DALHOUSIE UNIVERSITY

Title: Trunk neuromuscular pattern alterations during a controlled functional task in a low back injured group deemed ready to resume regular activities

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Abstract

Background: Trunk neuromuscular alterations have been found in those with chronic low back pain, but less well studied are whether responses are altered in those deemed recovered following an injury. Furthermore, coordinated trunk muscle responses are deemed important for normal spinal function, but there are no studies of temporal patterns early after a low back injury. Determining whether altered trunk muscle patterns exist early after injury could improve our understanding of recovery by providing an objective assessment of functional recovery and risk of re-injury.
Objective: To determine if amplitude and temporal characteristics of trunk neuromuscular patterns differ during a dynamic functional task in a group of participants with recent (within 12 weeks) low back injury (LBI), but deemed ready to resume normal activities, when compared to those with no similar history of injury (ASYM).
Participants: 35 participants in each group (17 females) were matched for age and body mass index (BMI); (ASYM 36 yrs, BMI 26, LBI 39 yrs, BMI 27).
Methods: Participants performed a controlled lifting task (2.9 kg) in a standing maximum reach position, which altered frontal and sagittal plane moments of force. Electromyographic activity of 24 trunk muscle sites, as well as thoracic and pelvis position via an electromagnetic sensor was collected. Principal component analyses extracted the temporal and amplitude waveform patterns. Mixed model ANOVAs tested for effects ($p < 0.05$) in the main patterns. Preliminary data regarding re-injury status after 1 year was included.
Results: Three principal patterns explained 97% of the variance, with the LBI group demonstrating increased amplitude and a more constant level of activity compared to the ASYM group. The LBI group also demonstrated more thoracic motion in all 3 axes during this highly constrained task. The no re-injury group had lower activation than the re-injury group, but similar temporal patterns.
Conclusions: Despite the perception of readiness to return to work and low pain scores, the temporal and amplitude muscle activation patterns were altered in this LBI group indicating that differences exist compared to a non-low back injured group. The differences are not just relative amplitude differences among muscles but include differences in the temporal response to the flexion moment.

Key Words: low back injury, neuromuscular patterns, principal component analysis, electromyography, temporal waveform patterns

1. Introduction

Disorders of the lower back continue to be a significant musculoskeletal problem worldwide [1] with a huge financial burden related to direct and indirect costs [2]. High indirect costs are partly due to the finding that lower back problems are the most frequently reported cause of activity limitations and sick leave in young to middle age adults [3]. Chronic low back disorders are the most costly with repeat injuries leading to these chronic conditions [4]. While 90% of low back injuries resolve themselves with respect to pain, up to 62 % will re-injure within a year [5]. Whether a predisposing condition exits or whether incomplete recovery leaves individuals vulnerable to re-injury is not clear. In either case, defining metrics that can guide rehabilitation or predict re-injury early after injury may improve return to work decisions.

At present, criteria for return to work are often based on self-reports of symptoms (pain) and functional disability. However, accurate self-report of function is difficult and even healthy individuals could not accurately estimate lifting loads [6]. Furthermore, when pain is present such as in a chronic low back disorder, the ability to perform tasks, rating of pain and exhibits of pain behaviors are altered [7]. Functional capacity tests [8], while more objective, have poor predictive capabilities for re-injury [9]. Furthermore, both approaches capture outcomes but do not assess physiological or biomechanical variations that are indicative of recovery or that reflect compensatory mechanics.

The present study focused on capturing physiological alterations in the trunk musculature as an objective marker of recovery that could provide insight into re-injury mechanisms. The rationale for examining the trunk musculature is three-fold. First, the trunk musculature generates forces

that provide stability and function to the spine and pelvis complex, with both theoretical and modeling work linking trunk neuromuscular alterations to re-injury [10,11]. Second, abdominal and back extensor muscle strength, cross sectional area and activation patterns, including amplitude and temporal characteristics, have been found to differ between healthy asymptomatic and chronic low back pain populations [12-20]. Third, while sufficient strength is needed, coordinated responses among the trunk musculature to the dynamic moments of force during fundamental tasks is considered important to maintain spinal stability and function [21,22]. Despite this, only a few studies have examined the temporal patterns among muscles. Prolonged activity, higher amplitudes and more variation in back extensors were found during walking [23] with asynchronous temporal patterns among abdominal muscles during therapeutic exercises reported for those with chronic low back pain [14]. If amplitude and temporal alterations exist in muscular responses early after injury and before a chronic condition develops, then muscle activation patterns should provide objective information for clinical and return to work decision making.

Minimal muscle activation data has been published that analyses those with a non-chronic low back disorder. MacDonald et al. found that onset times were similar and amplitudes differed in superficial and deep fibres of the multifidus muscle during upper limb and lower limb tasks for those with multiple low back pain episodes that were in remission [24-26]. Based on the analysis of this one muscle, they concluded that absence of pain was a poor indicator of the level of healing. A recent study showed that activation amplitude patterns from a comprehensive set of trunk muscles during a controlled bilateral lift and replace task differed between a low back injured group that was deemed recovered and a group of asymptomatic controls [27]. The

overall activation amplitudes and the relative amplitudes between muscles provide an indication of differences in demand and, to a certain extent, amplitude of co-activation; neither the task nor the analysis approach addressed the issue of coordination or the muscle responsiveness throughout the dynamic task where the external moment is continually changing. In asymptomatic individuals, a highly organized pattern of activity was found for abdominal and back extensor muscles in response to a right to left transfer task that systematically changed frontal and sagittal plane moments on the spine [28]. The task and the pattern recognition analysis provide a model to examine coordination among the trunk musculature and to establish whether trunk muscle activation synergies exist. Changes from a "typical" recruitment pattern, be they amplitude or temporal qualities in even one muscle, can impact spine motion and stability, altering the risk of low back pain [11,20]. So while our previous work found that relative amplitude differences existed which can reflect altered muscle strength, response to passive stiffness or an inhibition due to swelling or pain, we have limited knowledge as to whether the dynamic responses of these muscles (synergies) are different in those less than 12 weeks after injury, but now deemed recovered. Measuring muscle synergies as an objective assessment of recovery and evaluating their predictive capacity for re-injury could improve our ability to manage disorders of the low back before a chronic low back condition develops.

The primary purpose of this study was to compare the trunk muscle amplitude and temporal activation patterns in a group following a low back injury (LBI) that were deemed recovered, to those with no recent history of LBI or pain, during the performance of a controlled, asymmetrical lift and transfer task [28]. We hypothesized that the low back injured group would have increased amplitudes, altered shapes and more asynchronies among muscle sites indicative of

reduced coordination. A secondary purpose was to explore whether differences existed in muscle activation patterns between those that re-injure within one year and those that did not re-injure. We hypothesized that activation patterns from the group that did not re-injure would be different from the group that re-injured and would be similar to the asymptomatic controls. The goal is to provide objective information i) to help target interventions to specific problems and ii) for assessment of injured workers that has predictive capabilities.

2. Methodology

2.1 Participants

Similar to our previous study [27], participants were recruited via advertisements and electronic notices posted at Dalhousie University as well as from physiotherapy clinics. Inclusion criteria for the LBI group was an episode of low back (between ribs and gluteal folds) pain associated with an identifiable event within the 12 weeks prior to their session (mean (SD) = 6.8(±3) weeks) (that resulted in modification of daily activities). Excluded were those whose pain was related to a specific cause such as a fracture, serious disease process such as a tumour or infection, and who had radiological symptoms (i.e. pain) into the lower limbs [29]. At the time of testing, each participant self-reported minimal pain (mean Visual Analogue Score (VAS - 0=no pain, 100 =worst imaginable pain) score: 17.8(±20) mm out of 100), minimal disability (mean Roland Morris disability score: 4.5(±5) out of a possible 124) and perceived they were capable of returning to regular activities. Participants in the asymptomatic group (ASYM) had no recent history of low back pain (within one year) and no report of a LBI/pain resulting in time lost from

work and/or normal activities or requiring medical attention. All participants had no known cardiovascular, neurological or other serious orthopaedic conditions. 35 LBI and 35 ASYM participated in the study. The ASYM group was chosen from a larger group of 60 participants and was matched for sex (17 men, 18 women per group), age and mass with the LBI group (Table 1). Prior to testing, all participants signed an informed consent that was approved by the Health Sciences Research Ethics Board at Dalhousie University. In addition to the test session below, LBI participants were contacted one year later and completed a series of questions related to re-injury.

