Effects of Combined Motor Control and Isolated Lumbar Strengthening Exercises on Multifidus Muscle Morphology, Function, and Physical Disability in Chronic Low Back Pain

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Abstract

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Introduction
Low back pain (LBP) is the leading cause of disability and a major public health concern. Literature generally supports the effectiveness of exercise therapy for the treatment of chronic non-specific LBP, but there have only been modest effects. Considerable evidence suggests a link between lumbar multifidus degenerative changes and functional deficits. Recent preliminary evidence suggests that motor control and targeted lumbar extensor strength training may lead to hypertrophy and reduce fatty infiltration, however the optimal exercise intervention for reversing these changes is unknown.

Objectives
To study the effects of combined motor control and extensor strengthening on multifidus morphology and function, and to determine how physiological changes relate to pain, function, and psychological factors.

Participants
The study included 25 participants aged 18 to 65 with moderate-to-severe chronic non-specific LBP.

Intervention
Participants completed a 12-week supervised motor control and isolated lumbar extension intervention at a frequency of two times per week.

Measures
Primary: multifidus morphology
Secondary: multifidus function, lumbar extensor strength, pain, self-reported outcomes

Results
There were significant increases in the paraspinal CSA at L4-5 and L5-S1 between all timepoints and significant decreases in %fat fraction of multifidus and erector spinae at L5-S1 between baseline and both 6 and 12-weeks. There were significant increases in multifidus thickness and lumbar extensor strength between all timepoints.

Conclusion
Our results suggest that an intervention combining motor control and isolated lumbar extensor training has the potential to be successful in improving a large variety of outcomes, both physiological and clinical, in patients with chronic LBP.

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Introduction

Background

Low back pain (LBP) is a prevalent and persistent problem all over the world, with 60-90% of the North American population alone at risk of developing LBP in their lifetime.[1–3] This common musculoskeletal disorder is one of the leading causes of disability worldwide, costing billions of dollars in Canada alone each year, including health care costs and work absenteeism.[1,4] In 2015, it was estimated that about 540 million people were affected by LBP at any time, which is about 7.3% of the population worldwide.[4] A recent surge of research on LBP has expanded our knowledge and now includes psychological and social dimensions, as they interact with the standard biomedical model[5]. As the cause of LBP is usually multifactorial, it remains a challenge to treat as there is no single treatment that works for all patients. There have been many proposed treatments from exercise, manual treatments, to medication.[6] However, all reported and tested interventions have had minimal lasting long-term significant effects.[7] Furthermore, even those who recover from a first episode of LBP have a high chance of recurrence.[2] Given the lack of consistent results and complexity in the diagnostic evaluation and management of this musculoskeletal condition, identifying the most effective therapeutic approaches remains a primary research priority in order to improve not only short term, but also long term outcomes.

Definition of LBP

LBP is defined as pain, muscle stiffness, or tension between the 12 thoracic vertebrae and the gluteal folds.[4] It can be centralized or focused more on one side of the lower back. Individuals may or may not have concurrent leg pain in one or both lower limbs.[4] There is no single cause of LBP across all cases, rather there is a heterogenous pool of potential causes and risk factors that vary among individuals. Approximately only 10% of all LBP cases are caused by specific pathology, such as nerve root compression, fractures, or stenosis.[8] The other 90% of the cases have no identified cause (e.g. pain of unknown origin) and varying clinical manifestations, and are usually classified as non-specific LBP.

Many spinal structures could contribute to pain including joints, bones, ligaments, tendons, muscles, and nerves.[2,8] Repetitive mechanical stimulation of these structures can lead to continuous strain, degeneration, microtrauma, overload, or inflammation, any of which can lead to pain.[4,8,9] The acute stage of LBP is pain occurring for one month or less. Most cases of acute LBP will recover quickly and with minimal treatment.[6] Pain that lasts between two and three months is classified as sub-acute LBP. While pain lasting for longer than three months is labelled as chronic LBP.[2,10] Although approximately 10% of people with non-specific LBP typically develop chronic LBP, it remains a challenge to predict who will progress to the chronic stage.[4] However, age, gender, obesity, smoking, physical inactivity, occupation, genetics, certain medical conditions, and some psychological factors have been identified as possible risk factors for LBP.[9,10]

Risk factors

The main modifiable risk factors for LBP are obesity, smoking, physical inactivity, posture, and repetitive stresses such as a physically demanding job.[10,11] Obesity may increase the risk of LBP, especially large amounts of visceral fat, as it can increase the loads placed on the spine[11]. Furthermore, adipose tissue may secrete inflammatory mediators which can start a chronic cycle of pain.[11] Physical inactivity can lead to atrophy of key muscles that stabilize the spine, decreases in strength and endurance. This can allow excess movement of the spine and increase the risk of tissue injury.[12,13] Sedentary activity, especially prolonged sitting can lead to changes in lumbar lordosis, increased erector spinae activity, increased pressure on the disks and other structures, all of which can be a source of pain.[10] Conversely, individuals with physically exerting jobs, such as those requiring repetitive heavy lifting, repetitive bending, or twisting in unusual positions, have a higher risk of developing LBP.[2,4] Psychological factors including depression, anxiety, kinesiophobia, and pain catastrophizing, have been linked as predictors of someone transitioning to chronic low back pain[14]. Anxiety and depression have been linked with increased pain intensity, increased disability, poorer responses to exercise, and increased recurrence rates of injury or pain. Those with high kinesiophobia and catastrophizing react by avoiding activities that they are afraid will make their condition worse[5]. Some or all these factors combined can lead to poor responses to acute back pain, leading to avoidance of activity and increased pain intensity and duration. Many of these factors occur simultaneously and their effects on LBP is cumulative.

The main non-modifiable risk factors for LBP are age, gender, genetics, and certain medical conditions. Several studies have linked increasing age to the development of LBP.[2,4,7] With aging, muscles lose elasticity, bones start to weaken and intervertebral discs degenerate.[2] The degree to which this natural aging process occur varies between individuals, either due to genetics, lifestyle or a combination of both. Genetics can account for 21% to 67% of the potential to develop LBP.[4] Females seem to have a higher rate of LBP than males. This could be due to hormones, the stress from pregnancy and childbirth, or differences in pelvic structure.[15] Other chronic conditions may also increase the risk of developing LBP, such as asthma, diabetes and chronic headaches.[4]

Anatomy of the Spine

Non-specific mechanical LBP may originate from the musculoskeletal system and research has shed light on a few important muscles and how they relate to the spine and LBP. The local muscles attach directly to the spine and their main function is to stabilize and provide fine control during movement. Conversely, global muscles are larger muscles attaching to the pelvis and ribs, spine, and fascia. Supported by the local muscles, the large global muscles are mainly responsible for trunk movements, such as walking or bending, as well as postural control.

The erector spinae muscle, which is composed of three intermediate layers of muscle (e.g. illiocostalis, longissimus and spinalis muscles), is a large global muscle that mainly assist with general movement of the trunk. These muscles extend the full length of the spine and generate most of the force during lumbar extension.[16] On the other hand, the most important local and lumbar stabilizer muscle is the multifidus. Each fascicle of the multifidus muscle spans between two to five spinal levels and has a large cross-sectional area, making it ideal for segmental stability due to its ability to generate large forces over a small area.[13] Studies have

shown that the multifidus muscle also consist of a sensory component that detects perturbations of the spine.[16,17] Stretching of the tendons and ligaments of the spine cause reflexive contraction of the multifidus in order to add precision and stability during movements.[17] Working in conjunction with the multifidus to increase spinal stability, is the transverse abdominus and the pelvic floor muscles. The transverse abdominis is attached to the spine indirectly through the thoraco-dorsal fascia. When contracted, tension increases throughout the trunk and reduces the risk of spinal buckling, excess rotation and translation of the vertebra. Furthermore, when the transverse abdominis is contracting simultaneously with the pelvic floor and diaphragm, intra-abdominal pressure increases and creates a force into the direction of lumbar extension.[16,18] Finally, the pelvic floor muscles stiffen the sacroiliac joint during contraction, which further stabilizes the spine and pelvis.[19] As all these muscles are interrelated, dysfunction or injury to one can lead to issues with the other muscles and increase one's susceptibility to LBP.[18]

Imaging Techniques

Magnetic resonance imaging

Over the past decades, imaging studies examining the morphology and composition of the paraspinal muscles and its relationship with LBP have received increased attention. While both magnetic resonance imagining (MRI) and CT scan can be used to assess paraspinal muscle morphometry, MRI is the goal standard as it provides higher resolution images, and thus better detection of soft tissue such as muscle and fat. Furthermore, MRI is also preferred as it does not expose the patient to radiation. Previous studies have shown that MRI provides reliable measurements of muscle cross-sectional area (CSA), with intra-rater and inter-rater intra-correlation coefficient (ICCs) varying between 0.89 to 0.99, and 0.88 to 0.99 for the multifidus muscle, respectively.[20–22] Fortin and Battié[20] also reported excellent intra-rater reliability (ICC from 0.87 to 0.99) and agreement (ICC from 0.81 to 0.99) between two commonly used software program (OsiriX and ImageJ) for the assessment of paraspinal muscle CSA and composition. Figure 1A shows CSA measurements of the multifidus, erector spinae, psoas and quadratus lumborum from axial T2-weighted MR image.

Although a few studies have looked at the relationship between paraspinal muscle CSA (e.g. size) and LBP[23,24], atrophy of the paraspinal muscle (e.g., decrease in muscle quality) can occur without an actual decrease in total muscle CSA.[21] As such, the focus of research has shifted to not only consider the size or CSA of the muscle, but also the quantity of lean muscle tissue, defined as functional cross-sectional area (FCSA) (Figure 1D).[20–22] Indeed, chronic LBP has been associated with increased intramuscular fatty infiltration, where lean muscle tissue is replaced by fat and fibrous tissue.[22] This is an important distinction as muscle quality may affect functional contractile ability.[21,22,25] While several qualitative[26,27] (e.g., visually grading the degree of fatty infiltration) and quantitative MRI techniques have been used to assess paraspinal muscle composition, threshold techniques are one of the most widely used approaches. Specifically, a thresholding technique consists of identifying the range of pixels that represent lean muscle (low intensity) tissue and fat tissue (high intensity) on T1 or T2-weighted images. The threshold is then applied to the image to reflect either fat or muscle fibers within the muscle, after the muscle CSA is traced. Several studies have examined the reliability of measuring FCSA with MRI, with intra-rater and inter-rater intra-correlation coefficient (ICCs) varying between 0.77 to 0.98, and 0.68 to 0.99 for the multifidus muscle, respectively.[20–22,28]

While this thresholding method is highly reliable, it is very time consuming, and not always feasible in clinical settings. In a recent study, Fortin et al[28] developed a novel semi-automated thresholding algorithm for the assessment of paraspinal muscle composition. The algorithm was validated against the standard manual thresholding technique; the agreement between the two methods was excellent with inter-method reliability ICCs varying between 0.79 and 0.99, and intra-rater reliability coefficient varying between 0.95 to 0.99. The development of such algorithm greatly simplifies the tenuous aspects of MR imaging assessment of paraspinal muscle composition and facilitate comparison among studies. Indeed, in a recent review by Crawford et al[29] the variations in measurement techniques used by different group worldwide to assess paraspinal muscle composition was highlighted; such variability likely contributes to some of the divergence in study results. As such, the use and development of automated and standardized assessment tool is warranted to allow for more accurate comparison of study findings.[29]

Lastly, with advances in MRI technology, water and fat images and % fat-signal fraction derived from multi-echo acquisitions (e.g., DIXON, IDEAL) are now the contemporary standard for the assessment of soft tissue composition (Figure 1B and 1C).[30–32] However, water and fat sequences are rarely obtained clinically, consequently, routine T1- and T2-weighted images remain widely used and represent a valuable resource for researchers.[28,33]

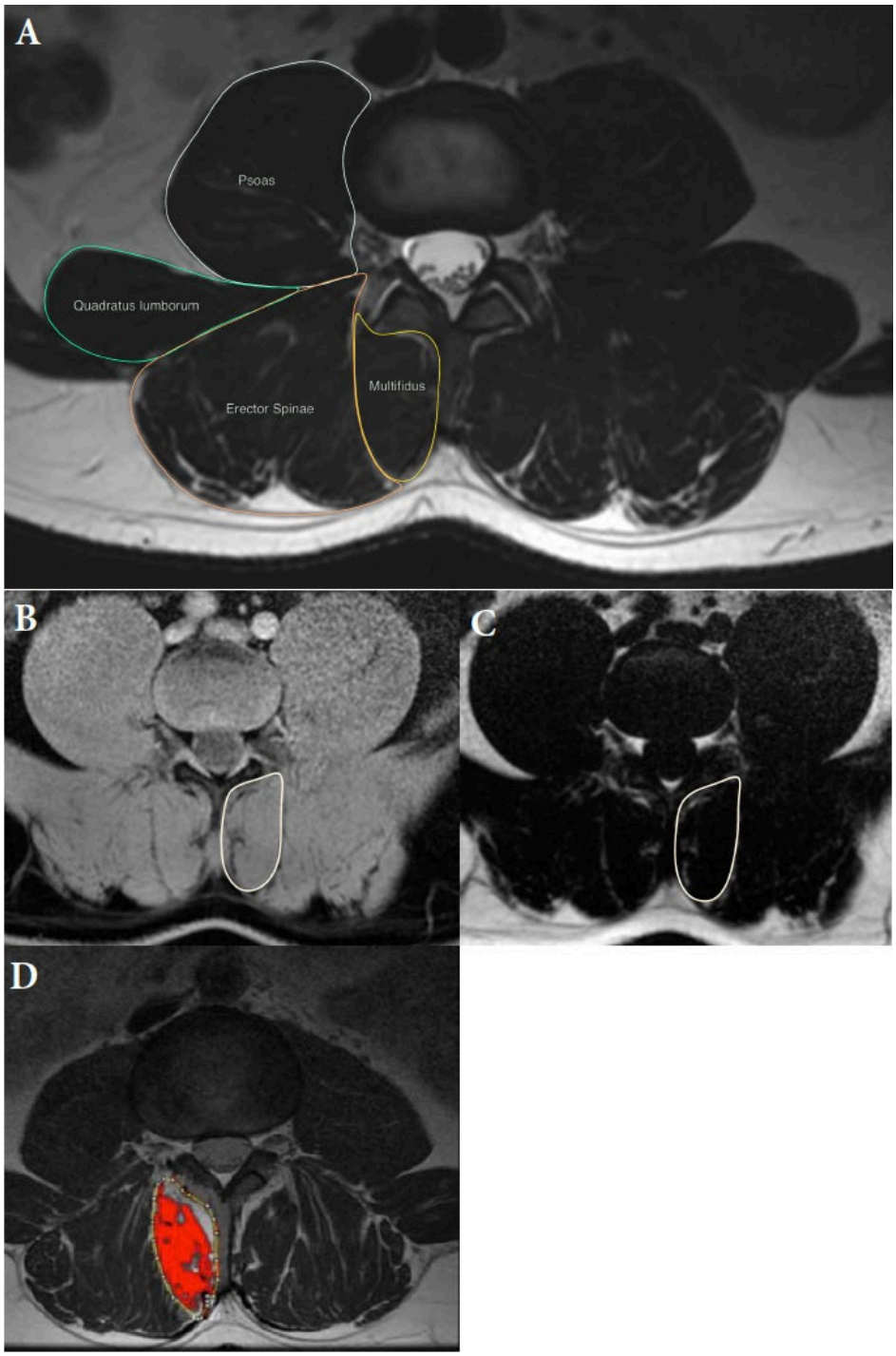

Figure 1: A) Axial T2-weigthed image and cross-sectional area (CSA) measurements of the multifidus, psoas, quadratus lumborum, erector spinae muscles. B) IDEAL water axial image and Region of Interest (ROI) of the multifidus CSA belonging to the lean muscle water index. C) IDEAL fat axial image and ROI of the multifidus CSA belonging to the fat index. D) Illustration of the thresholding technique used to quantify the multifidus muscle area of lean muscle mass.

