find out about any other physical activities that you participate in. Below is a table with examples of different activities. Please list (by number) any other sports that you regularly participate in.

Type of Sport	Months per year	No. of sessions per week	Duration of each session (mins)	Total hours per week

Examples of sporting activities

1. Jogging
2. Swimming
3. Cycling
4. Walking
5. Squash
6. Badminton
7. Netball
8. Soccer
9. Rugby
10. Aerobic dance/step
11. Martial arts
12. Volleyball
13. Strength training
14. Hiking
15. Rock climbing
16. Tennis
17. Golf
18. Skating
19. Yoga
20. Pilates
21. Dancing

b. Please indicate whether you have performed any of these specific strengthening exercises since January 2006

☐ Latissimus pull downs
☐ Behind-the-head military press
☐ Inclined flyes
☐ Supine flyes
☐ Wide grip bench press
☐ [illegible]
☐ Kayak (stationary) paddle with weight belt

____ Upright rows
____ Dumbbell rows
____ Triceps dips with weight belt
____ Triceps dips without weight belt
____ Chin ups with weight belt
____ Chin ups without weight belt

Thank you for completing the first part of the questionnaire. Please move on to the next part [illegible] to complete the second (and last) part of the questionnaire.

SECTION SIX: PHYSICAL ACTIVITY PARTICIPATION

a. We would like to find out about any other physical activities that you participate in. Below the table are examples of different activities. Please specify (number) any other sports that you regularly participate in.

Type of Sport	Months per year	No. of sessions per week	Duration of each session (minutes)	Total hours per week

Examples of sporting activities:

1. Jogging
2. Swimming
3. Cycling
4. Walking
5. Squash
6. Badminton
7. Netball
8. Soccer
9. Rugby
10. Aerobics/dance/step
11. Martial arts
12. Volleyball
13. Strength training
14. Hiking
15. Rock climbing
16. Tennis
17. Golf
18. Skating
19. Yoga
20. Pilates
21. Canoeing

b. Please indicate whether you have performed any of these specific strength training exercises since January 2005

☐ Lat palms up pull downs
☐ Behind the head military press
☐ Inclined flyes
☐ Supine flyes
☐ Wide-grip bench press
☐ Hamstring curls
☐ Squats (short range) whilst with weight belt
☐ Upright rows
☐ [illegible] rows
☐ Triceps dips with weight belt
☐ Triceps dips without weight belt
☐ Chin ups with weight belt
☐ Chin ups without weight belt

On behalf of the UCT Division of Physiotherapy and the MRC/UCT Research Unit for Exercise Science and Sports Medicine, thank you for taking the time to complete this questionnaire. The information will provide further insight into training, performance and injuries among marathon paddlers. It could in turn provide a basis for future studies that aim to decrease the risk of injuries and improve performance in marathon paddlers.

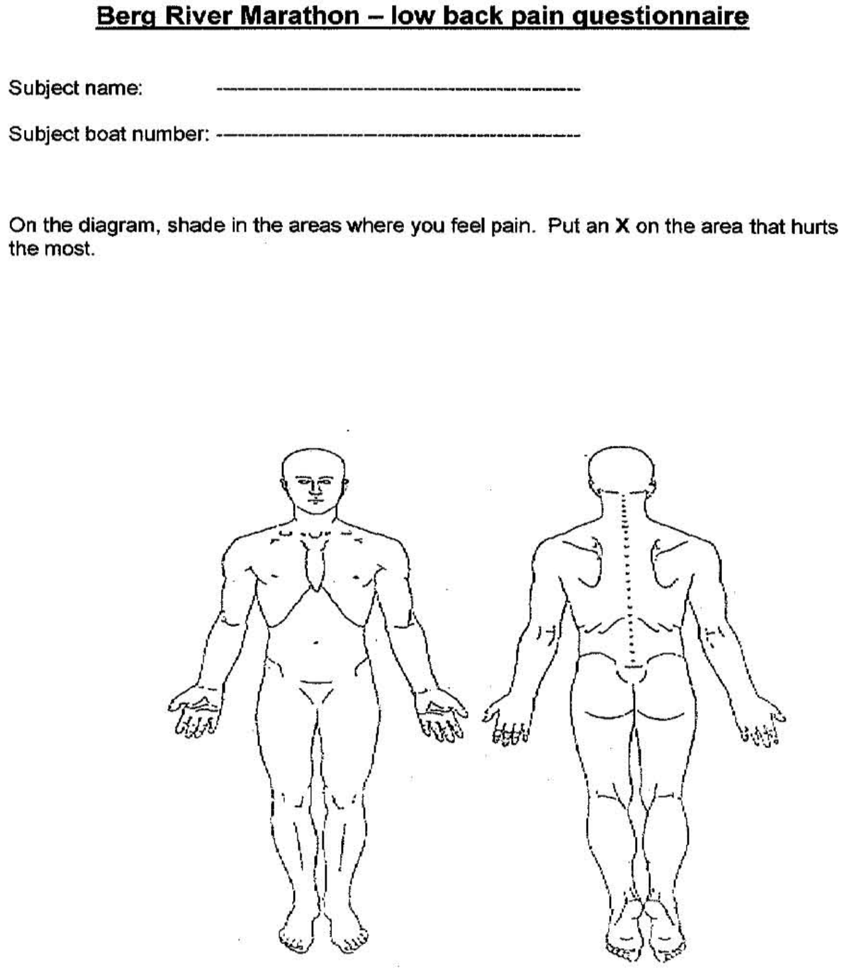
SECTION SEVEN: PADDLING RELATED INJURIES