Table 1 (see below)

2.2 Test procedure

Health screening was done initially over the telephone and then confirmed during testing. Participants attended an initial session, which took place within 2 weeks of the testing session to familiarize them with the protocol. The number of weekly aerobic activity and abdominal training sessions they undertook [30]. Abdominal function ability was assessed using a modified curl-up protocol where the spine was flexed and held for approximately a second. Grading criteria was defined by manipulating arm position to modify task intensity [31]. Participants were given a score of normal (100%), good (80%), and fair (60%) if they could complete the task with hands clasped behind the head, folded across the chest, or by their side respectively. The LBI group underwent postural and neurological assessments as well as a series of tests for instability [32] performed by a physiotherapist; the latter was used to define control impairment. Participants were instructed in the transfer task and collection procedure at this introductory session. The second session took place within 12 weeks post injury for the LBI group. All participants performed a highly controlled right-to-left transfer task, described in previous

studies [28,33]. Briefly, participants stood with their body midline aligned with the centre of the table that was height adjusted to their measured standing elbow height. They performed three trials of the lift, transfer and replace task (2.9 kg) from a standard lift position (60^o right from centre), height lifted (5 cm), and replace position (60^o left from centre) within a standardized 4 second count: lift on 1, midline on 3, replace on 5. Pressure sensors on the bottom of the mass indicated time of lift and replace, with an optoelectric light sensor indicating when the load passed across the midline. These defined 3 phases: right hand transfer (RHT), hand transition (HT) and left hand transfer (LHT). Time to complete each phase and the total time were recorded. Participants maintained a maximum reach (elbow extended) position, and were required to minimize pelvis motion and maintain contact with a tactile feedback sensor placed at the mid thoracic spine, thus minimizing trunk motion [33]. If the timing count was not adhered to or motion was detected (either visually by the tester or from the recorded event and motion traces), the trial was repeated. Quantitative motion was later determined from electromagnetic motion sensor data (described below) to confirm the subjective observation. These constraints resulted in a dynamic task that produced continuously changing flexion and lateral flexion moments on the trunk that were primarily created by the external load.

2.3 Normalization trials

Following the test trials, a series of 8 maximum voluntary muscle contractions (MVCs) were elicited to normalize electromyographic (EMG) amplitudes as a percentage of maximum voluntary effort. These were consistent with previous work [33] and included restrained sit-up, resisted lateral bend (left/right), resisted trunk extension, resisted trunk extension with left/right rotation, and resisted seated rotation (left/right). Exercises were performed in a randomized

order with two trials of the same exercise performed in succession. Each contraction was held for a count of 3-s with a 2-min rest interval between trials.

2.4 Surface EMG data collection and processing

Surface electrodes (Ag/AgCl, 10mm circular electrodes; Meditrace, Graphics Control Canada Ltd) were placed in a bipolar configuration (interelectrode distance of 25mm) over 12 muscle sites bilaterally following standard skin preparation (ratio of skin/amplifier impedance: < 0.1%). Abdominal muscle sites included placement over upper rectus abdominis (URA), lower rectus abdominis (LRA), internal oblique (IO) and 3 sites over external oblique (EO1-3), representing the anterior, lateral and posterior fibers of this muscle, respectively. Posterior sites included erector spinae (ES) at the L1 and L3 levels, both 3cm and 6cm from the midline to represent the longissimus and iliocostalis sites, respectively (L13, L16, L33, L36); as well as over quadratus lumborum (L48) and multifidus (L52). Specific anatomical landmarks used for these electrode sites and supporting literature have been previously described in detail [33]. Electrode placement was validated using a series of manual muscle tests [31,34] with slight changes in placement to accommodate individual anthropometry when necessary. EMG signals were pre-amplified (500X) and further amplified using three AMT-8 EMG systems (band pass 10 – 1000 Hz; CMRR = 115db, input impedance 10GΩ; Bortec Inc., Calgary Alberta). Raw EMG signals and event markers (a step voltage change) were digitized at 2000 samples per second, using a 16 bit resolution National Instruments analog-to-digital conversion board (National Instruments, CA-1000) and Labview^{TM} software (version 7). EMG signals were high pass filtered at 30Hz to remove electrocardiographic artifact [35] then full wave rectified and low pass filtered at 6 Hz using a second order recursive Butterworth filter to yield a linear envelope profile. Data were

time normalized from lift off to replace (100%) using a linear interpolation algorithm, then amplitude normalized to the 500 ms peak amplitude from the normalization exercises [14]. EMG ensemble average waveforms for each participant, muscle and condition (1680 X 101) were entered into a PCA model (Appendix 1) as previously described [28]. Briefly, eigenvector decomposition was performed on the covariance matrix of the original waveform data matrix, resulting in a set of principal components (PCs) that explained the principal patterns of variation in the measured EMG waveforms. For each waveform, a PC score was calculated providing a weighting as to how much that PC contributed to the original waveform. Basically, ensemble average waveforms that are similar in amplitude and shape will have similar PC scores, thus allowing statistical testing of quantitative scores rather than qualitative descriptions of the temporal and amplitude patterns. PCs explaining at least 95% of the variance or greater than 1% were included in the statistical analysis [28].

2.5 Motion Capture

An electromagnetic Flock of Birds™ (FOB) Motion Capture system (Ascension Technology Inc., Burlington, Vermont) recorded the angular motion of the pelvis throughout the exercise task in 3D with respect to a global coordinate system (x = transverse, y = sagittal, z = frontal plane). One sensor was placed superior to the left anterior superior iliac crest, the second over the T8 spinous process, thus measurements were not directly related to anatomical references. Maximum angular displacement for each of the three phases was calculated, providing a quantitative measure of how much motion occurred and validating the visual observation during testing.

2.6 Re-injury / no re-injury status

The participants in the LBI group were contacted by telephone approximately one year after their initial session with a follow-up questionnaire. Specifically, they were asked if they experienced a re-injury episode to their low back resulting in lost time from work and/or normal activities or requiring medical attention during the year.

2.7 Statistical Analysis

Student *t*-tests were performed to compare between-group differences in demographic and anthropometric data. Kinematic data for each FOB sensor in 3 orthogonal planes, and the time to complete each movement phase, were compared between groups using a mixed model analysis of variance (ANOVA) (Group*Phase). Mixed model ANOVA analyzed Group*Muscle differences of the PC scores. Pairwise comparisons were made on significant effects using a Tukey simultaneous test. Normality was confirmed using a Kolmogorov-Smirnov test, with non-normal data being transformed using a Johnson Transformation. Statistical analyses were performed in Minitab (Minitab Inc, State Collage, PA, version 16), with $\alpha = 0.05$. Abdominal and back muscle PC scores were analyzed separately (Appendix 1). For the follow-up data, descriptive statistics were calculated for the LBI participants that reported a re-injury and those that did not re-injure after 1 year. A Group*Muscle ANOVA was also performed on these data, comparing the two LBI groups with the ASYM to provide a preliminary analysis.