Ultrasound

Studies have also used ultrasound to examine the morphology of the paraspinal muscles, specifically the lumbar multifidus. Benefits to using ultrasound compared to MRI include affordability, portability, and the fact that images can be taken real-time in different positions. While recent advancement in ultrasound technologies greatly improved the image quality, this modality does not allow for the clear distinction of fat and muscle tissue.[22] The reliability of measuring multifidus CSA with ultrasound techniques is also lower as compared to MRI studies. Belavy et al[25] found an average ICC for all lumbar spinal levels to be between 0.61 and 0.89. Kiesel[34] suggested that repeating the measure three times and averaging the values will increase reliability. Another concern with ultrasound imaging is with inter rater reliability when multiple technicians take the measurements. Results from ultrasound imaging are somewhat dependant on the machine used and the location of probe placement. Therefore, it is ideal that the same person takes all the measurements that will be compared. Belavy et al[25] also examined the correlation between MRI and ultrasound measurements of multifidus CSA for all lumbar spine levels and had a Pearson Correlation coefficient of 0.52 (95% CI of 0.14 to 0.77), indicating low to moderate correlation.

Ultrasound has also been used to measure muscle function. By comparing muscle thickness at rest and during contraction, one can calculate the percent thickness change as an indicator of muscle function.[35] A review from Taghipour et al[36] examined the reliability of using ultrasound to measure thickness change in the lumbar multifidus and found that high quality studies generally had ICCs above 0.70, which is an acceptable level. Variation between studies could be due to transducer placement, muscle state, amount of pressure, and patient position.[36] While fewer studies have examined the reliability of multifidus thickness during contraction, those that have measure reported good results.[34,37] A study by Kiesel et al[34] compared a standardized protocol using ultrasound imaging with the gold standard measurement of intramuscular EMG. They found there was a high correlation ($r = 0.79$, $P < 0.001$) between the EMG measurements and the change in multifidus thickness during contraction. Another study by Sions et al[37] compared the reliability of ultrasound measurements of multifidus thickness between older adults and younger adults. Measurements from younger adults had ICCs of 0.80 to 0.95, both during resting state as well as during contraction using a contralateral limb lift. In the older adult group, they found similar reliability with ICCs of 0.74 to 0.94 during rest and 0.79 to 0.95 during contraction using a contralateral limb lift. This indicates that this method can be used reliably with all ages, which is important as the prevalence of LBP increases with age. Other benefits of ultrasound include the ability to test in different postures and using the real-time images as biofeedback during rehabilitation. Taken together, these results demonstrate that ultrasound is a valid, low-risk, and non-invasive alternative to measure function of the lumbar multifidus. Figure 2 shows the multifidus muscle thickness measurements at rest and during contraction.

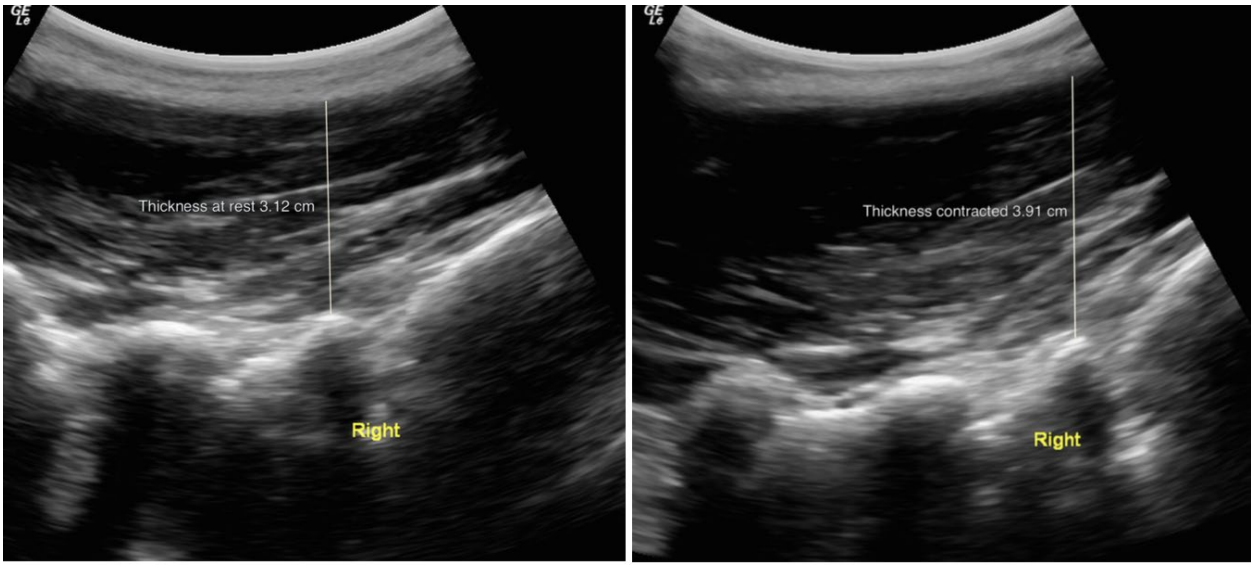

Figure 2. Multifidus muscle thickness measurement at rest (left image) and during submaximal contraction via a contralateral arm lift (right image) at L5-S1 level.

Muscular Adaptations

Structural Changes

Imaging studies have shown that individuals with LBP are more likely to have paraspinal muscle atrophy, fatty infiltration, and asymmetries, especially of the multifidus muscle.[24,38,39] A recent systematic review by Goubert and Daneels[40] summarized the current literature on imaging and histological studies that have examined structural changes of the lumbar muscles in people with non-specific LBP and healthy controls. There was moderate evidence to support a decrease in multifidus CSA in individuals with LBP, at one or more lumbar levels. Of the six studies showing multifidus atrophy, only two showed atrophy above L3, while all studies reported atrophy at L4 and L5. In comparison, individuals with acute or recurring LBP showed no consistent evidence of multifidus atrophy (e.g., reduced CSA). They found less conclusive evidence with regards to muscle side-to-side asymmetries. Two studies found asymmetry of the multifidus muscle at the lower spinal levels, while one study found no apparent differences. Hides et al. reported that individuals with unilateral LBP were more likely to develop asymmetries on the symptomatic side, compared to those with bilateral LBP. Lastly, two studies found the presence of fatty infiltration of the multifidus in individuals with chronic LBP.[40] Furthermore, some noted that not all cases of fatty infiltration were associated with a decrease in CSA.[21,22,40] It is intuitive that such increase in fatty infiltration negatively affect muscle function and contractile ability.[22]

Functional Changes

Along with the morphological changes, decreases in muscular endurance, strength and activation/contraction pattern have also been observed.[41,42] As the multifidus is one of the most important stabilizers of the spine,[13] functional impairments can lead to an increased risk of injury.[13] Atrophy and fatty infiltration of the multifidus muscles likely compromise its contractile ability, by limiting the amount of force that can be generated during contraction.[22,43] A study by

Van Dieen et al[41] assessed multifidus contraction between individuals with LBP and healthy controls. They found lower multifidus activation in patients with LBP, suggesting a decreased in muscle function. A recent systematic review by Prins et al[44] also reported moderate evidence that individuals with LBP demonstrate larger response times to perturbations of the spine, although this was not found in all studies. It is theorized that these changes occur due to splinting behaviour to protect the spine, however research remains inconclusive.

Causes of Adaptations

Possible mechanisms that may explain the degenerative muscle changes and decreased function observed in patients with LBP include poor motor control, deconditioning, denervation, and reflex inhibition.[45] Loss of motor coordination can lead to repetitive mechanical stress on surrounding structures, increasing the risk of instability, joint overloading, and pain.[46,47] Many individuals with LBP are more likely to avoid painful movements, which can lead to deconditioning of the lumbar muscles due to disuse.[43] Furthermore, changes in muscle activation can further exacerbate the deconditioning of these muscles.[12,41,43] Some studies have proposed that denervation of the multifidus muscles could be a potential cause of multifidus atrophy and degeneration,[17,43] although more commonly seen in individuals with disk herniations or stenosis. Proposed mechanisms are chemical irritation from inflammatory mediators[17] and stretching or entrapment of nerves during excessive or poor movements.[17,43] Lastly, there is support for reflex inhibition of the multifidus with LBP.[40,43] Nociceptive input from the various underlying spinal tissues as the multifidus shares an innervation with associated spinal segments.[13,41] This can lead to delayed and diminished activity of the muscles.[40] It is unclear whether these changes are cause or symptoms, or a combination of both in people with LBP. Regardless, they are key issues that need to be addressed as part of the treatment.

Exercise Interventions

Effects of different exercise interventions on LBP

Currently, exercise therapy is the most common conservative treatment for LBP as it is easily accessible and can be individually tailored to patients' needs.[38] A common finding that is shared among most exercise interventions is a statistically significant decrease in pain and increase in function compared to control groups with no exercise intervention component.[3] Hayden et al. found that core stabilization and functional restoration exercises such as motor control were more successful at reducing pain and disability.[48] A systematic review by Gordon and Bloxham[49] examined the effectiveness of various types of exercise such as aerobic training, core stabilization, and muscular strengthening programs for the treatment of non-specific chronic LBP. Included in the review. All three types of training had mixed results with some studies finding significant improvements in pain and disability post-treatment while other studies reported no effect. Overall, their conclusions were that since LBP can have a multitude of causes and risk factors, no single treatment will work for all LBP patients. Instead, we need to individualize treatments based on the patients' needs. Similarly, in another systematic review, Searle et al[50] found that exercise interventions were more effective than conservative therapies in treating LBP, although the effect size between different exercise intervention groups were small. Interestingly, different interventions had benefits in varying outcomes. For example, resistance training had better functional outcomes while flexibility was more likely to lead to improvements in pain.[50] In accordance with Gordon and Bloxham[49], Searle et al[50] concluded that additional

research is warranted to determine whether certain groups of LBP patients would benefit more from one intervention than others.

Effect of exercise interventions on multifidus muscle morphology

There is mixed evidence as to whether current exercise interventions can lead to significant improvement in paraspinal muscle morphology (e.g., hypertrophy, reversal of fatty infiltration) and function (e.g., strength, activation, contraction), and whether such changes are associated with improvement in patient-related outcomes. A recent study examined the effect of a high-intensity isolated lumbar extensor exercise protocol and its effect on cross-sectional area and fatty infiltration for both the erector spinae and multifidus muscles.[51] The study found variability in morphological changes, with some individuals experiencing increases in CSA and decreases in fatty infiltration, while others had no change. The authors suggest further exploration into identifying what types of resistance exercise works best.[51] A review by Shahtahmassebi[52] examined various exercise protocols and their effects on paraspinal muscle morphology. They found mixed results in studies using motor control interventions. Generally, groups that completed motor control exercise alone had little to no improvements in muscle morphology, while groups that combined motor control and resistance training interventions saw greater improvements in multifidus CSA. Furthermore, when comparing machine-based and non-machine-based resistance programs, there were mixed results across all categories. Conversely, no studies of cardiovascular exercise indicated any changes in paraspinal muscle morphology. However, the exercise intervention studies included in this review were generally of low quality, with high risk of bias and were severely critiqued for their lack of transparency regarding the exercise's protocols, not providing enough detail to allow for replication or accurate comparisons among studies.

Only a few studies have examined the effects of strength-based exercise on paraspinal fatty infiltration in people with chronic LBP.[51,53] Welch et al[53] found that, along with decreases in pain and disability, a 16-week full-body general resistance training protocol decreased fatty infiltration and increased FCSA of the multifidus. Currently there is a lack of comprehensive studies that have thoroughly examined the effect of different exercise interventions on overall paraspinal muscle health (e.g., morphology, composition, and function), and how such variations may correlate with improvement in pain and disability. In a preliminary study by Berry et al,[51] while no significant differences in multifidus and erector spinae muscle size and fatty infiltration were detected following a 10-week high-intensity machine-based resistance exercise program (n=14), improvement in muscle size and fatty infiltration of both muscles were correlated with improvements in disability, strength, and depression/anxiety. Unarguably, further studies are needed to provide a better understanding of the relationships between exercise, muscle morphology, and functional outcomes in patients with chronic LBP.

Motor Control

Background

In recent years specific motor control interventions have been examined to see if there is a better way to target paraspinal muscles directly. Motor control consists of the neural mechanisms that regulate posture, movement, and spinal stability. Receptors in the muscles, tendons, and ligaments relay information to the nervous system, which signals the muscles to contract or relax in order to achieve optimal functioning.[54] Motor control exercises are used to

retrain control of trunk muscles activation, alignment, and movement in order to restore proper function.[55] It is believed that patients lose proper motor control of the core stability muscles due to altering their posture or limiting certain movements in order to avoid painful positions.[47] This can lead to deconditioning of core muscles, overuse of certain muscles and structures, or a combination of both.[47] Over time the brain and body adapt to these maladaptive processes, and this harmful cycle will continue until muscles are properly retrained. Motor control training seeks to correct the order of activation and endurance of the key spinal stabilizers, specifically the lumbar multifidus and transverse abdominus muscles, and involves motor, sensory, and central processing.[38]

Patient Classification

Two studies by Van Dieen et al[9,12] have described a range of motor control dysfunctions, where both ends of the spectrum could be causes for LBP. One end of the spectrum, labelled tight control, presents as splinting or bracing to decrease the risk of excess spinal movement. Increased activity of the large trunk muscles (erector spinae) occurs, and movement becomes slower and more rigid. Although seemingly beneficial in the short-term, chronic overactivation of the erector spinae can lead to inhibition of the multifidus and increases the forces on the spine. This leaves the individual more susceptible to injury through repetitive malalignment and stress on the spine and surrounding structures.[9,12]

On the opposite end of the spectrum, loose control seeks to mitigate the forces and tissue loading of the spine due to muscle forces by decreasing muscular activity. However, this leads to less control over spinal stability, leaving the spine vulnerable to larger movement and displacement of the spine during perturbations.[12] This reduced control leads to higher risk of tissue injury and other adaptations in attempts to stabilize the spine.[9,12]

There is currently no consensus on the best way to classify patients based on motor control.[9] A few classification systems have been used, although not originally created for motor control classifications, they share many similarities to the model of Van Dieen et al[9]. The treatment-based classification by Alrwaily et al[56] split patients into one of four groups based on which type of treatment they were most likely to respond to. The manipulation category, where the main clinical finding is hypomobility, correlates with tight-control, whereas the stabilization category correlates with the loose-control model. Another classification system is the multidimensional clinical model from O'Sullivan et al[57] that separate people with LBP into adaptive/protective or maladaptive/provocative groupings. The former, also called movement impaired, correlates with tight-control, while the later correlates with loose-control. While a few studies have examined the effectiveness of matching subgroupings with targeted treatments compared to a generalized treatment, results remain inconclusive.[8,58] Van Dieen et al[9] hypothesized that this may be because the categories are not mutually exclusive. Rather, individuals might change their motor control patterns depending on the situation. Further research needed to increase our knowledge on developing screening techniques to identify which treatment each individual will respond to best.

Motor control interventions

Many studies have looked at the effectiveness of specific motor control treatments compared to traditional muscle strengthening. However, there are mixed results as to which

intervention would be more beneficial to individuals with chronic LBP.[52] A study by Shamsi et al[59] compared the effectiveness of motor control exercises with general strengthening exercises on disability and pain. Both groups completed 16 sessions over 6 weeks with measures being taken before and after the interventions. The motor control group had 3 phases; the first was isometric activation of the local trunk stabilizers, second was continuing these isometric contractions with low loads from limb movement, and the third and final phase was achieving these contractions while performing functional tasks. They found that both groups had statistically significant decreases in pain and disability as measured by the Oswestry Disability Index (ODI). Similarly, Macedo et al[55] also compared a motor control protocol with a graded exercise program over 8 weeks, with follow up at 6 and 12 months after completion. While the motor control group focused on regaining control and coordination of spinal muscles and the graded exercise group emphasized on increasing activity tolerance, both groups had similar reductions in pain and improvements in disability in function following the completion of the exercise program. These findings suggest that some people may benefit more from one type of intervention than others. It appears that an individual is more likely to respond positively to motor control training if they have poor control of their multifidus muscles.[38,55]

A comprehensive Cochrane review by Saragiotto et al[60] examined 16 studies that compared motor control exercises to other forms of exercise therapy for the treatment of chronic non-specific LBP. They found that across the studies there were minimal differences between motor control exercises and other exercise interventions in pain and disability outcomes. However, when compared to other interventions that do not include exercise, such as standard care or the use of electrophysical agents, there are clinically significant reductions in pain and disability with motor control exercises. Ultimately, in all comparisons from the review, the effect sizes were small, indicating that some people in all categories showed improvements while others did not. In contrast, a network meta-analysis by Hayden et al. found that core stability and motor control type exercises may be better for improving pain and disability than other types of exercise. However, they also agreed that any type of exercise had better outcomes that traditional conservative treatments[48].