a. Have you ever experienced a paddling related injury? ☐ Yes ☐ No

If Yes, please complete the following table.

Date of injury	Area of injury	Type of injury	Injury occurred during	Diagnosed by	Special investigations	Time off training (weeks)	Management of injury	Did the injury resolve? Y/N	If no, for how long did it persist? (weeks)
	Examples: Lumbar spine Thoracic spine Neck Shoulder Elbow Wrist/hand Hip Knee Ankle/foot	Examples: Muscle strain Ligament sprain Muscle cramp Stress fracture Tendon injury Nerve Dislocation Fracture Contusion	Examples: Indoor Portage Boat	Examples: Self-diagnosed Orthopaedic surgeon Physiotherapist Biokineticist GP	Examples: X-ray Bone Scan MRI		Examples: Self-managed Orthopaedic surgeon Physiotherapist Biokineticist GP		

APPENDIX IV- Low back pain visual analogue scale



Berg River Marathon – low back pain questionnaire

Subject name: --

Subject boat number: --

On the diagram, shade in the areas where you feel pain. Put an **X** on the area that hurts the most.

Rating of pain

Please rate your pain by drawing a vertical line along the axis below, in the position that best indicates your pain at its *worst* in the last week.

No pain — Unbearable pain

Please rate your pain by drawing a vertical line along the axis below, in the position that best indicates your pain at its *least* in the last week.

No pain — Unbearable pain

Please rate your pain by drawing a vertical line along the axis below, in the position that best indicates your pain on the *average*.

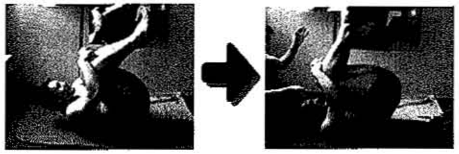
No pain — Unbearable pain

Please rate your pain by drawing a vertical line along the axis below, in the position that best indicates the pain you have *right now*.

No pain — Unbearable pain

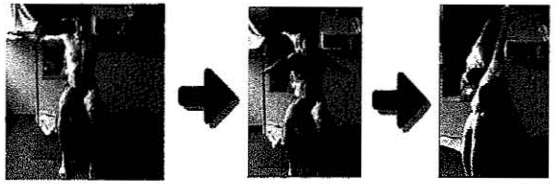
Lower back and hamstring stretches

Hold all stretches for 30 sec x 2

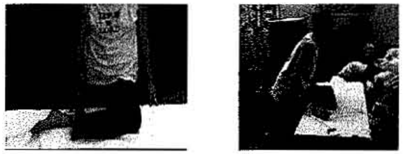


pull knees into chest and lift coccyx off floor

Sit in position as above, bottom leg straight, keep arm straight and allow side to sink towards the ground

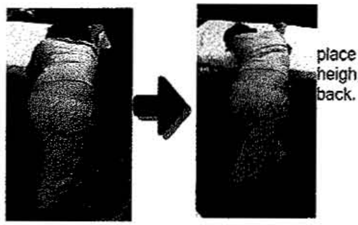


trunk rotations while seated on ball; repeat with touching the floor (can use a chair)