3. Results

3.1 Timing and motion variables

The total time to complete the task was 4±0.3s for the ASYM and 4±0.4s for the LBI group. There were no significant (p>0.05) differences in time to complete the task or phases between the two groups (Table 2). Motion for each phase for both sensors is found in Table 3. The only significant group main effect for the iliac crest marker indicated greater motion for the LBI group in the transverse plane (axial rotation). However, both groups had minimal pelvic motion in all three directions (<2°) within each phase. The LBI group demonstrated a main effect of greater frontal plane motion at the T8 marker (p<0.05). There was a significant Group*Phase interaction for the T8 marker in the sagittal and transverse planes. Post-hoc analysis indicated increased sagittal and transverse motion during the hand transition phase in the LBI group compared to the ASYM group. The between group differences were all less than 1° and the motion within each phase was less than 3°. Hence, these motions would have minimal effect on the external moments in the sagittal and frontal planes.

Table 2 and Table 3 (See below)

3.2 EMG waveform patterns

Example EMG waveforms for the abdominals and back extensors (Figure 1) for both groups show qualitatively that LBI participants had higher overall EMG activation levels compared to ASYM for both abdominals and back extensors. Qualitative differences among muscle sites for each group indicate muscle specific responses to the changing external moment (Figure 1). For example, RA had a constant activation level throughout the task whereas REO3, the most lateral abdominal muscle site, demonstrated high activity near the end of the task, responding to the increased left lateral flexion moment (Figure 1a). This increased activity near the end of the task was also more pronounced in the right lateral back extensors RL16 and RL36 (Figure 1b),

whereas the more medial back extensor sites had higher activation levels mid-task (L52, L33), responding to the increased flexion moment during hand transition.

Figure 1 (See below)

Three principal patterns explained 97% of the variance of the combined back and abdominal muscle activity (Figure 2). The linear combination of these three scores and corresponding PCs capture salient features with low reconstruction error compared to the original measured waveforms. Means and standard deviations of PC scores for all abdominal and back extension muscles for both groups are found in Tables 4 and 5 respectively. ANOVA results are in Table 6. Significant group main effects ($p<0.05$) were found for PC1 and PC3 scores for both the abdominal and back extensors and significant muscle effects ($p<0.05$) for all three PC scores for both muscle groups. There were no significant group*muscle interactions ($p>0.05$).

Figure 2 and Table 4, 5 and 6 (See below)

PC1, explaining 85.5% of the variance, captured the main pattern, with low activation at the beginning and end of the task, with a gradual rise and decline consistent with the flexion moment pattern mid-task (Figure 2a). Higher scores are associated with higher overall amplitudes of activity (see high-low scores in Figure 2b). For both abdominal and back muscles, PC1 scores were significantly higher for the LBI participants (Tables 4 and 5). Muscle main effects are illustrated on Figure 2c. Post hoc results showed that for the back extensors, the multifidus (L52) had the highest amplitudes, with the lower and more lateral sites (L36 and 48) significantly lower than most back extensor sites (Figure 2c, Table 7). For the abdominal sites, the IO PC1 scores were significantly higher than all other abdominal sites for both groups, and the EO sites had

higher PC1 scores than RA (Figure 2c,Table 7). Variability for the LBI group for most abdominal and back extensor sites was higher than the ASYM group, with the abdominal variability twice as high in the LBI group for all muscles except IO (Tables 4,5).

Table 7 (See below)

PC2 captured the muscle response to the changing lateral flexion moment (Figure 2d). A positive score depicts a pattern of low activity initially and then a gradual increase in activity as the lateral flexion moment moves from right to left, as illustrated for the high-low scores in Figure 2e. A negative PC2 score indicates the opposite pattern. There was a muscle main effect in both the back and abdominal muscles ($p<0.05$). Post hoc analysis revealed significant differences between sides for each back extensor muscle site, with all left sided PC2 scores negative, whereas the right sites had positive scores (Figures 2f, 3d). L16 PC2 scores were significantly greater in absolute magnitude than all other ipsilateral sites, whereas the medially situated L52 sites were the lowest (Figure 2f, Table 7). PC2 abdominal scores were lower and less asymmetrical than those in the back, with some right-left differences for the oblique sites (Figure 2f, Table 7).

Figure 3 (See below)

PC3, explaining 1.5% of the variance, captured a feature that corresponds to the change in flexion moment (Figure 2g). Positive PC3 scores would result in an increased relative response to the flexion moment during mid-task, thus the response differential between early/late and mid-task would be greater (Figure 2h). Figure 3a shows the differences in the shape of curves with a positive PC2 score (RL48) versus a positive PC3 score (RL52). A negative PC3 score would

result in the opposite effect and in linear combination with PC1 and PC2 would flatten the response as shown for the low score in Figure 2h and the REO2 example in Figure 3b. For both abdominals and back extensors, the LBI group had lower scores, as illustrated for the RL33 in Figure 3c. The 3 medial back extensor sites (L13, L33, L52) had significantly higher PC3 scores than most of the more lateral sites (L16, L36, L48) (Figure 2i, Table 7). All PC3 scores in the abdominal muscles were negative, with the more medial RAs and REO1 being significantly higher than the EO2,3 or IO sites (Figure 2i).

3.3 Re-injury / no-re-injury comparison

Follow up data were obtained from 27 participants (77%) in the LBI group. These data show that the group that re-injured within a year of testing (n = 14) were older than those that did not re-injure (n = 13), but the difference was not significant, and had higher VAS pain scores and Roland Morris Disability scores at time of testing, although these values still indicate low pain and minimal disability. The re-injury group also had higher percentages of men as well as those who were classified with control impairment [32] (Table 1).

The mean and SD for the PC scores for the abdominals and the back extensors are in Tables 4 and 5, respectively. There were significant group and muscle main effects for PC1 and PC3, and significant muscle effects for PC2 for both muscle groups. For PC1, all three groups were different from each other, with the ASYM group having the lowest and the re-injury group the highest PC1 scores for both muscle groups. PC3 scores were only different between the ASYM and the two LBI groups. No interactions were significant, although PC2 for back extensors approached significance ($p=0.116$) (Table 6).

4. Discussion

The two samples were well matched based on demographic characteristics, with the mean ages of 35 and 39 years directly within the age range where low back complaints are most prevalent in the general population. Since previous work found sex differences in activation patterns during this transfer task [28], matching for sex minimized confounding factors for between group differences. Overall, the pain levels and disability reported by the LBI group were low (Table 1), supporting the recovered classification. Finally, the highly controlled task minimized differences in task performance, with both groups demonstrating compliance with the time and motion constraints. The greatest motion (less than 3°) was in the transverse plane (axial rotation), which would have minimal effect on the magnitude of the moments of force acting on the spine in the sagittal and frontal plane. The significant differences between the groups for the transverse and sagittal plane motions during the transfer (HT) phase only, were small (<1degree), and the low variability associated with the task constraints explains why this small difference was significant. The latter should have minimal effect on the flexion moment magnitude, as similar anthropometrics and timing characteristics between groups should result in similar moments of force. However, what was interesting was that the LBI group muscle patterns did not respond to the flexion moment with a similar relative increase in activity as the ASYM group (i.e. consistently lower PC3 scores).

The general shape of PC1 illustrates moderate activation at the beginning of the task in response to the lateral flexion/flexion moments. As the load is transferred at the mid-line, the flexion moment peaks, which is reflected by an increased PC1. This is followed by a more distinct drop in activity while lowering the load as it is moved to the left (Figure 2a). The overall higher

muscle activation amplitudes (PC1 scores) for both abdominals and back extensors is consistent with the higher amplitude pattern reported for a recovered LBI group during the lifting phase of a bilateral lift and replace task [27]. In fact, the pattern for the PC1 scores among muscles is consistent for the most part with the normalized root mean squared amplitude pattern presented in that paper [27]. This increased activity in the LBI group could be in response to decreased passive trunk stiffness [22,43-46] requiring increased muscle activation to produce a given level of stiffness. While this need for increased active stiffness is consistent with the three subsystem theory proposed by Panjabi [10], it could also be the result of decreased muscle strength in both muscle groups as previously reported in people with chronic low back pain [18]. We did not measure muscle strength, although both groups did have similar abdominal function test results. However, the increased axial and sagittal plane thoracic motion during the hand transfer phase and overall for the frontal plane in the LBI group, suggests that the control was not evident for the thoracic spine. Control of the thorax has been shown to rely on different muscles and activation levels than pelvis control, with IO and the medial aspect of EO having more of an effect on the pelvis than the thorax, irrespective of directionality [47].