Lumbar Strengthening

Isolated Strengthening

A large body of evidence has confirmed the presence of lumbar multifidus degenerative changes (e.g., atrophy and fatty infiltration), particularly at the levels of L4 and L5,[13] in individuals with chronic LBP.[40] These deficits have also been correlated with increased pain and disability as well as decreased function.[61] Since the primary function of the multifidus is spinal stability, it is very important they are functioning properly. Based on current evidence, motor control interventions alone do not appear to provide enough overload to stimulate muscular hypertrophy.[52] However, when paired with lumbar extensor training, positive changes in paraspinal morphology were observed.[52] In order to be effective, the strength training needs to directly target the lumbar extensors. Shahtahmassebi et al[62] found that healthy individuals who completed lumbar extensor training on an isokinetic dynamometer had greater increases in multifidus CSA and thickness at L4/L5 and L5/S1 than individuals who completed general exercise training, with a concomitant increase in strength and functional ability.

Traditional trunk extension exercises, labelled TEX by Steele et al[63], were the early focus of lumbar muscular strengthening studies. Included in this category are exercises where the primary movement is trunk extension, such as roman chair extensions, without external fixation. Although studies have found improvements in strength or endurance for that specific movement, there has been no convincing evidence that TEX training causes any morphological changes of the multifidus.[61] It has since been hypothesized that the larger hip and thigh muscles may be overcompensating for weak lumbar extensors. The extensor muscles, specifically the erector spinae and multifidus, only account for approximately 72 degrees of extension. The remainder comes from the glutes and hamstring muscles, pulling the pelvis into posterior rotation.[64] A study by Smith et al[65] looked at the effect of pelvic stabilization during lumbar extension strengthening in patients with LBP. They found greater increases in strength and function and decreases in pain when completing the exercises with pelvic restraints compared to individuals completing the same exercises without pelvic restraints. The addition of pelvic restraints eliminate the activity/activation of the other large muscles of the posterior chain such as the glutes and hamstrings, which tend to provide most of the force in lumbar extension.[64] Therefore, the addition of the pelvic restraints isolates the lumbar extensors, can likely provide enough overload to lead to physiological adaptations such as muscle hypertrophy and increase muscular strength. Exercises that use these restraints have been defined as isolated lumbar extension (ILEX) by Steele et al[63]. As such, the lack of morphological changes in paraspinal muscles reported in previous studies may be due to the compensation of the other posterior chain muscles.[65]

MedX Isokinetic Dynamometer

There are currently only a few isokinetic dynamometers that allow to isolate the lumbar extensor muscles. The MedX lumbar extension machine has been highlighted in many recent studies as a method to test and train lumbar extension strength in a safe and standardized manner.[51,61,65,66] The machine allows for the full 72 degrees of lumbar extension that has been attributed to the lumbar extension muscles and can be split up into increments as small as 3 degrees. A preliminary study by Pollock et al[66] found that compared to a control group, healthy individuals who completed a 10-week training program on the MedX machine had significant increases of lumbar extensions strength. A study by Smith et al[65] found that completing a 12-week training program with pelvic stabilization on the MedX had significant increases in lumbar strength compared to individuals completing the same program without pelvic stabilization. These findings further support the theory that large muscles of the pelvis need to be eliminated in order to achieve increases in lumbar extensor strength, and that the MedX machine is able to successfully isolate the lumbar extensors.

Training Parameters

Several other studies have been conducted to compare key aspects of exercise training programs, including frequency, intensity, and range of motion parameters. Patients with chronic LBP are very likely to have limited range of motion and decreased strength, but most importantly fear that certain exercises or positions may worsen their condition. Therefore, it is important to find the balance between patient comfort and peace of mind while simultaneously stressing the muscles enough to see beneficial changes. Steele et al[67] compared the effects of partial versus full range of motion on lumbar extensor strength using a MedX machine. One group trained with the full lumbar range while the other completed the same protocol with 50% of their full range. They found that both groups showed substantial increases in lumbar extensor strength after a 12-

week intervention. This indicates that even individuals with limited range of motion can still benefit from this type of training. Ultimately the goal is to regain full range of motion but gains in strength are not limited to only those with full range. Another important factor to consider is the intensity of the training. Berglund et al[39] compared the effects of low load body weight and high load free-weight strength training. The focus on the low-load group was to improve coordination and activation of the spinal stabilizers, while the high-load group sought to increase strength and hypertrophy of the lumbar extensors. Both groups showed hypertrophy of the multifidus muscles, suggesting that individuals who start with lower strength level can begin exercising at a lower intensity and still see improvements.

Psychological Factors

Kinesiophobia and Catastrophizing

Given the fact that many exercises and therapeutic interventional studies reported minimal to no improvement in patients with chronic LBP regardless of what treatment they received, researchers began to look for other factors that may influence recovery. Based on the Fear Avoidance Model of Pain, it is hypothesized that individuals can be classified as either active copers or passive copers.[68] The model deviates from the standard biomedical models previously used with injury rehabilitation. Instead, it also includes psychological and social constructs relating to how an individual may recover from injury. It states that individuals with negative views towards pain are more likely to suffer from increased pain and are less likely to recover. These negative views are labelled as kinesiophobia and pain catastrophizing. Kinesiophobia is the fear of movement due to the belief that movement will increase their pain and may make their injury worse.[69,70] Catastrophizing is the tendency for an individual to ruminate on their pain and experience it in more exaggerated terms than normal.[69] Those with high kinesiophobia and catastrophizing are described as passive copers, as they react by avoiding activities that they are afraid will make their condition worse. This can lead to activity limitations, deconditioning, and increased disability, which in turn leads to increased negative views and continues as unending cycle. In contrast, active copers are those who remain physically active, do not avoid the activities they enjoy and have higher self-reported function as well as decreased pain and disability.[71]

Exercise has been shown to improve these negative feelings. A study by Ferrari et al[72] found that individuals with chronic LBP and higher level of kinesiophobia, measured by the Tampa Scale of Kinesiophobia (TSK), were more likely to have a higher pain intensity and disability. Whereas individuals with higher self-efficacy ratings had lower pain intensities and disability.

Depression and Anxiety

In addition to the physical effects of LBP, individuals afflicted are 3-4 times more likely to experience clinical depressive symptoms compared to the general population.[73] A study by Oliviera et al[14] found that 51.4% of their chronic LBP participants had significant depression and anxiety symptoms. Furthermore, anxiety and depression have been linked with increased pain intensity, increased disability, poorer responses to LBP interventions, and higher chances of recurrence.[14] Marshall et al[74] found that individuals with LBP who had more significant fear avoidance belief and depression reported greater disability relative to their pain levels. However, this relationship has been shown to be mitigated by regular bouts of physical activity.[74] There are

a lack of studies comparing the effect of resistance training on lumbar muscle morphology while also considering possible mediators of treatment effect such as anxiety and depression. To the best of our knowledge, the only study that has evaluated this relationship was a recent preliminary investigation by Berry et al[51]. Indeed, individuals with lower levels of depression and anxiety were found to have significant improvements in erector spinae CSA and fatty infiltration. However, this relationship was not seen for the multifidus muscle. There is an urgent need to further explore the impact of psychological factors on pain, disability, and muscle morphology as well as the effect of exercise interventions on these parameters.

Sleep

LBP also has adverse effects on sleep quality, although there is limited data currently available. Sezgin et al[75] found that patients living with LBP scored significantly higher in the Pittsburgh Sleep Quality Index (PSQI) as compared to healthy controls indicating a higher level of sleep disturbance. They also found a positive relationship with sleep disturbance, LBP intensity, and reported disability. This is also an important area for future research.

Rationale

Chronic, non-specific LBP is a highly prevalent and disabling condition. Firstly, while the literature generally supports the fact that exercise therapy has a positive effect on pain and disability, the effect size between different exercise interventions remained very small. A meta-analysis by Hayden et al found core and stabilization exercises may be more effective than other exercise interventions for improving pain and disability in LBP patients, although when adjusted for dose and co-interventions, some interventions were no longer more successful.[48] Furthermore, while paraspinal muscle morphology (e.g., atrophy and fatty infiltration) and functional (strength, activation/contraction) deficits have been linked to LBP, there are mixed results as to what type of exercise is effective to elicit positive morphological and physiological adaptations. Although promising preliminary findings suggest that a combination of motor control training and lumbar strengthening may improve quality and function of the multifidus muscle,[52] additional high-quality research is needed. It remains also unclear whether such physiological muscular changes and adaptations in muscle morphology and function, or both, are associated with improvements in clinical symptoms and functional outcomes.

Secondly, it is recognized that psychological factors such as kinesiophobia, catastrophizing, anxiety, depression, and sleep disturbances can play a negative role on patients' level of pain and related LBP- disability. However, very few studies have specifically examined how these factors may mediate changes in paraspinal muscle health and qualitative pain/functional outcomes in the context of exercise therapy interventions. Identifying such barriers to physical activity and improvement in function and disability is critical to mitigate the persistence of chronic LBP and warrants further attention.

Objectives

1) The primary objective of this study is to examine the effects of combined motor control and strengthening exercises (targeted exercise intervention) on i) paraspinal muscle morphology (e.g., size, fatty infiltration) and ii) paraspinal muscle function (e.g., % thickness change during contraction, strength) patient with non-specific chronic LBP.

2) Our second objective is to examine whether paraspinal muscle physiological changes/adaptations following the intervention are associated with improvement in pain, disability/function, kinesiophobia, catastrophizing, anxiety, depression, and sleep.

Hypotheses

Primary hypothesis: We hypothesize that after the exercise intervention, patients will have a significant increase in paraspinal muscle size, decrease in fatty infiltration, with concomitant increase in muscle strength and contraction.

Secondary hypothesis: We also hypothesize that patients will show significant improvements in disability/function status, pain related fear (catastrophizing), kinesiophobia, depression, anxiety, and sleep outcomes.

Methods

Study Design

This study was a pre-test post-test study and was conducted at the PERFORM Centre, Concordia University. Though the focus of this study was on a combined motor control and isolated lumbar strengthening intervention, it is part of a larger randomized control trial (RCT) that compares the effects of this intervention with another group completing a general exercise program. The project was approved by the Research Ethical Committee of the Institution and the Central Ethics Research Committee of the Quebec Minister of Health and Social Services (# CCER-19-20-09).

Participants

A total of 25 participants were recruited for this project. This sample size was determined based on power calculation for the larger RCT (see sample size calculation below – p.24). The intervention occurred over 12 weeks with 2 supervised visits per week at the PERFORM Centre, Concordia University.

Table 1: Inclusion and exclusion criteria for participants.

Inclusion Criteria	Exclusion Criteria
· Chronic, non-specific LBP for a minimum of 3 months (with or without leg pain), · Between the ages of 18 and 60 years old · Speak either French or English · Are currently seeking care for LBP · Score either "moderate" or "severe" on the modified Oswestry Low Back Pain Questionnaire · Do not engage in any sport or fitness training specifically for the lower back muscles either currently or 3 months before the start of the trial	· Any evidence of nerve root compression or reflex motor sign deficits. · Previous spinal surgery or vertebral fractures. · Major lumbar spine structural abnormalities. · Health conditions that prevent the safe participation in physical exercise as determined by the Physical Activity Readiness Questionnaire

Recruitment

Participants were recruited from the local university community through advertising as well as from the Quebec LBP Consortium. Participants expressing interest in the study were contacted by the study coordinator to confirm interest and enroll them in the study. Inclusion and exclusion criteria for participants are outlined in Table 1.

Exercise Intervention

The degenerative changes seen in the muscles of patients with LBP can be caused by poor motor control, which can lead to repetitive mechanical stress on surrounding structures, increasing the risk of instability, joint overloading, and pain.[46] Therefore, the targeted intervention consisted of exercises that directly target the deep lumbar muscles, aiming to retrain proper motor control and coordination between the deep and global muscles, and to restore proper function and posture.[59] The intervention was split into two phases: activation of the deep spinal muscles and functional rehabilitation.

Phase 1:

The initial phase started with an assessment of posture, breathing patterns, pelvic control, and muscle activation. The posture evaluation consisted of a visual scan from all sides with the participants standing and looking at the spinal curvatures, muscular imbalances, the plumb line, and any other forms of compensations. The movement of the spine during flexion and extension was also examined by asking the patient to tuck in their chin and slowly roll their spine as they reach to touch the floor.

Participants were then guided through the process of finding their neutral pelvis and neutral spine. As most exercises will begin in a neutral pelvis position, it is important for the participants to find this position accurately so that muscles are in the optimal position to work.

Breathing was assessed supine to examine and teach proper techniques. The participants were given the following instructions: breathe from your abdomen instead of your chest, abdomen should rise before the chest, both sides should expand equally and symmetrically, there should be no or minimal activation of accessory muscles (sternocleidomastoid and scalenes). This breathing pattern was incorporated into the exercises of both phases of the intervention.

The motor control program was then guided by deficiencies found during the assessment. The primary focus was on activating or increasing the activity of multifidus and transverse abdominis muscles, both of which are important for proper function and dynamic stability of the spine. The goal was for the patients to be able to regain control and independent activation of the multifidus, transverse abdominis, pelvic floor muscles, and diaphragm (e.g., muscle commonly found to have a poor control) and reduce the activity of any global muscle identified to be overactive (e.g,. obliquus externus abdominis). This was completed in a variety of starting positions that progressed in complexity as the patient improved. Before progressing to stage two the patient must have been able to meet the following criteria: complete ~10 repetitions while holding for 10 seconds, achieving activation with minimal feedback or cues, while maintaining a normal breathing pattern throughout the exercises.

Table 2: Transverse Abdominus Training Cues

Transverse Abdominis Activation	
Positions	· Start supine or crook-lying · Find neutral pelvis · Place fingers slightly medial and inferior to ASIS
Cues	· Try to pull your belly button down to the table · Try to move your fingers together (medially)
Ideal Response	· Gradual increase in tension; 10-15% effort · Symmetrical contraction · No global muscle activation · Normal breathing · Able to hold 10 x 10seconds

Table 3: Multifidus Training Cues

Multifidus Activation	
Positions	· Prone or on hands and knees (some people are better in one position to start) · Fingers on either side of spinous process; evaluation of different spinal levels from T1/T2 to T5/S1
Cues	· Try to swell muscle up into my fingers · Think about tilting pelvis without actually moving it · Imagine tensing a cable from your pelvis up through your spine
Ideal Response	· Symmetrical contraction · No global muscle activation · Normal breathing · Able to hold 10 x 10seconds

Phase 2:

This phase began once participants were able to adequately activate the multifidus and transverse abdominus muscles while starting to add low loads to the muscles, first into static positions and eventually into dynamic positions. The goals of this phase were to control lumbar position during movement, coordinate between deep and superficial muscles, and to progress to automatic activation of the deep muscles. Exercise difficulty and progression was achieved by moving participants into a more challenging position (e.g., supine to sitting), increasing the load (movement of limbs), and introducing the need for dynamic stability (moving to unstable surface such as sitting on an exercise ball).