90/90 stretch
keep hip and knee at 90 degrees
lean forward, should stretch buttock

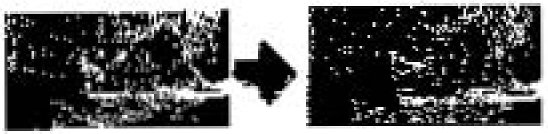
hamstring stretch
bend forward at the hip



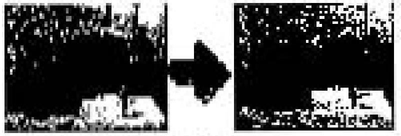
place hands on a surface which is hip heigh, hold on, lean backward and flatten back. Curve body to left and right.

Stretches must be done without feeling pain.
If there is pain with the stretch then leave it for a while.

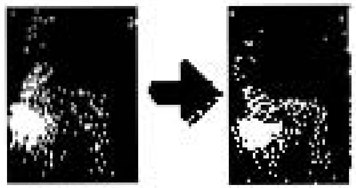
LOWER BACK EXERCISES



PELVIC TILTING
Hollow back and flatten back by rocking the pelvis. 2x 20
BACK ROTATION
Keep knees bent and drop them slowly to the right and then to the left. 10 reps on each side.



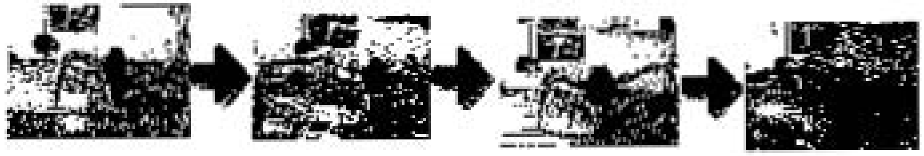
CAT STRETCH
Knees under hips, hands under shoulders. Arch and hollow your back by rocking your pelvis. 2x 20



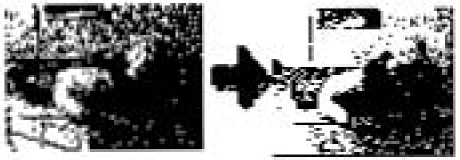
PELVIC TILTING (sitting on ball)
Hollow back and round back by rocking your pelvis. 2x 20

Advanced exercises

The following require you to activate your stabilisers. Do this by relaxing all the stomach muscles, tighten the very lowest part of the stomach by imagining you have to stop yourself from urinating. Hold the lower muscle tight while following the pictures