More important to this study were the temporal patterns that captured the responsiveness of the muscle throughout the dynamic task and hence coordination of activity among muscles. The two main features were the response to the lateral flexion moment (PC2) and an additional response to the flexion moment (PC3) (see Figures 2d,e,g,h). Since there were no significant interactions, the LBI and the ASYM groups responded similarly although the values in Tables 4 and 5 show how specific muscle sites influenced these significant differences.

For this transfer task, activation patterns for both groups were related to specific regions of the back extensors and with precise fiber orientations of the abdominal muscles, as previously reported for healthy no low back injured men and women [28]. The task was similar to what is used in functional capacity tests and is a basic load transfer and placement task fundamental in occupations, such as cashier or assembly line worker [48]. In many occupational tasks, the loads are not necessarily heavy, but the work can be repetitive, requiring the trunk musculature to continually adjust to the changing moment direction [49,50]. Constraining this task in the laboratory by minimizing trunk and pelvis motion, while standardizing the timing and mass lifted, reduced the kinematic and kinetic variables as confounders to the muscle responses. In contrast to our hypothesis based on findings from a chronic low back pain group [14], the LBI group did not have more asynchronous patterns than the ASYM group. The lack of significant differences for the PC2 scores, the significant group difference for the PC3 scores and no significant interactions indicate that the LBI group used a similar coordinated temporal pattern of activation as the ASYM group to complete the task. Also while the load was low, the task in maximal reach increased the degree of difficulty, thus reducing the degrees of freedom in activation patterns to complete the task. Perhaps a lower demand task, such as was used for the abdominal challenge in the previous study [14], would result in more differences among muscles. What was noted was that the variability was greater in the LBI group for all PC1 scores for both muscle groups, and for the abdominals this was twice as high for most sites (Tables 4,5). For the temporal features (PC2 and PC3 scores), variability was similar between groups, with only specific muscles (more cephalaud right sided back muscles for PC2 and L52 for PC3) having higher variability in the LBI group. This suggests that perhaps a range of demands should be examined, including tasks with lower demands to determine responses across a

spectrum. For example, subtle alterations in patterns such as the highly synchronized REO2 and REO3 waveforms for the ASYM (Figure 1a) but less so in the LBI group (Figure 1c) could be detected with a larger or more homogeneous LBI sample (as supported by the similar PC1, PC2 and PC3 scores in the ASYM group compared to the LBI in Table 4). Similarly, the lateral back extensors (RL36 and RL48) were highly synchronized in the ASYM group (Figure 1b) but not the LBI group (Figure 1d), as evidenced by the differences in PC1 and PC2 scores (Table 5). This could also reflect a power issue, given the size of the ANOVA model and variability in the LBI group.

In response to the changing flexion moment, the relative increase in activation mid-task for the ASYM group occurred when the flexion moment was highest, whereas the lower PC3 scores for the LBI group indicates a reduced response for both the abdominals and back extensors. This is illustrated by a longer duration of a constant level of activation (see Figure 2h and examples in Figures 3b,c) throughout the task even though the moment demands were changing. This difference was primarily influenced by the more medial sites longissimus (RL33) and multifidus (RL52) (Table 5) with the more pronounced drop off in activity from 60-100% time in the ASYM group compared to the constant level of activation from 60%- approximately 90% time for the LBI group (Figures 3b,c). While the between-group differences in thoracic motion were small (Table 3), the required task was highly constrained, with participants coached to minimize trunk motion with tactile feedback provided to minimize trunk motion. Thus, one would expect that this small change in motion could be larger in an unconstrained situation such as the work place. The diminished response to the flexion moment in the LBI group could reflect reduced proprioception, as previously reported in people with chronic low back pain [39]. Given the

repetitive nature of many workplace situations, the cumulative effect of this increased spine motion, paired with a constant level of activation, could lead to increased risk of injury, be it first time or repeat [40-42]. Whether this lack of response is negative and could threaten spine stability, which relies on precise neuromuscular control of all trunk muscles [10,36-38], or whether it is an adaptive response, requires further exploration. The follow up data provides some initial insights to address this issue.

The variability in the activation pattern data can in part be explained by the follow-up data and the clinical instability classification. More importantly, it provides preliminary findings that specific muscle activation pattern differences may be related to recovery and prediction of re-injury risk. Although the follow-up groups were small, with large variability reducing the statistical power, significant group effects were found for the overall amplitude (PC1) among all three groups, for both abdominal and back extensor muscles (Table 6). This suggests that the higher relative amplitudes, which could relate to decreased muscle strength and increased magnitude of co-activity, may not be an adaptive response. Increased muscle activity, in turn, places the trunk muscles at risk of fatigue as they are working at a higher percentage of maximum. Furthermore, there were trends for significant interactions in the temporal scores. No systematic differences between the re-injury and no re-injury groups for the two temporal patterns suggests that specific muscles may be affected differently, warranting further examination in a larger sample. For PC3, the group differences were between the ASYM and the two LBI groups for the abdominals only. For the back extensors, however, at muscle sites such as the more medial right sided RL13, RL33, RL52, the ASYM PC3 magnitudes were two-three times those of the no-re-injury group, whereas the left sided sights showed minimal differences.

A power analysis for the back extensors showed that a sample size of 56 would be needed for a significant difference between the ASYM and re-injury group. The re-injury group had the highest mean amplitudes (PC1), but their back extensor response to the flexion moment (PC3) was more similar to ASYM than the no re-injury group (Table 5). Thus, the no re-injury group had lower back extensor amplitudes but a more constant response in specific muscles, which perhaps provides evidence that this is an adaptive response. In contrast, the PC2 scores for the no re-injury group back extensor sites were closer to the ASYM group than the re-injury group for most muscles indicative of a similar temporal response to the lateral moment. Of note is that the re-injury group had a higher percentage of those classified with control impairment (46% vs 17%) than the no re-injury group (Table 1). Thus, the control impairment status should be further examined as a factor in recovery assessment and predictive models. There is minimal data quantifying objective physiological metrics early after a low back episode. These results therefore provide foundational data suggesting that muscular activation features provide objective information that should be assessed during recovery and included in predictive models of re-injury.

4.1 Study limitations

There is potential for EMG cross-talk, but care was taken in electrode placement, validation and heart-rate removal, according to published protocols [33,35]. While the ability to produce true maximal activation when an individual is in pain has been questioned, at the time of the testing, all LBI participants perceived that they were recovered from their injury, reported low pain scores, low disability scores and none reported pain during the MVC protocol. The LBI group had 13% lower raw amplitudes for the maximum normalization exercises, but raw amplitudes

should be interpreted with caution as other factors such as tissue volume conducting differences can affect amplitude. Furthermore, a systematic difference in normalization amplitudes for the low back injured group would only affect the amplitude values during the task (i.e. PC 1 scores). While the percentage differences in the amplitudes (PC1 scores) during the task between groups were significant the differences were larger than 13% for most muscles, and they were not systematic. Thus PC1 score differences could not all be explained by differences during normalization. Muscle strength measures for both groups and control impairment assessment for the ASYM group would have helped with interpreting the differences between groups.