Isolated Lumbar Extension Exercise - ILEX:

All participants also completed lumbar extensor strength training running concurrently with the motor control exercises (MC+ILEX). This training was completed on the MedX machine at the PERFORM Centre. Their one repetition maximum (1RM) was recorded during the baseline testing. They performed 2 sets of 15 to 20 repetitions of lumbar extension at 55% of their 1RM as determined by the baseline testing. Progressions occurred once the patient was able to complete 20 repetitions without failure. For each progression, the load was then increased by 5%.[67] The unique pelvic stabilization system of the MedX lumbar extensor machine, shown in figure 1, allowed for isolated testing and strengthening of the lumbar extensor muscles through full range of motion in the flexion-extension plane of movement. Pelvic stabilization and lower body restraints eliminated the activation of synergistic and compensatory muscles, such as the glutes and hamstrings, allowing for true lumbar extensor strengthening.

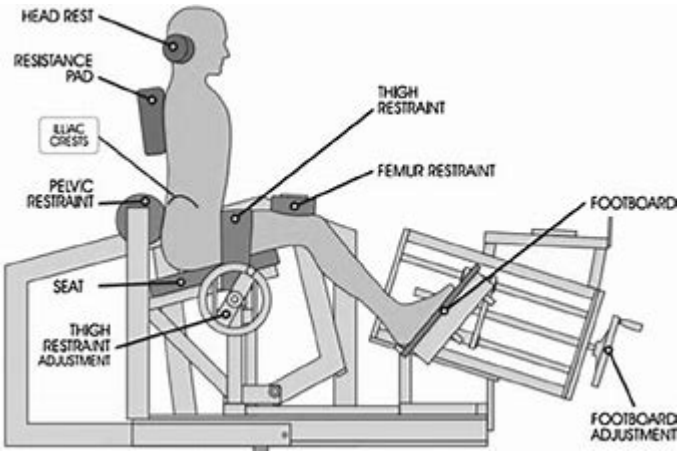

Figure 3: MedX Lumbar Extension machine

Primary outcome

The primary outcome measures were collected from all participants at baseline, 6 weeks, and at the conclusion of the study (12 weeks):

MRI assessment of paraspinal muscle morphology

The cross-sectional area (CSA) and fatty infiltration of the multifidus and erector spinae muscles from lumbar vertebrae of L4-L5 and L5-S1 levels was examined with an axial IDEAL fat-water (lava-flex, 2-echo) sequences obtained using a 3-Tesla GE MRI machine. The cross-sectional

area of the muscle was measured on 3 slices at each level: upper endplate, mid disk, and lower endplate. Once obtained, we calculated the left and right mean CSA of the paraspinal muscles.[20,28] Similarly, muscle composition was assessed on 3 slices for each level (e.g., L4-L5 and L5-S1) using IDEAL axial water and fat images. The percent-fat signal fraction of the muscles using the following equation: $\%FF=(Signal_{fat}/[Signal_{water}+Signal_{Fat}]x100)$ was calculated for each muscle at both spinal levels, and the mean value from the 3 analyzed slides per level was used in the analysis.

Secondary Outcomes

Ultrasound assessment of multifidus function

The multifidus muscle thickness at rest and during submaximal were evaluated by examining the changes in the multifidus muscle during contralateral arm lifts. The thickness of the multifidus was assessed using an Aixplorer (Supersonic Imagine, Aix-en-Provence, France) ultrasound machine with and SL10-2 curvilinear transducer with 5 MHz frequency. Measurements at rest and during submaximal contractions were performed 3 times on each side in a prone position. Submaximal contraction was assessed by instructing the participant to lift their arm while holding a handheld weight (e.g., based on subject's body weight) while the evaluator examined the contralateral multifidus using the ultrasound.[24,34] The thickness change in the multifidus between rest and contracted was calculated using the following equation: %thickness change=$(thickness_{contracted}–thickness_{rest})/thickness_{rest})x100$. All three acquired images for each side were analysed and the mean value was used in the analysis. This method of assessing multifidus using ultrasound is both reliable and valid as demonstrated by previous studies.[34,76]

Lumbar extensor muscle strength

Lumbar extensor muscle strength was assessed with the use of the MedX lumbar extension machine. The participants hips, knees, and pelvis were secured to the machine ensuring isolation of the lumbar extensor muscles with the axis of movement being fixed between vertebral levels L5-S1. This dynamometer assessed isometric lumbar extension muscular strength (torque) in a seated position and accommodates the dynamic resistance through a full 72° range of motion (ROM). Therefore, maximum lumbar extension torque was assessed as maximum voluntary isometric contraction (MVIC) in lumbar extensor muscle strength in seven positions: 72°, 60°, 48°, 36°, 24°, 12° and 0° of flexion.[77,78] Participants will be seated and positioned in the equipment; initial testing was performed to verify any limitations in their ROM and adjustment for the counterweight.[78] Participants first performed a slow controlled warm up for ~1 minute, and then the maximum strength test began.[78] The examiner provided verbal encouragement to encourage participants to generate maximum torque. The movement arm of the MedX machine was attached to a load cell that was interfaced with a computer, that calculated and recorded torque measurements at each angle.

Patient/Self-reported Outcomes

Self-reported measures were obtained at baseline, 6-weeks and 12-weeks with the use of self-reported questionnaires as outlined in Table 4.

Table 4: Secondary measures and their corresponding questionnaires.

Measure	Questionnaire
Disability/function status	Oswestry Low Back Pain Disability Index
Health status	SF-12 Item Health Survey
Level of physical activity	The International Physical Activity Questionnaire
Pain	Numerical pain rating scale (visual analogue scale)
Pain related fear including catastrophizing	The Pain Catastrophizing Scale
Kinesiophobia	Tampa Scale of Kinesiophobia
Depression and anxiety	The Hospital Anxiety and Depression Scale
Sleep Quality	Insomnia Severity Index

Oswestry Low Back Pain Disability Index (ODI)

The ODI is used to measure the patient's level of self-reported disability in relation to LBP. It is a 10-item scale in which each item is rated from 0-5, where 0 means that their pain does not influence that situation and a score of 5 indicates severe disability. The categories included in the questionnaire are pain, walking, lifting, sitting, standing, personal care, sleeping, travel, sex life, and social life. Scores are categorized as minimal, moderate, severe, crippled, or bed bound. The ODI has shown good reliability and validity, and therefore is considered to be the gold standard of measuring disability related to low back pain.[79]

SF-12 Item Health Survey

The 12-item Short Form Health Survey (SF-12) is the condensed form of the previous 36-item survey and is used to assess a patient's health-related quality of life. The 12-item survey consists of 8 domains that assess both physical and mental components of health.

1) Limitations in physical activities because of health problems.
2) Limitations in social activities because of physical or emotional problems
3) Limitations in usual role activities because of physical health problems
4) Bodily pain

5) General mental health (psychological distress and well-being)
6) Limitations in usual role activities because of emotional problems
7) Vitality (energy and fatigue)
8) General health perceptions

Scores from each of the twelve questions are combined to give an overall score between 0 and 100, with a score of 100 indicating the highest level of health. Given that this is a condensed version of a longer and established questionnaire, it has been extensively tested and shown to be both reliable and valid.[80,81]

The International Physical Activity Questionnaire (IPAQ)

The IPAQ is a self-reported log of physical activity (METs based on intensity) in minutes per week over a span of 7 days. The level of physical activity is rated either vigorous (8 MET), moderate (4 MET), walking (3.3 MET) and sitting/rest (1 MET) and must be assigned to the right category. The number of minutes per category is then added up and results are classified as high, moderate, or low physical activity based on the total MET minutes. This measure has been deemed both reliable and valid.[82]

Numerical pain rating scale (NPR):

The NRP for pain is a self-reported rating system for pain intensity. Ratings range from 0 to 10 with 0 being no pain, 1-3 being mild pain, 4-7 being moderate pain, and 8-10 being extreme pain. This is a very basic scale that has excellent reliability and validity in addition to being very user friendly. It is also able to detect statistical and clinically significant changes in perceived pain.[83]

The Pain Catastrophizing Scale (PCS)

The PCS is a 13-item questionnaire that assesses the participant's level of catastrophizing. Each item is rated from 0-4 for a possible total of 52. The questionnaire focuses on three domains that have been used to describe catastrophizing: attentional focus on pain related thoughts (rumination), exaggeration of painful stimuli (magnification), and adopting a hopeless orientation with coping (helplessness). The higher the score, the higher the level of catastrophizing, with scores above 30 being clinically significant. This scale is both reliable and valid.[84]

Tampa Scale of Kinesiophobia (TSK)

The TSK measures the intensity of ones' fear of movement or reinjury in the presence of pain. It contains 11 phrases related to kinesiophobia, such as "I'm afraid I might injure myself if I exercise", with each rating as a Likert scale from 1 to 4. The scores range between 11 and 44 with increasing scores showing increased levels of kinesiophobia. This measure has been shown to have high reliability and validity.[83]

The Hospital Anxiety and Depression Scale (HADS)

The HADS is a 14-item questionnaire used to assess a patient's level of depression and anxiety and was designed for a hospital outpatient setting. 7 items are related to depression while the other 7 relate to anxiety. Cognitive, behavioural, and emotional symptoms are covered in the questionnaire. Each item is rated from 0-3 with either depression or anxiety having scores

between 0 and 21 with 21 being the highest level possible. Scores for either domain between 0 and 7 are classified as normal, 8 to 10 as borderline, and 11 to 21 as abnormal or elevated. The HADS has been found to be both reliable and valid.[85]

Insomnia Severity Index (ISI)

The insomnia severity index is used to assess self-reported quality of sleep. It contains 7 questions than consider the ability to fall asleep, the ability to stay asleep, and effects on daily life. Each question is rated with a Likert-scale from 0-4, with lower ratings indicating a higher quality of sleep. Scores between 0 and 7 indicating no clinically significant insomnia, 8 to 14 indicating subthreshold insomnia, 15 to 21 indicating moderate insomnia, and 22 to 28 indicating severe insomnia. Fourteen has been commonly used as the cut-off score to detect primary insomnia. The ISI has shown to be reliable and valid.[86]

Analysis

Sample size

Sample size calculation was determined by using the effect size (e.g., multifidus muscle CSA) measurements following a motor-control exercise intervention) from a previous study.[55] The mean effect size (over 4 different spinal levels – see below) was calculated and used for sample size estimation (e.g. previously reported effect size (95% CI) for each spinal level; L2=0.87(0.20,1.54); L3=0.90 (0.19,1.62); L4=1.00(0.32, 1.67), and L5=0.81(0.10,1.53)).[55] Pre-post results were considered as independent (independent from two groups) to concur with the study design and analysis of the larger RCT (e.g. between-group factor). Therefore, sample size estimation of n=25 per group was calculated with the G*power software (mean difference, independent t-test) on the basis of a mean effect size 0.90, 80% power and a significance level of alpha 0.05, and allowing for a 10% lost to follow-up and 10% treatment non-adherence.

Statistical Analysis

Descriptive statistics were obtained for both the primary and secondary measures. A one-way repeated measure analysis of variance (ANOVA) was used to examine the changes in paraspinal muscle morphology, extensor muscle strength and secondary outcome measures (e.g., self-reported questionnaires) over the 3 times points. The level of significance was set at p=0.05. Pearson correlation and Spearman correlations (e.g., for data that was not normally distributed) were used to examine if changes in paraspinal muscle morphology and function were associated with variations (changes) in secondary outcome measures. Interpretation of the strength in correlations was based on Cohen's conventions, where 0.10, 0.30, and 0.50 are considered small, moderate, and strong correlations, respectively.[87]

Results

Demographics

195 potential participants expressed interest to take part in the larger two-arms RCT. Thirty-eight chose not to participate for personal reasons or did not maintain contact to be screened. 103 people were excluded due to not meeting the criteria, with the most common reason being having too low scores on the ODI, with second most common being having a known spine abnormality. Four were initially enrolled but then excluded due to spinal abnormalities appearing in baseline MR imaging.

A total of 50 participants were recruited and randomly assigned to each group (n=25 in each group). All 25 participants (Table 5) assigned to the motor control and isolated lumbar extension strengthening (MC+ILEX) intervention completed 12 weeks (e.g. no drop-out). As a group, there was a large variability in self-reported low back pain (LBP), with the average length of LBP being reported as 73.5 ± 82.8 months. Over the course of the intervention, participants averaged an attendance of 22 sessions out of the expected 24. Four participants reported unrelated pain/injury during the intervention and 10 participants reported exercise related muscle soreness after training sessions. Two participants reported receiving treatment for other conditions unrelated to LBP, and 4 participants reported receiving massage therapy at least once over the course of the intervention.

Table 5: Mean ± standard deviation of demographic measures

N=25	Baseline	6-week	12-week
Age (years)	45.12 ± 10.6		
Gender (M:F)	4:21		
BMI (kg/m^2)	26.1 ± 5.01		
LBP Length (months)	73.5 ± 82.8		
NPR (0-10)	5.23 ± 1.7	3.58 ± 1.8[a]	2.81 ± 1.8[a]
ODI (%)	29.4 ± 9.8	22.96 ± 11.5[a]	19.08 ± 10.9[a,b]
PCS	18.4 ± 12.6	13.0 ± 7.92[a]	10.3 ± 9.5[a]
TSK	26.9 ± 7.5	23.76 ± 7.4[a]	21.83 ± 7.2[a,b]
SF-12 Physical	38.26 ± 8.0	42.40 ± 7.4	45.20 ± 7.8[a]
SF-12 Mental	48.84 ± 8.2	46.99 ± 9.1	49.34 ± 12.8
HADS – Anxiety	9.08 ± 3.8	7.96 ± 4.1[a]	7.17 ± 5.28[a]
HADS – Depression	5.76 ± 3.1	6.48 ± 3.8	4.75 ± 4.19[b]
ISI	11.88 ± 6.8	9.56 ± 6.8	9.33 ± 6.20

[a] Significant difference from baseline

[b] Significant difference from 6 to 12-week measurements

Effect of MC+ILEX on Self-Reported Outcomes

There were significant improvements between baseline and the 6 and 12-week timepoints in self-reported pain levels (NPR; $p<0.001$), function (ODI, $p<0.001$), catastrophizing (PCS, $p=0.009$, $p=0.001$), kinesiophobia (TSK; $p=0.002$, $p=0.001$) and anxiety (HADS Anxiety;

p=0.048, p=0.036) (Table 1). There were also significant improvements in physical health (SF-12 physical; p=0.004) from baseline to 12 weeks and depression (p=0.012) between the 6- and 12-week measurements.

Effect of MC+ILEX on Muscle Cross Sectional Area (CSA)

There was a significant increase in multifidus muscle CSA between baseline and 6-week, baseline and 12-week, and between 6-week and 12-week measurements at L4/5 on the left and right (all p<0.001) side (Table 6). Results were similar at S1-L5 level, with significant improvement in CSA on the left and right sides, apart from the right multifidus muscle where no change was found between baseline and 6-week measurements.

With regards to the erector spinae muscle, there was a significant increase in CSA at each time point at L4/5 on the left side (p=0.013, p<0.001, p=0.003) and between baseline and the 6 and 12-week on the right side (p=0.024, p=0.006). There was a significant increase in CSA at each time point at L5/S1 on the right (p<0.001) and left (p=0.007, p<0.001, p=0.005) sides (Table 7). The overall change in multifidus and erector spinae CSA (mean of right and left side) from baseline to post-intervention at each level are presented in Figure 4 and 5.

Table 6: Change in Multifidus CSA (cm^2) (mean±SD, range) at each time point.

	Left			Right		
	Baseline	6-week	12-week	Baseline	6-week	12-week
L4-L5	9.57 ± 1.68[a, b, c] (8.86 to 10.28)	9.90 ± 1.75 (9.16 to 10.64)	10.32 ± 1.83 (9.55 to 11.09)	9.41 ± 1.61[a, b, c] (8.74 to 10.09)	9.75 ± 1.69 (9.04 to 10.47)	10.13 ± 1.73 (9.40 to 10.86)
L5-S1	11.48 ± 2.15[a, b, c] (10.57 to 12.38)	11.76 ± 2.18 (10.84 to 12.68)	12.22 ± 2.24 (11.29 to 13.17)	11.23 ± 1.73[b, c] (10.50 to 11.96)	11.45 ± 1.75 (10.71 to 12.19)	11.94 ± 1.77 (11.20 to 12.69)

[a] Significant difference (p≤0.05) between baseline and week 6.
[b] Significant difference (p≤0.05) between baseline and post intervention
[c] Significant difference (p≤0.05) between week 6 and post intervention

Table 7: Erector Spinae CSA (cm^2), (mean±SD, range) at each time point.