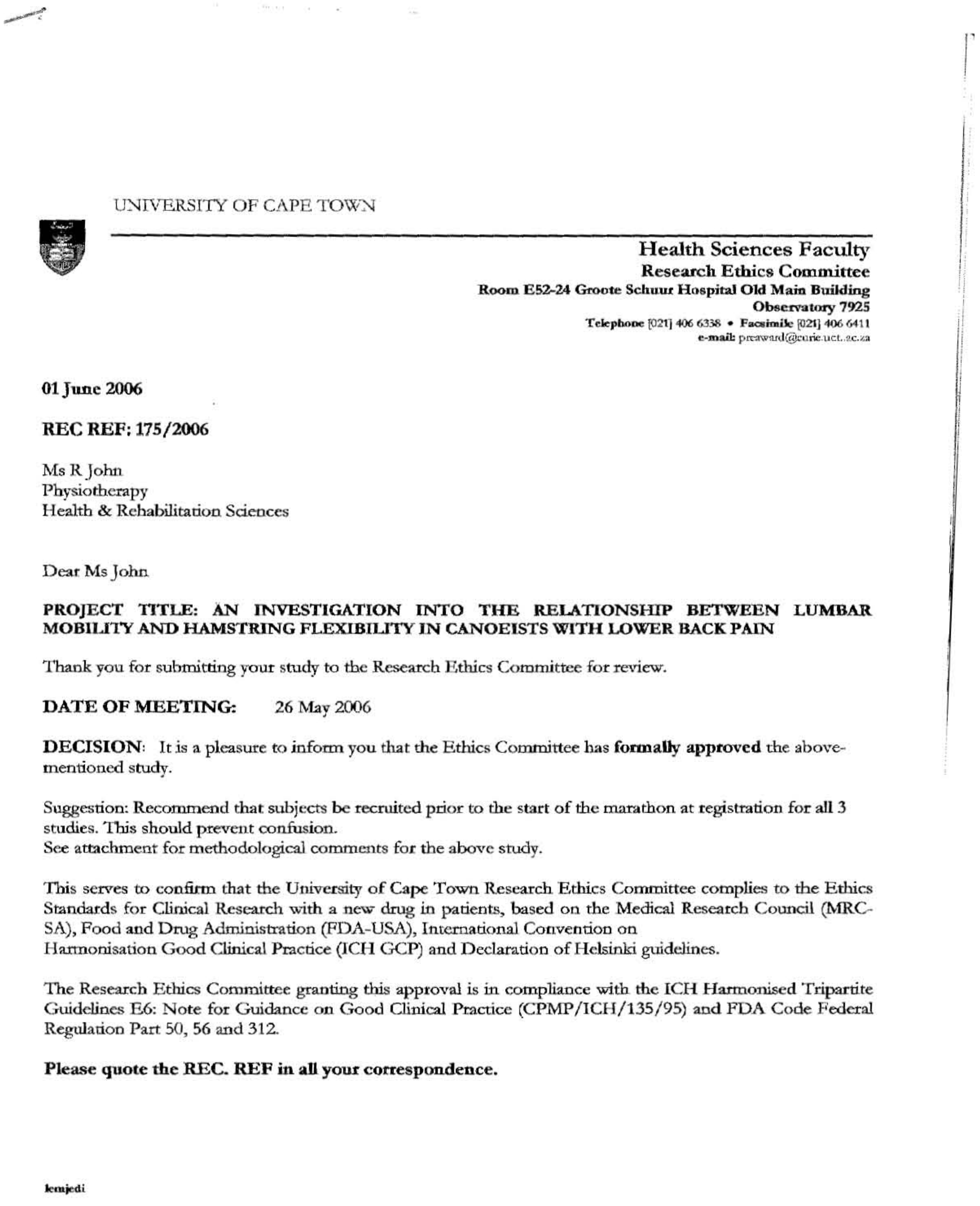
Only move onto the next picture when you are able to hold the position for 10x 30sec on each side while keeping the stabiliser tight.



Tighten lower stomach and squeeze buttocks while lifting. Hold 5secs x 12

Helyn John
[illegible] Physiotherapy [illegible]
[illegible]

APPENDIX VI – Ethical clearance



UNIVERSITY OF CAPE TOWN

Health Sciences Faculty
Research Ethics Committee
Room E52-24 Groote Schuur Hospital Old Main Building
Observatory 7925
Telephone [021] 406 6338 • **Facsimile** [021] 406 6411
e-mail: preaward@curie.uct.ac.za

01 June 2006

REC REF: 175/2006

Ms R John
Physiotherapy
Health & Rehabilitation Sciences

Dear Ms John

PROJECT TITLE: AN INVESTIGATION INTO THE RELATIONSHIP BETWEEN LUMBAR MOBILITY AND HAMSTRING FLEXIBILITY IN CANOEISTS WITH LOWER BACK PAIN

Thank you for submitting your study to the Research Ethics Committee for review.

DATE OF MEETING: 26 May 2006

DECISION: It is a pleasure to inform you that the Ethics Committee has **formally approved** the above-mentioned study.

Suggestion: Recommend that subjects be recruited prior to the start of the marathon at registration for all 3 studies. This should prevent confusion.
See attachment for methodological comments for the above study.

This serves to confirm that the University of Cape Town Research Ethics Committee complies to the Ethics Standards for Clinical Research with a new drug in patients, based on the Medical Research Council (MRC-SA), Food and Drug Administration (FDA-USA), International Convention on
Harmonisation Good Clinical Practice (ICH GCP) and Declaration of Helsinki guidelines.

The Research Ethics Committee granting this approval is in compliance with the ICH Harmonised Tripartite Guidelines E6: Note for Guidance on Good Clinical Practice (CPMP/ICH/135/95) and FDA Code Federal Regulation Part 50, 56 and 312.

Please quote the REC. REF in all your correspondence.

lcmjedi

Yours sincerely

DR. M. BLOCKMAN
CHAIRPERSON, FHS HUMAN ETHICS

hrayedi