5. Conclusion

This study found that trunk muscle amplitudes and temporal activation patterns differed from asymptomatic controls in a group of participants who had reported a low back injury within the past twelve weeks, but self-reported recovery and readiness to resume normal activities. While both groups were well matched for demographics, evidence of altered muscular activation patterns in the LBI group was found. Although small, the LBI group demonstrated more thorax motion in all three planes and decreased muscular responses to the mid-task increased flexion moment. Higher overall trunk musculature activation amplitudes in the LBI group and the more constant level of activity throughout the task (decreased response to the flexor moment) were the key differences from the ASYM group. Temporal responses to the lateral flexion moment (PC2) were similar between groups, thus the low back injured group produced the task with highly coordinated temporal patterns among trunk muscles similar to controls. Preliminary findings showed that the no-re-injury group had lower overall abdominal and back extensor activation and more constant activation levels than the re-injury group, with a trend for reduced

coordination that should be further examined. Thus, alterations in neuromuscular activation patterns exist even when pain and functional outcomes appear to be recovered, shedding light on adaptations to injury and potential re-injury mechanisms that might be useful to populate guidelines for return to work decisions.

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Figure Captions

Figure 1: Ensemble average waveforms for the *right* sided back extensor (a,c) and abdominal (b,d) muscles. The upper two graphs represent data from the ASYM group (no recent history of low back injury within the past year), and the lower two graphs are from a group of individuals who recently suffered a low back injury, but now were deemed ready to return to work (LBI).

Figure 2: Principal components (feature) for (a) PC1, (d) PC2 and (g) PC3. PC1 explains 85.5% of the waveform variance with PC2 and 3 explaining 10.1% and 1.5%, respectively. Ensemble average waveforms of the 5 highest (black line) and 5 lowest (grey line) scores for PCs 1 – 3 (b,e,h, respectively) are shown to aide with interpretation. PC 1 – 3 scores for muscle main effect are graphed in c,f,i, respectively.

Figure 3: Sample ensemble average waveforms of specific muscle comparisons; solid lines indicate the ASYM group, dashed lines the LBI group. (a) RL48 (black lines) compared to RL52 (grey lines): demonstrates the difference in curve shape between a muscle with a positive PC2 score (RL48) versus one with a positive PC3 score (RL52). (b) REO2: note the LBI group demonstrated a lack of rise in amplitude in the latter part of the task compared to the ASYM group, in keeping with their lower PC2 score. (c) RL33: the LBI group demonstrated more constant activation in the latter half of the task, in keeping with higher PC2 and lower PC3 scores compared to the ASYM. (d) RL16 (black) and LL16 (grey): demonstrates the sidedness of contralateral muscle activations, as evidenced by the opposite polarities of their PC2 scores.