	Left			Right		
	Baseline	6-week	12-week	Baseline	6-week	12-week
L4-L5	16.28 ± 4.20[a, b, c] (14.51 to 18.06)	16.86 ± 4.09 (15.13 to 18.59)	17.60 ± 4.13 (15.86 to 19.35)	16.11 ± 3.84[a,b] (14.488 to 17.73)	16.74 ± 4.07 (15.02 to 18.46)	17.15 ± 3.79 (15.55 to 18.75)
L5-S1	11.06 ± 4.10[a, b, c] (9.33 to 12.79)	12.11 ± 4.05 (10.39 to 13.82)	12.93 ± 4.38 (11.08 to 14.77)	10.77 ± 4.07[a, b, c] (9.05 to 12.48)	11.85 ± 4.01 (10.16 to 13.55)	12.77 ± 4.11 (11.05 to 14.51)

[a] Significant difference (p≤0.05) between baseline and week 6.
[b] Significant difference (p≤0.05) between baseline and post intervention
[c] Significant difference (p≤0.05) between week 6 and post intervention

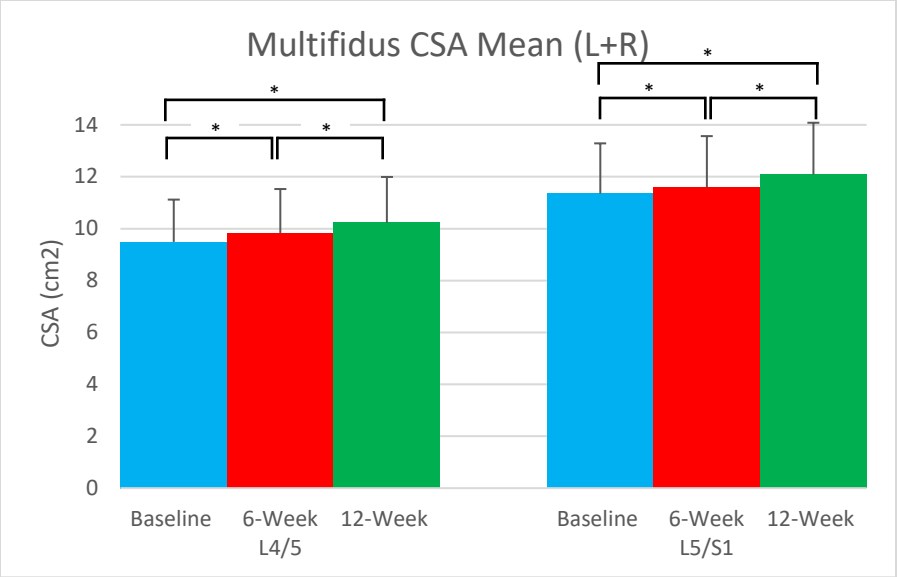

Figure 4: Overall changes in multifidus CSA (mean right and left sides) at L4-L5 and L5-S1 levels. Significant changes (* $p<0.05$) were identified from baseline to 6-week, 6-week to 12-week and from baseline to 12-week. Data is presented as mean±standard deviation.

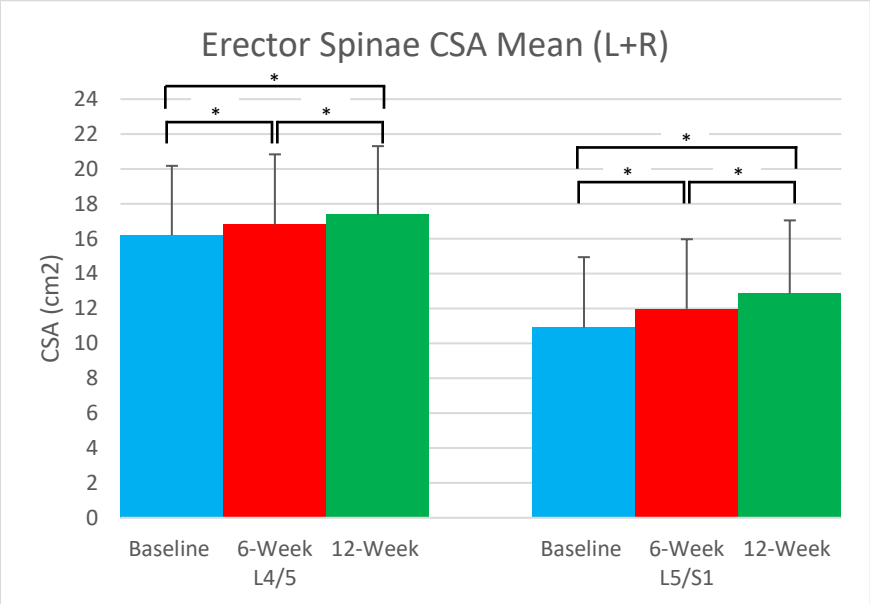

Figure 5: Overall changes in erector CSA (mean right and left sides) at L4-L5 and L5-S1 levels. Significant changes (* $p<0.05$) were identified from baseline to 6-week, 6-week to 12-week and from baseline to 12-week. Data is presented as mean±standard deviation.

Effect of MC+ILEX on Fatty Infiltration (%Fat Fraction)

There was no significant change in multifidus %FF at L4-L5 (Table 8) over the course of the intervention. However, a significant increase in multifidus %FF was observed at L5/S1 on the right side between baseline and 6-week (p=0.021) and between baseline and 12-week (p=0.017). With regards to the erector spinae muscle (Table 9), there was a significant decrease in %FF fatty at L5/S1 on the left side between baseline and 6-week (p=0.006) and 12-week (p=0.002) and on the right between baseline and 12-week (p=0.012). Due to technical issues, 4 sets of IDEAL images were unable to be reconstructed, and therefore fat fraction was unable to be calculated, limiting the sample size to 21 for this measure. The overall change in multifidus and erector spinae %FF (mean of right and left side) from baseline to post-intervention at each level are presented in Figure 6 and 7.

Table 8: Multifidus %FF, (mean±SD, range) at each time point.

	Left			Right		
	Baseline	6-week	12-week	Baseline	6-week	12-week
L4-5	27.12 ± 11.66 (21.66 to 32.58)	27.01 ± 11.88 (21.45 to 32.57)	26.32 ± 11.46 (20.95 to 31.68)	27.59 ± 11.74 (22.07 to 33.06)	27.69 ± 12.03 (22.06 to 33.32)	26.37 ± 11.41 (21.03 to 31.71)
L5-S1	28.81 ± 10.67 (23.82 to 33.80)	28.97 ± 10.54 (24.04 to 33.91)	29.33 ± 10.34 (24.49 to 34.17)	29.31 ± 11.30[a, b] (24.02 to 34.60)	31.10 ± 10.49 (26.19 to 36.01)	31. 49 ± 10.14 (26.75 to 36.24)

[a] significant difference (p≤0.05) between baseline and week 6.
[b] Significant difference (p≤0.05) between baseline and post intervention
[c] Significant difference (p≤0.05) between week 6 and post intervention

Table 9: Erector Spinae %FF, (mean±SD, range) at each time point.

	Left			Right		
	Baseline	6-week	12-week	Baseline	6-week	12-week
L4-L5	35.93 ± 10.67 (30.94 to 40.93)	35.47 ± 9.99 (30.80 to 40.15)	36.30 ± 9.73 (31.75 to 40.86)	38.64 ± 12.07 (32.99 to 44.29)	38.53 ± 12.38 (32.74 to 44.32)	37.67 ± 11.80 (32.15 to 43.19)
L5-S1	44.94 ± 8.46[a,b] (40.98 to 48.90)	42.29 ± 9.35 (37.92 to 46.67)	42.69 ± 9.02 (38.48 to 46.92)	44.71 ± 9.55[a,b] (40.24 to 49.17)	42.63 ± 10.18 (37.87 to 47.40)	42.58 ± 9.62 (38.08 to 47.08)

[a] significant difference (p≤0.05) between baseline and week 6.
[b] Significant difference (p≤0.05) between baseline and post intervention
[c] Significant difference (p≤0.05) between week 6 and post intervention

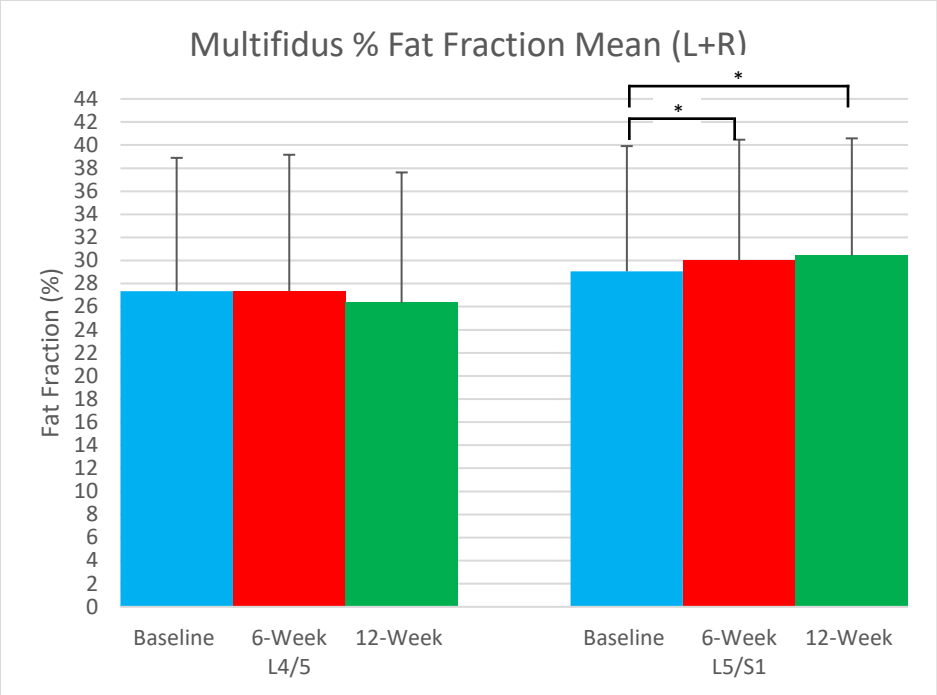

Figure 6: Overall changes in multifidus % fat fraction (mean right and left side) at L4-L5 and L5-S1 levels. Significant changes (* $p<0.05$) were identified from baseline to 6-week and from baseline to 12-week at L5-S1. Data is presented as mean±standard deviation.

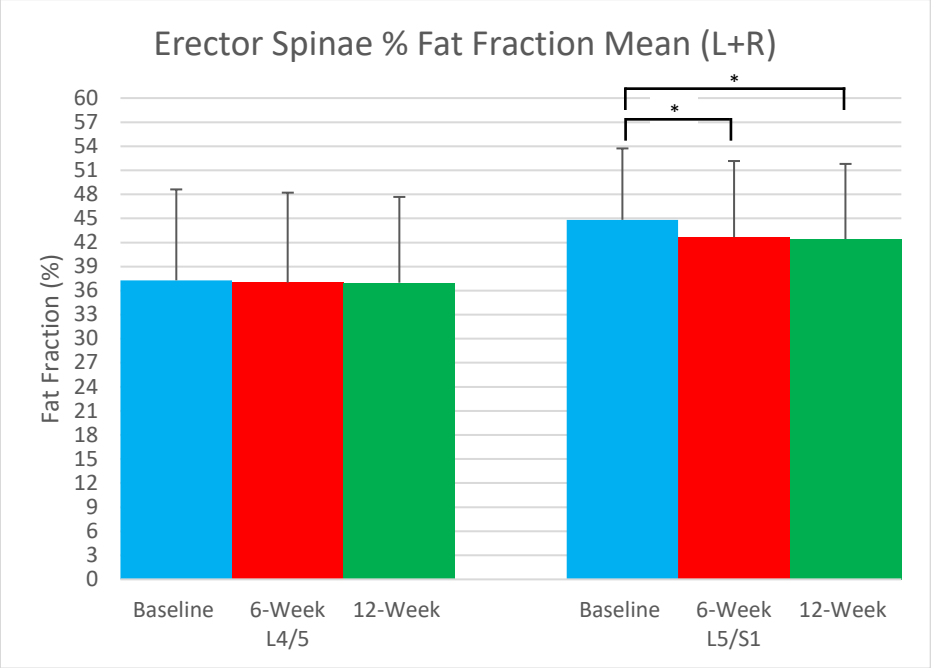

Figure 7: Overall changes in multifidus % fat fraction (mean right and left side) at L4-L5 and L5-S1 levels. Significant changes (* $p<0.05$) were identified from baseline to 6-week and from baseline to 12-week at L5-S1. Data is presented as mean±standard deviation.

Effect of MC+ILEX on Multifidus Thickness and Function

There was a significant increase in multifidus thickness at L4 on the left side between baseline and 6-week ($p=0.001$) and 12-week ($p<0.001$), and on the right side between baseline and 6-week ($p<0.001$) and 12-week ($p<0.001$) (Table 10). At the L5 level, significant improvements in thickness were observed between every time point, on both sides ($p<0.001$). There were no significant differences in thickness % changes between resting and contracted states of the multifidus muscle at any timepoints (Table 11). The overall change in multifidus thickness and % thickness change (mean of right and left side) from baseline to post-intervention at each level are presented in Figure 8 and 9.

Table 10: Multifidus Thickness (cm), (mean±SD, range) at each time point.

	Left			Right		
	Baseline	6-week	12-week	Baseline	6-week	12-week
L4	2.91 ± 0.45[a, b] (2.73 to 3.11)	3.02 ± 0.47 (2.84 to 3.22)	3.11 ± 0.42 (2.93 to 3.28)	2.80 ± 0.40[a, b] (2.64 to 2.97)	2.96 ± 0.38 (2.81 to 3.12)	3.07 ± 0.44 (2.88 to 3.25)
L5	2.79 ± 0.43[a, b, c] (2.61 to 2.97)	2.94 ± 0.45 (2.75 to 3.13)	3.02 ± 0.48 (2.83 to 3.22)	2.68 ± 0.47[a, b, c] (2.48 to 2.87)	2.85 ± 0.47 (2.66 to 3.04)	2.95 ± 0.44 (2.77 to 3.13)

[a] Significant difference ($p\leq0.05$) between baseline and week 6.
[b] Significant difference ($p\leq0.05$) between baseline and post intervention
[c] Significant difference ($p\leq0.05$) between week 6 and post intervention

Table 11: Multifidus Thickness Change (%), (mean±SD, range) at each time point.

	Left			Right		
	Baseline	6-week	12-week	Baseline	6-week	12-week
L4	15.13 ± 8.29 (11.71 to 18.55)	15.24 ± 6.83 (12.42 to 18.06)	16.49 ± 7.68 (13.32 to 19.66)	17.07 ± 7.19 (14.10 to 20.04)	15.91 ± 6.89 (13.07 to 18.75)	16.03 ± 7.00 (13.14 to 18.92)
L5	12.95 ± 8.07 (9.61 to 16.28)	12.63 ± 6.25 (10.05 to 15.21)	12.84 ± 7.04 (9.94 to 15.75)	12.61 ± 9.06 (8.87 to 16.35)	10.72 ± 5.52 (8.44 to 13.00)	11.44 ± 4.97 (9.39 to 13.49)

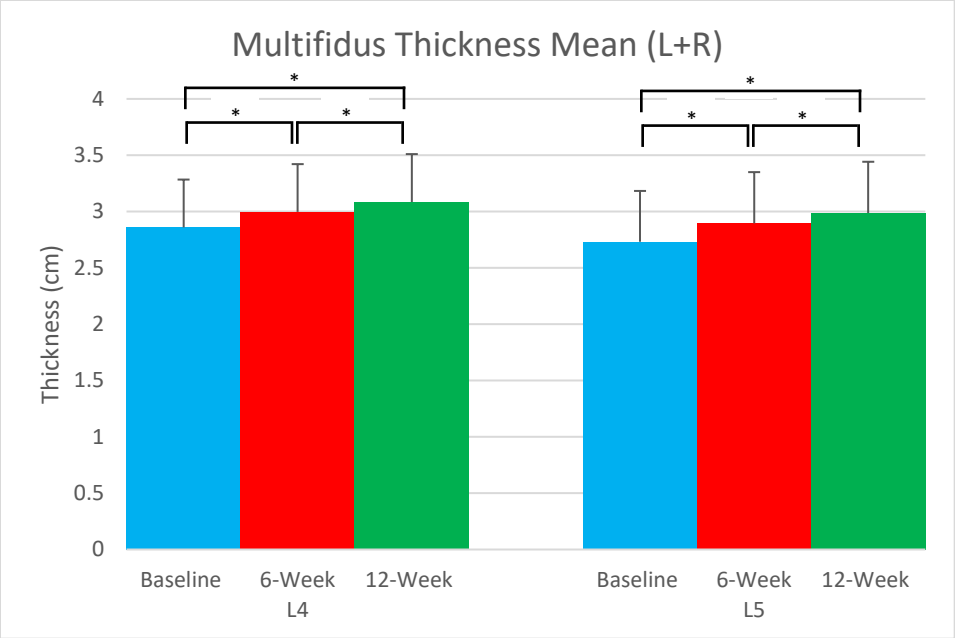

Figure 8: Overall changes in multifidus thickness (mean right and left side) at L4 and L5 levels. Significant changes (* $p<0.05$) were identified from baseline to 6-week, 6-week to 12-week and from baseline to 12-week. Data is presented as mean±standard deviation.

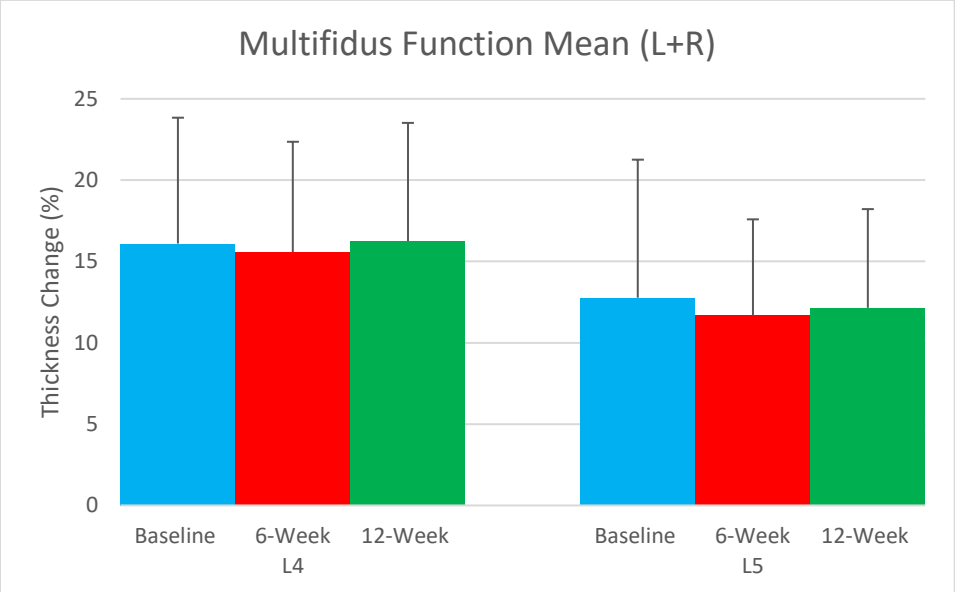

Figure 9: Overall changes in multifidus % thickness change (mean right and left side) at L4 and L5 levels. No significant changes (* $p<0.05$) were identified between any timepoints. Data is presented as mean±standard deviation.

Effect of MC+ILEX on Lumbar Extensor Strength

There were significant increases in lumbar extensor strength between all timepoints ($p=0.008$, $p=0.005$, $p<0.001$). However, due to technical difficulties with the load cell of the MedX, we were unable to complete strength tests for some participants over a period of a few weeks,

therefore our sample size for strength measurements were limited to 15 participants who had complete strength measurements at all time points.

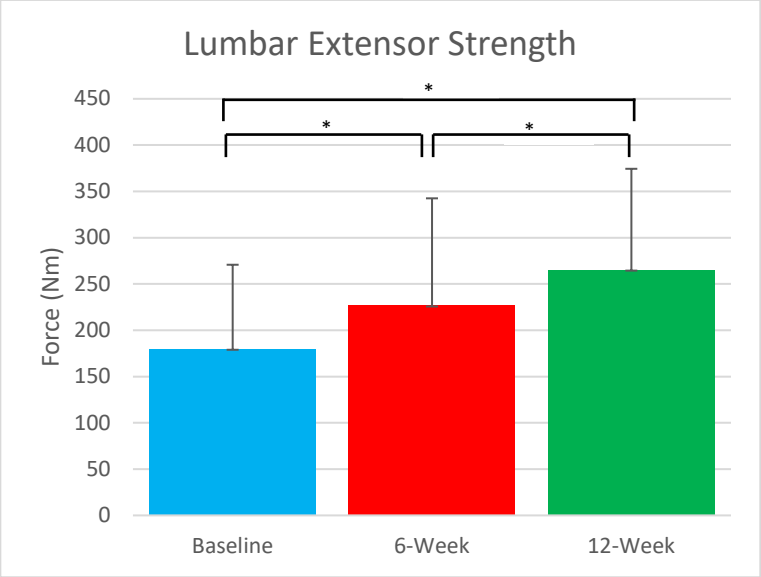

Figure 10: Overall changes in lumbar extensor strength. Significant changes (* $p<0.05$) were identified from baseline to 6-week, 6-week to 12-week and from baseline to 12-week. Data is presented as mean±standard deviation.

Correlation between muscle morphology and clinical outcomes

Pearson correlations between changes (from baseline to 12-week) in muscle morphology (primary outcomes) and changes in self-reported measures (secondary outcomes) are reported in table 12.

Table 12: Correlations between changes in muscle morphology and changes in secondary outcomes

	ΔNPR	ΔODI	ΔPCS	ΔTSK	ΔHADS-A	ΔHADS-D	ΔSF12-M	ΔSF12-P	ΔISI
ΔMF CSA L4/5	-0.49[a]	-0.15	-0.20	-0.09	0.06	-0.34	0.12	0.34	-0.24
ΔMF CSA L5/S1	-0.12	-0.21	-0.24	-0.16	-0.03	-0.07	0.03	0.01	-0.37
ΔES CSA L4/5	0.11	0.07	-0.07	-0.07	-0.08	-0.02	0.24	-0.16	0.46[a]
ΔES CSA L5/S1	-0.22	-0.35	-0.17	-0.31	-0.41[a]	-0.38	0.03	0.23	-0.42[a]
ΔMF FF L4/5	-0.06	0.11	0.03	0.13	-0.08	-0.08	0.19	-0.27	0.26
ΔMF FF L5/S1	0.06	0.57[b]	0.08	-0.03	0.01	-0.12	-0.17	-0.57[b]	0.22
ΔES FF L4/5	-0.15	0.31	-0.09	0.04	-0.07	-0.35	0.05	-0.13	0.35
ΔES FF L5/S1*	-0.03	0.06	0.09	0.30	0.21	0.13	-0.17	0.01	-0.32
ΔMF Thickness L4	-0.15	-0.15	-0.23	0.04	0.03	-0.09	-0.04	0.13	0.20
ΔMF Thickness L5	-0.11	0.04	-0.12	-0.11	0.03	0.03	-0.12	0.10	-0.04
ΔStrength	-0.55[a]	0.22	0.03	-0.05	-0.33	-0.33	0.03	-0.01	-0.03

* Indicates the data is not normally distributed and spearman correlation is used

[a] Indicates $p<0.05$

[b] Indicates $p<0.01$

Discussion

The purpose of this study was to examine the effects of a combined motor control and targeted lumbar extension strengthening intervention on paraspinal muscle morphology in people with chronic, non-specific low back pain. We hypothesized that after the intervention, participants would have a significant increase in paraspinal muscle CSA, decrease in fatty infiltration, and increased thickness and function. Our data demonstrated mixed results with these variables, which is overall consistent with the findings from previous studies.[51–53]

Cross Sectional Area (CSA)

In accordance with our hypothesis, significant increases in multifidus CSA were observed on both the left and right sides across all measured timepoints. Similar increases were seen in the erector spinae muscle except for at L4/5 on the right side. Previous studies have found varying results when it comes to how exercise interventions influence the size of the multifidus and erector spinae muscles in the LBP population.[51–53] Our results demonstrated consistent increases in paraspinal muscle size, which may be due to the unique nature of our intervention. Increases in erector spinae muscle size fall above the minimal detectable change of 100mm^2 indicating it was a true change and not due to possible measurement error.[88] For the multifidus, although the changes did not pass the level of minimal detectable change, many were close, falling just below that threshold so it is still possible to be true change and not measurement error.

Most studies used free-weight or machine-based exercises that did not include pelvic stabilization. Smith et al (2011) found that adding pelvic stabilization to lumbar extension training significantly increases lumbar strength compared to individuals completing the same training without the stabilization, indicating that pelvic stabilization allows for greater isolation and loading of the lumbar extensor muscles.[65] As such, pelvic stabilization may be a key component to lead significant gains in muscle strength and related CSA.

A study by Berry et al. examined the effects of an isolated lumbar extension training program with the MedX machine on lumbar paraspinal muscle morphology. They found no significant increases in multifidus CSA, which is contradictory to our findings.[51] The main difference between their protocol and ours was the inclusion of motor control training for the multifidus and transverse abdominus. It has been well documented that chronic LBP can lead to deactivation and dysfunction of the multifidus muscles along with concurrent overcompensation of the erector spinae and other large trunk muscles.[13] We combined motor control training at the same time as the strength training with the goal of restoring muscle function, therefore improving recruitment and allowing for adequate load on the muscles to hypertrophy. Solely completing resistance training of the trunk or lower body may not be enough to overload the multifidus if they aren't being actively and properly recruited. Berry et al. did not look at the effect of MedX training on the size and composition of the erector spinae. It is possible that in their study, since there was no motor control training, the erectors were doing most of the work during the training instead of having a balanced contribution between both sets of muscles.

Although there are no studies to our knowledge that combined motor control and isolated lumbar extension exercises, a review by Shahtahmassebi et al. did examined the effects of other

exercise interventions on paraspinal morphology across different populations, including LBP.[52] They found positive evidence of increased multifidus size when looking at interventions combining motor control exercises with some form of strength training. However, many studies focused on young and/or athletic populations who were also competing in other sports at the time of the intervention, and thus the results cannot be generalized to the general LBP population.[52]

Fatty Infiltration

In contrast to the CSA and thickness measurements, there was relatively few significant differences in fat fraction values across all muscles and time points. There were only significant decreases in fatty infiltration in the left and right erector spinae at L5/S1 between baseline and post intervention measurements. Our higher baseline values in the erector spinae muscles, especially at L5/S1 may be the reason for our significant findings. As there was a higher percentage of fat in the muscles, there was a greater room for improvement and therefore a higher likelihood of significant changes. Some previous studies have reported similar %fat fraction measurements[51] while others had lower values[53]. Possible reasons for these large variations could be measurement protocol. Inclusion or exclusion of epimuscular fat bands can greatly influence %fat fraction measurements. Age, sex, and weight (BMI) can also affect the amount of fat in the erector spinae muscles, so variations in study populations could also be a factor[26]

The lack of significant findings in generally consistent with the few limited number of studies examining the effect of resistance training on paraspinal muscles, where negligible changes were seen in fatty infiltration of the multifidus muscles. There was large variability in baseline values, with some participants starting as low as 9% and as high as 46%. Overall, some participants saw larger decreases in fatty infiltration while others did not, which may have led to a muted overall effect.

A study by Welch et al. looked at the effects of a free-weight exercise program on paraspinal CSA and fatty infiltration.[53] To our knowledge, they are the only study that has reported significant decreases in multifidus fatty infiltration using T2-weighted images. In comparison, Berry et al. found no significant differences in multifidus fatty infiltration using T1-weigthed images.[51] A possible cause in the variability of findings between studies could be the measurement protocol used. Currently there is no standard method for measuring the amount of fat in the paraspinal muscles and the use of different methods could account for the varying results. To our knowledge, this is the first study to use IDEAL fat/water images to measure fatty infiltration of the paraspinal muscles, and it is currently the best-known method. Furthermore, by using the mean of three slices per level rather than a single slice provides a more accurate assessment of overall changes.

Another consideration when comparing to other studies is how the region of interest was measured. We included the epimuscular fat band when present, while other studies have not, which may lead us to having a higher fat percentage than other studies. Furthermore, there are many other factors that are known to potentially modulate the morphology and quality of these muscles. Our population was primarily female, and our mean age was higher than the previous studies, both which can influence muscle composition and how the muscles adapt to an exercise intervention. Larger sample sizes with a more balanced population will help clarify if our results

are due to the variation in our sample and if they can be generalized to both males and females. Furthermore, transparency on imaging and measurement techniques in other studies will allow for direct comparison in results, as currently many studies use a large variety of techniques, and they frequently aren't described in the articles.

Multifidus Thickness and Function

In accordance with our results for the CSA measurements, we observed significant increases in multifidus thickness at L4 and L5 on both sides between baseline and post-intervention. These changes were also significant at the midpoint mark as well, indicating that morphological changes start to occur early in the exercise program. These values fell above the minimal detectable change of 3.6mm for resting thickness, indicating it was a true change and not due to measurement error.[88] This is consistent with previous studies examining the effect of motor control exercises on multifidus thickness. Kehinde et al. found increases in multifidus thickness after completion of an 8-week stabilization intervention and that these changes were similar to groups completing stabilization exercises paired with other modalities at the same time.[89] A review by Pinto et al. examined 4 studies that looked at changes in multifidus thickness after motor control exercises and found significant increases, although they failed to reach minimal detectable change.[88]

When calculating the percent thickness change between rest and contraction, we found no significant differences. Very few studies have examined the effect of motor control training on percent thickness change of multifidus muscles in LBP populations.[88] One study by Lariviere et al. also found no increases in multifidus activation following an 8-week lumbar stabilization program. However, during measurement they had participants complete a voluntary contraction of the multifidus rather than an involuntary contraction, which could account for the lack of change as it is a difficult task to accomplish after a short period of training.

Generally, the thickness change in a muscle is an indication of muscle function, however, since the multifidus muscles are dynamic stabilizers, they normally function in a submaximal state, similar to the contraction during the image acquisition. A combination of increased muscle size, as seen with increased CSA and thickness, as well as more efficient recruitment and activation that was targeted by the motor control portion of the intervention, it is possible the muscles are working more efficiently despite having an insignificant difference in the measure for muscle function. Furthermore, measurements were taken from the best quality images. Therefore, this data does not indicate the timing of the muscle activation, which has been shown to be an important factor in muscle function and LBP.

Strength

As predicted, our participants saw significant increases in lumbar extensor strength between baseline and the conclusion of the intervention. Many studies have looked at changes in lumbar extensor strength after completing a training program on the MedX and seen improvements in strength. To our knowledge, only one other study has looked at the effects of isolated lumbar extensor training with the MedX machine on lumbar paraspinal muscle morphology. Berry et al. also reported significant increases in strength after a 10-week high

intensity training program on the MedX.[51] Our findings highlight that higher intensity exercise may not be necessary to see improvements in strength and function of the lumbar extensors, as we saw similar increases in strength while completing exercise starting at 55% of our participants 1RM, compared to the 80% 1RM completed in the previous study.[51] This may be important for patients with chronic LBP who are more likely to be in a deconditioned state and may not be able to complete higher intensity training initially. Whether this specific training is more effective than traditional resistance training has yet to be seen, but our findings for the efficacy of ILEX are promising. As we also included a motor control portion to our intervention, it is also a possibility that these exercises could influence our strength outcomes. Currently we are unaware of any studies that have examined the effect of motor control exercises on lumbar extensor strength.