Figure 1

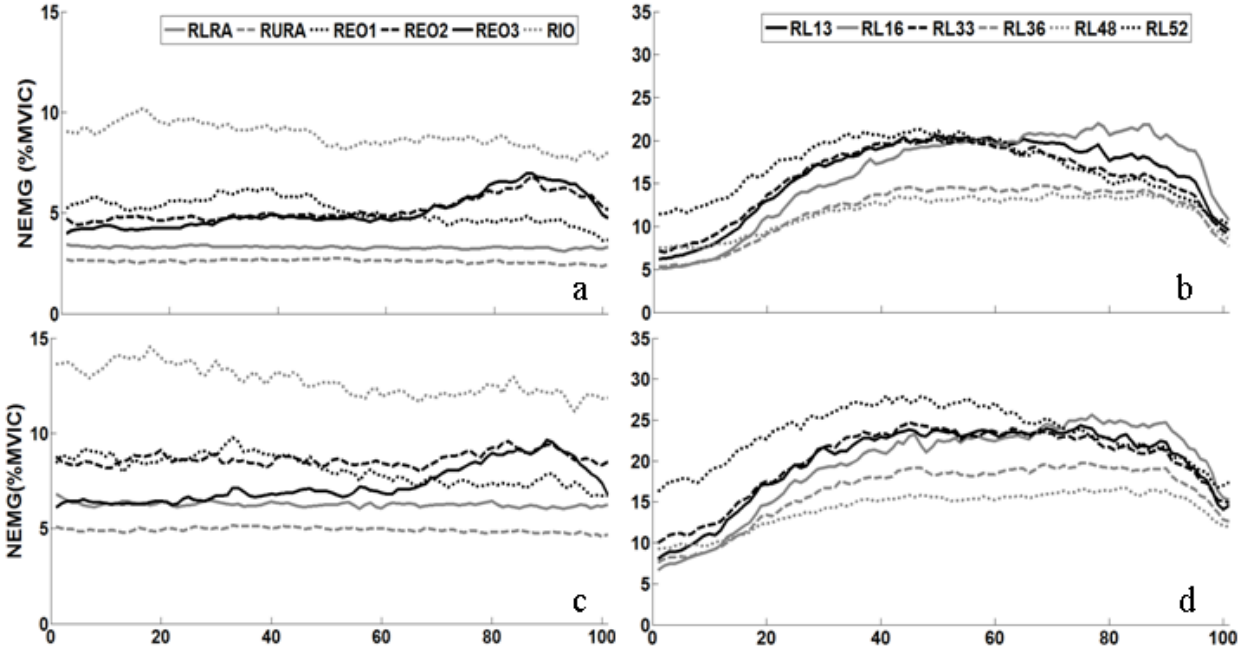

RLRA
RURA
REO1
REO2
REO3
RIO
NEMG (%MVIC)
a
RL13
RL16
RL33
RL36
RL48
RL52
b
NEMG(%MVIC)
c
d

Figure 2

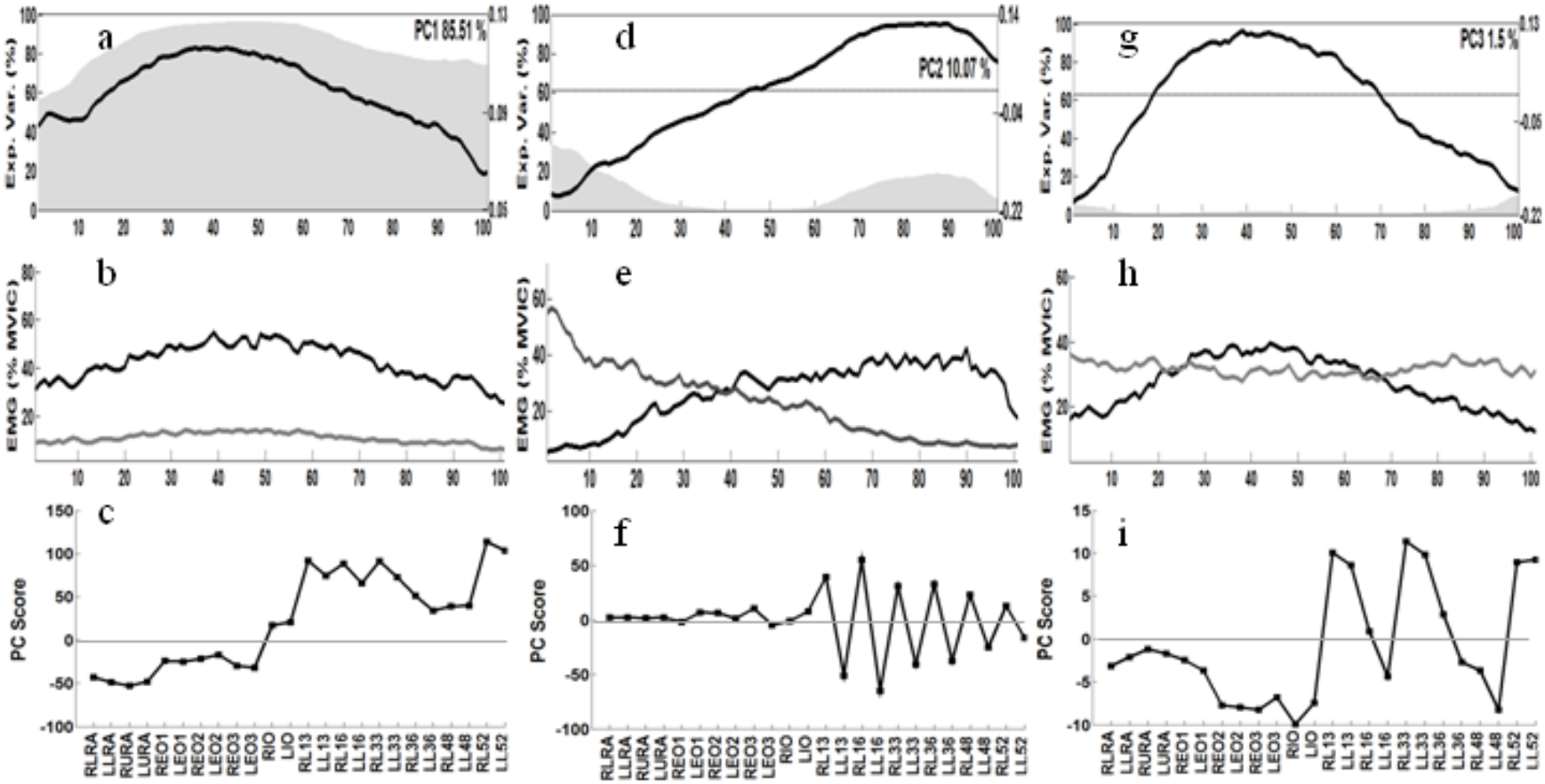

a
PC1 85.51 %
Exp. Var. (%)
b
EMG (% MVIC)
c
PC Score
d
PC2 10.07 %
e
f
g
PC3 1.5 %
h
i
RLRA
LLRA
RURA
LURA
REO1
LEO1
REO2
LEO2
REO3
LEO3
RIO
LIO
RL13
LL13
RL16
LL16
RL33
LL33
RL36
LL36
RL48
LL48
RL52
LL52

Figure 3

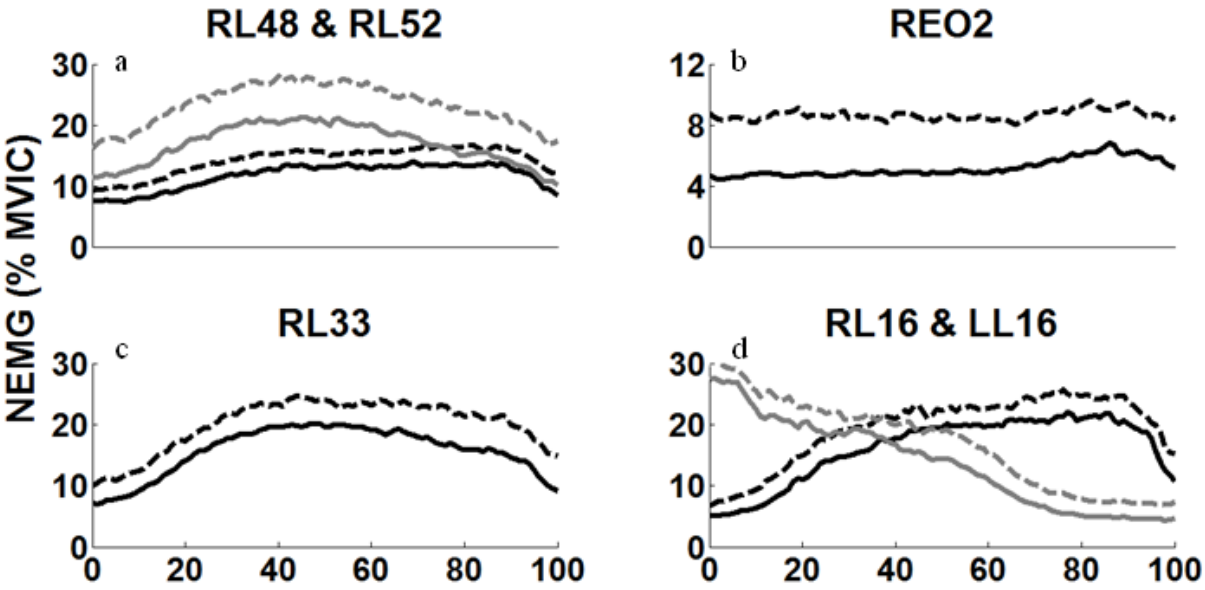

RL48 & RL52
REO2
RL33
RL16 & LL16
a
b
c
d
NEMG (% MVIC)
30
20
10
0
12
8
4
0
0
20
40
60
80
100

Table 1: Demographic table comparing the low back injured (LBI) group to one which had no history of low back injury (ASYM). "Re-injury" and "No Re-injury" indicate verbal indication of whether these subsets of the LBI group re-injured their back within the year follow-up. BMI = body mass index; RMD = Roland Morris Disability Index; Pain Cat = Pain Catastrophizing scale; VAS = Visual Analog scale. Shading = significant between group difference ($p < 0.05$) with post hoc showing that the ASYM was younger than the LBI: Re-injury group.

Variable	**ASYM (n = 35)**	**LBI (n = 35)**	***p*-value**	**LBI: Re-injury (n = 14)**	**LBI: No-Re-injury (n = 13)**	***p*-value**
Age	35.5(10)	39.6(12)	0.16	44.5(10)	38.8(14)	0.04
Mass	76.7(15)	79.3(21)	0.46	81.3(22)	77.1(23)	0.72
Height	171.7(8)	170.3(9)	0.55	171.0(10)	168.3(5)	0.52
BMI	25.9(4)	27.2(6)	0.24	27.6(6)	27.2(8)	0.45
Aerobic Activity (per wk)	3.4(3)	2.3(2)	0.06	2.3(3)	2.4(2)	0.33
Abdominal Training (per wk)	2.3(3)	2.5(3)	0.74	2.3(3)	2.9(3)	0.82
RMD	NA	4.5(5)		6.3(5)	2.3(3)	
Pain Cat	NA	13.2(11)		13.7(11)	11.8(7)	
VAS	NA	17.8(20)		25.7(22)	16.1(21)	
Norm. Ab Function Test (%)	70	67		60	67	
Control Impairment (%)	0	34.2		46	17	
Female (%)				53	69	

Table 2: Mean (SD) for total time to complete the task and for individual phases. LBI = low back injured group, ASYM = no recent history of low back injury. RHT = right hand transfer; HT = hand transition; LHT = left hand transfer.

	Total(s)	RHT(s)	HT(s)	LHT(s)
LBI	4.0(0.4)	1.4(0.3)	1.0(0.3)	1.6(0.3)
ASYM	4.0(0.3)	1.5(0.2)	0.8(0.2)	1.6(0.2)

Table 3: Maximum angular displacement in degrees for the three orthogonal axes: mean (SD). LBI = low back injured group, ASYM = no recent history of low back injury. RHT = right hand transfer; HT = hand transition; LHT = left hand transfer. * = significant between-group difference from post hoc of interaction; shading indicates a significant group main effect (p<0.05).