Clinical Outcomes

In addition to improvements in muscle size, thickness, and strength, our participants also had significant improvements in pain, disability, and psychological health. The significant improvements in physical health scores from the SF-12 by 6.94 points were above the minimal detectable change (MDC) of 3.77 and minimal clinically important difference (MCID) of 3.29[90]. For anxiety and depression with the HADS, the MCID is 1.7[91], and we had improvements above that cut-off for anxiety but not depression. However, our baseline values were quite low that not much change was expected. There is limited information on what changes in ODI (MDC=13.5-16.7)[92,93], TSK (MDC=8.4-12.3)[94], and PCS (MDC=12.8; MCID=3.2-4.5)[95,96] would have clinical significance for patients. Some studies indicate it depends on the subpopulation or baseline values, but overall has been mostly unreported[97]. Our values for all fell below their respective MDCs.

The correlation between muscular strength improvements and clinical outcomes has been previously reported in a few studies. A review by Steele et al. found that most studies examined had significant correlations between increased extensor strength and decreases in pain and disability measured by the VAS and ODI, respectively.[42]

Our study found a moderate negative correlation between changes in multifidus CSA at L4-5 and changes in pain ($r=-0.49$, $p=0.016$), and a strong negative correlation between changes in extensor strength and changes in pain ($r=-0.55$, $p=0.035$). This indicates that as muscle size and strength increases, participants were reporting lower pain levels. These were similar findings in a review by Steele et al, where many but not all studies found correlations between extensor strength and pain levels[42]. The difference is that we looked at the correlation between changes in these values, whereas Steele et al. only had studies that compared the mean or peak values.

There was a strong positive correlation between changes in multifidus %fat fraction at L5-S1 and changes in disability ($r=0.59$, $p=0.009$). To our knowledge, no studies have looked at the correlations between muscle morphology and disability. Limited previous studies have primarily focused on functional and performance outcomes and how they correlate with pain and disability[42].

There was also a strong correlation between changes in SF-12 physical scores and changes in multifidus %fat fraction at L5-S1 ($r=-0.57$, $p=0.008$). There were moderate

correlations between changes in erector CSA at L5-S1 and anxiety (r=-0.41, p=0.045). Finally, there were moderate correlations between changes in sleep scores (ISI) and changes in erector CSA at L4-5 (r=0.46; p=0.025) and L5-S1 (r=-0.42; p=0.042). To our knowledge we are not aware of any other studies that have looked at these correlations for us to compare our results to.

The benefits of exercise on mental health and related psychological factors are well known. Our findings further support that, as our participants saw significant improvements in catastrophizing, kinesiophobia, and anxiety. Relationships between changes in these factors and muscle health in LBP is still unknown. Our results indicate there may be a relationship between the changes fatty infiltration of both the multifidus and erector spinae muscles and LBP related disability, where individuals with larger decreases in %fat fraction measurements reported greater improvements in disability. Furthermore, it appears there may be a link between lumbar extensor strength and pain levels, as seen in the few previous studies that have examined this relationship.[42] Our preliminary findings revealed significant relationships between paraspinal muscle health and other psychological factors that are known to be possible mediators in LBP.

Implications of Findings

Expanding our knowledge on how different exercise interventions affect muscle morphology in patients with LBP is important for clinical decision making when treating these individuals. Much of prior research on LBP focuses on pain and functional outcomes. Despite clear links between multifidus morphological degeneration and LBP, there is limited research assessing which exercise interventions may be best to combat these negative changes.

Current research has consistently shown mixed results due to the complex nature of non-specific LBP. Finding an optimal treatment has been unsuccessful so far. Our results suggest that an intervention combining motor control and isolated lumbar extensor training has the potential to be successful in improving a large variety of outcomes, both structural and clinical, in patients with chronic LBP.

Future research with larger sample sizes and a better balance between males and females needs to be conducted to confirm and expand our results. Furthermore, our population consisted of solely physically inactive individuals, and while a large portion of people suffering from chronic LBP are physically inactive, there are also many active individuals such as athletes that can also suffer from chronic LBP as well. As detraining is a huge factor in whether an individual will undergo hypertrophy with resistance training, we are unable to generalize our results to that population. Further studies should be completed to see if active individuals will see the same results with this intervention. Furthermore, as we are one of the few studies to examine the correlation between extensor strength and pain/disability, and one of, if not the first to compare paraspinal morphology with functional outcomes and psychological factors, more studies need to be completed to further expand on our results.

Limitations

We were limited to a small sample size, which may have an influence on the lack of statistically significant results for certain variables. Furthermore, our sample was primarily female. It has

been well established that the muscular characteristics between males and females are different, so it will be harder to generalize our findings, particularly to males, as only four were included in this study. Due to technical errors, data was missing for some strength and %fat fraction measurements, further reducing our sample size for that data.

Conclusion

Overall, our results indicate that a LBP intervention combining motor control exercise and isolated lumbar extensor strength training can be successful in reducing pain and disability, increasing paraspinal muscle size and strength, and improving other psychological factors that have been linked with LBP. However, due to the novelty of this intervention and the many factors it encompasses, further research needs to be completed to confirm our results and strengthen our knowledge on how exercise affects muscle morphology and how it relates to the many outcomes related to LBP.

References

1. Dagenais S, Caro J, Haldeman S. A systematic review of low back pain cost of illness studies in the United States and internationally. *Spine J*. 2008;8(1):8-20. doi:10.1016/j.spinee.2007.10.005

2. Delitto A, George SZ, Van Dillen L, et al. Low Back Pain: Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopaedic Section of the American Physical Therapy Association. *J Orthop Sports Phys Ther*. 2012;42(4):A1-A57. doi:10.2519/jospt.2012.42.4.A1

3. van Middelkoop M, Rubinstein SM, Kuijpers T, et al. A systematic review on the effectiveness of physical and rehabilitation interventions for chronic non-specific low back pain. *Eur Spine J*. 2011;20(1):19-39. doi:10.1007/s00586-010-1518-3

4. Hartvigsen J, Hancock MJ, Kongsted A, et al. What low back pain is and why we need to pay attention. *The Lancet*. 2018;391(10137):2356-2367. doi:10.1016/S0140-6736(18)30480-X

5. Hapidou EG, O'Brien MA, Pierrynowski MR, de las Heras E, Patel M, Patla T. Fear and Avoidance of Movement in People with Chronic Pain: Psychometric Properties of the 11-Item Tampa Scale for Kinesiophobia (TSK-11). *Physiother Can*. 2012;64(3):235-241. doi:10.3138/ptc.2011-10

6. Foster NE, Anema JR, Cherkin D, et al. Prevention and treatment of low back pain: evidence, challenges, and promising directions. *The Lancet*. 2018;391(10137):2368-2383. doi:10.1016/S0140-6736(18)30489-6

7. Nascimento PRC do, Costa LOP, Araujo AC, Poitras S, Bilodeau M. Effectiveness of interventions for non-specific low back pain in older adults. A systematic review and meta-analysis. *Physiotherapy*. 2019;105(2):147-162. doi:10.1016/j.physio.2018.11.004

8. Saner J, Kool J, Sieben JM, Luomajoki H, Bastiaenen CHG, de Bie RA. A tailored exercise program versus general exercise for a subgroup of patients with low back pain and movement control impairment: A randomised controlled trial with one-year follow-up. *Man Ther*. 2015;20(5):672-679. doi:10.1016/j.math.2015.02.005

9. van Dieën JH, Reeves NP, Kawchuk G, van Dillen LR, Hodges PW. Analysis of Motor Control in Patients With Low Back Pain: A Key to Personalized Care? *J Orthop Sports Phys Ther*. 2019;49(6):380-388. doi:10.2519/jospt.2019.7916

10. Karunanayake AL, Pathmeswaran A, Kasturiratne A, Wijeyaratne LS. Risk factors for chronic low back pain in a sample of suburban Sri Lankan adult males. *Int J Rheum Dis*. 2013;16(2):203-210. doi:10.1111/1756-185X.12060

11. Shiri R, Falah-Hassani K, Heliövaara M, et al. Risk Factors for Low Back Pain: A Population-Based Longitudinal Study. *Arthritis Care Res*. 2019;71(2):290-299. doi:10.1002/acr.23710

12. van Dieën JH, Reeves NP, Kawchuk G, van Dillen LR, Hodges PW. Motor Control Changes in Low Back Pain: Divergence in Presentations and Mechanisms. *J Orthop Sports Phys Ther*. 2019;49(6):370-379. doi:10.2519/jospt.2019.7917

13. Freeman MD, Woodham MA, Woodham AW. The Role of the Lumbar Multifidus in Chronic Low Back Pain: A Review. *PM&R*. 2010;2(2):142-146. doi:10.1016/j.pmrj.2009.11.006

14. Oliveira DS, Vélia Ferreira Mendonça L, Sofia Monteiro Sampaio R, Manuel Pereira Dias de Castro-Lopes J, Ribeiro de Azevedo LF. The Impact of Anxiety and Depression on the Outcomes of Chronic Low Back Pain Multidisciplinary Pain Management—A Multicenter Prospective Cohort Study in Pain Clinics with One-Year Follow-up. *Pain Med*. 2019;20(4):736-746. doi:10.1093/pm/pny128

15. Wáng YXJ, Wáng JQ, Káplár Z. Increased low back pain prevalence in females than in males after menopause age: evidences based on synthetic literature review. *Quant Imaging Med Surg*. 2016;6(2):199-206. doi:10.21037/qims.2016.04.06

16. MacKenzie JF, Grimshaw PN, Jones CDS, Thoirs K, Petkov J. Muscle activity during lifting: Examining the effect of core conditioning of multifidus and transversus abdominis. *Work*. 2014;47(4):453-462. doi:10.3233/WOR-131706

17. Haig AJ. Paraspinal denervation and the spinal degenerative cascade. *Spine J*. 2002;2(5):372-380. doi:10.1016/S1529-9430(02)00201-2

18. Bussey MD, Aldabe D, Ribeiro DC, Madill S, Woodley S, Hammer N. Is Pelvic Floor Dysfunction Associated With Development of Transient Low Back Pain During Prolonged Standing? A Protocol. *Clin Med Insights Womens Health*. 2019;12:1179562X1984960. doi:10.1177/1179562X19849603

19. Bernard S, Gentilcore-Saulnier E, Massé-Alarie H, Moffet H. Is adding pelvic floor muscle training to an exercise intervention more effective at improving pain in patients with non-specific low back pain? A systematic review of randomized controlled trials. *Physiotherapy*. Published online March 2020:S0031940620300195. doi:10.1016/j.physio.2020.02.005

20. Fortin M, Battié MC. Quantitative Paraspinal Muscle Measurements: Inter-Software Reliability and Agreement Using OsiriX and ImageJ. *Phys Ther*. 2012;92(6):853-864. doi:10.2522/ptj.20110380

21. Hu ZJ, He J, Zhao FD, Fang XQ, Zhou LN, Fan SW. An Assessment of the Intra- and Inter-reliability of the Lumbar Paraspinal Muscle Parameters Using CT Scan and Magnetic Resonance Imaging: *Spine*. 2011;36(13):E868-E874. doi:10.1097/BRS.0b013e3181ef6b51

22. Ranson CA, Burnett AF, Kerslake R, Batt ME, O'Sullivan PB. An investigation into the use of MR imaging to determine the functional cross sectional area of lumbar paraspinal muscles. *Eur Spine J*. 2006;15(6):764-773. doi:10.1007/s00586-005-0909-3

23. Ranger TA, Cicuttini FM, Jensen TS, Heritier S, Urquhart DM. Paraspinal muscle cross-sectional area predicts low back disability but not pain intensity. *Spine J*. 2019;19(5):862-868. doi:10.1016/j.spinee.2018.12.004

24. Fortin M, Rizk A, Frenette S, Boily M, Rivaz H. Ultrasonography of multifidus muscle morphology and function in ice hockey players with and without low back pain. *Phys Ther Sport*. 2019;37:77-85. doi:10.1016/j.ptsp.2019.03.004

25. Belavý DL, Armbrecht G, Felsenberg D. Real-time ultrasound measures of lumbar erector spinae and multifidus: reliability and comparison to magnetic resonance imaging. *Physiol Meas*. 2015;36(11):2285-2299. doi:10.1088/0967-3334/36/11/2285

26. Kjaer P, Bendix T, Sorensen JS, Korsholm L, Leboeuf-Yde C. Are MRI-defined fat infiltrations in the multifidus muscles associated with low back pain? *BMC Med*. 2007;5(1):2. doi:10.1186/1741-7015-5-2

27. Kader DF, Wardlaw D, Smith FW. Correlation Between the MRI Changes in the Lumbar Multifidus Muscles and Leg Pain. *Clin Radiol*. 2000;55(2):145-149. doi:10.1053/crad.1999.0340

28. Fortin M, Omidyeganeh M, Battié MC, Ahmad O, Rivaz H. Evaluation of an automated thresholding algorithm for the quantification of paraspinal muscle composition from MRI images. *Biomed Eng OnLine*. 2017;16(1):61. doi:10.1186/s12938-017-0350-y

29. Crawford RJ, Cornwall J, Abbott R, Elliott JM. Manually defining regions of interest when quantifying paravertebral muscles fatty infiltration from axial magnetic resonance imaging: a proposed method for the lumbar spine with anatomical cross-reference. *BMC Musculoskelet Disord*. 2017;18(1):25. doi:10.1186/s12891-016-1378-z

30. Fischer MA, Nanz D, Shimakawa A, et al. Quantification of Muscle Fat in Patients with Low Back Pain: Comparison of Multi-Echo MR Imaging with Single-Voxel MR Spectroscopy. *Radiology*. 2013;266(2):555-563. doi:10.1148/radiol.12120399

31. Reeder SB, Hu HH, Sirlin CB. Proton density fat-fraction: A standardized mr-based biomarker of tissue fat concentration. *J Magn Reson Imaging*. 2012;36(5):1011-1014. doi:10.1002/jmri.23741

32. Yoo YH, Kim HS, Lee YH, et al. Comparison of Multi-Echo Dixon Methods with Volume Interpolated Breath-Hold Gradient Echo Magnetic Resonance Imaging in Fat-Signal Fraction Quantification of Paravertebral Muscle. *Korean J Radiol*. 2015;16(5):1086. doi:10.3348/kjr.2015.16.5.1086

33. Crawford RJ, Fortin M, Weber KA, Smith A, Elliott JM. Are Magnetic Resonance Imaging Technologies Crucial to Our Understanding of Spinal Conditions? *J Orthop Sports Phys Ther*. 2019;49(5):320-329. doi:10.2519/jospt.2019.8793

34. Kiesel KB, Uhl TL, Underwood FB, Rodd DW, Nitz AJ. Measurement of lumbar multifidus muscle contraction with rehabilitative ultrasound imaging. *Man Ther*. 2007;12(2):161-166. doi:10.1016/j.math.2006.06.011

35. ShahAli S, Shanbehzadeh S, ShahAli S, Ebrahimi Takamjani I. Application of Ultrasonography in the Assessment of Abdominal and Lumbar Trunk Muscle Activity in Participants With and Without Low Back Pain: A Systematic Review. *J Manipulative Physiol Ther*. 2019;42(7):541-550. doi:10.1016/j.jmpt.2019.05.003

36. Taghipour M, Mohseni-Bandpei MA, Behtash H, et al. Reliability of Real-time Ultrasound Imaging for the Assessment of Trunk Stabilizer Muscles: A Systematic Review of the Literature. *J Ultrasound Med*. 2019;38(1):15-26. doi:10.1002/jum.14661

37. Sions JM, Velasco TO, Teyhen DS, Hicks GE. Ultrasound Imaging: Intraexaminer and Interexaminer Reliability for Multifidus Muscle Thickness Assessment in Adults Aged 60 to 85 Years Versus Younger Adults. *J Orthop Sports Phys Ther*. 2014;44(6):425-434. doi:10.2519/jospt.2014.4584

38. Hides JA, Donelson R, Lee D, Prather H, Sahrmann SA, Hodges PW. Convergence and Divergence of Exercise-Based Approaches That Incorporate Motor Control for the Management of Low Back Pain. *J Orthop Sports Phys Ther*. 2019;49(6):437-452. doi:10.2519/jospt.2019.8451

39. Berglund L, Aasa B, Michaelson P, Aasa U. Effects of Low-Load Motor Control Exercises and a High-Load Lifting Exercise on Lumbar Multifidus Thickness: A Randomized Controlled Trial. *SPINE*. 2017;42(15):E876-E882. doi:10.1097/BRS.0000000000001989

40. Goubert D, Danneels L. Structural Changes of Lumbar Muscles in Non-Specific Low Back Pain. *Pain Physician*.:16.

41. van Dieën JH, Selen LPJ, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. *J Electromyogr Kinesiol*. 2003;13(4):333-351. doi:10.1016/S1050-6411(03)00041-5

42. Steele J, Fisher J, Perrin C, Conway R, Bruce-Low S, Smith D. Does change in isolated lumbar extensor muscle function correlate with good clinical outcome? A secondary analysis of data on change in isolated lumbar extension strength, pain, and disability in chronic low back pain. *Disabil Rehabil*. 2019;41(11):1287-1295. doi:10.1080/09638288.2018.1424952

43. Hodges PW, Danneels L. Changes in Structure and Function of the Back Muscles in Low Back Pain: Different Time Points, Observations, and Mechanisms. *J Orthop Sports Phys Ther*. 2019;49(6):464-476. doi:10.2519/jospt.2019.8827

44. Prins MR, Griffioen M, Veeger TTJ, et al. Evidence of splinting in low back pain? A systematic review of perturbation studies. *Eur Spine J*. 2018;27(1):40-59. doi:10.1007/s00586-017-5287-0

45. Hodges P, Holm AK, Hansson T, Holm S. Rapid Atrophy of the Lumbar Multifidus Follows Experimental Disc or Nerve Root Injury: *Spine*. 2006;31(25):2926-2933. doi:10.1097/01.brs.0000248453.51165.0b

46. Nowotny AH, Calderon MG, de Souza PA, et al. Lumbar stabilisation exercises versus back endurance-resistance exercise training in athletes with chronic low back pain: protocol of a randomised controlled trial. *BMJ Open Sport Exerc Med*. 2018;4(1):e000452. doi:10.1136/bmjsem-2018-000452

47. Saner J, Kool J, de Bie RA, Sieben JM, Luomajoki H. Movement control exercise versus general exercise to reduce disability in patients with low back pain and movement control impairment. A randomised controlled trial. *BMC Musculoskelet Disord*. 2011;12(1):207. doi:10.1186/1471-2474-12-207

48. Hayden JA, Ellis J, Ogilvie R, et al. Some types of exercise are more effective than others in people with chronic low back pain: a network meta-analysis. *J Physiother*. 2021;67(4):252-262. doi:10.1016/j.jphys.2021.09.004

49. Gordon R, Bloxham S. A Systematic Review of the Effects of Exercise and Physical Activity on Non-Specific Chronic Low Back Pain. *Healthcare*. 2016;4(2):22. doi:10.3390/healthcare4020022

50. Searle A, Spink M, Ho A, Chuter V. Exercise interventions for the treatment of chronic low back pain: a systematic review and meta-analysis of randomised controlled trials. *Clin Rehabil*. 2015;29(12):1155-1167. doi:10.1177/0269215515570379

51. Berry DB, Padwal J, Johnson S, Englund EK, Ward SR, Shahidi B. The effect of high-intensity resistance exercise on lumbar musculature in patients with low back pain: a preliminary study. *BMC Musculoskelet Disord*. 2019;20(1):290. doi:10.1186/s12891-019-2658-1

52. Shahtahmassebi B, Hebert JJ, Stomski NJ, Hecimovich M, Fairchild TJ. The Effect of Exercise Training on Lower Trunk Muscle Morphology. *Sports Med*. 2014;44(10):1439-1458. doi:10.1007/s40279-014-0213-7

53. Welch N, Moran K, Antony J, et al. The effects of a free-weight-based resistance training intervention on pain, squat biomechanics and MRI-defined lumbar fat infiltration and functional cross-sectional area in those with chronic low back. *BMJ Open Sport Exerc Med*. 2015;1(1):e000050-e000050. doi:10.1136/bmjsem-2015-000050

54. Panjabi MM. The Stabilizing System of the Spine. Part I. Function, Dysfunction, Adaptation, and Enhancement: *J Spinal Disord*. 1992;5(4):383-389. doi:10.1097/00002517-199212000-00001

55. Macedo LG, Latimer J, Maher CG, et al. Effect of Motor Control Exercises Versus Graded Activity in Patients With Chronic Nonspecific Low Back Pain: A Randomized Controlled Trial. *Phys Ther*. 2012;92(3):363-377. doi:10.2522/ptj.20110290

56. Alrwaily M, Timko M, Schneider M, et al. Treatment-Based Classification System for Low Back Pain: Revision and Update. *Phys Ther*. 2016;96(7):1057-1066. doi:10.2522/ptj.20150345

57. O'Sullivan P. Diagnosis and classification of chronic low back pain disorders: Maladaptive movement and motor control impairments as underlying mechanism. *Man Ther*. 2005;10(4):242-255. doi:10.1016/j.math.2005.07.001

58. Azevedo DC, Ferreira PH, Santos H de O, Oliveira DR, de Souza JVL, Costa LOP. Movement System Impairment-Based Classification Treatment Versus General Exercises for Chronic Low Back Pain: Randomized Controlled Trial. *Phys Ther*. 2018;98(1):28-39. doi:10.1093/ptj/pzx094

59. Shamsi M, Sarrafzadeh J, Jamshidi A, Arjmand N, Ghezelbash F. Comparison of spinal stability following motor control and general exercises in nonspecific chronic low back pain patients. *Clin Biomech*. 2017;48:42-48. doi:10.1016/j.clinbiomech.2017.07.006

60. Saragiotto BT, Maher CG, Yamato TP, et al. Motor control exercise for chronic non-specific low-back pain. Cochrane Back and Neck Group, ed. *Cochrane Database Syst Rev*. Published online January 8, 2016. doi:10.1002/14651858.CD012004

61. Steele J, Bruce-Low S, Smith D. A Review of the Clinical Value of Isolated Lumbar Extension Resistance Training for Chronic Low Back Pain. *PM&R*. 2015;7(2):169-187. doi:10.1016/j.pmrj.2014.10.009

62. Shahtahmassebi B, Hebert JJ, Hecimovich M, Fairchild TJ. Trunk exercise training improves muscle size, strength, and function in older adults: A randomized controlled trial. *Scand J Med Sci Sports*. Published online April 7, 2019:sms.13415. doi:10.1111/sms.13415

63. Steele J, Fisher J, Bruce-Low S, Smith D, Osborne N, Newell D. Variability in Strength, Pain, and Disability Changes in Response to an Isolated Lumbar Extension Resistance Training Intervention in Participants with Chronic Low Back Pain. *Healthcare*. 2017;5(4):75. doi:10.3390/healthcare5040075

64. Mayer JM, Graves JE, Udermann BE, Ploutz-Snyder LL. Development of lumbar extension strength: Effect of pelvic stabilization during resistance training. *J Back Musculoskelet Rehabil*. 2002;16(1):25-31. doi:10.3233/BMR-2002-16105

65. Smith D, Bissell G, Bruce-Low S, Wakefield C. The effect of lumbar extension training with and without pelvic stabilization on lumbar strength and low back pain1. *J Back Musculoskelet Rehabil*. 2011;24(4):241-249. doi:10.3233/BMR-2011-0301

66. Pollock ML, Leggett SH, Graves JE, Jones A, Fulton M, Cirulli J. Effect of resistance training on lumbar extension strength. *Am J Sports Med*. 1989;17(5):624-629. doi:10.1177/036354658901700506

67. Steele J, Bruce-Low S, Smith D, Jessop D, Osborne N. A Randomized Controlled Trial of Limited Range of Motion Lumbar Extension Exercise in Chronic Low Back Pain: *Spine*. 2013;38(15):1245-1252. doi:10.1097/BRS.0b013e318291b526

68. Summers SJ, Higgins NC, Te M, Byrne A, Chipchase LS. The effect of implicit theories of pain on pain and disability in people with chronic low back pain. *Musculoskelet Sci Pract*. 2019;40:65-71. doi:10.1016/j.msksp.2019.01.013

69. Gatchel RJ, Neblett R, Kishino N, Ray CT. Fear-Avoidance Beliefs and Chronic Pain. *J Orthop Sports Phys Ther*. 2016;46(2):38-43. doi:10.2519/jospt.2016.0601

70. Larsson C, Hansson EE, Sundquist K, Jakobsson U. Psychometric properties of the Tampa Scale of Kinesiophobia (TSK-11) among older people with chronic pain. *Physiother Theory Pract*. 2014;30(6):421-428. doi:10.3109/09593985.2013.877546

71. Zale EL, Ditre JW. Pain-related fear, disability, and the fear-avoidance model of chronic pain. *Curr Opin Psychol*. 2015;5:24-30. doi:10.1016/j.copsyc.2015.03.014

72. Ferrari S, Chiarotto A, Pellizzer M, Vanti C, Monticone M. Pain Self-Efficacy and Fear of Movement are Similarly Associated with Pain Intensity and Disability in Italian Patients with Chronic Low Back Pain. *Pain Pract*. 2016;16(8):1040-1047. doi:10.1111/papr.12397

73. Pinheiro MB, Ferreira ML, Refshauge K, et al. Symptoms of Depression and Risk of New Episodes of Low Back Pain: A Systematic Review and Meta-Analysis: Depression and Risk of Low Back Pain. *Arthritis Care Res*. 2015;67(11):1591-1603. doi:10.1002/acr.22619

74. Marshall PWM, Schabrun S, Knox MF. Physical activity and the mediating effect of fear, depression, anxiety, and catastrophizing on pain related disability in people with chronic low back pain. Yang J, ed. *PLOS ONE*. 2017;12(7):e0180788. doi:10.1371/journal.pone.0180788

75. Sezgin M, Hasanefendioğlu EZ, Sungur MA, et al. Sleep quality in patients with chronic low back pain: A cross-sectional study assesing its relations with pain, functional status and quality of life. *J Back Musculoskelet Rehabil*. 2015;28(3):433-441. doi:10.3233/BMR-140537

76. Koppenhaver S, Kniss J, Lilley D, et al. Reliability of ultrasound shear-wave elastography in assessing low back musculature elasticity in asymptomatic individuals. *J Electromyogr Kinesiol*. 2018;39:49-57. doi:10.1016/j.jelekin.2018.01.010

77. Mayer JM, Childs JD, Neilson BD, Chen H, Koppenhaver SL, Quillen WS. Effect of Lumbar Progressive Resistance Exercise on Lumbar Muscular Strength and Core Muscular Endurance in Soldiers. *Mil Med*. 2016;181(11):e1615-e1622. doi:10.7205/MILMED-D-15-00543

78. Conway R, Behennah J, Fisher J, Osborne N, Steele J. Associations between Trunk Extension Endurance and Isolated Lumbar Extension Strength in Both Asymptomatic Participants and Those with Chronic Low Back Pain. *Healthcare*. 2016;4(3):70. doi:10.3390/healthcare4030070

79. Saltychev M, Mattie R, McCormick Z, Bärlund E, Laimi K. Psychometric properties of the Oswestry Disability Index: *Int J Rehabil Res*. 2017;40(3):202-208. doi:10.1097/MRR.0000000000000226

80. Huo T, Guo Y, Shenkman E, Muller K. Assessing the reliability of the short form 12 (SF-12) health survey in adults with mental health conditions: a report from the wellness incentive and navigation (WIN) study. *Health Qual Life Outcomes*. 2018;16(1):34. doi:10.1186/s12955-018-0858-2

81. Ware J, Kosinski M, Keller S. A 12-Item Short-Form Health Survey: Construction of Scales and Preliminary Tests of Reliability and Validity. *Med Care*. 34(3):15.

82. Tran DV, Lee AH, Au TB, Nguyen CT, Hoang DV. Reliability and validity of the International Physical Activity Questionnaire-Short Form for older adults in Vietnam: D. V. Tran *et al* . *Health Promot J Austr*. 2013;24(2):126-131. doi:10.1071/HE13012

83. Alghadir A, Anwer S, Iqbal A, Iqbal Z. Test–retest reliability, validity, and minimum detectable change of visual analog, numerical rating, and verbal rating scales for measurement of osteoarthritic knee pain. *J Pain Res*. 2018;Volume 11:851-856. doi:10.2147/JPR.S158847

84. Sullivan MJL, Bishop SR, Pivik J. The Pain Catastrophizing Scale: Development and Validation. :9.

85. Villoria E, Lara L. Assessment of the Hospital Anxiety and Depression Scale for cancer patients. *Rev Médica Chile*. 2018;146(3):300-307. doi:10.4067/s0034-98872018000300300

86. Alsaadi SM, McAuley JH, Hush JM, et al. Detecting insomnia in patients with low back pain: accuracy of four self-report sleep measures. *BMC Musculoskelet Disord*. 2013;14(1):196. doi:10.1186/1471-2474-14-196

87. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. 2nd ed. Routledge; 1988.

88. Pinto SM, Boghra SB, Macedo LG, et al. Does Motor Control Exercise Restore Normal Morphology of Lumbar Multifidus Muscle in People with Low Back Pain? - A Systemic Review. Published online August 2021.

89. Kehinde AA, Sra A, Do O. Effect of Stabilization Exercise on Lumbar Multifidus Muscle Thickness in patients with non-specific Chronic Low Back Pain. *Iran Rehabil J*. 2014;12(20):5.

90. Hägg O, Fritzell P, Nordwall A. The clinical importance of changes in outcome scores after treatment for chronic low back pain. *Eur Spine J*. 2003;12(1):12-20. doi:10.1007/s00586-002-0464-0

91. Lemay KR, Tulloch HE, Pipe AL, Reed JL. Establishing the Minimal Clinically Important Difference for the Hospital Anxiety and Depression Scale in Patients With Cardiovascular Disease. *J Cardiopulm Rehabil Prev*. 2019;39(6):E6-E11. doi:10.1097/HCR.0000000000000379

92. Maughan EF, Lewis JS. Outcome measures in chronic low back pain. *Eur Spine J*. 2010;19(9):1484-1494. doi:10.1007/s00586-010-1353-6

93. Copay AG, Cher DJ. Is the Oswestry Disability Index a valid measure of response to sacroiliac joint treatment? *Qual Life Res*. 2016;25(2):283-292. doi:10.1007/s11136-015-1095-3

94. Ostelo RWJG, Swinkels-Meewisse IJCM, Vlaeyen JWS, Knol DL, de Vet HCW. Assessing pain and pain-related fear in acute low back pain: what is the smallest detectable change? *Int J Behav Med*. 2007;14(4):242-248. doi:10.1007/BF03002999

95. Fernandes L, Storheim K, Lochting I, Grotle M. Cross-cultural adaptation and validation of the Norwegian pain catastrophizing scale in patients with low back pain. *BMC Musculoskelet Disord*. 2012;13(1):111. doi:10.1186/1471-2474-13-111

96. Ogunlana MO, Odole AC, Adejumo A, Odunaiya N. Catastrophising, pain, and disability in patients with nonspecific low back pain. *Hong Kong Physiother J*. 2015;33(2):73-79. doi:10.1016/j.hkpj.2015.03.001

97. Lauridsen HH, Hartvigsen J, Manniche C, Korsholm L, Grunnet-Nilsson N. Responsiveness and minimal clinically important difference for pain and disability instruments in low back pain patients. *BMC Musculoskelet Disord*. 2006;7(1):82. doi:10.1186/1471-2474-7-82