	T8 sensor			Iliac crest sensor		
	frontal	**sagittal**	**transverse**	**frontal**	**sagittal**	**transverse**
LBI RHT	1.3(0.9)	1.7(1.3)	2.8(1.5)	1.2(0.8)	0.9(0.7)	1.8(1.0)
ASYM RHT	0.9(0.4)	1.6(1.3)	2.4(1.5)	1.4(1.2)	0.9(0.9)	1.6(1.0)
LBI HT	0.9(0.5)	1.4(0.9)	2.4(1.7)	0.5(0.6)	0.3(0.2)	1.1(0.6)
ASYM HT	0.6(0.4)	0.9(0.8)*	1.6(1.3)*	0.5(0.4)	0.4(0.2)	0.7(0.6)
LBI LHT	1.4(0.6)	1.8(1.4)	2.9(1.5)	0.8(0.6)	0.6(0.4)	1.7(0.9)
ASYM LHT	1.3(0.8)	1.7(1.3)	2.9(2.1)	0.8(0.7)	0.6(0.4)	1.4(1.0)

	T8 sensor			Iliac crest sensor		
	frontal	**sagittal**	**transverse**	**frontal**	**sagittal**	**transverse**
LBI RHT	1.3(0.9)	1.7(1.3)	2.8(1.5)	1.2(0.8)	0.9(0.7)	1.8(1.0)
ASYM RHT	0.9(0.4)	1.6(1.3)	2.4(1.5)	1.4(1.2)	0.9(0.9)	1.6(1.0)
LBI HT	0.9(0.5)	1.4(0.9)	2.4(1.7)	0.5(0.6)	0.3(0.2)	1.1(0.6)
ASYM HT	0.6(0.4)	0.9(0.8)*	1.6(1.3)*	0.5(0.4)	0.4(0.2)	0.7(0.6)
LBI LHT	1.4(0.6)	1.8(1.4)	2.9(1.5)	0.8(0.6)	0.6(0.4)	1.7(0.9)
ASYM LHT	1.3(0.8)	1.7(1.3)	2.9(2.1)	0.8(0.7)	0.6(0.4)	1.4(1.0)

Table 4: Mean (SD) PC scores for the abdominal muscles, by group and muscle. ASYM = no recent history of low back injury; LBI = low back injured group; "no re-I" = subgroup of the LBI group who had not re-injured one year later (n = 13); "re-I" = subgroup of the LBI group who re-injured within one year post-testing (n = 14). See Methods section for muscle definitions. Bold indicates pairs where the variance in the LBI group was ≥ twice that of the ASYM group, except when ASYM variance was = 1.

PC	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO	Mean (SD)
1 ASYM	**-57.6(38)**	**-61.5(27)**	**-64.4(19)**	**-60.0(25)**	**-38.7(32)**	**-40.7(29)**	**-39.3(30)**	**-40.4(30)**	**-40.5(28)**	**-45.3(31)**	-2.1(46)	7.6(57)	-40.2(40)
1 LBI	**-28.1(77)**	**-36.5(62)**	**-41.3(46)**	**-36.7(51)**	**-9.1(67)**	**-9.2(68)**	**-4.4(78)**	**6.1(97)**	**-18.4(59)**	**-18.8(66)**	36.7(76)	34.2(78)	-10.4(73)
1 no re-I	-36.6(75)	-36.3(83)	-51.2(35)	-44.5(41)	-13.4(56)	-16.0(48)	-7.8(77)	-1.8(80)	-25.9(32)	-30.1(43)	28.3(83)	25.0(82)	-17.5(61)
1 re-I	-12.1(94)	-36.2(49)	-29.9(61)	-29.3(66)	9.9(83)	8.5(89)	2.9(78)	17.6(107)	-8.5(81)	3.7(91)	57.0(79)	58.8(87)	3.5(84)
2 ASYM	1.7(2)	1.7(1)	1.4(1)	1.9(1)	-1.6(9)	7.6(8)	7.3(10)	-1.0(8)	9.4(9)	-2.9(6)	-0.9(5)	7.5(6)	2.7(7)
2 LBI	2.3(2)	2.4(1)	2.0(2)	2.6(3)	-2.6(10)	6.1(9)	4.8(10)	3.2(6)	11.3(11)	-6.4(9)	-1.7(9)	7.4(8)	2.6(9)
2 no re-I	2.4(2)	2.5(2)	1.7(1)	2.1(2)	-0.4(8)	4.2(9)	7.5(14)	0.9(6)	14.6(15)	-7.7(10)	-0.8(10)	5.9(10)	2.7(7)
2 re-I	2.9(2)	2.6(1)	2.3(2)	3.1(3)	-2.7(13)	8.9(11)	3.3(9)	5.5(7)	10.6(9)	-7.8(9)	-2.6(11)	8.7(7)	2.9(9)
3 ASYM	**-1.2(5)**	**-0.4(3)**	**0.2(2)**	**-0.3(3)**	**-0.3(4)**	-3.6(6)	-6.2(8)	-5.2(8)	-6.4(7)	-5.0(6)	**-6.9(6)**	-4.5(6)	-3.3(6)
3 LBI	**-5.1(10)**	**-3.8(8)**	**-2.5(6)**	**-3.1(7)**	**-4.5(8)**	-3.7(11)	-9.2(11)	-10.7(15)	-10.1(10)	-8.5(8)	**-13.0(12)**	-10.3(9)	-7.0(10)
3 no re-I	-4.1(9)	-3.9(11)	-1.6(4)	-2.6(5)	-4.5(7)	-5.6(8)	-11.2(12)	-9.6(10)	-11.7(10)	-9.1(8)	-12.9(15)	-10.2(11)	-7.2(9)
3 re-I	-7.2(13)	-3.7(6)	-3.8(8)	-3.8(9)	-5.5(10)	-1.3(16)	-8.4(8)	-12.3(19)	-9.5(11)	-9.6(9)	-15.2(12)	-12.2(8)	-7.7(12)

Table 5: Mean (SD) PC scores for the back extensor muscles, by group and muscle. ASYM = no recent history of low back injury; LBI = low back injured group; "no re-I" = subgroup of the LBI group who had not re-injured one year later (n = 13); "re-I" = subgroup of the LBI group who re-injured within one year post-testing (n = 14). See Methods section for muscle definitions. Bold indicates pairs where the variance in the LBI group was ≥ twice that of the ASYM group, except when ASYM variance = 1.

PC	RL13	LL13	RL16	LL16	RL33	LL33	RL36	LL36	RL48	LL48	RL52	LL52	Mean(SD)
1 ASYM	73.4(65)	54.2(60)	71.5(81)	49.5(68)	69.7(94)	55.4(84)	30.0(82)	17.8(70)	26.6(76)	22.4(68)	81.7(97)	74.8(85)	52.2(80)
1 LBI	110.7(101)	94.3(79)	104.9(104)	82.2(78)	112.6(113)	89.9(98)	72.7(95)	50.1(88)	52.0(81)	57.2(98)	146.0(123)	132.2(137)	92.1(104)
1 no re-I	102.7(100)	106.4(102)	98.8(94)	89.7(95)	116.4(120)	107.6(120)	70.2(107)	55.2(98)	54.6(89)	45.3(102)	161.4(149)	132.5(145)	95.1(110)
1 re-I	139.7(107)	101.4(58)	134.9(111)	91.6(52)	145.4(112)	109.4(85)	105.7(96)	76.7(88)	66.4(84)	89.3(108)	156.1(107)	171.3(148)	115.7(101)
2 ASYM	36.5(20)	-50.2(27)	53.8(32)	-65.8(43)	**26.4(13)**	-42.1(29)	29.1(17)	-35.6(25)	21.8(15)	-22.7(23)	10.4(13)	-18.7(19)	-4.8(45)
2 LBI	42.2(31)	-52.2(29)	55.9(36)	-64.5(34)	**34.9(29)**	-39.7(22)	36.4(25)	-40.3(23)	24.4(21)	-27.6(29)	15.3(15)	-14.0(18)	-2.4(48)
2 no re-I	35.3(21)	-51.7(29)	50.6(27)	-61.2(31)	29.5(20)	-42.9(29)	30.9(13)	-39.8(26)	23.5(13)	-28.2(29)	13.3(19)	-7.6(23)	-4.0(23)
2 re-I	50.7(37)	-56.8(28)	64.5(42)	-71.1(26)	45.1(36)	-42.4(15)	48.1(34)	-47.4(23)	32.1(27)	-36.0(30)	17.7(15)	-20.2(14)	-1.3(55)
3 ASYM	13.1(14)	9.2(13)	2.5(14)	-4.6(16)	15.7(14)	12.1(10)	5.1(7)	-1.3(9)	-1.7(7)	-7.4(14)	**12.9(13)**	11.4(11)	5.6(14)
3 LBI	7.0(11)	8.0(12)	-0.7(13)	-4.2(13)	7.1(16)	7.4(13)	0.7(8)	-4.1(13)	-5.7(8)	-9.0(11)	**5.0(23)**	7.1(17)	1.6(15)
3 no re-I	5.7(14)	6.7(14)	-2.8(16)	-2.5(11)	5.5(15)	5.5(15)	0.6(11)	-4.3(12)	-6.0(12)	-10.0(13)	1.6(31)	2.0(22)	0.2(16)
3 re-I	9.3(11)	7.5(13)	2.1(13)	-8.0(15)	9.5(13)	9.5(12)	2.0(8)	-5.2(17)	-4.2(5)	-11.5(10)	6.5(21)	10.3(15)	2.3(15)

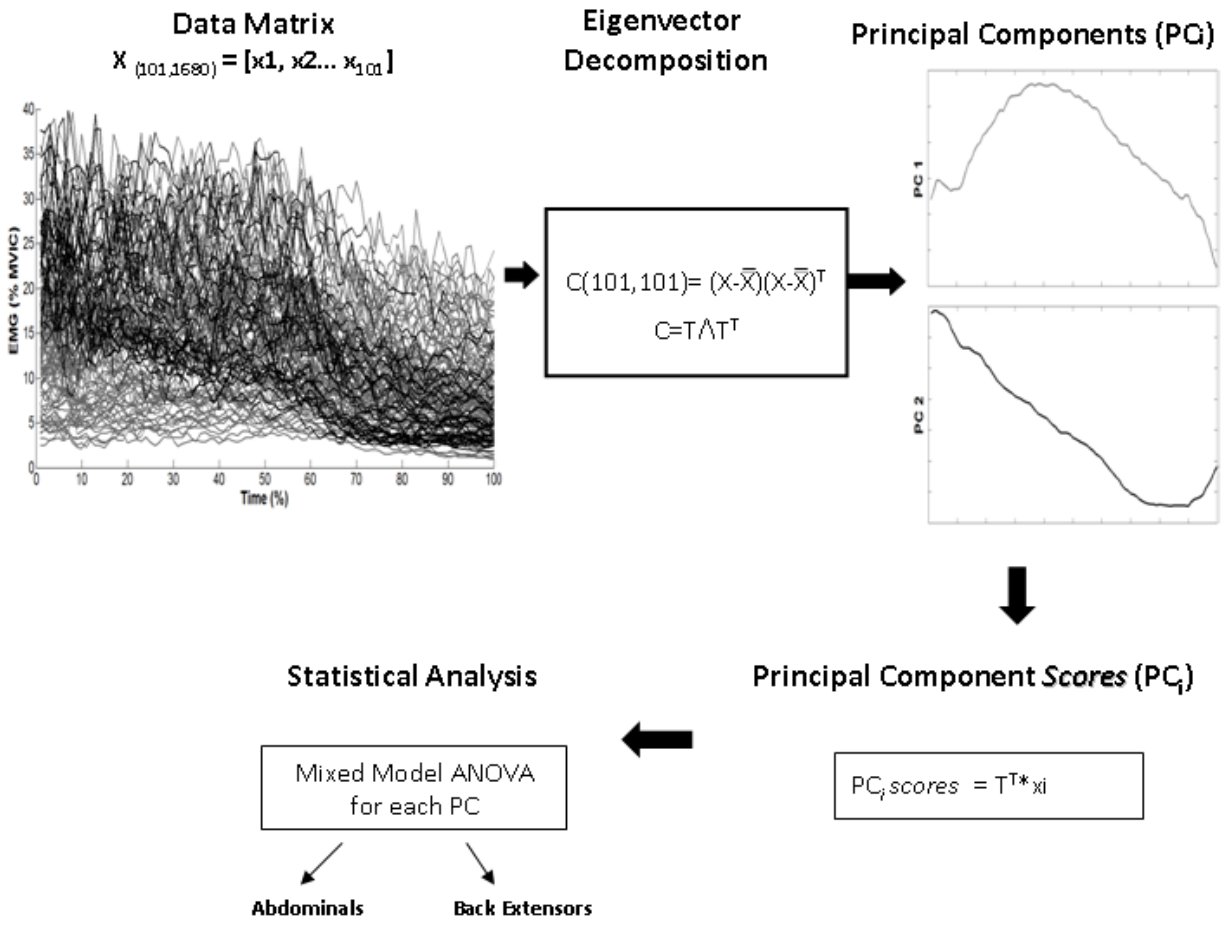
Table 6: Significance results for the Group*Muscle ANOVAs. Post-hoc results for group main effects comparing ASYM/re-injury/no re-injury indicate the following: PC1abs: ASYM < no-re-injury < re-injury; PC3abs: ASYM < no-re-injury & re-injury; PC1back: ASYM< no-re-injury <re-injury; PC back, while not significant, re-injury; showed a trend of ASYM<no-re-injury & re-injury.

	Abdominals			Back Extensors		
ASYM/LBI (n=70)	**PC1**	**PC2**	**PC3**	**PC1**	**PC2**	**PC3**
Group	p=0.014	p=0.920	p=0.008	p=0.020	p=0.640	p=0.056
Muscle	p<0.001	p<0.001	p<0.001	p<0.001	p<0.001	p<0.001
Group*Muscle	p=0.632	p=0.148	p=0.214	p=0.742	p=0.134	p=0.830
ASYM/re-injury/no re-injury (n=62)						
Group	p=0.014	p=0.939	p=0.022	p=0.022	p=0.634	p=0.075
Muscle	p<0.001	p<0.001	p<0.001	p<0.001	p<0.001	p<0.001
Group*Muscle	p=0.996	p=0.147	p=0.133	p=0.650	p=0.116	p=0.930

Table 7: Muscle main effects for abdominal and back muscle PC scores for each of the 3 PCs. See Methods section for muscle definitions. Muscles with the same letter are not significantly different from each other (p>0.05).

Abdominals				Back Extensors			
	PC1	**PC2**	**PC3**		**PC1**	**PC2**	**PC3**
RLRA	C	D	A	**RL13**	ABC	B	A
LLRA	C	D	A	**LL13**	BCD	G	AB
RURA	C	DE	A	**RL16**	ABCD	A	CD
LURA	C	CD	A	**LL16**	DE	H	DE
REO1	B	EF	A	**RL33**	ABCD	BC	A
LEO1	B	AB	AB	**LL33**	CD	G	A
REO2	B	BC	C	**RL36**	EF	BC	BC
LEO2	B	DE	C	**LL36**	F	FG	CDE
REO3	B	A	C	**RL48**	F	CD	DE
LEO3	B	F	BC	**LL48**	F	EF	E
RIO	A	DEF	C	**RL52**	A	D	AB
LIO	A	AB	C	**LL52**	AB	E	A

Appendix 1


Data Matrix
X (101,1680) = [x1, x2... x101]
EMG (% MVIC)
Time (%)
Eigenvector Decomposition
C(101,101)= (X-X̄)(X-X̄)T
C=TΛTT
Principal Components (PCi)
PC 1
PC 2
Principal Component Scores (PCi)
PCi scores = TT*xi
Statistical Analysis
Mixed Model ANOVA for each PC
Abdominals
Back Extensors